The Self-Concept

and

Persecutory Delusions

Thesis submitted in accordance with the requirements of the University of Liverpool for the degree of Doctor of Philosophy by Peter Kinderman

October 1996

PAGE NUMBERING AS ORIGINAL

Acknowledgements

Thanks are due primarily to the patients who participated in the studies described in this thesis. Thanks are also due to the Consultant Psychiatrists in Liverpool and the surrounding districts who allowed access to their patients as well as the staff of psychiatric units in which the majority of interviews took place. I am grateful also to those individuals who completed questionnaires as 'normal' controls.

I should like also to thank my colleagues in the Department of Clinical Psychology at the University of Liverpool who tolerated my behaviour during the research, especially the secretarial staff.

I am indebted to a vast number of colleagues who have helped me through constructive discussions and critical comments on conference presentations and submitted journal articles.

This thesis, of course, could only have been possible due to the help, advice, encouragement and supervision of Professor Richard Bentall. Any strengths in the thesis are a credit to his guidance, any weaknesses are due to me ignoring his recommendations.

Finally, I should like to thank both Sarah and Eleanor, whose ability swiftly to learn the function of the 'delete' button served to keep me on my toes.

Abstract

Despite the use of the term 'schizophrenia' for over 100 years, the scientific validity of the concept appears dubious. The problems commonly leading to a diagnosis of schizophrenia are serious, yet there does not appear to be a single consistent pattern of problems underlying schizophrenia. For this reason research targeting specific problems has recently been initiated. One such specific problem is paranoia or delusions of persecution. Delusions of persecution are known to be associated with an abnormal attributional style whereby negative events tend to be explained as arising from external causes. A detailed model of paranoia hypothesises that such attributions serve a defensive function, limiting the accessibility of self-actual:self-ideal discrepancies. Hypotheses stemming from this model were tested in the studies described in this thesis.

Paranoid individuals were found to attend preferentially to self-referent material, and to exhibit a pattern of low self-actual:self-ideal discrepancies coupled with high self-actual:other-actual discrepancies. Both these findings support the attributional, defensive, model of paranoid ideation.

Investigation of the dynamic relationship between causal attributions and self-discrepancies revealed that external attributions led to a reduced accessibility of self-actual:self-ideal discrepancies. However, contrary to predictions, external attributions were not associated with increased accessibility of self-actual:other-actual discrepancies. Further analysis revealed a distinction between two forms of external attribution, personal (due to the actions of other individuals) and situational. Further studies revealed that external-personal attributions for negative events were uniquely associated with the pattern of self-discrepancies described above. Moreover, such external-personal attributions were characteristic of paranoid patients.

Finally, such external-personal attributions appeared to be associated in non-patient participants with deficits in mentalizing ability or 'theory-of-mind'. Such theory-of-mind deficits are also characteristic of paranoid individuals, suggesting a possible link between these two psychological processes.

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1.1 Introduction

This thesis concerns delusions of persecution and their relationship with the self-concept. It seems important, however, to begin with a brief discussion of schizophrenia. Although the validity and utility of this broad concept will be criticised later in this thesis, delusions have always been central to the concept of schizophrenia, and the relationship between schizophrenia and delusional ideation is necessarily close. Research into the individual symptoms of schizophrenia is relatively novel, and discussion of previous pertinent research must involve studies conducted on patients with a broad diagnosis of schizophrenia. Similarly, discussion of possible aetiological theories of delusions must address theories of schizophrenia. Finally, whatever the eventual verdict on the wisdom of using a classification of schizophrenia, the vast majority of people experiencing delusions are diagnosed as suffering that disorder, and treated accordingly. For these reasons, schizophrenia requires examination.

Richard Bentall (Bentall, Jackson, & Pilgrim, 1988) remarked that almost all variables known to influence human behaviour have been suggested as potential causes of schizophrenia. It has also been noted that the impact on schizophrenia on an individual affects almost all aspects of their life (Birchwood, Hallett, & Preston, 1988). As I will discuss later, it seems unlikely that schizophrenia will be revealed to be an illness with a single cause and simple effects. A complex interaction between variables is likely, with complexity to be expected and the sufferer caught in the middle.

1.2 History

To begin in early recorded western history, it has been claimed that Socrates (469-399 BC) suffered from schizophrenia. He was guided by intense personal inspirations that apparently took the form, on occasion, of auditory hallucinations (Russell, 1961). He is also reported to have occasionally slipped into catatonic states "He has a way of stopping anywhere and losing himself without any reason" (quoted by Russell, 1961; p.108 from Xenophon's *Symposium*). The tendency of ancient philosophers, authors and mystics to refer to inner voices or the voices of Gods guiding their thoughts and actions was a powerful piece of evidence used by Jaynes (1982) to conclude that early cultures were shaped, in part, by mental activity that was less evolved than today's and prone to hallucinations and psychotic phenomena.

References to people who would now be described as suffering from a mental illness are common in early and mid medieval vernacular literature (Doob, 1974). It has been suggested that the prevalence of psychosis was high because of frequent contamination of the staple peasant food, bread, with hallucinogenic substances (Camporesi, 1989). Biblical reports, and reworkings of these stories by medieval authors, frequently refer to madness, as do medieval physicians. For example, Bartholemeus Anglicus (an early medieval physician) reports that:

> "Ye pacient is feynt, ferdful in herte without cause... He dremyth dredful dremes of derkness and ferdfull to se, and of stynkynge sauoure and smelle... Also it comyth in a madnes and of dysposycion of malencoyle whan suche hath lykynge and laughe alwaye of sorowfull thynges, and make sorrowe and doloure for ioyefull thynges. Also suche holden theyr peas whan they shoulde speke and speke tomoche whan they shold be styll. Also some trowe that they ben erthyne vesselles and drede to be towched leest they breke... And some wene that they haue noo heedes, and some that haue leden heedes or asse

> heedes, or some other euyll shapen faccion... Also some fall in to full euyll suspeccion without recour and therfore they hate and blame theyr frendes and somtyme smite and slee theim." (Bartholemeus, 1260/1495; book 4, ch. 11; cited in Doob, 1974).

Translated into modern medical jargon, Bartholemeus could well be listing: depression and anxiety, nihilistic delusions, hallucinations both visual and olfactory, flattened and inappropriate affect, delusions similar to the 'glass delusion' (a common delusion of physical fragility (Speak, 1990)), and somatic and persecutory delusions.

Explanations of madness in medieval Europe are a confusion of animism, religion and humours. Madness was seen as a part of the natural world in which the transmigration of souls was conceivable, daemonic possession likely and the daily intervention of God was taken for granted. At the same time humoural explanations of human behaviour were also common (see Doob, 1974). The explanation, and presumably the treatment, a patient received may have differed widely.

One extreme example of medieval care in the community was the witch hunt. The dominican inquisitors Jakob Sprenger and Heinrich Kraemer (taking the ecclesiastical name Henricus Institoris) published Malleus Maleficarum "The Hammer of Witches" in the late 15th century. On 9th December 1484 Pope Innocent VIII published a bull commanding his "beloved sons" Sprenger and Kraemer to extirpate witchcraft in Germany, and the University of Cologne issued an approval of their work on 19th May 1487. Malleus Maleficarum (Institoris & Sprenger, 1492) describes the wiles of witches and the methods of their detection. It is a comprehensive manual for conducting witch hunts. If the men and women described in Malleus Maleficarum and other accounts of witch hunting (Lumby, 1995) were not possessed by daemons, some form of psychotic or delusional experiences may have been confused for veridical accounts of supernatural phenomena. Zilboorg (1941) for one believed that while "not all accused of being witches and sorcerers were mentally

sick, [nevertheless] almost all mentally sick were considered witches, or sorcerers, or bewitched" (p.153). If this is true, there were probably more in the latter category, people who were either seen as possessed by virtue of their odd behaviour or claimed they were possessed because of psychotic perceptions or delusions. Lumby (1995) gives a relatively clear description of a dying man's paranoid ideas implicating a woman later hanged as a witch. It is to be hoped that modern psychiatric and psychological approaches to psychopathology are less judgemental.

The modern story of schizophrenia begins with Kraepelin (1919/1973), who attempted to reclassify the confusing contemporary array of mental conditions into two broad illnesses. Assuming that the previous conditions of démence précoce, hebephrenia, catatonia, and dementia paranoides were all parts of a single picture of presenile mental deterioration with chronic and deteriorating course, Kraepelin coined the term dementia praecox (Kraepelin, 1919/1973). Kraepelin contrasted this condition with manic-depression, which he regarded as having a more favourable prognosis. With dementia meaning intellectual decline, madness or insanity, and praecox implying an early onset, Kraepelin had identified a mental condition striking young adults. Kraepelin viewed hallucinations, delusions, distortions of emotional expression, attention deficits, negativism, stereotypical movements and posturing, reduced vocational, and impaired intellectual abilities as characteristic of dementia praecox. One of the most fascinating aspects of Kraepelin's approach towards dementia praecox was his view of the interplay between psychological and organic factors. This interplay continues in most elegant commentaries on schizophrenia to the present. Thus, although Kraepelin viewed dementia praecox as an illness primarily affecting the patient's personality, he based his classification on clinical course, commenting that dementia praecox had its onset in early adulthood, and had an invariable chronic course with a poor prognosis. He noted many symptoms

considered characteristic of schizophrenia today. He also (Kraepelin, 1919/1973) suggested that the illness was organic, possibly caused by metabolic disorders that effectively poisoned the brain, despite its effects on the psychology and personality of the sufferer.

The bases of Kraepelin's dementia praecox were further developed by Eugene Bleuler (1950). He suggested that there exists a related group of conditions - the group of schizophrenias - which encompassed dementia praecox. As the term indicates, Bleuler regarded schizophrenia as a condition characterized by a fragmentation of thought, a loosening of associations. In contrast to an organised, rational, purposeful individual, a patient with schizophrenia would find their emotions, thoughts and behaviour dissociated one from another. This fundamental deficit, which Bleuler believed to be of organic origin, led to a number of primary symptoms. These included: the expression of logically unrelated ideas, associated only in the patient's mind; the use of symbolic and alliterative language and the condensation of thought processes. Bleuler believed that the loosening of associations in the patient's mind led to specific alterations in cognition and emotion. These alterations were believed to be particularly significant for the patient's relationship with the external, real, world.

Fragmentation of thought processes, and a splitting of emotional and rational aspects of personality were emphasised in Bleuler's model. Delusions, hallucinations, autism, negativism, stereotypical behaviours and catatonia were felt to be to be secondary symptoms. For Bleuler, delusions could be either basic - a fundamental belief driven by emotional disturbances, or elaborative - an elaboration or extension from the basic delusional theme. A basic delusion might be one of persecution; an elaborative delusion might concern a particular individual believed by the patient to be a secret agent.

Psychiatrists based at the University of Heidelberg, Jaspers, Mayer-Gross and Schneider, extended Bleuler's approach to the classification and definition of

schizophrenia in the 1950's. Their approach tended to focus explicitly on observable symptoms. Schneider (1959) therefore placed less emphasis on such intangibles as lack of volition and dissociative processes than Bleuler and Kraepelin. In contrast, he produced a list of 'first rank' symptoms of schizophrenia - bizarre delusions, third person hallucinations (voices discussing a person's actions or thoughts), delusions of external control of thought (thought insertion, thought broadcasting or thought withdrawal) or movement (delusions of control or passivity). Presentation with these symptoms virtually guaranteed a diagnosis of schizophrenia.

The specificity of the Schneiderian approach has proved practically very useful. In the 1930's and 1940's a significant difference was observed between the schizophrenia diagnosis rates in the United Kingdom and the United States (Bellack, 1958). Comparison of the frequency and circumstances of schizophrenia diagnoses in Europe and America, suggested that the term 'schizophrenia' was being used in different ways in different places. More pertinently it suggested that clinicians in America were using diagnostic criteria much looser than their European counterparts. Beyond the clinical implications, this had major theoretical implications. How, for instance could researchers compare results if they were conducting research on people with 'schizophrenia' who would not be so labelled across the Atlantic? The statistical reliability of the diagnosis of schizophrenia was found to be poor (kappa = 0.6, Spitzer & Fliess, 1974) with only 32% of the disagreement being due to poor measurement of symptoms, but 63% due to unclear criteria (Beck, Ward, Mendelson, Mock, & Erbaugh, 1962).

Careful and collaborative revision of the diagnostic criteria for schizophrenia followed. These tended to closely follow the Heidelberg approach. The Present State Examination (PSE; Wing, Cooper, & Sartorius, 1974b) comprises a structured interview and a rating system that explicitly incorporate Schneider's 'first rank' symptoms. The use of such a system markedly improves agreement between raters

(Wing, et al., 1974b). The use of the PSE, for example, revealed very similar rates of schizophrenia in America and the UK, if the condition was reliably defined (Cooper, Kendell, Gurland, Sharpe, Copeland, & Simon, 1972). The World Health Organization's International Pilot Study of Schizophrenia (World Health Organization, 1973; 1979) surveying over a thousand patients in nine different countries, also revealed good agreement between the research team's diagnosis and the local psychiatrists using PSE criteria. Such technological and methodological improvements have improved the reliability of the diagnosis of schizophrenia to the point that it has been suggested that the problem of unreliable classification has been "largely solved" (Birchwood, et al., 1988; p.19). Such a claim, even if true, does not necessarily mean that such reliable diagnoses are valid.

1.3 Classification and diagnosis

There exist two major classificatory systems for mental illness. One, the World Health Organization's International Classification of Disease, is regularly revised. The latest revision - ICD-10 (World Health Organization, 1992b) - is technically the universal standard classification, recommended for administrative and epidemiological purposes and forms the basis of UK National Health Service statistical procedures. The American Psychiatric Association's Diagnostic and Statistical Manual is also regularly revised (American Psychiatric Association, 1980; 1987), and is currently in its fourth revision - DSM-IV (American Psychiatric Association, 1994). DSM-IV is, of course, widely used in the USA. It is recommended for epidemiological and statistical purposes, but also for research classification. In consequence, it has become standard for researchers, even in Europe, to use DSM-IV criteria.

The distinction between the two approaches is small. Practically, each manual

makes explicit its similarities and differences with the other. Theoretically, potential conflict is reduced as both use Kraepelin's basic distinction between affective and schizophrenic disorders, and both emphasize Schneider's 'first rank' symptoms.

1.3.1 ICD-10 Diagnostic criteria

The International Statistical Classification of Diseases and Related Health Problems, 10th revision (World Health Organization, 1992b) is an attempt to classify all medical conditions, including but not exclusive to, psychiatry. ICD-10 classifies schizophrenia with a number of related conditions, all involving fundamental and characteristic distortions of thinking and perception and disturbance of affect. ICD-10 (like DSM-IV) concentrates on observable, current symptoms, and offers diagnoses that can be applied at a patient's first presentation. Although the diagnosis of schizophrenia is a serious one, a protracted, deteriorating, or chronic course is not a necessary part of the diagnosis.

As a direct legacy of Schneider, an ICD-10 diagnosis of schizophrenia can be made if at least one of a core group of symptoms: a) thought echo, thought insertion, or thought withdrawal, b) delusions of control, influence or passivity or delusional perception, c) third-person hallucinatory voices either keeping up a running commentary on the patient's behaviour or discussing the patient, or d) persistent delusions are present. Alternatively, the presence of at least two of the following symptoms will lead to a diagnosis of schizophrenia: a) persistent hallucinations (either every day for a month, or accompanied by delusions), b) thought disorder or incoherence, c) catatonic behaviour, or d) negative symptoms (apathy, poverty of speech, blunted or incongruent affect) (World Health Organization, 1992a). To exclude brief psychotic states, temporary reactions, or the effects of psychoactive

compounds, the core symptoms must have been present for at least one month to warrant a diagnosis of schizophrenia.

The general diagnosis of schizophrenia is further divided into various sub-types, each of which is assigned a specific code-number (see Appendix 1).

1.3.2 DSM IV Diagnostic criteria

As with ICD-10, DSM-IV criteria for schizophrenia include the presence of characteristic psychotic negative and positive signs and symptoms (such as delusions or hallucinations) for an active phase of at least one month. In keeping with the historical legacy of broad inclusive criteria in American psychiatry, a diagnosis of schizophrenia in DSM-IV is associated with social and occupational consequences. Beyond a one-month history of major psychotic phenomena, some signs of the disorder must have been present for at least six months. Kraepelin's concept of a prolonged and deteriorating course is therefore still evident. In DSM-IV the core diagnostic symptoms - positive symptoms of delusions, hallucinations, disorganized speech and grossly disorganized or catatonic behaviour and negative symptoms of flattened emotions, alogia and avolition - are less clearly related to Schneiderian 'first rank' symptoms. To attract a diagnosis of schizophrenia, a person would show at least two of the following symptoms over a 1-month period: delusions, hallucinations, disorganized speech (frequent derailment or incoherence), grossly disorganized or catatonic behaviour or negative symptoms (emotional flattening, alogia or avolition). In addition, social and occupational dysfunction must be evident, such as poor self-care, productivity or interpersonal relations. There must have been continuous signs of the disorder for at least six months.

A characteristic feature of DSM-IV diagnoses is the use of exclusion criteria.

This means that, to attract a diagnosis of schizophrenia, the patient must not meet the criteria for mood disturbance or developmental disorder, and there must be no possibility that substance abuse or a medical condition might have led to the symptoms. These aid research by reducing the likelihood that phenomena with different aetiologies are invalidly grouped together. In schizophrenia a diagnosis can only be made if there is no reason to suppose that the psychotic symptoms are caused by organic factors.

As with ICD-10, there are a number of subtypes of DSM-IV schizophrenia. These are distinguished by symptomatology (see Appendix 2).

1.3.3 Other classifications

In addition to ICD-10 and DSM-IV, other classification systems for schizophrenia exist. The CATEGO (Wing, Cooper, & Sartorius, 1974a) computer program uses data from the present state examination, PSE, to produce a diagnosis. Since this relies on algorithmic rules rather than clinical judgements, it is very reliable for given data.

Brockington, Kendell and Leff (1978) reviewed ten established definitions of schizophrenia (Feighner, Robins, Guze, Woodruffe, Winokur, & Munoz's diagnostic criteria, (1972), Forrest and Hay's definitions for schizophrenia in young adults and for middle life (Forrest & Hay, 1973), Taylor's definition (Taylor, Abrams, & Gaztanaga, 1975), the CATEGO system (Wing, et al., 1974a), Schneider's first rank symptoms (Schneider, 1959), Carpenter's 'Flexible System' (Carpenter, Strauss, & Bartko, 1973), the New Haven Schizophrenia Index (Astrachan, Harrow, Adler, Brauer, Schwartz, Schwartz, et al., 1972), Langfeldt's (Langfeldt, 1960) criteria and Spitzer's Research Diagnostic Criteria, (Spitzer,

Endicott, & Robins, 1975)). Each of these systems appeared individually valid and reliable, with coefficients of inter-rater agreement (Kappa) above 0.65 (Brockington, et al., 1978). However, there was only moderate overlap across the definitions, with a coefficient of concordance (Kappa) of between 0.29 and 0.59 depending on the patient sample. On the basis of findings such as these, Birchwood and colleagues (1988) concluded that there is a "core group of patients" (p.20), who possess the diagnostic criteria for almost all definitions of schizophrenia. However they also conclude that some 70% of patients have less definite symptoms, leading to diagnostic uncertainty. The need for clear agreement, especially in research cannot be overstated.

1.4 The problem of schizophrenia

1.4.1 Scale of the problem

Different estimates of the incidence of schizophrenia have been recorded in different populations by different researchers. Generally there is a consensus that slightly fewer than ten people in a thousand will develop the disorder during their lifetime (Birchwood, et al., 1988). The prevalence of the disorder (a slightly different statistic reflecting not an individual's risk, but the proportion of people currently sufferers) is similarly estimated at between 0.2 and 1% (Torrey, 1987). It tends to be first diagnosed in early adulthood (late teens and early twenties) (Cooper, 1978). This means that, in the UK, between one hundred thousand and half a million people are sufferers. It is usually reported as more common (Hambrecht, Maurer, & Hafner, 1992; Lynge & Jacobsen, 1995), and possibly more severe (Shtasel, Gur, Gallacher, & Heimburg, 1992), in men, although these data may be in doubt (Castle, Abel, Takei, & Murray, 1995; Hambrecht, Riecher-Rossler, Fatkenheuer, Louza, & Hafner, 1994).

As previously mentioned, following the revisions of the diagnostic criteria for schizophrenia, very similar rates of schizophrenia have been revealed in America and the UK (Cooper, et al., 1972; World Health Organization, 1973; 1979; Wing, et al., 1974b). More interestingly, it has been suggested that the incidence and prevalence of schizophrenia, using reliable criteria, are relatively consistent despite cultural differences (Jablensky & Sartorius, 1975). Many different cultures have, it is claimed, terms broadly denoting madness that describe the common psychotic symptoms (Leff, 1982; Murphy, 1978).

1.4.2 Personal impact

Clearly an individual diagnosed as suffering from schizophrenia is faced with a set of changes that are likely to prove major and long-lasting. Less than a third of patients can expect to return to their expected level of personal health and social functioning (Keith, Gunderson, Reifman, Buchsbaum, & Mosher, 1976). Although to many people the main feature of schizophrenia is its complexity, apparently consistent patterns of clinical course have been reported (Ciompi, 1980; World Health Organization, 1979; Watt, Katz, & Shepherd, 1983). Five different clinical histories are distinguished: Single episode with full recovery (25% of sufferers), Episodic course with full remission between episodes (20% of sufferers), Episodic course with partial remission between episodes (25% of sufferers), Episodic course merging into chronic illness (15% of sufferers), Chronic deterioration (25% of sufferers) (Birchwood, et al., 1988).

Impairments intrinsic to the disorder (such as the symptoms themselves) and so-called extrinsic problems (such as unemployment, prejudice or lack of confidence and poor social skills) (Wing, 1978) encompass all areas of life. Patients suffer a

range of disturbing, frightening and debilitating conditions encompassed by the disorder such as hallucinations, delusions, thought disorder, withdrawal, apathy, emotional blunting, attention and intellectual problems and the fear of further or future episodes (Birchwood, et al., 1988). Because of the disorder they are likely to suffer unemployment or at least reduced employment prospects, with a consequent reduction in material quality of life. Social, housing and accommodation, and economic, difficulties are likely (Serban, 1975), and the consequence of lengthy stays in hospital can be negative (Fan, Huang, Wu, Jiang, & Phillips, 1994). Many patients experience a reduced social network, losing touch with friends (Brockington, et al., 1978; Pattison, DeFrancisco, Wood, Frazier, & Crowder, 1975; Tolsdorf, 1976).

Birchwood and colleagues (1988) comment that schizophrenia affects not only the sufferer herself or himself, but also has a negative effect on the family. Relatives report high levels of negative symptoms (Creer & Wing, 1973; Gibbons, Horn, Powell, & Gibbons, 1984) that they find distressing. In addition, caring for a family member with schizophrenia places economic, emotional and practical pressures on carers, often for many years (Winefield & Harvey, 1994).

Johnstone and colleagues (Johnstone, Owens, Gold, Crow, & MacMillan, 1984) found that only 18% of patients discharged from hospital had no residual symptoms and were functioning satisfactorily. Thirty-eight percent had impairments in personal care, and the same proportion were experiencing social problems. Given that schizophrenia comprises severe and frightening symptoms and that it has a prognosis so grave, it should not be surprising that schizophrenia has a high suicide rate even when compared to other psychiatric diagnoses such as depression (Caldwell & Gottesman, 1992). About one in ten people with a diagnosis of schizophrenia take their own lives, with the suggestion that suicide is the chief cause of premature death for patients (Caldwell & Gottesman, 1992). Young, single people living alone are at highest risk, especially if they fear further mental disintegration (Caldwell &

Gottesman, 1992).

This major negative impact of schizophrenia occurs in spite of the widespread use of neuroleptic medication. By far the most common treatment regimes for people with schizophrenia involve the use of neuroleptic medication. These are significantly effective in acute episodes of schizophrenia (Cole, Kleberman, & Goldberg, 1964). They are also effective in preventing relapse, reducing the relapse rate by between 70% and 40% (Hogarty, 1993). However, neuroleptic medication does not prove effective in all cases. While some two-thirds of patients will relapse on placebo medication, a third will relapse even if prescribed neuroleptics (Davis, 1975; Johnstone & Geddes, 1994; Kane & Lieberman, 1987; King, Blomqvist, Cooper, Doherty, Mitchell, & Montgomery, 1992; Rifkin, Quitkin, Rabiner, & Klein, 1977).

Despite the obvious effects of schizophrenic symptoms, the side-effects of neuroleptic medication may be as pertinent in terms of the negative impact on quality of life (see Day & Bentall, 1996, for a review). Neuroleptic drugs are associated with many highly distressing adverse reactions. Parkinsonian side effects (changes in gait, tremor, rigidity, hypersalivation, akinesia, dysphagia and expressionless appearance) occur in some 15 to 25% of patients (Ayd, 1961; Kennedy, Hershon, & McGuire, 1971). Acute dystonic reactions (acute muscular spasms of the head and neck) affect about 10% (Swett, 1975) of mainly younger patients treated with neuroleptics. Akathisia (a restlessness or discomfort) has been estimated to affect from 20% (Ayd, 1961; Braude, Barnes, & Gore, 1983; Swett, 1975) to 75% (van Putten, May, & Marder, 1984) of patients taking neuroleptics.

So-called anti-cholinergic side effects (such as dry mouth, constipation, blurred vision and urinary retention) are also common, plaguing about 40% of treated patients (Lingjaerde, Ahlfors, Dech, Dencker, & Elgen, 1987). Other side-effects include sexual effects (orgastic dysfunction and reduced libido in both sexes and erectile dysfunction, ejaculatory dysfunction and priapism (permanent erection) in

males (Segraves, 1988; Sullivan & Lukoff, 1990), and elevated prolactin levels, resulting in amenorrhea, gynaecomastia (swollen and tender chest, which may occur in both men and women) and galactorrhoea (milk production, also in both sexes). Weight gain, often significant, (Gordon & Grotte, 1964) is common. Neuroleptic drugs have been implicated in causing several allergic reactions, including rashes and photosensitivity reactions (Edwards, 1986) and excessive urination and drinking (Lawson, Karson, & Bigelow, 1985).

Tardive dyskinesia is a frequently irreversible movement disorder affecting tongue and jaw movements (Jeste & Wyatt, 1979). Between 0.5% and 65% of patients suffer this adverse effect (Simpson, Pi, & Stramek, 1982), with older women, and patients on larger doses of neuroleptics being particularly at risk (Kane & Smith, 1982; Muscettola, Pampallona, Barbato, Casiello, & Bollini, 1993). The risk of agranulocytosis (a frequently fatal (Edwards, 1986) drop in the numbers of white blood cells) may be as high as 1 in 50 for patients taking clozapine (Fischer, Haar, Greiner, Lloyd, & Mason, 1991). Rapid dose changes of neuroleptic medication can also trigger epileptic convulsions (Toone & Fenton, 1977).

Finally, neuroleptic malignant syndrome affects some 0.5 to 1% of patients with a mortality rate of about 20 percent (Caroff, 1980), and neuroleptic induced deficit syndrome (a state of emotional indifference and lethargy) may affect an unknown proportion of patients taking sedative neuroleptics (Crow, MacMillan, Johnson, & Johnstone, 1986). The latter is a major reason for non-compliance with neuroleptics (Van Putten & May, 1978; Weiden, Shaw, & Mann, 1986).

Clearly, well treated schizophrenia is likely to have a less significant negative effect on an individual than an untreated condition. Nevertheless the adverse effects of neuroleptic medication contribute to the major threat that schizophrenia poses to quality of life. Bollini, Pampallona, Orza, Adams and Chalmers (1994), reviewing a number of studies, concluded that higher doses of neuroleptics were associated with more (or

more severe) side-effects, but not with better clinical response. Meanwhile, Finn, Bailey, Schultz and Faber (1990) suggested that, for patients at least, the side-effects of neuroleptic medication may be as distressing as the symptoms of schizophrenia themselves.

1.4.3 Economic Aspects of Schizophrenia

Wasylenki (1994) has concluded that schizophrenia demands the largest proportion of mental health care expenditure in the US. As this expense is due to the high prevalence of this illness, chronicity and early age of onset, features that are common to both the UK and USA, it may be expected that the economic costs of schizophrenia are similarly high in Britain.

In Britain the 1992 Government White Paper "Health of the Nation" (Department of Health, 1992) recognizes mental illness in general and schizophrenia in particular as significant health costs. The cost of treating schizophrenia for many years with expensive medication, the use of medical and social day services, frequent hospitalization is recognised, as is the cost to the community of losing productive members (Department of Health, 1992). The fact that mental illness accounts for more than 14% of certified sickness leave was a major factor in the development of specific mental health targets: the improvement of health and social functioning of mentally ill people, the reduction of the general suicide rate by at least 15%, and of the suicide rate of severely mentally ill by at least 33% by the year 2000 (Department of Health & Social Services Inspectorate, 1994).

Despite the difficulty of assessing all the direct and indirect costs of schizophrenia (Andreasen, 1991), a number of researchers have attempted to estimate the true financial costs of the condition. Davies and Drummond (1994), calculated the

cost of one year's treatment at £2,138 per person (1992 prices). Assuming a population of 185,400 people in the UK with schizophrenia, they concluded that medical and social services cost £397 million per year, 1.6% of the total health care budget. These costs do not include indirect costs (unemployment and time off work) which Davies and Drummond (1994) estimate as £1.7 billion. They also exclude the financial impact on family and carers and the financial costs of suicide. Although the majority of patients receive medication, only 5% of direct costs are accounted for by drug treatments, leading Davies and Drummond (1993) to conclude that more expensive drugs that reduce other direct and indirect costs would benefit patients and save money. It is also worth noting that McGuire (1991) estimated that the costs of mental illness are rising much faster than other indices of expenditure.

1.5 Causes and aetiology

As this thesis concerns the phenomenon of delusions, rather than schizophrenia in general, only the major aetiological approaches to the disorder will be discussed. In principle, three broad areas can be identified as possible causes, organic or biological factors, social or environmental factors and psychological factors. These will be addressed in turn.

1.5.1 Biological approaches

1.5.1.1 Genetics

The simplest approach to the investigation of genetic influences on schizophrenia is to examine whether the relatives of schizophrenic patients are at a

higher risk of developing the disorder than unrelated individuals. Neale and Oltmanns (1980) reviewed a variety of studies to conclude that the risk faced by general population (the likelihood that a person will develop schizophrenia) is 0.85%. Gottesman and Shields (1982) reviewed a number of studies examining the relationship between genetic relatedness and morbidity risk. They concluded that there exists a strong genetic relationship, with a risk of 2.4 % for first cousins, 3% for nephews or nieces and 12.8% for the children of schizophrenic patients. Research in this area has been conducted for many years, and the studies have differed in quality. Overall, however a relatively consistent pattern is seen. More recent studies, employing operationalized diagnostic criteria such as the PSE, DSM-IV or ICD-10, ratings of mental health made independently of knowledge of familial status and a normal comparison group have indicated that the first-degree relatives of people with schizophrenia are more likely to be diagnosed as suffering from schizophrenia themselves than are the children of non-sufferers (Baron, Gruen, Rainer, Kane, Asnis, & Lord, 1985; Gershon, DeLisi, Hamovit, Nurnberger, Maxwell, Schreiber, et al., 1988; Kendler, Gruenberg, & Tsuang, 1985). These data are not conclusive, however, with some inconsistencies. For example, Baron, Gruen and Romo-Gruen (1992) found that the risk of first-degree relatives was low for patients with negative symptoms, but raised for patients with positive symptoms.

This pattern is usually interpreted as clear evidence of a genetic aetiological role (Gotteman & Shields, 1972); one's risk increases as one shares genes with a sufferer. However, a close familial relationship between two people implies that environmental, as well as genetic, similarity is high. Schizophrenia may run in families, but not necessarily through genes.

An alternative method of study involves the investigation of identical and non-identical twins. From as early as 1929 (see Rosenhan & Seligman, 1984) researchers

have used such studies to investigate the possibility that schizophrenia has a genetic basis. The logic of this approach is that differences between monozygotic (MZ) twins (who share 100% of their genetic information) and dizygotic (DZ) twins (who share 50% of their genes) are indicative of the genetic loading of the illness. Because twins are seen as "valuable experiments [of] nature" (Torrey, Bowler, Taylor, & Gottesman, 1994, p.5), researchers as diverse as the notorious Dr Mengele (Lifton, 1986) and Sir Francis Galton (Galton, 1875) have been keen to pursue this line of investigation.

Since 1929 many studies have investigated the schizophrenia concordance rates in pairs of MZ and DZ twins. A concordance rate of 100% means that every person with a diagnosis of schizophrenia has a twin sibling who also has the diagnosis, a concordance rate of 0% means that none of the identified patients has a twin with the illness, while 50% concordance means that half of the sufferers have patient twins. Since the prevalence of schizophrenia is around 1%, if no familial of genetic loading existed, a concordance rate of 1% would be expected. Statistical and methodological arguments rage over whether concordance rates should be calculated as the probability of finding a twin of a patient with schizophrenia who also has the illness (proband-wise) or as the probability of finding a pair of twins who both have the illness (pair-wise). For abstruse statistical reasons, proband-wise approaches result in slightly higher but perhaps more valid concordance rates (Birchwood, et al., 1988).

Early researchers seem to have used loose diagnostic criteria, including twins with such conditions as manic depression and personality disorder as concordant for schizophrenia (Marshall, 1984). Kallmann (1938; 1946; 1953) in fact seemed to go further, diagnosing latent schizophrenia in seemingly happy and normal individuals. Moreover, many researchers, again including Kallmann, failed to ensure that the researchers assessing mental state in one twin were blind to the status of the sibling (Marshall, 1984). As importantly, early researchers often judged the genetic

relatedness of the twins subjectively (Marshall, 1984). This is important, because an unconsciously (or deliberately) biased researcher might be tempted to classify twins concordant for schizophrenia as MZ and a discordant pair as DZ. Perhaps as a result of these dubious practices, Kallmann's data were impressive, with concordance rates of 86% in MZ twins compared to 15% in DZ twins (Kallmann, 1953), entirely consistent with schizophrenia being caused by a recessive gene.

More recently, and more reliably, Gottesman and Shields (1982) reviewed five recent twin studies to calculate concordance rates. Overall, the concordance rate for MZ twins was 46% and 14% for DZ twins. Both general surveys of similar research (Birchwood, et al., 1988) and more recent studies (Onstad, Skre, Torgersen, & Kringlen, 1991) have replicated these data which are taken as being strongly indicative of genetic factors in schizophrenia.

Although, in the 1930's scientists confidently advocated the eugenic sterilization of patients with schizophrenia on the basis of twin studies, a practice adopted by the functionaries of the Nazi state (Lifton, 1986; Torrey, et al., 1994), it has been argued that, if blind rating is employed and only clear diagnoses are used, the difference in concordance rates between the schizophrenic and control subjects appears greatly reduced (Lidz, Blatt, & Cook, 1981; Marshall, 1984; Rose, Lewontin, & Kamin, 1984). Jackson (1960) also suggested that comparing MZ and DZ twins is invalid, as MZ twins are raised differently. Specifically MZ twins are more likely to be treated as identical than are the DZ twins. If being treated as identical to another increases your risk for developing schizophrenia, this could undermine the premise of twin studies generally. The data from twin studies are, moreover, strongly indicative of environmental factors. As concordance rates for MZ twins are less than 100%, it is clear that some environmental factor or factors must exist that affects the occurrence of the disorder.

An alternative source of information on the relative weights of inherited and environmental factors in schizophrenia is the study of adopted children. If you are born to a parent (or parents) with schizophrenia, but are brought up by a healthy couple, any future illness could be argued to be inherited. On the other hand, if you are born to a healthy couple, but brought up by an adoptive parent with schizophrenia, future illness would appear to be environmentally determined. Systematic studies following this model have generally found that the prevalence of schizophrenia is higher in the children of schizophrenic parents than the children of parents without the illness (Heston, 1966; Kety, Rosenthal, Wender, Schlushinger, & Jacobsen, 1978; Rosenthal, Wender, Kety, Schlushinger, Welner, & Reider, 1968; Rosenthal, Wender, Kety, Schlushinger, Welner, & Reider, 1975; Wender, Rosenthal, Kety, Schlushinger, & Welner, 1974). For example, in a more recent study, Kety and Ingraham (1992) reported a survey of 14,427 Danes. In Denmark over the past several years, careful records have been made concerning births and adoptions. They claim classical chronic schizophrenia was found almost exclusively in the biological relatives of chronic schizophrenic probands, and its prevalence was 10 times greater than that in the biological relatives of controls. These findings seem to be easily replicable (Tienari & Wynne, 1994; Tienari, Wynne, Moring, & Lahti, 1994).

As with the twin studies, however, methodological problems may be important. Sample sizes tend to be low, as there are few adopted children of parents with schizophrenia. Very broad diagnoses tend to be employed (Thaker, Adami, Moran, & Lahti, 1993), with cases being recorded (and assumed to be support for a genetic theory of schizophrenia) in the event of affective or personality disorders - so-called schizophrenic spectrum disorders - which might include such things as 'inadequate personality' (Rosenthal, et al., 1975). It is also the case that all children in the studied groups, whether the biological children of parents with or without schizophrenia, showed a high level of psychological problems (Kety, et al., 1978;

Rosenthal, et al., 1975; Wender, et al., 1974). There are also oddities in some of the adoption details, with the independence of adoptive and biological families occasionally in question (Birchwood, et al., 1988; Lidz, et al., 1981; Rose, et al., 1984).

Moreover, the picture is not perfectly clear even from the data already gathered. Kendler, Gruenberg and Kinney (1994) reported a study in which, although the frequency of schizophrenia was greater in the biological relatives of adopted children with what was termed the schizophrenic spectrum disorders than in the relatives of non-affected adopted children, this difference was not statistically significant. More interestingly, there is evidence (Tienari, Wynne, Moring, & Wahlberg, 1993) that any genetic effect (a higher rate of serious psychiatric disorder in the adopted children of patients) is only expressed if there was a disturbed adoptive family environment following adoption and was not seen in a healthy, possibly protective, adoptive family. Nevertheless, these studies do tend to suggest that genetic relatives of people who have received a diagnosis of schizophrenia run a greater than average risk also to receive such a diagnosis.

The biological basis of genetic investigations of schizophrenia is itself complex. There are a number of difficulties in carrying out genetic studies in schizophrenia. As I shall discuss below, there are a number of conflicting models of schizophrenia. Findings supportive of one view (for example that schizophrenia is a broad, unitary disorder) may undermine a conflicting model (such as a heterogeneous model). More importantly, there may be genetic differences between two individuals suffering from schizophrenia such that an aetiologically important genetic abnormality in one person may not be seen in a second, who may possess an equally important abnormal gene, but a different one. As noted by McGue & Gottesman (1991), despite the powerful general evidence outlined above, researchers have not identified a single

major gene responsible for schizophrenia. If one uses data from a number of families, the presence of a small number of such important genes may be overlooked. Moreover, of course, even the most strident conclusions from the most convincing data suggest that environmental influences play an essential role in most cases. As Birchwood and colleagues (1988) have pointed out, the interesting question may be not whether genetic factors may contribute to schizophrenia, but how.

The simplest model of genetic transmission is single gene transmission. As mentioned above, Kallmann (1953), viewed schizophrenia as being caused by a single (recessive) gene. Such a simple model of transmission is, of course, essential if one advocates sterilizing sufferers. A slightly more complex model of genetic transmission is polygenetic, where a number of chromosomes act together to produce a particular characteristic. Even more complex is the issue of variable penetrance, where the microcellular genetic mechanisms may be altered by environmental (or biochemical) factors such that the presence of a chromosomal abnormality need not necessarily lead to the full expression of the phenotype.

Recent developments in theoretical molecular genetics and novel experimental techniques have allowed empirical studies of individual gene sequences in extremely small sample populations. Thus scientists have attempted to link individual chromosomal abnormalities with particular hypothesised illnesses. Kendler and Diehl (1993), despite concluding that there was evidence of a genetic element in schizophrenia, also conceded that we have no idea what the genetic element of schizophrenia might be. Campion, Damato, Bastard, Laurent, Guedj, Jay, Dollfus, Thibaut, Petit, Gorwood, Babron, Waksman, Martinez and Mallet (1994) and Nothen, Korner, Lannfelt and Sokoloff (1993) studied the genetic bases of four (D1, D2, D3 and D4) dopamine receptor genes, and found no differences between sufferers and non-patients. Sanders, Rinconlimas, Chakraborty, Grandchamp, Hamilton, Fann

and Patel (1993) investigated six different genetic loci, each of which might reasonably be expected to be implicated in schizophrenia. Only one of these showed more abnormalities in patients suffering from schizophrenia than in control subjects. Clearly, as a scientific test of an hypothesis, this is inadequate. There seem to be at least as many published papers reporting a failure to find specific genetic abnormalities as there are papers reporting a positive finding. This suggests strongly that, at the very least, there is no simple (single mutation) genetic basis of schizophrenia. It is interesting that Mulcrone and colleagues (Mulcrone, Whatley, Marchbanks, Wildenauer, Altmark, Daoud, Gur, Ebstein and Lerer (1995), after examining the genetic bases of the D2 receptor gene and finding nothing, concluded that it was "still possible, however, that a gene of major effect exists in this region, either with low penetrance or with heterogeneity" (p.103).

Taken together, studies of the genetic aspects of the aetiology of schizophrenia provide considerable evidence that some inherited factors play a role in the development of the disorder. However, it seems very difficult to conclude how much weight such factors play. Opinions remain divided. Although no-one seriously suggests that genetic factors play no role in the development of schizophrenia, Rose, Lewontin and Kamin (1984) provide what seems convincing evidence that genetic factors are overvalued for cultural and political reasons. E. Fuller Torrey (see Torrey, et al., 1994) has extensively examined the role of genetic factors in schizophrenia, but concluded that the role of inherited characteristics may be over emphasised (Torrey, 1992), suggesting viruses (Yolken & Torrey, 1995) possibly carried by house pets (Torrey & Yolken, 1995) may be important! Peter McGuffin (McGuffin, Asherson, Owen, & Farmer, 1994; McGuffin, Owen, & Farmer, 1995), on the other hand, suggests that schizophrenia is probably only genetic in origin. It also remains unclear as to how genetic differences might lead to psychiatric abnormalities. More pertinent for the purpose of this thesis, studies examining genetic factors in schizophrenia may

be unable to shed light on the aetiology of delusional ideation.

1.5.1.2 Dopamine hypothesis

Many psychoactive recreational drugs, especially LSD, amphetamines and cocaine, seem to induce states similar to acute schizophrenia. In particular the use of dopamine agonists - substances that cause or facilitate a release of the neurotransmitter dopamine - such as amphetamine can result in psychotic conditions that trained clinicians occasionally find almost impossible to distinguish from acute paranoid schizophrenia (Brady, Lydiard, Malcolm, & Ballenger, 1991; Mitchell & Vierkant, 1991). It has also been found that amphetamine can exacerbate psychoses in a person already diagnosed as schizophrenic (Iversen & Iversen, 1975).

On the other hand, it is also widely recognised that dopamine antagonists reduce or ameliorate psychotic symptoms. Substantial support for the dopamine hypothesis comes from examination of the neurochemistry of dopamine and neuroleptic medication. Sunahara, Seeman, van Tol and Niznik (1993) suggested that the dopamine hypothesis is supported by the fact that clinically effective antipsychotic medication invariably blocks dopamine D2 receptors when administered at conventional dose levels. Moreover, when radio-labelled neuroleptics are applied to post mortem brain tissues from schizophrenic patients, an increased number of dopamine receptors is revealed, an increase which is also seen in living neuroleptic naïve patients using radio-labelled binding agents and positron emission tomography.

Although neuroleptic drugs were developed essentially on a trial and error basis (Delay & Denker, 1952) to reduce schizophrenic symptoms, it is clear that one of their major pharmacological effects is to affect dopamine metabolism in a way that

reduces the effects of dopamine on the brain but might actually lead to higher dopamine levels in the brain. This effect is probably achieved by blocking dopamine receptors (Crow, 1981; Snyder, 1982). Johnstone, Crow, Frith, Carney, and Price (1978) compared the therapeutic efficacy of different optical isomers of the neuroleptic drug flupenthixol. They found that the isomer with the greater in-vitro antidopaminergic activity also possessed the greater clinical efficacy, suggesting that antidopaminergic activity and clinical effectiveness correlate.

Another area of evidence purported to support the dopamine hypothesis also demonstrates the complexity of the topic. A number of studies (Lee, Seeman, Tourtellotte, Farley, & Hornkiewicz, 1978; Owen, Cross, Crow, Longden, Poulter, & Riley, 1978) have reported an increased number of dopamine (D2) receptors in the basal ganglia of people with schizophrenia. An increase in D2 receptor binding efficiency has also been described in a number of studies (Hess, Bracma, Kleinman, & Creese, 1987; Mackay, Bird, Spokes, Rossor, Iversen, Creese, et al., 1980). If dopamine activity is related to schizophrenic symptoms, then large numbers of efficient dopamine receptors would appear to be support for the model. However, Farde, Wiesel, Hall, Halldin, Stone-Elander and Sedvall (1987) did not find an increase in D2 dopamine receptors in previously untreated patients with schizophrenia.They suggested that one consequence of treatment with neuroleptic medication was a reactive increase in the number (and possibly efficiency) of dopamine receptors.

As a consequence of these lines of research, the dopamine hypothesis is now widely accepted. Despite almost universal acceptance in popular literature, however, several lines of evidence shed doubt on a simple dopaminergic model. As mentioned above, dopamine agonists used as recreational drugs are commonly thought to exacerbate or mimic psychotic symptoms. However in a review of 12 studies, van Kammen, Docherty, Marder, Schulz, Dalton and Bunney (1982) found that psychotic

symptoms worsened in only 25% of cases following administration of amphetamine. In an apparently direct challenge to the dopamine hypothesis, in 46% of cases there was no change and in 29% of cases patients' symptoms improved. Similarly, both Wyatt, Karoum, Suddath and Hitri (1988) and Unnithan and Cutting (1992) have criticised the idea that cocaine intoxication acts as a model of schizophrenic psychosis on clinical and phenomenological grounds.

Crucially, no convincing link has been established between hyperactivity in a person's dopamine metabolism and psychotic symptoms (Jackson, 1990). Similarly, one of the tenets of the dopamine hypothesis is that neuroleptic medication achieves its effects via the blocking of dopamine receptors (Sunahara, et al., 1993). However, although neuroleptics are clearly effective (as outlined above), a failure to respond to neuroleptic medication does not imply that dopamine receptor blocking has failed (Wolkin, Barouche, Wolf, Rotrosen, Fowler, Shuie, et al., 1989). Rather, as mentioned above, antipsychotic medication invariably blocks dopamine receptors (Creese, Burt, & Snyder, 1976; Sunahara, et al., 1993). This is an important point, as it suggests that clinical symptoms may persist even when the supposed malfunctioning dopamine system is comprehensively addressed.

A novel development in therapy for schizophrenia has been the increased use of atypical neuroleptics. These drugs, such as respiridone and clozapine, have been specifically developed to have a weaker blocking effect on dopamine than on serotonin (5HT) receptors. Such drugs (serotonin-dopamine antagonists) are popular because of their lower rate of 'extrapyramidal' side-effects (He & Richardson, 1995; Kerwin, 1994; Meltzer, Lee, & Ranjan, 1994). However, they are also effective at treating schizophrenia, despite the fact they are designed not to block dopamine metabolism. The central role of dopamine in the aetiology of schizophrenia has consequently been reviewed (Kerwin, 1994), with other neurotransmitters - including serotonin, phenylethylamine, noradrenaline, transmethylated amines, acetylcholine, gamma-

aminobutyric acid (GABA), endorphins and prostaglandins - becoming suspects.

1.5.1.3 Other neurotransmitters

Serotonin is an obvious candidate as an aetiological agent in schizophrenia following examination of the dopamine hypothesis. LSD (and other recreational drugs) affects serotonin as well as dopamine, while Clozaril and the other novel antipsychotic drugs are powerful serotonin antagonists, and are more clinically effective than dopamine antagonists. However, evidence of serotonin abnormalities is patchy. DeLisi, Neckers, Weinberger and Wyatt (1981) reported high levels of serotonin in patients with schizophrenia who also exhibited abnormal CT scans, but studies examining the levels of serotonin or its metabolites have generally been inconclusive (Freedman, Belendiuk, Belendiuk, & Crayton, 1981; Gattaz, Waldmeler, & Beckmann, 1982; Joseph, Owen, Baker, & Bourne, 1977; Post, Fink, Carpenter, & Goodwin, 1975; Sedvall & Wode-Helgodt, 1980).

Phenylethylamine (PEA) is a substance very similar to adrenalin, with similar neurochemical action. Fischer, Spatz, Saavedra, Reggiani, Miro and Heller (1972) and Sandler and Reynolds (1976) suggested that receptors for PEA exist in the brain, which are supersensitive in schizophrenia, while Wyatt, Gillin, Stoff, Mojo and Tinklenberg (1977) suggested that PEA is not adequately metabolized by people suffering from schizophrenia, creating the equivalent of chronic amphetamine poisoning. High PEA levels have been reported in the urine of people with schizophrenia (Jeste, Doongaji, Panjwani, Datta, Potkin, Karoum, et al., 1981; Wyatt, Potkin, Bridge, Phelps, & Wise, 1980). As with serotonin (and dopamine) however, no conclusive evidence of abnormal PEA activity has been reported (Meltzer, Jackman, & Arora, 1980; Wyatt, Bigelow, & Gillin, 1979).

Wise and Stein (1969) suggested that psychological reward was mediated by noradrenaline. This led to the suggestion that a deficit in noradrenaline might be responsible for some symptoms of schizophrenia (Stein & Wise, 1971). Specifically they suggested that a combination of noradrenaline and dopamine released by neurones associated with reward might be toxic. As with other theories, some supportive (Bird, Spokes, & Iversen, 1979; Sternberg, van Kammen, Lake, Ballenger, Marder, & Bunney, 1981; Wise, Baden, & Stein, 1974) but many unsupportive (Castellani, Ziegler, van Kammen, Alexander, Siris, & Lake, 1982; Crow, Baker, Cross, Joseph, Lofthouse, Longden, et al., 1979) findings have been reported.

One of the older biochemical theories is that of the involvement of transmethylated amines, chemicals similar to normal brain chemicals but created by methylation rather than hydroxylation. As such they might mimic, but distort, brain function. Friedhoff and van Winkle (1962) reported methylated dopamine derivatives in the urine of people with schizophrenia, but this result has not been replicated (Wyatt, Termini, & Davis, 1971). Once again, mixed or inconclusive results are reported (Wyatt, et al., 1971). Similar stories can be told about theories involving acetylcholine (Crow, 1981), GABA (Garbutt & van Kammen, 1983), endorphins (Watson, Akil, Berger, & Barchas, 1979) and prostaglandins (Rotrosen, Miller, Mandio, Traficante, & Gershon, 1980). See Birchwood, et al., (1988) for a review.

1.5.1.3 Brain structure and function

On July 25, 1989, President George Bush designated the 1990s the 'Decade of the Brain'. He was swiftly followed by the European Community Council of Ministers (Editorial; Nature, 1992). Recent advances in imaging techniques such as

computed tomography (CT), magnetic resonance imaging (MRI) and positron emission tomography (PET) have clearly aided neuroanatomical investigations of many disorders, including schizophrenia. Enlarged cerebral ventricles (resulting in smaller brain volumes) have frequently been reported (Jones, Harvey, Lewis, Toone, Van Os, Williams, et al., 1994b; Weinberger, 1984).This increased ventricular size has been associated with deficit symptoms, cognitive impairment and poor outcome in schizophrenia (van Os, Fahy, Jones, Harvey, Lewis, Williams, et al., 1995), and seems not to be a simple consequence of the disorder (Jaskiw, Juliano, Goldberg, Hertzman, Urow-Hamell, & Weinberger, 1994).

In addition other neuroanatomical changes, mainly effective reductions in brain size, have been identified in people diagnosed as suffering from schizophrenia. Thus Raz (1993) reported a widening of cerebral fissures and sulci while Andreasen (1988) reported the results of MRI studies demonstrating reduced cerebral volume. Cerebellar dysfunction (Taylor, 1991) and medial temporal lobe hypoplasia (Roberts, 1991) have also been reported.

These findings are evidence to some researchers that schizophrenia is associated with neurodevelopmental abnormalities and may be of genetic origin (Bogerts, 1993), to others that brain damage caused by viruses is involved (Lim, Beal, Harvey, Myers, Lane, Sullivan, et al., 1995).

Meta-analyses and reviews have generally supported the general idea of minor neuroanatomical abnormalities in the brains of some of the people diagnosed as having schizophrenia. However, within the reported differences there is usually considerable overlap with the characteristics of the brains of individuals with no psychiatric diagnoses. The changes reported are usually small and only 6 to 40% of people with schizophrenia may exhibit such changes (Syvalahti, 1994). Such changes are not exclusive to schizophrenia (Elkis, Friedman, Wise, & Meltzer, 1995), occurring in mood disorders, although possibly to a slightly smaller degree. Chua and McKenna

(1995) reviewed CT, MRI, post mortem and functional imaging studies and concluded that lateral ventricular enlargement is "is modest and there is a large overlap with the normal population" and "may be better understood as a risk factor than a causative lesion" (p.563).

In addition to neuroanatomical abnormalities in the brains of people diagnosed as suffering from schizophrenia, some studies have reported unusual patterns of functional activity of the brain. Investigators have used electroencephalograms (EEG's) (Barrett, McCallum, & Pocock, 1986), evoked potentials (Brecher & Begleiter, 1983; Strik, Dierks, Franzek, Stober, & Maurer, 1994) to investigate neuro-electrical activity. Similarly, single photon emission computerised tomography (SPECT) (Sauer, Schroder, Henningsen, & Wilhelm, 1980) and positron emission tomography (PET) (Gur, Resnick, Alavi, Gur, Caroff, Dann, et al., 1987) have been used to investigate changes in cerebral blood flow.

Overall, studies seem to produce frequent findings of oddities that are extremely difficult to form into a coherent whole. One finding which has been reported as being "consistent" (Birchwood, et al., 1988; p.90) is that there is low relative glucose metabolism in the frontal lobes of the brain - so-called 'hypofrontality'. This claim is supported by evidence (Ariel, Golden, Berg, Quaife, Dirksen, Forsen, et al., 1983; Ingvar & Franzen, 1974) from chronic patients. However it has been claimed by other neurofunctionality researchers that there is no evidence of hypofrontality in neuroleptic-naive subjects (Early, Haller, Posner, & Raichle, 1994). Decreased regional cerebral blood flow in schizophrenic patients as compared to controls has been replicated in a number of studies (Gur & Pearlson, 1993). More specifically, psychomotor poverty and disorganization have been associated with complex alterations of blood flow in the prefrontal cortex, and reality distortion was associated with alterations in the medial temporal lobe (Liddle, 1994; Liddle, Friston, Frith,

Jones, Hirsch, & Frackowiak, 1992).

As in the neuroanatomical studies, one of the main criticisms of these studies is that often subjects who take part in the research have been exposed to neuroleptic medication over many years. It is therefore therefore difficult to determine causality. Overall, some abnormalities seem to be reliable. Nevertheless, as Chua and McKenna (1995) concluded, "The ... findings ... suggest that, as a disorder, schizophrenia shows complex alterations in regional patterns of activity rather than any simple deficit in prefrontal function" (p.563). It is also fascinating that at least one study (Verleger, Bode, Arolt, Wascher, & Kompf, 1994) revealed that evoked P3 potential amplitudes varied according to the relevance of the subjective task associated with each event. This is interesting, as it suggests that apparent neurobiological events may be closely related to psychological factors.

Neurological and neuropsychological approaches to the understanding of schizophrenia is a large area. David and Cutting (1994) review a number of theories accounting for schizophrenic syndromes and symptoms by referring to neurological processes. Of particular interest for this thesis are models of individual psychotic phenomena which are substantively psychological, but which may also interact with neuropsychological processes (Bentall, 1994; Slade, 1994).

Overall, I believe, it is wise to echo the more limited conclusions of Chua and McKenna (1995), and conclude that neurological, neuroanatomical and neurofunctional abnormalities are present in people diagnosed as schizophrenic. They seem easier to discover in chronic patients, but there is no reason to suppose they do not exist in neuroleptic naive and early diagnosed patients. However, despite elegant theories, we have no real idea of how such abnormalities might cause, or even be related to, schizophrenia. One of the conclusions might be that it is via the study of individual psychotic symptoms that such relations will be determined. We also have no idea of what might cause such abnormalities, although once again elegant theories

have been developed.

1.5.1.4 Birth seasonality

Since the 1960's (Barry & Barry, 1961) it has been observed that babies born in late winter and early spring are more likely to develop schizophrenia later in life than children born at other times (Dalen, 1974; Hare, Price, & Slater, 1974; Machon, Mednick, & Schlusinger, 1983; Odegard, 1974; Shimura, Nakamwa, & Mivra, 1977; Torrey, Torrey, & Peterson, 1977). This is apparently specific to schizophrenia; other psychiatric disorders do not share this pattern (Hare & Price, 1968). If you are born in the early months of the year, you are more likely to develop schizophrenia. However, if you are born early in the year, you are a little bit older than if you were born in the autumn. The 'age incidence effect' suggests that, if you are older, you will have had more time in which you might have developed schizophrenia before the time of investigation. Although the birth seasonality effect is modest, it is replicable; even when controlling for this age incidence effect (Rodrigo, Lusiardo, Briggs, & Ulmer, 1992; Takei, Sham, O'Callaghan, Glover, & Murray, 1995b).

As with most of the biological findings in schizophrenia, the reason for the season of birth effect is unknown. Many factors may be involved, such as infectious agents (more germs being present in the winter), nutritional factors (foodstuffs, especially vitamins, fresh fruit and vegetables being seasonal), temperature variations at the time of conception, and an interaction of these factors with genetic influences (DeLisi & Crow, 1986). The season of birth effect has not always been replicated, with a suggestion that the effect is present predominately in the Northern hemisphere (Berquier & Ashton, 1991). For example, Kim, Lee, Lim and Noh (1994) failed to replicate the birth seasonality effect in a study of 1,606 patients with schizophrenia and

4,582 controls in Korea. The fact that the season of birth effect appears to affect only the northern hemisphere has been argued to support a theory of viral aetiology in the development of schizophrenia (DeLisi & Crow, 1986), the argument being that viral pandemics in northern Europe adversely affect unborn or neonatal children.

1.5.1.5 Viral infections in pregnancy

In 1957 a major, worldwide, pandemic of influenza occurred. A number of years later, several researchers reported that people who were born to mothers in the second trimester of pregnancy during the epidemic were more likely to receive a diagnosis of schizophrenia when adults (Barr, Mednick, & Munk-Jorgensen, 1990; Mednick, Huttunen, & Machon, 1994; Takei, Murray, Sham, & O'Callaghan, 1995a).

Not all studies have been unequivocal, however. McGrath, Pemberton, Welham and Murray (1994) examined the birth data of 7,858 Australian patients with schizophrenia with respect to influenza epidemics in 1954, 1957, and 1959. They found an excess of diagnoses of schizophrenia following the 1954 epidemic for men, and following the 1957 epidemic for women. The 1959 epidemic was not associated with any significant excess in diagnoses of schizophrenia. Although it is possible that mutations in the influenza viruses lead to different effects, the data are not conclusive proof. Similarly, Kunugi, Nanko, Takei, Saito, Hayashi and Kazamatsuri (1995) found an excess of diagnoses of schizophrenia only in women.

More pertinently, there are also clear negative findings. Crow and Done (1992) investigated a sample of 945 mothers who were known to have contracted influenza during the 1957 epidemic. They did not show an excess of children diagnosed with schizophrenia. Similarly, Selten and Slaets (1994) and Susser, Lin, Brown and

Lumey (1994) concluded that Dutch patients showed no relative increase in risk of developing schizophrenia following the 1957 influenza epidemic and Erlenmeyer-Kimling, Folnegovic, Hrabak-Zerjavic and Borcic (1994) reported a similar null result with respect to the 1957 epidemic in Croatia. Moreover, despite the fact that the 1944 and 1951 influenza epidemics were more serious (in terms of fatalities) than the 1957, no relationship has been demonstrated between development of schizophrenia and the earlier infections (Crow, 1994). Crow (1994) also described inconsistencies in the linkage of influenza with schizophrenia, pointing out that initial estimates of an 87% increase in incidence following influenza epidemics were later reduced to 1-2%, while the nature of any causal link is difficult or impossible to prove. Finally, although some research seems to suggest that schizophrenia is linked only with influenza, rather than other infectious diseases (O'Callaghan, Sham, Takei, Murray, Glover, Hare, et al., 1994), other researchers have directly implicated other infectious agents, most notably cat viruses (Torrey & Yolken, 1995).

Kirch (1993), reviewing more general theories of infection as possible causes of schizophrenia, concluded that there is no clear evidence of an infectious cause of schizophrenia. On the other hand, both Kunugi and colleagues (1995) and Mednick and colleagues (1994) conclude that there is substance in the hypothesis that exposure to influenza during gestation is associated with later schizophrenia. Nevertheless, this link appears to be that of a risk factor rather than a direct causal agent.

1.5.2 Social and psychosocial approaches

1.5.2.1 Social deprivation

The social environment generally is a vital part of the aetiological picture of many diseases, both physical and mental (Radley, 1994). In 1939 Faris and Dunham found that people living in deprived inner-city areas were up to seven times more likely to be given a diagnosis of schizophrenia than were people living in more affluent suburban areas. Faris and Dunham's social isolation theory (Faris, 1944) suggested that poverty and social isolation were stressors likely to trigger schizophrenia in vulnerable individuals.

A competing theory, the social drift theory (Fox, 1990), suggests that people who develop schizophrenia might be born (and develop their illness) in any socio-economic environment. However, the development of psychotic symptoms (and the effects of treatment) are, as I discussed above, likely to prove hugely disadvantageous for social functioning and employment opportunities. People therefore are likely to drift into lower socio-economic circumstances and end up in the poor housing and employment noted by Faris and Dunham. What is clear is that poverty and schizophrenia are related, but whether as cause or effect is unknown.

In this context, it is worth noting that being an immigrant (again a disadvantaged social and economic condition) is a risk factor for schizophrenia. Caribbean born immigrants and second generation Caribbeans (Cochrane & Bal, 1989) (Harrison, Owens, Holton, Neilson, & Boot, 1988) and Irish immigrants (Cochrane & Bal, 1989) have been reported as experiencing higher rates of schizophrenia and other related diagnoses. Harrison and colleagues (1988) found that the increased rates of schizophrenia were particularly high in second generation Afro-Caribbeans.

One interesting observation is that people from more advantageous

socioeconomic backgrounds appear to experience the same number of negative experiences as people in less fortunate social circumstances. Disadvantaged people, however, encounter fewer positive experiences that might be expected to buffer the negative life events and protect against psychological distress (Myers, 1975). With this background, the role of life events in the aetiology of a number of mental illnesses has been investigated.

1.5.2.3 Stress

The occurrence of negative life events (changes and losses) have been reliably linked to the onset and development of depression (Brown & Harris, 1978; Glassner & Haldipur, 1983; Tennant, Bebbington, & Hurry, 1981).

The relationship between stressful life events and schizophrenia is less clear. Early attempts to stimulate and rehabilitate withdrawn patients with primarily negative symptoms were noted to occasionally lead to a reemergence of acute psychotic symptoms (Goldberg, Schooler, Hogarty, & Roper, 1977; Kazdin, 1977). However, there does not seem to be a consistent relationship between life events (such as bereavement, divorce or being made redundant) and the development of the symptoms of schizophrenia (Paykel, 1979). Certainly such a relationship is weaker in the case of schizophrenia than depression (Tennant, et al., 1981).

Although the link between schizophrenia and life events is weaker, and possibly of a different nature, than depression, such a link does seem to exist. A number of studies (Beck & Worthen, 1972; Jacobs & Myers, 1976; Jacobs, Prusoff, & Paykel, 1974; Schwartz & Myers, 1977) have found that, compared to non-patient controls, people admitted to hospital with a diagnosis of schizophrenia reported a greater number of life events over the previous year. Such findings are, however, not conclusive. Negative findings have been reported (Al Khani, Bebbington, Watson, &

House, 1986). Moreover, as schizophrenia has an extended prodromal period, it is entirely possible that people later to be diagnosed with schizophrenia were, in the year before their admission, acting oddly. Such odd behaviour may mean that the occurrence of negative life events are partial consequences of the disorder.

Light can be shed on these relationships by examining the occurrence of life events in a more limited period immediately preceding the onset of acute symptoms. (Bebbington, Wilkins, Jones, Foerster, Murray, Toone, et al., 1993; Brown & Birley, 1968; Ventura, Nuechterlein, Lukoff, & Hardesty, 1989) have reported a significantly increased number of life events in the six week period preceding onset or relapse of psychotic symptoms. If the relationship between stressful life events and schizophrenia is limited to the few weeks before the onset of symptoms, negative findings such as those of Al Khani and colleagues (1986) may be explained. Jacobs and Myers (1976) found such a relationship over the immediate three week period, but this effect disappeared when averaged out over an entire year.

Once again, the effect of stressful life events seems to be one of a general risk factor rather than a direct causal agent. However, in the case of such life events, a more elegant psychological process may be hypothesised. If, as I shall attempt to outline below, psychological processes are important in schizophrenia generally and delusional ideation in particular, negative, stressful or ambiguous life events are likely to be a source of material that the individual needs to accept, explain and process. If the life events are salient, personally relevant and resonant with maladaptive cognitive processes, distress seems almost inevitable.

Perhaps the most powerful way to investigate this hypothesis would be to follow up observations by Goff, Brotman, Kindlon, Waites and Amico (1991), who found that 43% of 61 chronically psychotic in-patients reported abuse in their childhoods and Greenfield, Strakowski, Tohen, Batson and Kolbrener (1994), who found that 20 out of 38 patients admitted for first episode psychosis reported

childhood abuse. Since abuse is known to be associated with dissociative symptoms and personality disorders (Chu & Dill, 1990), the association of abuse with psychosis and schizophrenia is unsurprising.

1.5.2.4 Expressed Emotion

A considerable amount of work has now been focussed on the role of potentially stressful family processes in schizophrenia. In particular the concept of Expressed Emotion has been central. Brown, Carstair and Topping (1958) found that, contrary to expectations, hospital patients discharged to live with their spouse or parents were more likely to remain ill or relapse than patients who were discharged to independent accommodation. Although it turned out that this finding could not be replicated (Blumenthal, Kreisman, & O'Connor, 1982), as living alone is a risk factor itself, it did lead Brown and colleagues to examine why living with parent might be dangerous. Brown, Monck, Carstair and Wing (1962) coined the term Expressed Emotion to describe the overall emotion (positive and negative) emotional over-involvement or criticism concerning the patient elicited from a near relative or spouse. The Camberwell Family Interview (Brown & Rutter, 1966) was explicitly designed to measure Expressed Emotion. Studies using the Camberwell Family Interview have systematically found that close contact with relatives showing high Expressed Emotion is a major risk factor in schizophrenia. For example, Vaughn and Leff (1976) found that close contact (more than 35 hours a week of face to face contatct)with high Expressed Emotion was associated with higher relapse rates.

The causes and nature of high Expressed Emotion remain unclear. The level of observed Expressed Emotion is not necessarily stable (Tarrier, Barrowclough, Vaughn, Bamrah, Porceddu, Watts and Freeman, 1988), and may reflect a response

by carers to a family member with schizophrenia (Birchwood & Smith, 1987; Macmillan, Gold, Crow, Johnson, & Johnstone, 1986). Of particular interest for this thesis is the observation that Expressed Emotion reflects the causal attributions of family members. Brewin, MacCarthy, Duda and Vaughn (1991) found that high, negative, Expressed Emotion was correlated with a tendency by family members to attribute the blame for symptomatic problems within the patient. Thus, if abnormal behaviour is attributed to characteristics of the patient, and seen as potentially controllable, blame and negative emotions will follow.

Because the research findings linking family difficulties and relapse are so strong, a large number of family interventions have attempted to reduce Expressed Emotion and thus reduce relapse rates. These have been highly successful (Falloon, Boyd, McGill, Razani, Moss, & Gilderman, 1982; Leff, Kuipers, Berkowitz, Ebstein-Vries, & Sturgeon, 1982; Tarrier, Barrowclough, Porceddu, & Fitzpatrick, 1994), with new variants of interventions being developed each year (Mueser, Gingerich, & Rosenthal, 1994). Such findings are not particularly good evidence to support a claim that such family processes cause schizophrenia, but they do indicate that such dynamics might maintain or exacerbate difficulties.

1.5.3 Psychological approaches

1.5.3.1 Freud and Freudians

Freud's personality theory is relatively well known. Of particular interest in the discussion of schizophrenia are his theories of defense mechanisms. Essentially Freud regarded schizophrenia as a disorder caused by regression to an early narcissistic stage in which the difference between oneself and the external world is lost (Freud, 1914/1957). Such regression was thought to be precipitated by a defence against

traumatic experiences. In such a state normal object relations are lost, replaced by fantasies and denial. In some circumstances, according to Freud (1924/1957), the patient attempts to re-build object relations, leading to 'world reconstruction fantasies' such as some hallucinations and delusions. Freud discussed delusions of persecution in much greater detail than he did schizophrenia. Freud's theories of paranoia will be discussed later.

Other psychoanalysts (Ferden, 1952) differed from Freud in several theoretical details concerning the relationship between the id and the ego. In Fernden's view, schizophrenia is typified by a breakdown of ego boundaries. This means that psychological distinctions between the self and other people or between fantasy and reality are lacking, leading to psychotic phenomena. Klein (1946), in a similar argument, viewed schizophrenia as a regression to a 'schizoid' position. In such a state, the ego tends to fragment.

As with almost all other Freudian models of psychology, these accounts of schizophrenia can be criticised on scientific grounds, as they fail Popper's (1968) criterion of testability. That is, they do not, as a rule, contain within them any means by which they could be proved false. As such, Popper would have contended, they are worthless. The other problem with such approaches, even if one finds them attractive, is that they must remain descriptive. To talk of 'a breakdown of ego boundaries' is an implicit evocation of a process, but is not an explanation.

1.5.3.2 Jung

Jung was a student of Freud (described by the latter as his most promising pupil) who later left the Freudian circle following theoretical differences over the nature of libido - seen by Jung as a non-sexual life-force. This life-force echoes

Jung's concept of a collective unconscious. That is, Jung (Jung, 1964) believed that the depths of human unconscious are common to all. He believed that the dreams and fantasies reflecting a person's deep unconscious are shared, believing that we are (symbolically and literally) joined in one collective unconscious entity, harking back to our primitive proto-human past.

Jung's personality structure was importantly different from Freud's. In particular he believed that we each possess an 'anima' or 'animus'. These are our inner selves, masculine for women and feminine for men, and reflect our complex and multifaceted personality. Personality can develop unevenly, Jung believed, causing distress and psychiatric disturbance. Putting the patient in touch with the collective unconscious, and their anima/animus, represents a restoring of healing harmony and balance, evening out the facets of the personality (Jung, 1964).

Although Jung's writings are particularly difficult to interpret and paraphrase, he has suggested two (possibly not mutually exclusive) routes to schizophrenia. First, the unconscious contains symbolic material, including multiple magical figures, from our proto-human past. If the personality disintegrates, this material is visible in parallel with consciousness interpreted as schizophrenia (Jung, 1968) (pp 88-89). Jung, however also appears to view schizophrenia as process which can be either "functional or real" (p.337) that leads to a disintegration of the personality and reveals the "psychic substratum" (p.336). This latter process appears to be a surprising acceptance of traditional psychiatry.

1.5.3.3 Laing

Laing's psychology was primarily phenomenological - to Laing the concept of external reality was of lesser importance. In schizophrenia, in particular, whether an

individual is perceiving the world 'correctly' is less important than understanding the deeper structure and meaning of how that individual sees the world (Laing, 1961; 1967). Central to Laing's theory of schizophrenia was the concept of 'alienation' (Laing, 1965). He believed that conflicting demands (often stemming from the patient's family) lead to the creation of a false self. Consequently the individual loses touch with their real self, loses touch with reality and therefore exhibits psychotic feelings about the real self - hallucinations and delusions.

As an indirect result of such a model, Laing regarded conventional psychiatric diagnosis as a flawed attempt to be 'objective', and merely reflected social judgements made about unconventional individuals (Laing, 1967). Rather than psychiatric hospitals and attempts to persuade the individual to forget and repress their psychotic experiences, Laing believed sufferers need to close the split between false and real selves. Therefore they need a supportive, warm, environment where their feeling can be understood and tolerated. In conventional treatment settings, abnormal behaviour is not tolerated, patients cannot be themselves, and vitally for Laing, cannot talk about their delusions and hallucinations. This means they can't express themselves, can't close the gap between real and false selves (this gap perhaps being increased), and therefore can't recover. Laing therefore established therapeutic communities, treatment settings where tolerance and acceptance were used to allow integration rather than repression of psychosis.

In his later writing, Laing clearly viewed a person experiencing psychosis as a pioneer, preempting modern 'New Age' approaches. Laing believed psychotic experiences offer an insight to deeper levels of experiences, and cited outstanding figures from history - such as Joan of Arc, saints, mystics and Socrates - who all seemed to benefit from psychosis (Laing, 1965).

Laing made a distinction between ontologically secure and ontologically insecure individuals (Laing, 1965), where security was felt to reflect a set of life

circumstances leading to a person having a positive sense of self and their place in the world. He further defined three forms of insecurity (Laing, 1965); engulfment, being swallowed into other people's personality, implosion, being empty inside and unable to stand up to others, or petrification, being unable to resist other people's demands. From such ontological insecurity alienation ensues.

1.5.3.4 Bateson's Double Bind

The most popular or well-known of the alleged routes to ontological insecurity is Bateson's Double Bind (Bateson, Jackson, Haley, & Weakland, 1956). This is defined as involving: i) two or more people, ii) repeated experience, iii) a primary negative injunction (such as "do this or I will punish you"), iv) a secondary injunction (such as "and don't see me as a punishing parent") and v) an injunction forbidding escape. The consequence of being trapped in such a double bind is that one cannot react normally, and one cannot escape. Madness is the only sane response.

In support of such an account, Wynne, Ryckoff, Day and Hirsch (1958) reported how disturbed families pretend to be well, pretend to be normal, while (Laing, 1961) commented that family members who don't join in with the game of pretending to be well are labelled as "sick". Although such approaches have the historical benefit of leading to the development of family therapy, they lack the vital component of supportive evidence. Both Wynne and colleagues (1958) and Laing (1961) seem to be presenting their own double bind. The family is disturbed, therefore lack of obvious pathology is evidence that they are 'pretending" to be well. Disturbance would, presumably, be evidence of pathology, sanity is also confirmation.

1.5.3.5 Behaviourism

In addition to psychoanalytical and psychodynamic approaches, behavioural models of mental illness have also been popular. As seems quite typical for behavioural approaches, therapeutic success seems greater than explanatory power. Although a number of behavioural models have been proposed, two in particular are worthy of study.

Salzinger (1973) noted how animals (especially pigeons) can become superstitious. That is, when rewarded on a schedule unknown to the recipient, animals may exhibit idiosyncratic patterns of behaviour that are extremely resilient. Behaviours, occurring by happenstance at the same time as non-contingent reward, become reinforced by what seems to be a straightforward application of the Law of Effect. Salzinger suggests that this may explain a lack of subjective or objective contact with reality. Similarly, delusional beliefs were believed to be the result of a conditioned link between a (real) reinforcer and a (chance) stimulus. An example of this would be the presence of a nun in full religious regalia at a time when an individual slipped and fell. A consequent delusional belief may arise in this context, such as 'God is punishing me for my sins". Finally, Salzinger suggests that hallucinations occur when a conditioned stimulus triggers perception in the absence of the perceptual object.

Ullmann and Krasner (1975) proposed a less basic behavioural model of schizophrenia. The premise behind their model was the what they termed a 'failure of reinforcement' (p.357). This means that, in schizophrenia, the normal discriminant stimuli, that signal when reinforcement is likely (and when it is not), are absent or dysfunctional. Normality, it is assumed, is synonymous with responding normally to discriminant stimuli. Individuals who do not respond in the normal manner to such stimuli, might appear uninterested or unresponsive. Normal behaviour will, of course,

be distorted as cues for action (discriminant stimuli) will be distorted. For the individual, normal behaviour will be difficult to understand, and may become the subject of delusional interpretations.

Since discriminant stimuli are signals for reinforcement, Ullmann and Krasner also attempted to explain later, chronic and negative symptoms. Normal contingencies for reinforcement will be lost, and patients will settle into an alternative pattern of behaviour. Because of the common treatment approaches, Ullmann and Krasner suggest that these new patterns of reinforcement will lead to both idiosyncratic and socially withdrawn behaviour. Finally, in a withdrawn and isolated state, the patient will further lose or respond abnormally to discriminant stimuli, losing external stimuli and developing idiosyncratic internal ones.

As a model, this is efficient and elegant, but proof is difficult to find. Both Salzinger (1973) and Ullmann and Krasner (1975) support their theses with reference to previous studies of behavioural learning and the epidemiology and phenomenology of schizophrenia, reworking these data in a behavioural framework. More telling evidence comes from a number of behavioural interventions in schizophrenia. Thus desensitization, (on the rationale that hallucinations are precipitated by stress and anxiety) (Nydegger, 1972; Slade, 1972), self-management (Alford, 1986; Alford, Fleece, & Rothblum, 1982; Falloon & Talbot, 1981), contingency management (removing reinforcers following symptom expression, offering reinforcers following incompatible positive behaviours) (Ayllon & Kandel, 1976; Nydegger, 1972) and others have been used (see Birchwood, et al., 1988 for a review).

Such interventions are often successful, but results are not always generalized or maintained (Davis, Wallace, Liberman, & Finch, 1976; Lambley, 1973; Siegel, 1975). Moreover, Wincze, Leitenberg and Agras (1972) and Marzillier and Birchwood (1981) comment that reductions in target behaviour cannot be considered synonymous with curing underlying psychopathology. In particular, Wincze and

colleagues (1972) commented that, while operant techniques reduced spontaneous expressions of pathology, delusions were still evident if patients were asked directly about their beliefs. Behavioural techniques and models alone therefore appear to be useful therapeutically, but do not offer a complete picture of the aetiology or phenomenology of schizophrenia.

1.5.3.6 Cognitive Processes

Despite the popularity of psychoanalytical and psychodynamic theories in the post-war period, and the subsequent popularity of behaviourism, such approaches have largely decreased in popularity. More recent studies have focussed on cognitive processes (attention, perception, memory, etc.) and have attempted to link these with schizophrenia. In the main, such cognitive approaches to psychopathology will be discussed in Chapter 2, when I will discuss cognitive accounts of delusional thinking. However cognitive theorists have also attempted to explain schizophrenia as a disorder, or at least attempted to use cognitive approaches to explain major schizophrenic syndromes.

1.5.3.6.1 IQ

Generally, studies have found that IQ is measurably lower in people who will later develop schizophrenia than in their peers or siblings (Lane & Albee, 1968; Offord & Cross, 1971; Watt & Lubensky, 1976). Interestingly, this may only be true of men (Aylward, Walker, & Bettes, 1984). There is a suggestion that this pattern of low IQ is most marked in early-onset schizophrenia, associated with neurological signs and

brain abnormalities (Jones, Guth, Lewis, & Murray, 1994a). On other hand, several studies have failed to find an association between low IQ and later onset schizophrenia (Castle & Murray, 1992; Kay, 1963; Kay & Roth, 1961; Post, 1982). Jones and colleagues (1994a) suggest that these IQ findings are a reflection of a neurodevelopmental syndrome, accounting for at least some cases of schizophrenia. Once again, however, such findings seem to support the concept of general, non-specific risk factors.

1.5.3.6.2 Attention

Clinical observations of patients with schizophrenia have frequently focussed on attentional abnormalities (Cutting, 1989). Although perception and attention have always been closely related, Matussek's (1952) account of a patient who reported that "I look at a garden, but ... I can only concentrate on details" (p. 92) seems to be closer to an attentional than a perceptual problem. In particular a number of researchers (see Hemsley, 1987) have pointed to a tendency for the cognitive or intellectual performance of patients with a diagnosis of schizophrenia to be disrupted by the intrusion into consciousness of material normally ignored. It may be that such disruption could explain the pattern of low IQ referred to above. Hemsley (1994) has further suggested that, as well as intruding, items are brought to attentional awareness in an isolated or fragmentary form. This echoes earlier accounts of attentional difficulties in schizophrenia, worded in terms of Gestalt psychology (Matussek, 1952). Although the claim has been made for many other processes, there have been claims that deficits in selective attention are the major cause of cognitive dysfunction in schizophrenia (Neuchterlein & Dawson, 1984b).

One particular paradigm used to examine such attentional intrusions is latent

inhibition. In this technique a participant is first presented with repeated instances of, for instance, stimulus A, then is later asked to learn an association between A and C. Learning the second association is more difficult than would be the case without the previous presentation of A. Unusually, patients with schizophrenia do better in latent inhibition tasks than non-patient participants (see Hemsley, 1994). Hemsley and colleagues conclude that this success is due to the failure of patients with schizophrenia to use the "past regularities" (Hemsley, 1994; p.97) of experience to regulate cognitive processing. Out of this, a major theory of the genesis of a number of schizophrenic symptoms has been developed (Hemsley, 1987).

The idea of a failure in the use of "past regularities" of experience has some powerful aspects, especially in the area of abnormal cognitive processes. However as an explanation of hallucinations and delusions it seems somewhat lacking. In particular, if patients are presumed to be over-general in their focus of attention, and to fail to understand patterns and regularities of experience, it is a little difficult to explain their focussed and persistent delusional ideas, and their systematic selective attention for material related to their delusions (which I shall refer to later).

1.5.3.6.3 Perception

Disorders of perception have also been suggested as a possible cause of schizophrenia. Maher (Maher, 1988; 1992; Maher & Ross, 1984) has suggested that perceptual abnormalities might lead to delusional thinking, and this issue will be dealt with in Chapter 2. More generally, however, perceptual abnormalities have been implicated in the broader concept of schizophrenia.

Neuchterlein, Buschbaum and Dawson (1994) used a signal-detection paradigm to investigate possible basic perceptual abnormalities in patients with

schizophrenia. They reported that patients with schizophrenia were significantly less skilled at perceiving correctly stimuli in degraded form. The fact that this deficit was present even when symptomatology had lifted was interpreted as evidence of a persistent cognitive vulnerability factor.

In a series of experiments bridging attention and perception, Green and Neuchterlein (1994) used the technique of backward masking to investigate basic perceptual processes. Backward masking consists of presenting one (target) stimulus tachistoscopically, followed by another - the mask. The target stimulus is typically a number or letter, the mask is a pattern. Green and Neuchterlein (1994) report deficits by patients with schizophrenia whereby a longer interval between the target and the mask is required for successful perception. This has been interpreted as indicating that schizophrenia is associated with slowed information processing (Branff, 1981). Interestingly this deficit seems to be reduced following the administration of neuroleptic medication (Branff & Saccuzzo, 1982).

Clearly there are very close links between attention and perception. The model of selective attention deficits in schizophrenia (Hemsley, 1987; Hemsley, 1994) also encompasses perceptual abnormalities. If, it is hypothesised, the stored regularities of experience are not used to the same extent in directing attention, perception is likely to be structured more by the characteristics of the sensory input than by previous expectations and contextual cues. Stimuli are likely to enter consciousness and be assessed in terms of significance and implications for action on the basis of their phenomenological characteristics. Once again, however, this picture does not seem to fit with a clinical picture of hallucinations or delusions, where experiences are expressly not appraised anew on each occasion, but are routinely interpreted in terms of their delusional beliefs or explanatory frameworks concerning their hallucinations. The stored regularities of experience seem rather strong.

1.5.3.6.4 Memory

It has been claimed that evidence of episodic memory impairment in schizophrenia is not only "well established" (McKenna, Mortimer, & Hodges, 1994; p.163), but "the leading neuropsychological deficit [in schizophrenia]" (p.169). Specifically, impairments in episodic memory have been reported in both chronic (Cutting, 1985) and acute patients (McKenna, Tamlyn, Lund, Mortimer, Hammond, & Baddeley, 1990). These problems appear to be associated with recall and recognition, with a relative sparing of short-term memory (Tamlyn, McKenna, Mortimer, Lund, Hammond, & Baddeley, 1992).

In addition, schizophrenia seems to be associated with an impairment of semantic memory. Koh (1978) found that, when patients with schizophrenia were asked to categorize words (such as lake, hill, valley, canyon, book, table), they tended to use fewer categories. In a similar approach, Tamlyn and colleagues (1992) found that patients with schizophrenia were slow and error prone when judging the falsity of 'silly sentences' ("Desks wear clothes"). McKenna and colleagues (1994) also report a poor performance by patients with schizophrenia on a verbal categorical sorting task. This they interpret as a reflection of over-inclusive thinking. This is a tendency for concepts to be broad, vague and overlapping, and has been suggested as the defining feature of schizophrenic thought disorder.

Finally, working memory deficits have been hypothesised as central to schizophrenia (Flemming, Goldberg, & Gold, 1994). Working memory is a concept comprising a number of cognitive processes that involve the storage and processing of current information. It is supposed (Baddeley, 1986; Baddeley, 1992) to comprise a cental executive (coordinating information processing) and two "slave systems" a verbal articulatory loop and a visuospatial scratch-pad. Although few specific tests of working memory have been reported, Flemming and colleagues (1994) re-evaluate a

number of experimental studies in terms of deficits in working memory. In this context, attentional deficits such as those outlined above could reflect disturbance of the central executive - responsible for filtering information and allocating processing. More empirical studies of the nature of working memory deficits in schizophrenia may prove informative.

As with all other systems, memory seems affected in schizophrenia. However, once again the nature and significance of these disturbances are unclear. The paucity of such findings as explanations is illustrated by the claim by Cutting and Murphy (1988) - that symptoms of schizophrenia reflect deficiencies in "real world knowledge". Of course, if you believe that you are being followed by members of the KGB, and you are not, your real world knowledge is deficient. Similarly, if I hear, or remember hearing the voice of God, my knowledge of the real world is presumably lacking. To state the obvious in jargon, however, does not pass for an explanation.

1.5.3.6.5 Theory-of-Mind Deficits

Chris Frith (Frith, 1994) has suggested that a deficit or abnormality in what is termed Theory of Mind may explain a number of schizophrenic symptoms. We do not, as a rule, appraise other people's actions and conversation as simple behavioural or verbal patterns. Rather, we assume that we can use behaviour to divine wishes, hopes, beliefs, intentions etc. Such an understanding is termed Theory of Mind, and is believed to be vital for successful social intercourse (Premack & Woodruff, 1978).

Theory of Mind deficits have been seen as possible explanations of the difficulties in communication, imagination and social relationships characteristic of autism (Baron-Cohen, Tager-Flusberg, & Cohen, 1993; Happé & Frith, 1994; Leslie, 1987). Thus, even if an item is hidden from view, autistic children who are aware of

the hiding place tend to suggest that others will look for hidden objects in the correct location (Baron-Cohen, Leslie, & Frith, 1985). This is strongly suggestive of the fact that children with autism fail to understand correctly the mental processes of others.

The role of Theory of Mind deficits in schizophrenia itself has been less thoroughly explored. Pilowsky and Bassett (1980) report that people with schizophrenia tend to use more physical than temperamental descriptors, consistent with a lower level of use of Theory of Mind. Frith (1994) has also suggested that Theory of Mind deficits may be related to flattening and impoverishment of affect, as patients will be unable to recognize and address their own or other people's emotions. Rochester and Martin (1979) discuss how schizophrenic thought disorder may, in part, be caused by a lack of referents in speech. These referents (definite and indefinite articles such as; the, it, an, he, etc,) are used in normal speech to let listeners know to whom and to what the speaker is referring. A lack of referents in speech suggests that an understanding of the needs of the listener is lacking, perhaps because of Theory of Mind deficits (Frith, 1994).

Positive symptoms (hallucinations and delusions) have also been linked to Theory of Mind deficits. Frith (Frith, 1987; Frith & Done, 1989) has suggested that passivity experiences (delusions of being controlled or of thought disorder) reflect a deficit in one's central monitoring of action, essentially a Theory of Mind concerning one's own mental states. Similarly the experiences of thought insertion and hallucinations can be explained, it is believed, by Theory of Mind deficits concerning one's own mind (I don't realize that I am thinking) (Frith, 1994). Other psychotic phenomena, such as paranoia, are believed to reflect Theory of Mind deficits concerning the mental states of other people.

Overall, the Theory of Mind approach to schizophrenia is elegant and persuasive. Three problems remain however. One difficulty is the possible differences between a deficit in one's ability to understand one's own mental processes and a

deficit in understanding the mind of others. Frith (1994) does not discuss this issue in depth, but the distinction may be important. Secondly, in autism, where the presence of a Theory of Mind deficit seems highly likely (Frith, 1989; Frith, Morton, & Leslie, 1991; Leslie, 1991) clear and unambiguous schizophrenic symptoms are usually absent, while in schizophrenia, autistic problems are frequently absent. This suggests that putative Theory of Mind deficits must be partial or gradual. This is consistent with the observation that ToM abnormalities in schizophrenia appear to be present only during acute, symptomatic, phases of illness (Corcoran, Mercer, & Frith, 1995). This issue has not been further investigated. Finally, if Theory of Mind problems are important in schizophrenia, they are likely to achieve their effects through other cognitive processes. This point will be addressed later in this thesis.

1.5.4 Aetiological complexity

Despite this wealth of research, there seem few firm conclusions. One explanation for this lack of clarity may be the dubious nature of the concept of schizophrenia itself. If schizophrenia is an invalid concept, it may not be surprising that no single, clear, aetiological picture emerges.

There seem to be genetic, biological, social and psychological routes to schizophrenia. No one route dominates, rather many factors seem to raise the risk of developing schizophrenia. There are a number of possible models of explanation that may shed light on the issue. Before we can understand the precise relationship between the many possible aetiological factors and the many schizophrenic phenomena, a clear conceptual model of schizophrenia is vital.

1.6 Conceptual approaches to schizophrenia

Perhaps because there is little evidence of a single, clear, aetiological route to schizophrenia, the nature of the postulated disorder itself has come into question. A number of different theoretical models of schizophrenia have been proposed. These reflect differences between researchers in the favoured aetiological route(s), with some (such as Freud and the behaviourists) proposing that schizophrenia is a disorder caused by psychological processes, while others (such as the genetic and viral theorists) propose that schizophrenia is a purely biological disorder.

In addition, and orthogonally to this, there is debate over the concept of schizophrenia as a disorder at all. Some theorists maintain that schizophrenia is a unitary, single disease with a single aetiology. Others suggest that it is a single disease, but one with a heterogenous aetiology. Still others believe that 'Schizophrenia' does not exist; that there is no overall structure linking disparate symptoms and syndromes. The contrasts between such models are important, since, as I shall outline below, patterns of research methodology differ according to the conceptual model used.

1.6.1 Schizophrenia as a distinct illness

Most authorities accept that the aetiology of schizophrenia is both complex and, at present, unclear. Many also believe, however, that this confusion will soon be resolved (Johnstone, 1993). In such unitary disease models, schizophrenia is often seen as an organic, biological condition. Johnstone (1993) suggests that the evidence for a biological basis of schizophrenia is strong. She also argues that a biological illness model of schizophrenia, even if incomplete or simplistic, is useful for

psychiatrists, patients and relatives.

Despite this optimistic or confident approach, however, the issue of the validity of such an approach is important. It may be comforting to have a simple model of schizophrenia, and it may be comfortable to see schizophrenia as a primarily physiological illness, but such comfort must be weighed against the scientific doubts as to the correctness of such a unitary disease concept.

1.6.2 Validity issues

Scientific concepts are usually accepted if they are both reliable and valid, with reliability being a necessary but insufficient criterion for validity (Spitzer & Fliess, 1974). As discussed above (section 1.2) researchers and clinicians have addressed the issue of reliable diagnoses of schizophrenia by developing precise operational criteria such as DSM-IV and ICD-10. Although different operational criteria tend to lead to slightly different diagnoses, each system appears to be reliable (Brockington, et al., 1978; Farmer, Jones, Williams, & McGuffin, 1993; McGorry, Singh, Connell, McKenzie, van Riel, & Copolov, 1992; McGuffin, Farmer, & Harvey, 1991).

The reliability of psychiatric diagnosis of schizophrenia is welcome, but does not necessarily address the issue of validity. The fact that different authors have emphasized different symptoms of schizophrenia, suggests that there may be disjunctions within a single concept, threatening scientific validity. This pattern; highly reliable definitions of slightly different collections of symptoms each given the same name, led Brockington and colleagues (1978) to refer to "a babble of precise but differing formulations of the same concept" (p.387).

A number of researchers, notably Richard Bentall, have discussed the poor scientific validity of schizophrenia (Bentall, 1990; Bentall, 1993; Bentall, et al.,

1988). Three main arguments emerge weakening the construct validity of 'schizophrenia'. First, the common symptoms of schizophrenia do not seem to cluster together. Blashfield (1984) and Bentall and colleagues (1988) reviewed a number of classification systems for schizophrenia, finding that no general cluster consistent with the concept of schizophrenia emerged from studies using multivariate statistical techniques. Second, the course of schizophrenia appears to be extremely variable and dependent upon social factors rather than on presenting symptoms (Ciompi, 1984; Sartorius, Jablensky, Ernberg, Leff, Korten, & Gulibant, 1987). This is important since a valid concept should show uniform properties, but also because it weakens the claim that schizophrenia, whatever it is, is an organic illness. Finally, patients with a diagnosis of schizophrenia do not respond to neuroleptic medication in a uniform fashion. A limited number of studies have been conducted in which psychiatric medication has been randomly prescribed to all patients (i.e. those with diagnoses of schizophrenia and with other diagnoses). In such studies, response to neuroleptic medication is neither specific to schizophrenia, nor uniform for all those with such a diagnosis (Kendell, 1989).

1.6.3 Subtypes of schizophrenia

The idea that schizophrenia may comprise two or more subtypes is a potential response to such discussions of validity. Tim Crow has suggested that schizophrenia comprises two subtypes; Type I and Type II (Crow, 1980). In this formulation, Type I schizophrenia consists of positive symptoms: hallucinations, delusions, bizarre behaviour and thought disorder. In Type I schizophrenia, symptoms respond well to neuroleptic treatment and there is no intellectual impairment. Crow (1980) has suggested that Type I schizophrenia is caused by overactivity of dopamine receptors.

In contrast, Type II schizophrenia consists of negative and chronic symptoms: blunted affect, withdrawal, amotivation and poverty of speech. These symptoms do not to respond well to neuroleptic treatment (Angrist, Rotrosen, & Gershon, 1980; Angst, Stassen, & Woggon, 1989). Crow (1980) suggested that Type II schizophrenia is caused by cell loss and structural changes within the brain.

Although there seem to be subjective as well as objective differences between individual experiences of schizophrenia (Peralta & Cuesta, 1992), Crow's hypothesis has been criticised as simplistic (De Leon, Simpson, & Peralta, 1992). In fact, in a factor analysis of phenomenological reports from patients (Liddle, 1987), three factors were identified. Two of these might bee seen as supportive of Crow's model. However, a third factor - cognitive disorganization - also emerged. Such a three-way distinction has been supported in other studies with both clinical (Andreasen, Arndt, Alliger, Miller, & Flaum, 1995a; Andreasen, Arndt, Miller, Flaum, & Nopoulos, 1995b; Frith, 1992; Klimidis, Stuart, Minas, Copolov, & Singh, 1993; Liddle, 1987; Minas, Stuart, Klimidis, Jackson, Singh, & Copolov, 1992) and nonpatient samples (Bentall, Claridge, & Slade, 1989; Chapman, Chapman, & Miller, 1982), although von Knorring & Lindstrom (1995) suggested a five factor (positive, negative, excited, anxious/depressive and cognitive) model. As a two factor subtype model, Tim Crow's approach appears attractive. As the number of dimensions included in a model of schizophrenia grows, however, the more schizophrenia appears an invalid concept.

1.6.4 Stress-vulnerability model

In attempts to address the issues of causation and validity in schizophrenia, many researchers have suggested a stress-diathesis (Shields, 1978) or stress-vulnerability (Neuchterlein & Dawson, 1984a) model. In these models, schizophrenia

is not seen purely as a physical illness, although genetic, neurological or biochemical factors are accepted as important. Similarly schizophrenia is not seen as a purely psychological or social phenomenon, although such factors are also seen as important. The stress-vulnerability model proposes that schizophrenia develops when environmental or psychosocial stressors interact with personal vulnerability (Neuchterlein & Dawson, 1984a; Zubin & Spring, 1977). Although some psychologists have discussed this vulnerability in terms of psychological variables, in the original stress-vulnerability model vulnerability is seen as genetic, neurological and neurochemical.

Neuchterlein and Dawson (1984a) and Neuchterlein, Dawson, Ventura, Fogelson, Gitlin and Mintz (1991) have suggested that neurological and cognitive deficits can be characterised as 'Stable vulnerability indicators', 'Mediating vulnerability factors' and 'Episode indicators'. Stable vulnerability factors are factors which remain abnormal in individuals with a diagnosis of schizophrenia even if their illness has remitted. Episode indicators are abnormalities which are present during phases of active symptoms, but which are absent during times of remission. Such variables are seen as important markers of illness, and worthy of treatment, but are not seen as indicators of continuing vulnerability. Mediating vulnerability factors are perhaps the most complex, as they are thought to be abnormalities which, although most severely present during active symptom phases, do not disappear during remission.

Neuchterlein and colleagues have uncovered a number of factors that seem to fit into the general model of stress-vulnerability, especially vigilance and attention (Neuchterlein, 1991; Neuchterlein, et al., 1994; Neuchterlein & Dawson, 1984b). Rather than claiming that such abnormalities are direct causes of schizophrenia, Neuchterlein and his colleagues incorporate these findings within a wider context. Essentially the stress-vulnerability model seems to permit the integration of a wide

variety of factors. Of particular interest in this thesis is the idea that psychological, information processing variables (attention, attribution and self-representations) might be mediating vulnerability factors.

The stress-vulnerability model has potential clinical significance in addition to its theoretical utility. Clinically, both stable and mediating vulnerability factors could be identified in people before episodes of illness. If such factors can be identified, it is argued (Neuchterlein, et al., 1994) that people prone to schizophrenia may also be identified and offered remedial or prophylactic treatment, coping strategies or environmental changes to prevent relapse. It has also been suggested that psychological treatment of underlying cognitive deficits would be of benefit (Neuchterlein, et al., 1994).

1.6.5 Schizotypy

Meehl (1989; 1990) has suggested a conceptual approach very similar to the stress-vulnerability model. He proposed the existence of "schizotaxia", a genetic predisposition that could develop into schizophrenia when the individual was faced with stress. In an environment free from stress, however, this predisposition would display itself as schizotypy, cognitive or behavioural traits similar to psychotic symptoms in reduced intensity. Schizotypy may be present in individuals with no diagnosis of schizophrenia, highly creative individuals, religious or political fanatics or individuals who are highly mystic. The contrast between the stress-vulnerability model and the schizotaxia/schizotypy model is small. In the stress-vulnerability model, residual traces of the intrinsic biological or constitutional vulnerability is evidenced by cognitive abnormalities or information processing deficits, in the schizotaxia/schizotypy model, by oddities of behaviour and thinking.

Claridge (1990) has compared schizotypal personality with high blood pressure. With hypertension, no clear distinction can be made between the well and the ill, as blood pressure varies in a continuous manner. When blood pressure is high, however, the danger of developing a physical illness is also high. Moreover, in this example, environmental factors such as stress, a high fat diet, smoking and alcohol use can all lead to an increased risk of heart disease. Claridge (1990) compares this with schizotypy, where some biological vulnerability is evident as oddities of behaviour and thought, and where life stresses increase the risk of receiving a diagnosis of schizophrenia.

1.6.6 Szasz and Social Constructionism

The notion of social constructionism (the idea that concepts such as schizophrenia are better understood as socially constructed concepts rather than organic disease entities) is associated primarily with the name of Szasz. Szasz has taken the view that the concepts of mental illness in general and schizophrenia in particular, are not only invalid, but also positively harmful (Szasz, 1973). Szasz argues that conventional illnesses are physiological conditions with physical symptoms. Szasz suggests that psychiatric illnesses lack the central characteristics of illness. That is, they are primarily non-physiological conditions where the symptoms are an individual's feelings, beliefs and perceptions.

Szasz also points out that psychological or psychiatric symptoms are impossible to dissociate from social, cultural, ethical and even legal contexts. Moreover such symptoms are labelled by Szasz as value judgements, based on whether the perceptions, emotions and beliefs of the patient differ from that of the observer.

Szasz proceeds to argue that the function of a diagnosis such as schizophrenia is to allow social control of deviant individuals. It would be wrong to assume that Szasz does not think that a problem exists when a diagnosis of schizophrenia is made. Rather , Szasz views schizophrenia as a problem in living or coping with social intercourse. Szasz makes two points; that personal unhappiness and socially deviant behaviour certainly exist but that these phenomena should not be called an illness. It is perhaps unfortunate that his stated views that, for instance, to believe in mental illness is as ludicrous as to believe in daemonic possession, has clouded these more reasonable positions. In particular the view that labelling tends to deny the humanity of a person in distress seems important.

The views of Szasz have always been highly controversial. Stripping away the rhetoric, however, Szasz's basic point, that the symptoms of schizophrenia are entirely social in origin seems untenable given the enormous number of findings of real and important abnormalities outlined above.

1.6.7 Phenomenological approach

In contrast to the majority of this chapter, a powerful set of arguments have been marshalled supporting the idea that the lack of validity of diagnosis of schizophrenia has hindered research. Instead of researching vast and invalid concepts such as 'schizophrenia', it is argued that study of individual symptoms (Bentall, et al., 1988; Persons, 1986; Slade & Cooper, 1979) is likely to be more profitable.

The theoretical grounds for such an approach are the lack of validity of broader diagnostic concepts, and the knowledge that individual symptoms can be operationally defined with reliable precision. The value of such an approach can be evaluated by results. If a phenomenological approach is valid, interesting, replicable and coherent

results should follow. Many of the results of such an approach are outlined in subsequent chapters of this thesis.

1.7 Conclusions

Schizophrenia is a concept so massive in its scale, and subject to so much research, that simple conclusions are extremely unwise. A few things seem clear. Psychiatric problems falling under the broad umbrella of schizophrenia are common and serious. Such problems have been well known for centuries. They have a massive impact on the lives of individuals and cause a important social and personal problem. No clear causes or aetiologies have emerged from many years of study. It does seem that events which impact negatively on the brain lead to an increased likelihood of an individual developing such problems. Similarly many psychological, social and family problems seem to be risk factors. Finally, there is evidence that personal or constitutional factors are important, possibly genetically transmitted.

It has been suggested that the relative lack of progress despite the volume of research may be due to conceptual errors. If schizophrenia has multiple aetiologies, and is itself a loose collection of different phenomena, it is not surprising that no single conclusion about nature and cause can be made. For the reasons listed in each section above, it is my belief that the phenomenological approach is the most appropriate research strategy. The remainder of this thesis will concentrate on the phenomenological approach, studying delusions of persecution as objects of research in their own right.

Chapter 2.

Delusions

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2.1 Introduction

Chapter 1 reviewed the status of schizophrenia as a general syndrome. Doubt about the validity of such a concept suggests that individual symptoms should be the focus of study. Delusions of persecution and paranoia will be discussed in Chapter 3, this chapter is concerned with delusions in general.

2.2 History of the concept of delusion

Although Hare (1983) has suggested that psychotic illnesses are essentially a modern phenomenon, medieval writers clearly described psychotic phenomena. It seems clear, for instance, from the quotation from Bartholomaeous Anglicus in Chapter 1, that what we would now call delusions have been noticed and recorded for centuries. However it is important to distinguish between references in historical sources to phenomena that we would now term delusional beliefs, and contemporary descriptions of false beliefs recognised as such at the time. For example, Zilboorg (1941), discusses the accounts of psychotic phenomena in Greek and Roman sources. Although Zilboorg regards the contemporary descriptions as coherent and clear, there is little evidence that abnormal beliefs (rather than emotions or behaviour) were regarded as central. Most of these phenomena are described in behavioural and emotional terms.

A good example of early descriptions of delusions and of the manner in which beliefs and emotional issues were confounded comes from the writings of Aulus Cornelius (25BC-). Aulus Cornelius, a Roman gentleman and observer of contemporary life used the pen-name Celsus, and described what may be depressive delusions as follows:

> "But insanity is really there when a continuous dementia begins, when the patient, although up till then in his senses, yet entertains certain vague imaginings; the insanity becomes established when the mind becomes at the mercy of such imaginings.....Some need to have empty fears relieved, as was done for a wealthy man in dread of starvation, to whom pretended legacies were from time to time announced." ... "More often however, the patient is to be agreed with rather than opposed, and his mind slowly and imperceptibly turned from the irrational talk to something better." De medicina (Celsus, 15/1935; p.289)

Such an account appears reasonably clear, and appears to be a description of abnormal beliefs. However it is also clear that Celsus collapses a number of aspects of illness and treatment in a rather haphazard manner. Thus:

> "Beneficial for ... composing the mind itself, is saffron ointment with orris applied to the head. If in spite of this, patients are wakeful, some endeavour to induce sleep by draughts of decoction of poppy or hyoscyamus; others put mandrake apples under the pillow; other smear the forehead with cardamonum balsam or sycamine tears." (p.289) "If, however, it is the mind that deceives the madman, he is best treated by certain tortures, When he says or does anything wrong, he is to be coerced by starvation, fetters and flogging." (Celsus, 15/1935; pp.302-303)

Plutarch (46-120 AD) also described what we would now call delusional depression. When a man becomes depressed, comments Plutarch:

> "....Every little evil is magnified by the scaring sceptres of his anxiety. He looks on himself as a man whom the gods hate and pursue with their anger.... Asleep or awake, he is haunted alike by the spectres of his anxiety. Awake, he makes no use of his reason; and asleep, he enjoys no respite from his alarms.

> His reason always slumbers; his fears are always awake. Nowhere can he find an escape from his imaginary terrors." (Quoted in Buckhill & Tuke, 1879; p.230).

Celsus and Plutarch both appear to me to be describing what would now be described as delusions. Jackson (1983) suggested that early medieval physicians occasionally refered to disturbed behaviour and emotion as stemming from particular beliefs. Ackerknecht (1968) took this cognitive interpretation further, suggested that early writers advocated psychotherapy for delusions. Zilboorg (1941) quotes Caelius Aurelianus, who re-presented the works of Soranus, a first century physician, suggesting that: "Those patients who have nearly recovered may be permitted to listen to the disputations of philosophers. Sadness, fear and rage have often been dispelled in this way, and philosophers have thus contributed much to the reestablishment of health" (p.33). Such advice is, however, included in a set of recommendations including predominantly physical treatments, in no particular order.

Such accounts are, in my opinion, fascinating, but conclusions must be cautious. In particular it appears to be common for modern writers (including myself) to read texts very selectively. As an example, Zilboorg (1941) comments that "The sexual causation of hysteria almost universally accepted today was not consciously suspected by Hippocrates, but intuitively he must have sensed it, since he considered marriage the best remedy for this affliction in the case of girls." (p.47). Zilboorg was writing in 1941, when his comment about the universal acceptance of a Freudian model of hysteria may have been accurate. The changing nature of the models of psychopathology evident here should warn us about concluding too much on the basis of selective quotation.

It is perhaps important that I could find no evidence that Hippocrates, perhaps the primary early medical authority, made any mention of beliefs, abnormal or otherwise. It has been suggested that a distinction between the concepts of knowledge

(a reference to a supposed link between a mental construct and externally evidenced reality) and faith (a ontological model without an explicit reference to evidence) developed only in the later middle ages (Wirth, 1983). The current definition of a delusion: "a fixed false opinion with regard to objective things especially as a form of mental derangement" (Oxford English Dictionary: Simpson & Weiner, 1989) has been traced back to the sixteenth century (Garety, 1985), an observation consistent with Wirth's literary analysis.

In the seventeenth century the philosophers Thomas Hobbes and John Locke discussed delusional ideas, since their thoughts about the links between experience and knowledge made explanations for delusional, false, beliefs difficult. Locke, in particular, suggested that delusions should be classified as reflective ideas (ideas based on combinations of experiences) rather than sensible ideas (more direct experiences) since reasoning is preserved in insane people. This suggests that people are combining ideas reasonably, but the bases upon which such combinations are occurring are faulty. Such an argument is very similar to that of Berrios (1991).

In early medieval times delusions seem to have been poorly understood and generally unrecognised. By the eighteenth century, delusions had become a cardinal sign (perhaps the cardinal sign) of mental illness. In 1800 a certain James Hadfield tried to assassinate George III while suffering from delusional beliefs that he should sacrifice his life to safeguard the future of the world. His defending counsel, Lord Erskine, stated that "Delusions.... when there is no frenzy or raving madness, is the true character of insanity" (see Sims, 1988). During the eighteenth century, Berrios (1991) suggests, the major assumptions behind the scientific study of delusions were established :

(i) Delusions are beliefs and are therefore susceptible to reality-testing,

(ii) Delusions are symptoms or indicators of disease,

(iii) Causes of delusions are believed to include the individual's character,

past history, circumstances, and/or brain state,

(iv) Both 'form' and 'content' of delusional beliefs are important in developing criteria for defining delusions,

(v) Form, content and aetiology of delusions determine the clinical importance of delusions and suggest that different delusions reflect different disease.

As mentioned in Chapter 1, the nineteenth century psychiatrist Emil Kraepelin, developed the concept of dementia praecox (Kraepelin, 1919/1973). Kraepelin identified six subtypes of delusion: ideas of sin, ideas of persecution, ideas of influence (delusions in which people believe that they are being influenced or controlled), exalted ideas (grandiose delusions), sexual ideas and ideas of reference. As with Locke, and future researchers, Kraepelin suggested that delusions reflect perceptual abnormalities (Winters & Neale, 1983). Bleuler (1911/1950) considered dissociative processes to be characteristic of schizophrenia, with delusions and hallucinations as consequences of this disturbance. Bleuler therefore only discussed the distinction between primary delusions and secondary, elaborative delusions as mentioned in Chapter 1.

Although less central to the story of schizophrenia, (Jaspers, 1912/1963) made two important contributions to the study of delusions, mainly because he favoured a phenomenological approach. First, he suggested that some psychiatric phenomena may be explicable (explainable by people or researchers observing the phenomenon) but not understandable (meaningless and impossible to empathize with) (see Sims, 1988). Jaspers believed that delusions were essentially not understandable.Secondly, Jaspers advocated separating the form of a phenomenon from its content. The form of delusion would mean, for instance, whether a delusion was developed and elaborated slowly over time, or arrived fully-formed in one percept-like experience. Jaspers

believed the form of a delusion to be far more important than the content.

2.3 Phenomenology

2.3.1 Definitions

Jaspers (1912/1963) also developed the bases of definitional criteria for delusions, suggesting that they are held with extraordinary conviction, are impervious to other experiences or counter-arguments, and have bizarre or impossible content. However he also suggested that such criteria are true for over-valued ideas and delusion-like ideas. These latter, Jaspers argued, differed from true delusions in that they are related to, and understandable in the context of a person's personality, mood or other experiences (including other psychotic symptoms such as hallucinations). True, primary, delusions are essentially ununderstandable. These two types of experience were believed to have different phenomenological characteristics, with primary delusions occurring rapidly, while secondary delusions are elaborated slowly over time.

Other researchers and clinicians have not tended to focus on understandability of delusional ideas. They have, however, also included consideration of the patient's cultural and social background. Thus Hamilton (1985), revising Fish's 'Clinical Psychopathology' defined a delusion as "a false unshakeable belief, which is out of keeping with the patient's social and cultural background" (p.43). Hamilton also states that "A delusion is the product of internal morbid processes and this is what makes it unamendable to external influences. The fact that it is false makes it easy to recognize but this is not its essential quality. A very common delusion among men is that their wives are unfaithful to them. In the nature of things, some of these wives will indeed have been unfaithful; the delusion will therefore be true, but only by coincidence"

(p.43).

Hamilton therefore essentially includes three criteria, that the belief is false, that it is not supported by the individual patient's cultural doctrines, and that the belief is the product of pathological processes. Such a view is almost directly reproduced in DSM-IV (American Psychiatric Association, 1994) where the definitional criteria for delusion are:

"A false personal belief based on incorrect inference about external reality that is firmly sustained in spite of what almost everyone else believes and in spite of what usually constitutes incontrovertible and obvious proof or evidence to the contrary. The belief is not one ordinarily accepted by other members of the person's culture or subculture (e.g. It is not an article of religious faith). When a false belief involves a value judgement, it is regarded as a delusion only when the judgement is so extreme as to defy credibility. Delusional conviction occurs on a continuum and can sometimes be inferred from an individual's behaviour. It is often difficult to distinguish between a delusion and an overvalued idea (in which case the individual has an unreasonable belief or idea, but does not hold it as firmly as is the case with a delusion." (American Psychiatric Association, 1994; p.765)

As mentioned in Chapter 1, ICD-10 (World Health Organization, 1992a; 1992b) views delusions as central symptoms of psychopathology, yet no definition of a delusion is included.

The Present State Examination (Wing, Cooper, & Sartorius, 1974a; 1974b) identifies two classes of delusions, primary and secondary, echoing Jaspers closely. Specific definitions of delusional beliefs are outlined for individual delusional subtypes, categorized by content, of which there are 22. These are delusions of: alien

penetration, appearance, assistance, catastrophe, control, depersonalization, grandiose ability, grandiose identity, guilt, hypochondriasis, morbid jealousy, paranormal influence, persecution, pregnancy, physical intrusion (such as X-rays), reference, sexual, somatic, subcultural and that the patient smells (delusional halitosis) as well as religious and fantastic delusions. Also listed are delusional confabulation, delusional memories, delusional misinterpretation and delusional preoccupation.

Each delusional subtype has an individual definition. Delusions of persecution are defined as: where "the subject believes that someone, or some organization, or some force or power, is trying to harm him (sic) in some way: to damage his reputation, to cause him bodily injury, to drive him mad or to bring about his death." (Wing, et al., 1974a; p.170).

2.3.1.1 Criticisms of definitions

Such definitions have been criticised in respect to most of their components. Falsity does not necessarily mean that a belief is a delusion (Heise, 1988; Moor & Tucker, 1979). This is accepted in that subcultural beliefs are defined as not being delusional. There is also considerable variation in delusional content across cultures (Westermeyer, 1988).

Delusions are supposed to be impervious to counter-argument, yet cognitive therapy is sometimes successful with deluded patients (Chadwick & Lowe, 1990; Garety, Kuipers, Fowler, Chamberlain, & Dunn, 1994; Hartman & Cashman, 1983; Johnson, Ross, & Mastria, 1977; Milton, Patwa, & Hafner, 1978; Tarrier, Beckett, Harwood, Baker, Yusupoff, & Ugarteburu, 1993). This necessarily implies that the conviction of delusional beliefs is not absolute (Garety & Hemsley, 1987; Spitzer, 1992). Finally, the inclusion of value judgements, even if regarded as delusional only

when the judgements defy credibility, is dubious.

Nevertheless, the reliability of identification of delusional beliefs appears high. Flaum, Arndt and Andreasen (1991) found that the inter-rater reliability of distinguishing bizarre versus non-bizarre delusions was less than 0.40, regardless of whether or not a structured definition was applied. However, Spitzer, First, Kendler and Stein (1993) calculated the reliability of twelve individuals evaluating 180 delusions of separate psychiatric patients according to three alternative definitions of bizarre delusions, reporting kappas of 0.64, 0.65, and 0.45. Forgus and DeWolfe (1974) concluded that inter-judge reliability for categorizing delusional themes ranged from .73 to .93.

In an approach reviewed, with approval, by Walkup (1990), Garety and Hemsley (1987) reported a reliable self-report scale comprising 11 belief characteristics: conviction, preoccupation, interference, resistance, dismissibility, absurdity, self-evidentness, reassurance seeking, worry, unhappiness, and pervasiveness. There was considerable variability between patients in terms of all characteristics (except conviction, since delusions must have delusionally great conviction). Garety and Hemsley (1987) suggested a 4-way factor structure: distress, belief strength, obtrusiveness, and concern.

One of the potentially important aspects of the DSM-IV definition of delusions is that they are personal. Little research appears to have been done on this aspect, but it is possible that a qualifying criterion is that the belief has personal significance. For instance, a patient who believes in the presence of non-human alien intelligences may be described as delusional (rather than a member of a subcultural group believing in UFO's) if their belief was seen as being intimately related to them or their self-concept (such as being the conduit for alien messages or the human coordinator of a plan to spread the message of impending inter-galactic war).

Alternatively, delusions may be an example of fuzzy logic (Winkelgren,

1981), where strict dichotomous criteria are inadequate to describe a phenomenon, yet the phenomenon is real. I think it would be rather fitting if delusions were described as fuzzy concepts.

Oltmanns (1988) reviewed the status and nature of delusions, came to a similar conclusion, and suggested a number of possible characteristic features of a delusional belief. It is important to recognize that Oltmanns suggested that none of these should be regarded as either necessary nor sufficient conditions for the classification of a belief as delusional. I, however, consider that (allowing for some linguistic editing) all of these conditions come close to being necessary, although none are sufficient. They are:

i) The balance of evidence for and against the belief is such that other people consider it completely incredible.

ii) The belief is not shared by others

iii) The belief is held with firm conviction. The person's statements or behaviours are unresponsive to the presentation of evidence contrary to the belief.

iv) The person is preoccupied with (emotionally committed to) the belief and finds it difficult to avoid thinking or talking about it.

v) The belief involves personal reference, rather than unconventional religious, scientific, or political conviction.

vi) The belief is a source of subjective distress or interferes with the person's occupational or social functioning.

vii) The person does not report subjective efforts to resist the belief (in contrast to patients with obsessional ideas).

(Oltmanns, 1988; p.5)

In my opinion, Oltmanns' conceptualization of delusional beliefs is unlikely to prove perfect. However, in my opinion, it has fewer problems than other approaches,

and a number of benefits, especially the stress placed on the personal nature of the content of the kind of beliefs termed 'delusional'. It does, however, seem to rely on a general awareness of conventional definitions, and act as a qualification to them.

2.3.2 Classifications

Authorities differ in their preferred classifications as well as definitions of delusions. Most classifications of delusional beliefs conventionally focus on content. Therefore, in DSM-IV (American Psychiatric Association, 1994), delusions are classified as:

Bizarre:	a delusion that involves a phenomenon that the person's culture would regard as totally implausible.
Delusional Jealousy:	the delusion that one's sexual partner is unfaithful.
Erotomaniac:	a delusion that another person, usually of higher status, is in love with them.
Grandiose:	a delusion of inflated worth, power, knowledge, identity or special relationship to a deity or famous person.
Of being controlled:	a delusion in which feelings, impulses, thoughts or actions are experienced as being under the control of some external force rather than being under one's own control.
Of reference:	a delusion whose theme is that events, objects or other persons in one's immediate environment have a particular and unusual significance. These delusions are usually of a negative or pejorative nature, but also may

be grandiose in content. This differs from an idea of reference, in which the false belief is not as firmly held nor as fully organized into a true belief.

Persecutory: a delusion in which the central theme is that one (or someone to whom one is close) is being attacked, harassed, cheated, persecuted or conspired against.

Somatic: a delusion whose main content pertains to the appearance or functioning of one's body.

Thought broadcasting: the delusion that one's thoughts are being broadcast out loud so that they can be perceived by others.

Thought insertion: the delusion that certain of one's thoughts are not one's own, but rather are inserted into one's mind.

Such a classification is clearly content-based, although (half way through the list) the sub-types of Mood-congruent and Mood-incongruent are included. This content-based classification is also seen in the case of DSM-IV Delusional Disorder, with erotomaniac, grandiose, jealous, persecutory and somatic types, as well as the ubiquitous mixed and unspecified types.

Similarly the PSE (9th edition) (Wing, et al., 1974a; 1974b) includes delusions of: control, reference, persecution, assistance, grandiose ability, pregnancy, guilt, depersonalisation, and catastrophe. Also included are: religious delusions, delusional explanations in terms of paranormal phenomena, delusional explanations in terms of physical forces, delusions of alien force penetrating or controlling mind or body, morbid jealousy, sexual delusions, fantastic delusions, delusional memories, delusional confabulations, simple delusions concerning appearance, hypochondriacal delusions, subculturally influenced delusions and delusional misinterpretations or misidentifications.

To compound its lack of a definition of delusions, ICD-10 also lacks a clear taxonomy of delusional themes, although a discrimination between different beliefs on the basis of content seems implied. Thus delusional sub-types are apparent in the diagnostic criteria for schizophrenia and paranoid schizophrenia: delusions of control, influence and passivity, somatic delusions, culturally inappropriate or bizarre delusions and delusions of superhuman powers or ability (schizophrenia), delusions of persecution, reference, exalted birth, special mission, bodily change or jealousy (paranoid schizophrenia). Also mentioned are psychotic phenomena categorized by DSM-IV as delusions: thought echo, thought insertion or withdrawal, thought broadcasting and delusional perception. ICD-10 suggests that the most common sub-types of Delusional Disorder are persecutory, litiginous, self-referential, grandiose, hypochondriacal or somatic, jealous and erotomanic, reflecting sub-types of delusions.

In addition to classifications based on content, other approaches have been developed. As mentioned above, Kraepelin (1919/1973) classified delusions into six subtypes (sin, persecution, influence, exaltation, sex and reference), while Jaspers (1912/1963) described primary and secondary (elaborative) delusions.

Garety, Everitt and Hemsley (1988) examined phenomenological characteristics (conviction, preoccupation, interference, resistance, dismissibility, absurdity, self-evidentness, reassurance seeking, worry, unhappiness, and pervasiveness) of 55 delusions using a scale which excluded content information. They concluded that the patients clustered into three groups - hebephrenic, paranoid and depressed. Also examining phenomenological characteristics (conviction, extension, systematization, probability, pressure and global state), Jorgensen and Jensen (1994) found differences between clinical groups. While patients with different diagnoses reported delusions with identical content, other aspects of the delusional experience differed and appeared independent of content. Severity of delusional belief

(including, but not exclusively conviction) was greatest in patients with a diagnosis of schizophrenia.

Finally, Trower and Chadwick (Chadwick & Trower, 1996; Trower & Chadwick, 1995) have proposed a subdivision of paranoia, into the rather pejoratively called "poor-me" and "bad-me" paranoia. This categorization has the benefit of being based on theoretical distinctions between different cognitive processes believed to be aetiologically important. It appears to have two weaknesses, however. First, the evidence supporting the sub-division appears weak. Second, it may do little more than replicate the division into primary delusions and those that are secondary to affective processes (Gelder, Gath, & Mayou, 1983).

The complexity of delusional classification was emphasised by Gladis, Levinson and Mowry (1994), who commented that it is often difficult to determine the difference between unusual beliefs and full delusions, or between brief episodes and persistent delusions, or bizarre versus nonbizarre delusions. They suggested that continua rather than dichotomous classifications may be appropriate. Such an approach is not, of course, new (Strauss, 1969).

2.3.2.1 Common delusional themes

Although the classifications of delusional ideation outlined above are designed to be as complete as possible, it does seem that certain delusional themes are more common than others. Delusions of reference refer to the phenomenon in which a patient believes that chance or circumstantial events have special significance for them, or are directed at them. Hamilton (1985) suggested that delusions of reference are a form of persecutory delusion, as the personal significance is rarely benign.

Some patients express the belief that they are involved in some kind of special

project or mission, which may be important in determining the future of the country or even of the whole world. In their most extreme form, grandiose delusions may involve the claim that the patient is a celebrity, a member of the royal family, or even God. Grandiose delusions are commonly seen in two contexts, in manic-depression and in combination with delusions of persecution. Grandiose delusions are more common in manic-depression than in schizophrenia (Junginger, Barker, & Coe, 1992), with up to 76% of patients with mania reported as suffering from delusions of grandeur, as opposed to some 40% of patients with schizophrenia (Karson, 1980). Having said that, however, it is has also been claimed that grandiose delusions and persecutory delusions are very closely linked (Manschreck, 1979; Oxman, Rosenberg, Schnurr, & Tucker, 1988).

Many delusions appear to have religious or mystical themes (Ndtai & Vadher, 1984), causing some psychodynamic authors to suggest that a "messiah-complex" is a universal psychotic theme (Goldwert, 1993). That religious ideas surface in psychosis is to be expected. Religious ideas are widely held, with extreme conviction, by significant numbers (perhaps a majority) of the general population (Duncan, Donnelly, & Nicholson, 1992; Northover & Gonzalez, 1993; Winter & Short, 1993). At the same time, religious or mystical ideas explain human phenomena using non-rational, non-reductive, processes. It also seems likely that religious delusional ideas are very closely related to the self-concept (in that one believes ones-self to be God or Jesus Christ or having access to higher knowledge or powers). It seems that religious ideas in general may be important for maintaining self-representations (Lilliston & Klein, 1991). Finally, religious ideas, focussing on sin, guilt and transcendence, as well as powers, forces and the fearful half-known are strongly emotionally laden.

Cultural and doctrinal differences between the religious patient and a rationalist therapist may create difficulties for psychiatric treatment (Greenberg & Witztum, 1991) as may the difficulty in differentiating religious beliefs and rituals from

delusions and compulsions. Nevertheless, some authors have taken a direct and scientific approach to the area, confidently assigning religious and mystical beliefs to abnormality. Brugger, Dowdy and Graves (1994) suggest that mystical or religious experiences might be related to 'superstitious conditioning', when opportunistic events attain the subjective qualities of discriminant stimuli, leading animals to behave in odd ways for reinforcers that would occur without any need for such behaviour. Brugger and colleagues (1994) suggest that such experiences might be due to dysfunction of the hippocampus, suggesting that organic, neuropsychological processes are responsible for normal superstitions and delusional beliefs. From a different perspective, Hale and Pinninti (1994) report how the subculturally valid beliefs of supernatural possession 'successfully' treated with clopenthixol following a failure of traditional healers and exorcists.

One of the most fascinating approaches to mystical delusions and experiences is that of Persinger, who has suggested that feeling the presence of non-tangible others, mystical experiences and religious experiences can be explained as arising from cognitions generated in the right cerebral hemisphere but detected by the left (Persinger, 1993). These, Persinger argues, generate a wide range of mystical, alien or 'out-of-body' experiences. Persinger has suggested that such phenomena are linked to epileptic processes, and can even be induced by direct electrical stimulation of the temporal lobes; creating mystical experiences on demand (Persinger & Makarec, 1993)!

As can be seen from the classifications of delusions outlined above, a common theme is somatic, with up to 55% of psychotic patients expressing hypochondriacal delusions (McGilchrist & Cutting, 1995). McGilchrist and Cutting (1995) developed a comprehensive subclassification of 37 distinct somatic delusions. Their system involved a distinction into experiential and elaborative (similar again to Jaspers' primary and secondary) delusions, within which delusions might concern malfunction

or distortion of the body, loss of control over the body or alienation of the self from the body.

2.3.2.2 Less common delusions

There is an unfortunate but inescapable element of prurient, morbid or comedic interest in delusional beliefs. This is particularly evident in the case of less common delusional beliefs, where the undoubted theoretical and clinical importance of many conditions seems less important than their weirdness. Enoch and Trethowan (1979) and Franzini and Grossberg (1995) both discuss unusual delusional and psychotic phenomena in some depth.

With many of the more uncommon delusional beliefs, it is sometimes difficult to distinguish between over-valued ideas and true delusions. This is particularly true for delusional (or morbid) jealousy (Enoch, 1991; Soyka, 1995; Soyka, Naber, & Volcker, 1991) the so-called 'Othello syndrome' (Enoch & Trethowan, 1979). Delusional jealousy may be of significant clinical and societal importance, given that people with such beliefs pose a significant risk of violence (Leong, Silva, Garza-Trevino, Oliva, Ferrari, Komanduri, et al., 1994). The converse, delusions of love; erotomanic delusions or De Clerambault's syndrome, occur when an individual believes that someone (usually rich, successful or famous) loves them when this is, in fact not the case. The love object is, in fact, often remote and inaccessible (Rudden, Sweeney, & Frances, 1990; Seeman, 1978).

Other uncommon delusional themes include delusional parasitosis (Musalek, Bach, Passweg, & Jaeger, 1990), a belief which usually takes a sufferer to a dermatologists rather than a psychiatrist or psychologist, and which is occasionally shared by a second individual (Musalek & Kutzer, 1990). A very few patients believe

they are changing into animals (Franzini & Grossberg, 1995), a condition termed therianthropy or lycanthropy. Although usually temporary, in one case (Kulick, Pope, & Keck, 1990) for 15 years a man believed he was a cat.

Delusional misidentification syndromes have been extensively studied, probably because they have been conditions in which some hints at causal mechanisms have been seen. In the Capgras syndrome (Capgras & Reboul-Lachaux, 1923/1994), an individual believes that family or friends have been replaced by impostors. As might be expected, variants of this delusion (with different individuals replaced) have been noted. Again to be expected, these variants have been named: the Frégoli delusion (Coubon & Fail, 1927/1994) described recently by de Pauw, Szulecka and Poltock (1987), where persecutors act in fiendish and clever disguise, the delusion of intermetamorphosis (Coubon & Tusques, 1932/1994) where it is believed that one individual is in fact another, but with altered appearance (Young, Ellis, Szulecka, & de Pauw, 1990), and the delusion of inanimate doubles (Anderson, 1988) where objects rather than people are substituted. In many cases it appears that intense suspiciousness, or paranoid beliefs (Fleminger, 1994) precede the misidentification delusion. Misidentification syndromes have become a group of phenomena where reasonably coherent neuropsychological causal mechanisms, centring on facial recognition (Ellis & Young, 1990) have been proposed, and will be discussed below.

2.3.2.3 Delusions of persecution

Clearly a complete taxonomy of delusional beliefs would be exhausting as well as exhaustive. In a relatively simple five-theme approach, Garety and colleagues (1988) examined the relative frequency of delusions in 55 British psychiatric patients. Paranoid delusions were the most common (35.2% of patients) compared to abnormal

negative beliefs about the self (31.5%), grandiose beliefs (25.9%) and negative (5.5%) and positive delusions about the world (1.8%). Similarly, Ndtai and Vadher (1984) studied the frequency of different delusional themes in different cultural groups by examining hospital records of patients from different national backgrounds. There were clear differences in the relative frequency of different delusional themes, although persecutory delusions were the most common, at 26.3% of patients overall, and were the most common form of delusions in 8 out of 9 cultural groups (the exception being Far Eastern patients, where sexual delusions were common). Persecutory delusions were most common amongst African and Jamaican patients, an observation which the authors account for by stating that, "In the African culture there is a widespread belief in witchcraft and a tendency to explain events in terms of external causation... This tendency persisted and was preserved in Africans taken to the West Indies as slaves.." (p.75).

Studies such as this have generally concluded that delusional beliefs concern themes which are understandable in social and cultural context. Moreover, most of these themes reflect universal concerns (such as safety and identity) or relate to the individual's position in the social universe (Bentall, 1994; Musalek, Berner, & Katschnig, 1989).

2.4 Measurement and Assessment

There seem to be two broad approaches to the measurement and assessment of delusional experiences. As will be clear from the earlier sections in this thesis, delusions are commonly viewed as symptoms of schizophrenia, and are assessed as such. Delusions are, however, also occasionaly seen as phenomena worthy of study in their own right.

2.4.1 Assessment of of delusions as symptoms of schizophrenia

General psychiatric rating scales, such as the Brief Psychiatric Rating Scale, (Overall & Gorham, 1962) the Comprehensive Psychopathology Rating Scale, (Asberg, Montgomery, Perris, Shallig, & Sedvall, 1978), the Present State Examination (PSE; Wing, et al., 1974a; 1974b) and the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) include brief assessments of the presence or absence of symptoms of schizophrenia, including delusional beliefs.

More precise measures, stemming from the assumption that schizophrenia is a valid concept include the Scale for the Assessment of Positive Symptoms (SAPS), the Scale for the Assessment of Negative Symptoms (SANS) and the composite Positive and Negative Symptoms Scale (PANSS). The SANS (Andreasen, 1983), the SAPS (Andreasen, 1984) and the PANSS (Kay & Opler, 1987; Kay, Opler, & Fszbein, 1988; Kay, Opler, & Lindenmayer, 1989)assume that schizophrenia is a more multiplex syndrome characterized by at least two relatively distinct syndromes. The PANSS includes strict operational criteria, leading to highly reliable (interrater reliability between 0.83 and 0.87) and apparently valid syndrome scores.

Within such scales, however, delusional ideation is viewed as a part of a whole syndrome rather than a phenomenon worth examining alone. More specific rating scales and assessment devices have been developed for the assessment of delusions alone.

2.4.2 Assessment of delusions

Although traditionally delusions have usually been viewed as either being

present or absent, Strauss (1969) suggested delusions could, and should, be described on four parameters: conviction, cultural or stimulus determinants, proccupation, and implausibility. Kendler, Glazer and Morgenstern (1983) incorporated Strauss's four factors, with a fifth, to produce a scale assessing: conviction, extension (the degree to which the delusional belief involves various areas of the patients life), bizarreness, disorganization, and pressure (preoccupation and concern). Psychometric analysis showed that all dimensions were independent, supporting the idea that delusional beliefs are multidimensional

In a similar approach, Harrow, Rattenbury and Stoll (1988) used three dimensions; conviction, the patient's awareness of other people's views of his or her delusion, and emotional commitment. Again, these dimensions did not correlate with each other. Harrow and colleagues (1988) also found evidence that the content of patient's delusions reflected premorbid concerns. Brett-Jones, Garety and Hemsley, (1987) used a modified version of Shapiro's Personal Questionnaire (Shapiro, 1961) to measure conviction, preoccupation, reaction to hypothetical contradiction and interference in delusional beliefs. They reported that the PQ was an effective measure. The same authors (Garety & Hemsley, 1987) also developed the Characteristics of Delusions Rating Scale mentioned above. Finally, a recent approach to the assessment of delusions is the Maudsley Assessment of Delusions Scale (MADS: Buchanan, Reed, Wessely, Garety, Taylor, Grubin, et al., 1993). This scale, used to examine the relationship between delusions and violence will be discussed in the next chapter.

Finally, Emanuelle Peters and colleagues have developed the eponymous PDI (Peters et al Delusions Inventory; Peters & Garety, 1996; Peters, Joseph, & Garety, 1995). This scale appears similar in nature to many other scales, in particular the CDRS (Garety & Hemsley, 1987), although its psychometric properties have been reported to be good.

2.5 Prevalence

2.5.1 Delusions in general medical conditions

Delusions, although considered pathognomic of madness, are known to occur in a wide variety of general medical conditions. Maher and Ross (1984), Cummings (1985) and Manschreck (1989) have each attempted to list the many conditions associated with the presence of delusional beliefs. I do not claim that my catalogue is complete, as new case reports appear regularly, but Table 2.1 contains elements taken from all three sources and some case studies.

Table 2.1 Conditions associated with delusions and other paranoid features

Neurological Disorders

Arteriosclerotic psychoses
Blunt head trauma
Brain tumours
Cerebrovascular disease
Delirium
Dementia
Fat embolism
Hearing loss
Huntington's chorea
Hydrocephalus
Hypertensive encephalopathy
Idiopathic basal ganglia calcification
Idiopathic Parkinson's disease
Intracranial hemorrhage
Marchiafava-Bignami disease
Menzel-type ataxia
Metachromatic leukodystrophy
Migraine
Motor neurone disease
Multiple sclerosis
Muscular dystrophy
Narcolepsy
Postencephalitic parkinsonism
Presenile psychoses (Alzheimer's and Picks)
Roussy-Levy syndrome
Senile psychoses
Spinocerebellar degeneration
Subarachnoid hemorrhage
Subdural haematoma
Temporal lobe epilepsy

Metabolic and endocrine disorders

Acute intermittent porphyria
Addison's disease
Complication of portacaval anastomosis for cirrhosis
Cushing's syndrome
Folate deficiency
Haemodialysis
Hypercalcemia Hyponatremia
Hypopituratism
Liver failure
Malnutrition
Niacin deficiency
Pancreatic encephalopathy

Metabolic and endocrine disorders continued

Parathyroid disorders
Pellagra
Pernicious anaemia
Phenylketonuria
Systemic lupus erythromatosus
Thyamine deficiency
Thyroid disorders
Uraemia
Vitamin B12 deficiency
Wilson's disease

Sex chromosome disorders

47 XXY
Klinefelter's syndrome
Turner's syndrome

Infections

Acquired immune deficiency syndrome (AIDS)
Encephalitis lethargica
Creutzfelt-Jakob disease
Malaria
Syphilis
Toxic shock syndrome
Tryphanosomiasis
Typhus
Viral encephalitides

Psychiatric disorders

Brief reactive psychosis
Delusional disorder
Induced psychotic disorder
Mood disorders
Schizoaffective disorder
Schizophrenia (all sub-types)
Schizophreniform disorder

Table 2.1 Conditions associated with delusions and other paranoid features continued

Alcohol and drug abuse disorders

Alcohol withdrawal
Amphetamine
Anaesthetic nitrous oxide
Atropine toxicity
Barbiturate
Chronic alcohol hallucinosis
Chronic bromide intoxication
Cocaine
Ephedrine
Marijuana
Mescaline (and other hallucinogens)
Perbitine
Withdrawal from minor tranquilizers and hypnotic medications

Toxic agents

Arsenic
Carbon monoxide
Manganese
Mercury
Thallium

Pharmacologic agents

ACTH
Amphetamine and related compounds
Anticholinergic drugs
Antimalarials
Antitubucular drugs
Bromocriptine
Buproprion
Cimetidine
Cortisone
Diphynylhydantoin
Disulphiram
L-Dopa
Imiprimine and other tricyclic antidepressants
Mephentermine
Methyldopa and imiprimine (in combination)
Methyltestosterone
Pentazocine
Phenylpropanolamine
Prophylhexidrine

From: Manschreck, T. C. (1989). Delusional (Paranoid) Disorders. In: Kaplan, H. I. & Sadock, B. J. (Eds) Comprehensive Textbook of Psychiatry. 5th Edition. Baltimore: Williams & Wilkins.

With additional material from: Cummings, J. L. (1985). Organic Delusions: Phenomenology, anatomical correlations and review. British Journal of Psychiatry, 146, 184-197.

and

Maher, B. A., & Ross, J. S. (1984). Delusions. In: H. E. Adams & P. Suther (Eds.), Comprehensive handbook of psychopathology. New York: Plenum.

Delusions therefore occur in association with around 105 general medical conditions. I do not intend to discuss the presumed natural history of each, but a few common themes appear.

2.5.1.1 Delirium

In many of the non-psychiatric conditions outlined above, delirium (a state of disordered or impaired consciousness combined with alteration of perceptual functioning or affect which is generally of fluctuating course) seems important. (See American Psychiatric Association, 1994) for diagnostic criteria for delirium). Some 10% of medical-surgical inpatients and 40% of geriatric patients become delirious at some point in their hospitalisation (Horvath, Siever, Mohs, & Davis, 1989) as a result not of specific brain damage, but rather of a more general disruption of neurological function (Engel & Romano, 1959) consequent upon neurological, metabolic or endocrine disorders, following both systemic and encephalitic infections, or as a consequence of substance abuse or poisoning by toxic agents.

About 20% of patients with delirium present with clear delusions and hallucinations in the absence of general cognitive deterioration (Horvath, et al., 1989). Although it is therefore common for patients suffering from delirium to be misdiagnosed as suffering from a psychiatric disorder there may be subtle differences between organic and psychiatric delusions. Lishman (1978) and the authors of DSM-IV (American Psychiatric Association, 1994) both suggest that organic delusions are more haphazard and fleeting than those of the functional disorders, while Cummings (1985) found that general cognitive functioning correlated with the organization of delirious patients' delusions. When compared to those observed in functional illnesses, the delusions of organic conditions seem, "vague, poorly

systematized, incoherent, fleeting and changeable, or restricted and stereotyped in content" (Lishman, 1978; p.178) and, "transient and disorganized" (Gelder, et al., 1983; p.276).

2.5.1.2 Specific neurological conditions

Neurological conditions other than delirium are also associated with delusions, including degenerative dementias such as Alzheimer's disease, multi-infarct dementia and metabolic encephalopathies. In these cases the delusions are commonly persecutory and accompanied by considerable cognitive disturbance (for example, memory loss) (Deutsch, Bylsma, Rovner, Steele, & Folstein, 1991).

Delusions are common in metachromatic leukodystrophy, a rare inherited disorder which involves demyelination of the sub-frontal cortex of the brain (Waltz, Harik, & Kaufman, 1987), affecting 27% of patients (Hyde, Ziegler, & Weinberger, 1992) and leading to frequent misdiagnoses. It has been claimed that psychotic symptoms are associated with damage to the temporal lobes of the brain (Davison & Bagley, 1969; Hyde, et al., 1992), being reported in up to 83% of patients suffering from Huntington's chorea (Rosenbaum, 1941) and in approximately 10% of those who suffer from temporal lobe epilepsy (Roberts, Done, Bruton, & Crow, 1990).

2.5.1.3 The effects of psychoactive substances

Although many drugs may make people delirious, it is also clear that certain drugs are particularly likely to induce delusions. Amphetamines and cocaine, especially when they are taken in large doses (for a review of the psychological effects

of psychoactive drugs, see Jaffe, 1989) and many prescribed drugs which cause delusions. This is especially true of L-Dopa, which is metabolized to dopamine when it has crossed the blood-brain barrier, which is used in the treatment of Parkinson's Disease (Cummings, 1991; Friedman & Sienkiewicz, 1991).

As mentioned in Chapter 1, the actions of such drugs in mimicking the psychotic phenomena have led some to suggest that neurotransmitter dysfunction is responsible for schizophrenic symptoms (Crow, 1981; Wyatt, Kirch, & DeLisi, 1989). However, once again, the delusions seen during LSD use have important phenomenological differences to those in schizophrenia (Brady, Lydiard, Malcolm, & Ballenger, 1991; Mitchell & Vierkant, 1991). Although the effects of drugs in producing delusional beliefs pose a serious problem of explanation for psychologists, it has been argued that their neurochemical actions cannot provide a complete model of the development of delusional thought (Unnithan & Cutting, 1992).

2.5.1.4 Endocrine and metabolic conditions

As with other conditions, most of the reports of delusions in metabolic or endocrine abnormalities implicate delirium. In diabetes mellitus (Wilkinson, 1981), pellagra (Lishman, 1978) and pernicious anaemia (Ko & Liu, 1992) delusions occur, but in association with 'florid confusion', 'acute confusional states' or 'twilight states'. Thyroid problems are associated with the presentation of delusions. Hypothyroidism in particular has a reputation for producing psychiatric symptoms - 'myxoedema madness'. Depressive and persecutory delusions are especially common in this condition (Asher, 1949). Once again, however, the clinical picture will "in general be coloured by mental slowing, and often will include features indicative of organic mental impairment" (Lishman, 1978; p.604). The common presentation of

depressive and paranoid delusions is probably explicable simply because these themes are generally most common.

Occasionally, however, once again there are a number of metabolic syndromes in which delusions occur in clear consciousness. In acute intermittent porphyria (a disease associated with abnormalities in porphyrin metabolism (Stein & Tschudy, 1970, and allegedly suffered by King George III), psychological symptoms are reported in up to 75% of patients (Ackner, Cooper, Gray, & M., 1962). Psychotic symptoms severe enough to warrant 'mental certification' occur in up to 10% of patients (Goldberg, Brodie, & Moore, 1978). Although delusions are usually limited to the active phase of the somatic illness, delusions in the absence of delirium are occasionally the initial symptoms (Goldberg, et al., 1978).

2.5.1.5 Infections

Once again focussing on delusions occurring in clear consciousness as a result of systemic or encephalitic infections, neurosyphilis seems prototypical. Syphilis was known as 'general paresis of the insane' even after the infectious nature of the disorder was demonstrated (Noguchi & Moore, 1913) with delusions of grandeur seen as stereotypical (Gelder, et al., 1983). As the prevalence of neurosyphilis has dropped, the clinical presentation also changed (Hare, 1959). Although delusions remain common, these are now usually depressive, with delusions of persecution reportedly rare (Dewhurst, 1969). There is no adequate explanation for these changes.

2.5.2 Delusions in psychiatric disorders

In the medical conditions listed above, delusions are presumed to be secondary to the physical pathology. Although it seems difficult to understand the aetiology of the abnormal beliefs, it does seem that, when the underlying illness is treated, the delusions usually disappear. Delusions are far more common in psychiatric conditions, where they are often difficult to treat and tend to persistent.

2.5.2.1 Depression

Psychologists have often argued that abnormalities of thinking, particularly the presence of negative thoughts, play a central role in the origin and maintenance of depression (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978; Beck, 1976; Beck, Rush, Shaw, & Emery, 1979; Ellis, 1962). The negative thoughts of depressed patients (e.g. "I am a worthless and hateful person") are usually distinguished from 'true' delusions, which may be either mood-congruent or incongruent (Gelder, et al., 1983). Delusions occur in 10-20% of depressed inpatients (Leckman, Weissman, Prusoff, Caruso, Merikangas, Pauls, & Kidd, 1984) with mood-congruent delusions being more common.

The exact relationship between depression and delusions is not clear. However, it appears that delusions are associated with more severe forms of depression (Glassner & Haldipur, 1983) but that the presence of delusions also appears to depend upon characteristics of the individual patient. Charney and Nelson (1981) compared 54 delusional and 66 nondelusional unipolar depressed patients in terms of the course, symptoms and treatment response of their depression and on personality variables. They found that the delusional patients had more guilt feelings

and were more ruminative, agitated, and self-referential than the nondelusional patients. Patients with delusions responded poorly to tricyclic antidepressants, but better to a combination of antidepressant and antipsychotic medication. Depressed patients with delusions were also reported to respond well to Electro-Convulsive Therapy. Charney and Nelson (1981) believed that these findings supported the idea that delusional depression was a distinct subtype of depression.

2.5.2.2 Mania and bipolar affective disorder

Delusions occur in up to 70% of manic patients (Goodwin & Jamison, 1990) and are usually grandiose, concerning: wealth, importance, status or special personal powers. As in depression, separating mood-congruent, exaggerated and abnormal but non-delusional beliefs from 'true' delusions in mania is difficult and perhaps of little clinical value.

2.5.2.3 Schizophrenia, schizoaffective, delusional and related disorders

Schizophrenia and schizoaffective and delusional disorders have been extensively discussed in Chapter 1. The presence of delusions are highly likely to lead to a diagnosis of schizophrenia, and almost guarantee one of the other related diagnoses. Delusions of persecution have been reported in over 60% of patients with a diagnosis of schizophrenia (Sartorius, Shapiro, & Jablensky, 1974). Such delusions in schizophrenia are usually systematised and persistent as opposed to the fleeting and haphazard beliefs of people with organic diseases.

As mentioned in Chapter 1, schizoaffective disorder is essentially characterized

by a combination of the symptoms of schizophrenia and affective disorder (American Psychiatric Association, 1994). Delusional disorder is defined as a syndrome in which delusions are present in the absence of other psychotic symptoms (American Psychiatric Association, 1994).

The status of delusional disorder as a syndrome separate from other psychiatric disorders (and most especially schizophrenia) is also a matter of controversy (Kendler, 1984). Kraepelin (1919/1973) considered paranoia to be a rare condition distinct from dementia praecox, characterised by the (pathognomic) presence of delusions but without the deteriorating course. Bleuler (1911/1950), on the other hand, considered that paranoia was a form of schizophrenia. These contrasting views persist in the psychiatric literature (Manschreck & Petri, 1978), with some authors arguing that delusional disorder should be thought of as a subtype of schizophrenia, others that it is a subtype of affective illness, and still others claiming that it is a distinct diagnostic entity (Kendler, 1980; Kendler, 1982; Kendler, 1984; Munro, 1987; Opjordsmoen & Retterstol, 1991; Watt, 1985; Winokur, 1986).

Delusions are also listed as symptoms of various less common disorders described in DSM-IV: schizophreniform disorder, brief reactive psychosis' (also known as boufée délirante) and induced psychotic disorder. This later condition, commonly referred to as 'folie a deux' was briefly described in Chapter 1. Occasionally, such delusions are shared by more than two people (Myers, 1988). The importance of the relationship in determining this disorder is evident from the fact that, when the closeness of the relationship is reduced, the second partner's conviction in the delusion usually subsides (Bourgeois, Duhamel, & Verdoux, 1992; Enoch & Trethowan, 1979).

Finally, delusions may occur in circumstances which make any of the above DSM-IV diagnoses impossible yet it seems unreasonable to conclude that organic illness is implicated. DSM-IV resolves this dilemma by means of the useful category

of 'atypical psychosis'.

2.6 Causes and aetiology

2.6.1 Biological approaches

I intend to spend little time describing biological approaches to explaining the aetiology of delusional beliefs for two reasons. First, as can be deduced from Chapter 1, biological approaches to the aetiology of schizophrenia usually also advocate the study of delusions only as part of an assumed syndrome of schizophrenia. Explanations of delusions are, therefore, the same as explanations of schizophrenia, and I do not wish to be repetitive. Second, this thesis focusses on a phenomenological approach, and a psychological approach.

2.6.1.1 Genetics

Genetic studies of schizophrenia (see Chapter 1) seem to imply that inherited factors are important, but not directly causal. Genetic influences on delusional ideation have received little specific study. Where links between genetic factors and delusional beliefs have been made, these usually emerge as post hoc findings from research protocols designed to study schizophrenia.Thus Gladis and colleagues (1994) examined the relatives of people with a diagnosis of schizophrenia, and suggested that a variety of unusual beliefs, extending to frank delusions, are genetically inherited.

Dopamine (again as mentioned in Chapter 1) has frequently been associated with schizophrenia, and thus with delusions. The DRD4 gene codes for one of the neurological receptors for the neurotransmitter dopamine, and has been studied in

genetic-linkage studies of schizophrenia. Catalano, Nobile, Novelli, Nothen and Smeraldi (1993) found that an abnormality in the DRD4 gene was associated with the presence of delusions more closely than with the diagnosis of schizophrenia. In general, however, the genes coding for dopamine neurotransmitter receptors (the DRD1, DRD2, and DRD3 genes) have not been reliably associated with schizophrenia (reviewed in Chapter 1). This may indicate that Catalano and colleagues' (1993) finding is invalid, or that such abnormalities are specific to delusional disorder, not general to schizophrenia. In the same way that psychological research in psychosis has benefitted from the study of individual phenomena, genetic and biological investigations may also benefit.

2.6.1.2 Dopamine

As seen in Chapter 1, dopamine is no longer viewed as a likely contender for the status of single schizophrenogenic factor. However, dopamine-releasing illicit drugs are thought to trigger delusions, especially delusions of persecution. The Type I / Type II division of schizophrenia into subtypes suggested by Tim Crow (Crow, 1980; 1981), is argued, in part, to reflect the possibility of dopaminergic malfunction in schizophrenia typified by positive symptoms.

There is some evidence of dopaminergic abnormalities specific to delusional ideation (Bracha, Livingston, Clothier, Linington, & Karson, 1993; Krieckhaus, Donahoe, & Morgan, 1992). However biological researchers seem to have unwittingly hampered their own investigations in a potentially important area by studying broad constructs such as schizophrenia. It may be the case, for instance, that disturbance of mental health in general is too broad a concept to be related to specific neurotransmitter mechanisms even if individual psychotic phenomena, such as

hallucinations and delusions, may be. Dopamine functioning remains a possible avenue for investigation in delusions.

2.6.1.3 Neurological approaches

As seen in Chapter 1, a range of neurological abnormalities are associated with schizophrenia, although in a manner too general to be impressive or particularly useful. A number of researchers have attempted to clarify links between neurology and psychosis by examining delusional ideation. Horvath and colleagues (1989) proposed two classes of organic delusional disorder. The first implicates the temporal lobes and diencephalon while second broad category involves the potentiation of dopaminergic systems. This classification mirrors those of Cummings (1985), who suggested that delusions appear to involve either toxic metabolic processes or defects of the limbic system and basal ganglia and Crow's (Crow, 1980; 1981) referred to above.

Pavlov (1934), in a paper with a disarmingly honest title 'attempted' to explain delusions of reference in terms of pathological inertness. In an argument strikingly similar to that of Frith (Frith, 1994; 1987; 1992; Frith & Done, 1988; see below), he proposed that a perception of one's own inability to perform intended acts may lead to delusional explanations. Again preempting later, more psychological, approaches, Guy de Clerambault (1942) suggested that delusions stem from anomalous neurological experiences demanding explanation but unamendable to rational attribution. This general approach has been developed and argued assertively by Maher (Maher, 1974; 1988; 1992; Maher & Ross, 1984), an approach which will discussed below

Such neurological models are often entirely compatible with psychological accounts discussed below. However, most neurological models of delusions are

extremely general. One exception is the case of Capgras and other delusions of misidentification.

2.6.1.3.1 Misidentification syndromes

It has been claimed that delusional misidentification syndromes are more likely to have organic causes than other delusions (Cutting, 1991; Signer, 1992). In particular, it has been suggested that specific deficits in the processing of information involved in the recognition of familiar faces are responsible (Ellis & Young, 1990; Fleminger, 1992). These models suggest that delusional misidentification results from accurate perception combined with a failure to appreciate familiarity (de Haan, Young, & Newcombe, 1987; Ellis & Young, 1990; Ellis, Young, & Koenken, 1993). Faces are recognised, but felt to be unfamiliar.

Although some cases of Capgras' delusion do appear to have signs of neurological damage (Hay, 1986; Morrison & Tarter, 1984; Sylvia, Leong, & Wine, 1993; Wilcox, 1984) there are cases where no organic damage is observed (Fishbain & Rosomoff, 1986; Fleminger & Burns, 1993; Pellettier, Bartolucci, & Wallace, 1985). Psychological factors (drug-induced misinterpretation of normal events (Eva & Perry, 1993), psychodynamic conflicts (Berson, 1983; Pulman, Dupont, & Ananth, 1983; Sinkman, 1983) and developmental changes (de Leo, Galligioni, & Magni, 1985; Kourany, 1983; MacCallum, 1984)) have also been suggested as causal. Moreover, delusional misidentification syndromes seem to occur as a symptom of schizophrenia (McLauchlin & Sims, 1984), indicating that, even here, multiple routes to delusional beliefs exist (Fleminger & Burns, 1993; Pellettier, et al., 1985; Wright, Young, & Hellawell, 1993).

2.6.2 Psychological approaches

Both Arthur (1964) and Winters and Neale (1983) have reviewed psychological theories of delusional thinking. Winters and Neale (1983) distinguished between motivational and deficit theories. Deficit theories generally assume that cognitive or attentional abnormalities lead to delusions, while motivational theories in general assume that individuals develop delusions either to explain unusual perceptual experiences, or aversive psychological states.

2.6.2.1 Psychological deficit theories

Necessarily, psychological, neuropsychological and neurological (organic) deficit theories overlap. Since many (perhaps all) psychological processes have specific neurophysiological bases, the differences between an 'organic' deficit and a psychological deficit are often small.

2.6.2.1.1 Deficits (or excesses) in emotional expression or processing

A number of theorists have suggested that delusions stem from excessive or exaggerated emotion. Arthur (1964) discusses a number of psychiatrists speculating that strong emotions lead to delusional beliefs. Arthur cites Stocker (1940) as suggesting that delusional sub-types suggest four basic emotions: depressive, manic, anxious and suspicious, and Frankhauser in 1932 suggesting that paranoia be classified as a third affective psychosis (with mania and depression). However, Arthur (1964) also comments that no putative causal pathways were suggested

There is some evidence of disruption (or facilitation) of emotional processing in delusions, particularly paranoia. LaRusso (1978) reported that paranoid patients were more accurate than normal controls in discriminating between the real emotions of volunteers receiving electric shocks and the fake emotions of actors. More generally, research examining cognitive processes in persecutory delusions has examined the processing of information related to emotions and the self-concept. This research will be dealt with later in this thesis.

The most important theory relating emotions to delusions is that of Bleuler (1911/1950) - a theory that also implicates logical thinking.

2.6.2.1.2 Deficits in logical thinking

Because delusions are illogical, many authors have suggested that delusional beliefs occur as a result of deficits in an individual's ability to think logically. Bleuler (1911/1950) suggested that the central issue in schizophrenic thought disorder was a disruption of normal associative links. In the absence of coherent, logical, associations, Bleuler suggested that affective influences became dominant and hence delusional ideas generated. This model seems to fit with some clinical experience. The major reservation reported with respect to Bleuler's model is that the key affective state observed in patients with schizophrenia is flattened affect (Spitzer, Endicott, & Robins, 1978). It may be the case that, in the early stages of the development of delusional beliefs, affective influences are volatile, contrasting with chronic or non-delusional presentations.

Some researchers have linked hypothesised deficits in thinking with neurological problems. Benson and Stuss (1991) suggested that jumping to conclusions and an inability to properly evaluate evidence were associated with

delusions, symptomatic of abnormality in the frontal lobes. Similarly, Cutting and Murphy (1988) linked 'deficits in the utilization of real-world knowledge' to frontal lobe deficits and delusions, while Liddle (1994) suggested that frontal lobe dysfunctions were linked to deficits in executive functions and reality distortion. As discussed in Chapter 1, however, such models lack specificity.

More specifically there have been a number of models of delusions involving specific deficits in logical processes. von Domarus (1946) studied deductive, syllogistic thinking in schizophrenia, and suggested that patients mistake similar sounding premises for premises with logical equivalence. A development of this model was proposed by Arieti (1974), who suggested that, for people with a diagnosis of schizophrenia, association between concepts equals a causal relationship. Such models seem to explain a number of aspects of thought disorder, but their relevance to delusions is slightly less clear.

Inductive reasoning draws more information out of the situation than deductive reasoning, combining premises to conclude something not known before. It is, therefore, a better model for examining delusional beliefs (as they are new ideas by definition). In delusions, errors or biases in inductive reasoning have been investigated by a number of researchers (Garety, Hemsley, & Wessely, 1991; Hemsley & Garety, 1986; Huq, Garety, & Hemsley, 1988; John & Dodgson, 1994). These studies have often been cited as evidence of rapid, over-confident, less evidence-based patterns of inductive reasoning in deluded patients.

Moreover, Maher (1992), pointed out that there are so many possible forms of logical error that it would be surprising if we didn't see evidence of some. This is especially important because normal people are known to be bad at such formal logical tasks (see Elster, 1986; Sutherland, 1992; Tversky & Kahneman, 1974). Rather than perform logical functions, people use simple heuristic strategies to solve problems. These heuristics are clearly highly functional, but they are not logical. Rather, they are

are often associative. To find that deluded people use associative rather than logical rules is not terribly interesting. What is likely to be interesting is what kind of associations the deluded patients are making.

2.6.2.1.3 Perceptual deficits

I have already mentioned how John Locke discussed the possible role of perceptual as opposed to rational routes to abnormal belief. I have also mentioned how both Kraepelin (1919/1973) and De Cleranbault (1942) speculated that delusional beliefs in dementia praecox and misidentification syndromes might be due to perceptual deficits as opposed to reasoning errors. A perceptual deficit model of delusional beliefs is associated today with Brendan Maher.

Maher's position is essentially that delusions are, in every case, the products of healthy, rational, attempts to explain anomalous or disturbed perceptions (Maher, 1974; 1988; 1992; Maher & Ross, 1984). Much of the evidence Maher cites in support of his model is negative, in that he claims no real evidence of reasoning abnormalities in deluded subjects exists. As an example, Maher (Maher, 1974; Maher & Ross, 1984) suggests that a study of formal syllogistic reasoning (Williams, 1964) supports the claim of normal reasoning in deluded patients. However the participants in that study had a broad diagnosis of schizophrenia, possibly unrelated to the presence of delusions, and formal reasoning tests are unlikely to be very relevant to clinical and emotional processes. I have already discussed theories concerning abnormal logical reasoning in deluded subjects, and it is certainly the case that other authors would disagree with Maher's claim the the reasoning of deluded patients is normal.

More positive evidence in favour of Maher's model, albeit somewhat

unconventional, was provided by Zimbardo, Andersen and Kabat (1981). This study involved a group of university undergraduates. Some of this group were hypnotized to induce partial deafness with and without awareness; some were aware of the induction of partial deafness, while some were induced to scratch their ears. A projective test and two psychometric measures of paranoia suggested that the unaware partial-deafness group showed higher levels of paranoia than the other groups. This experiment is quite fun, but does little to support Maher's (1974) hypothesis. First, the measures may well be unrelated to clinical delusions. Second, the validity of hypnotically induced deafness is dubious (Wagstaff, 1986).

There is some clinical evidence of a relationship between deafness and delusions of persecution. Cooper, Garside and Kay (1976) found that 51% of a group of elderly patients with delusions of persecution suffered from hearing-impairment. Kay, Cooper, Garside and Roth (1976) found that six variables, one of which was deafness, discriminated patients with persecutory delusions from patients with an affective disorder. However, Moore (1981) found that sensory deficits, when age had been accounted for, had no effect on the prevalence of persecutory delusions, and Watt (1985) found no relationship between sensory deficits and paranoia in younger patients.

2.6.2.1.4 Perceptual excesses

Maher (Maher, 1974; Maher & Ross, 1984) has also argued that hallucinations may lead to rational delusional beliefs. It might seem quite rational to develop delusional explanations for inexplicable perceptual experiences. Despite the logic of this approach, there is little experimental support. Chapman and Chapman (1988) suggested that individual case-studies demonstrated a link between delusional beliefs

and hallucinations, and reported equivocal results in a large study of individuals showing schizotypal traits, while Hustig and Hafner (1990) reported that, in twelve psychiatric patients, delusional conviction was negatively correlated with the subjective intrusiveness of hallucinations. One group of perceptual excesses known to lead to delusions is drug-induced hallucinations (see Jaffe, 1989, and Chapter 1). However, as discussed in Chapter 1, the delusions seen in drug use seem qualitatively different from functional psychoses (Brady, et al., 1991; Lishman, 1978).

One area where perceptual excesses have been reliably associated with delusions is monosymptomatic hypochondriacal psychosis (Munro, 1980). There seem to be two routes to a monosymptomatic hypochondriacal delusion. One is a distorted body-image, while the other is an exaggeration of real physical stimuli (Munro, 1980). Munro (1980) and Berrios (1982) have suggested that this exaggeration is not hallucination, but a misinterpretation of relatively normal stimuli. Flynn, Cummings, Scheibel and Wirshing (1989) and Harper and Moss (1992) report cases where neurological damage is believed to lead to sensations suggestive of infestation, while Aizenberg, Schwartz and Zemishlany (1991) report a case where delusional parasitosis was apparently stimulated by drug-induced pruritus.

2.6.2.1.5 Theory-of-mind deficits

Theory-of-mind deficits have been discussed in Chapter 1 in the context of schizophrenia, and will be discussed later in this thesis. At this point it is simply worth mentioning that Frith's model (Frith, 1994; Frith, 1992) is particularly focussed on delusions. Delusions of reference are seen as stemming from an awareness that other people have different beliefs from themselves, but a lack of understanding as to what these beliefs might be. Delusions of alien control and hallucinations are understood as

resulting from a failure to understand that internal mental events (intentions or cognitions) are indeed internal. Although support for this model comes mainly from phenomenological discussions, a number of recent empirical studies have been reported (Corcoran, Cahill, & Frith, submitted; Corcoran, Mercer, & Frith, 1995; Frith & Corcoran, submitted).

2.6.2.1.6 Attentional deficits

Attention deficit models have been discussed in Chapter 1 in the context of schizophrenia. Again, however, delusions are primary in this model. Hemsley (1987; 1994) suggests that delusions, in particular, stem from a process involving abnormalities in selective attention (Gray, Feldon, Rawlins, Hemsley, & Smith, 1991; Hemsley, 1987; Hemsley, 1994) whereby attention is focussed on trivialities. This leads to a lack of influence from learned regularities. Clinically, abnormalities in selective attention seem common. As discussed above, however, certain details of the causal, psychological, linking processes seem vague and poorly related to clinical details of delusional beliefs.

2.6.2.2 Psychological motivational theories

2.6.2.2.1 Psychoanalytical theories

Chapter 1 addressed psychoanalytic theories of schizophrenia. Because psychoanalysis does not follow the established medical model, theories of psychopathology within this approach have tended to focus on individual symptoms. Psychoanalytic theories of delusions have tended to fall into two categories. Some

theorists (Ferden, 1952; Hartmann, 1953) have suggested that delusions result from weaknesses in an individual's ego boundaries. If one's distinction between self and world, and between self and fantasy is weak, the distinction between reality and falsehood is assumed to be similarly weak.

More commonly, psychoanalytic theories have invoked defense mechanisms, particularly projection. Freud (1911/1950; 1915/1956), Henderson and Gillespie (1936), Kretschmer (1927), Mayer-Gross, Slater and Ross (1954) and even Bleuler (1911/1950) suggested that delusions represent the externalization of desires, fears, or conflicts. Particularly important here is Freud's concept of projection in persecutory delusions. This model will be discussed more fully in the Chapter 3.

2.6.2.2.2 Personality theories

A number of authors, frequently from the psychoanalytic or psychodynamic persuasions, have linked the expression of delusions with particular personality types, and with how different people see their relationships with others. Kretschmer (1927) suggested that delusions stem from three personality types: aggressive, sensitive and wish-fulfilling. Aggressive types, he argued, were outwardly confident, but hiding secret insufficiency, leading to suspiciousness and paranoid delusions. Sensitive types were sensitive and conscious of their sense of insufficiency, leading to ideas of reference, while wish-fulfillers were likely to experience grandiose delusions. It is unfortunate that empirical studies have demonstrated no relationship between personality types and delusions similar to that predicted (Arthur, 1964).

Other personality theorists have focussed on relationships between self and others (Arthur, 1964). Serieux and Capgras (1909) suggested that grandiose delusions stem from a reluctance to indulge in any form of self-criticism. Schulte (1924)

regarded some delusions as the resolution of an awareness of a gulf between self and others, while Kahn (1940) suggested that other delusions serve the function of reducing loneliness and isolation (authors cited in Arthur, 1964).

2.6.2.2.3 Existential approaches

Existential psychology is concerned with an individual's experience and self-awareness. Binswanger (1958) suggested that an existential study of psychology should focus on the 'world design' (i.e. ontology) of an individual. This carries the implication that a delusion is an inappropriate world design. One interesting offshoot of the existential approach, cited in Winters and Neale (1983), it that of Gudeman (1966). He suggested that the balance of self-intentions and desires is disrupted in people with delusions, such that desires are misperceived. This approach mirrors that of Frith (1994).

More centrally within the existential tradition Ey (1955) (cited in Arthur, 1964) suggested that reality is a subjective notion, easily distorted within one's relationship with the world. In this context, it is worth reiterating that Forgus and DeWolfe (1974) reported that delusional themes matched the dominant themes in a person's world view. This is not surprising, but is hardly an explanation. Although Jaspers (1912/1963) suggested that delusions are essentially ununderstandable, one of the dominant themes of this review is that the individual expression and content of delusional ideas is shaped by personal experience and culture.

2.6.2.2.4 Learning theory

The development of learning theory as a major psychological discipline has, of course, led to behavioural theories of delusions. One general approach is to assume that psychotic phenomena are maintained by positive reinforcement. Ullman & Krasner's (1969) behavioural theory of schizophrenia has been mentioned earlier. As discussed by Winters and Neale (1983), although behavioural approaches to the treatment of delusions have frequently proved successful (Davis, Wallace, Liberman, & Finch, 1976; Liberman, Teigen, Patterson, & Baker, 1973; Nydegger, 1972), this tells us little about the origins of delusional beliefs.

Another behavioural approach to delusions is to assume that delusions are reinforcing through avoidance. Mednick (1958) suggested that patients with schizophrenia were sensitive to anxiety and stress (which may well be true (Day, Neilsen, & Korten, 1987; Neuchterlein & Dawson, 1984)), and therefore that avoidance through delusional ideation may be welcome. Dollard and Miller (1950) suggested that delusions might reduce aversive feelings of guilt or shame by approaching, but distorting the true state of affairs, while Shimkunas (1972) suggested that delusional talk ensures that people avoid you, lessening the danger of threatening intimate disclosure.

Overall, however, these models are unsatisfying. The fact that psychopathology gets worse in conditions of stress is not a good test of any model. Moreover, such models seem to invoke, but not address, cognitive variables. Such variables will be addressed in this thesis.

2.6.2.2.5 Theories of persecutory delusions and paranoia

Winters and Neale (1983) also comment on specific theories of persecutory delusions. These theories, and others, will be discussed in the next chapter.

2.7 Conclusions

This chapter has addressed a number of issues of importance to this thesis. Delusions have been described and classified, and the shortcomings of these approaches discussed. Although weaknesses remain, these are, I believe, much less important than the corresponding difficulties in the case of schizophrenia. It would be attractive if clearer aetiologies were apparent in delusions than in schizophrenia. Delusions themselves, however, may be heterogeneous. It is for this reason that delusions specifically of persecution are the focus of the empirical work reported in this thesis.

The causes of delusions are still unclear. It seems that physical, biological, aetiological approaches assume that delusions are ununderstandable (Jaspers, 1912/1963). The biological explanations are therefore reductive, playing down or ignoring relationships to personal qualities or to the individual's social world. On the other hand, psychological models assume that delusions are (in one sense or another) meaningful and relevant. This is even more true in the case of paranoia, which is frequently assumed to be to do with the self and the social world. Paranoia and delusions of persecution will be discussed in the next chapter.

Chapter 3.

Paranoia

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3.1 Introduction

In this Chapter, paranoia and persecutory delusions will be discussed in detail. The history and phenomenology of the concepts will be discussed, as will the relationship between the two. Measurement and assessment approaches will be reviewed. Finally, psychological theories of the causes of paranoia and persecutory delusions will be critically reviewed.

3.2 History of paranoia and persecutory delusions

Paranoia is a term which is widely used in common language. Its modern (OED) definition is "Mental derangement; specifically chronic mental unsoundness characterized by delusions and hallucinations" (Simpson & Weiner, 1989). Lewis (1970), has observed that the meaning of 'paranoia' and 'paranoid' has fluctuated over the centuries. The literal translation of the Greek "paranoid" is "by the side of the mind' (Hamilton, 1985) and was, Lewis (1970) suggests, used in a general manner to mean crazy or mad. This does not necessarily mean in a pejorative manner, but rather in a forensic sense, as a citizen judged paranoid would be disenfranchised and his (always his) property put in the care of his son (Bury, 1926). Lewis (1970) comments that this was not a medical diagnosis, as dementia might lead to 'paranoia' but so could "thinking amiss" (p.2).

Lewis (1970) suggests that paranoia had become used as a synonym for dementia by the eighteenth century. Nevertheless, as I have shown in Chapter 1, delusions of persecution (unfounded conviction that one's neighbours are plotting murder) have been described as symptoms of mental illness in medieval literature. The fact that the term paranoia was not used in conjunction with such beliefs of persecution

means that it is very difficult to track the development of the concept.

It appears that the use of the term paranoid subsided until a resurgence in Germany in the first half of the nineteenth century (Lewis, 1970). Heinroth (1818) pioneered the modern use of the term paranoia as a distinct, delusional, disorder. He suggested four subtypes: ecnoia (a single false idea resulting in gross distortions of person's relation to the world), paraphrosyne (supernatural delusions), moria (megalomania) and paranoia catholica (a mixture of emotional, cognitive and behavioural abberations). In contrast Kahlbaum (1863), writing slightly later, viewed paranoia as a single delusional condition (authors cited in (Lewis, 1970)). Kahlbaum's view formed the basis of Kraepelin's first classification of schizophrenia (in the 1896 German language edition of his textbook (Kraepelin, 1907) into dementia praecox, catatonia and dementia paranoides, later (in the 1899 edition) collapsed into one - dementia praecox. Later again, in 1912, Kraepelin suggested that paranoia should be used in the case of an illness where there are clear delusions in the absence of other psychopathology (cited in Lewis, 1970). Lewis (1970) suggests that Kraepelin's changing usage indicates his lack of confidence in the term.

In the English-speaking world a similar confusion of usage can be seen. In the UK, Tuke (1892) objected to the use of the word 'paranoia' while Norman (1899) used the term to describe systematized delusional insanity. In the United States, Meyer's (1951) acceptance of Freud's (1911/1950) psychodynamic model of paranoia led to the widespread use of the term, and to a diminution of any distinctions between delusional and non-delusional paranoia. This point will be returned to below. Lewis (1974) commented that paranoia was an "American-named psychosis" (p.34). This is, as I have just demonstrated, untrue although it is possible that paranoia is a very American phenomenon.

3.3 Relationship between paranoia and persecutory delusions

The links between paranoia, persecutory delusions and paranoid schizophrenia are close but confused. As mentioned in previous Chapters, in DSM-IV the term 'delusional disorder' is used instead of the term 'paranoia', to describe a syndrome characterised by delusions, usually of a persecutory theme, in the absence of other psychotic symptoms such as hallucinations or thought disorder. This view is mirrored in the official (OED) dictionary definition. In ordinary language, however, the word 'paranoid' has become general, applied to people who either have an unreasonable complaint of victimisation or unwarranted complaints, or unreasonable fears of threat.

Although the basis of many psychiatric classifications is that distinctions can be drawn between normality and abnormality, and between types of abnormality, it is clear that more general commentators have assumed that paranoid traits are seen in otherwise sane individuals. In fact, even within psychiatry, the spread of paranoid ideas is recognized in the diagnosis of paranoid personality disorder.

3.3.1 Paranoid Personality Disorder

Personality disorder is defined in DSM-IV as being an "enduring pattern" involving (at least two of) cognition, affectivity, interpersonal functioning and impulse control (P633). The paranoid subtype (301.0) is defined as a "pervasive distrust and suspiciousness of others such that their motives are interpreted as malevolent" and sufferers must display at least four of the following:

a) Unjustified suspicions that other people are exploiting, harming or deceiving them

b) Preoccupation with unjustified doubts about the loyalty or trustworthiness of friends

c) Reluctance to confide in others because of the unwarranted fear that information will be used maliciously against them

d) Interpretation of benign remarks or events as revealing hidden demeaning or threatening messages

e) Bearing of grudges

f) Perception of attacks on their character, not apparent to others, which are quickly reacted to angrily

g) Recurrent unjustified suspicions that their spouses are unfaithful.

Essentially, people with such a disorder are described as being "suspicious and cynical" and "bear grudges and take criticism extremely badly" (Chadwick, 1995) (p.9). If we accept the concept of 'over-valued ideas' as contrasted with delusions referred to above, paranoid personality disorder appears to be the emotional and behavioural concomitants of over-valued persecutory ideas.

Paranoid personality disorder appears to be relatively common in the general population (Turkat & Banks, 1987), and has been placed on a continuum, where "paranoid schizophrenia should be regarded as a more severe form of paranoia, which is a more severe form of paranoid personality" (Romney, 1987; p.651). It may well also be claimed that paranoid personality disorder is a severe form of a schizotypal trait (Bentall, Claridge, & Slade, 1989; Claridge, 1993; Claridge, 1987). It has been suggested (Kendler, Gruenberg, & Tsuang, 1985) that paranoid personality disorder is genetically linked to schizophrenia, although this association appears weak (Watt, 1985). Maier, Lichtermann, Minges and Heun (1994) in fact found that paranoid personality disorder was more frequent in relatives of people with depression (2.9%) than in relatives of people with a diagnosis of schizophrenia (1.7%).

Persisting doubts as to the validity of 'paranoid personality disorder' remain. For example, in a report of a cognitive-behavioural intervention, Williams (1988) reports a patient as having a paranoid personality disorder characterised by "interpersonal conflict, social withdrawal, difficulty relaxing, and dysphoric mood" (p.570).

3.4 Significance and social impact

Harper (1994) criticises Lewis's (1970) historical account of paranoia for two reasons. First that it overvalued modern 'objective' theories, but also that political and ideological factors influenced the development of paranoia as a concept in Western psychiatry. Certainly the term paranoia has been widely employed in social and political writings.

As an example, I conducted a limited search in the computerized data-base of a national UK newspaper (The Independent) for stories containing the terms paranoid or paranoia. For the period from 1st January 1995 to 31st December 1995, 51 such stories were recovered. Of these, 11 referred to patients of the mental health services, 12 referred to violence without reference to mental health (for example the reporting of the build-up to minor riots), 9 referred to politics and foreign affairs, and 19 referred to art, cinema and book reviews. I did not conduct a comprehensive analysis of these articles, but my impression was that the later category essentially consisted of reports of artistic activities referring to the other three categories (a review of 'Reservoir Dogs' and the paranoia and violence within that film, for example).

In particular, two areas seem important. First, paranoia is used repeatedly in political contexts. Secondly, paranoia and persecutory delusions are associated with violence.

3.4.1 Politics

Paranoia has been described as a characteristic of a vast variety of political systems. Glass (1988) suggested that the utopian systems of both Plato's 'Laws' and Hobbes's 'Leviathan' were characteristically paranoid. That is, Glass suggests, the systems advocated watchfulness and the suppression of dissident speech and action. The term paranoid has been used to describe a great number of political systems from Tudor England between the 15th and 17th centuries (Smith, 1986), the UK in the 18th century (Porter, 1989) to the 19th century (Wheatley, 1992). In more modern times, both American (Lockard, 1994) and Soviet (Gaddis, 1993) Cold War political systems have been described as paranoid.

Political leaders as individuals have also been described as paranoid. Robins (1986) identifies Hitler and Stalin as "charismatic paranoids" (p.15), combining paranoia with a charismatic personality. This analysis has been reflected in a number of historical analyses (Campo, 1977; Fusswerk, 1982).

However, three doubts remain. First, given that paranoid personality disorder may be as invalid a diagnosis as schizophrenia, making such assertive claims as to the mental health of powerful people long dead seems dubious. Secondly, what such authors seem to be describing is an authoritarian approach. While I can imagine links between this and paranoia, I know of no empirical studies supporting a claim that the two are identical. Finally, many of the people described as paranoid were the targets of numerous assassination attempts. Tudor statespeople, Hitler, and Stalin were all targets for assassination (Sifakis, 1991) (Hitler being physically injured by a bomb placed by a Lieutenant Colonel on his General Staff).

It seems that what such authors mean by paranoia is therefore not "Mental derangement; specifically chronic mental unsoundness characterized by delusions and hallucinations" as defined by the OED. Rather they mean a combination of fear of

threat or retribution with aggressive defences (by this I do not only mean psychological defences, but also murdering one's enemies) and a scapegoating, other-blaming approach. For example, Hitler is reputed to have achieved much of his political success by stressing the positive attributes of the German people, while (and by the means of) demeaning others, notably the Jews. As will become apparent in this thesis, I believe this combination of vigilance for threats and blaming others for failings is characteristic of delusions of persecution and will be discussed at length later in this thesis.

3.4.2 Violence

Referring to the so-called Jonestown suicide cult, where followers of a minor religious cult committed mass suicide, Lasaga (1980) identified Jim Jones as a 'paranoid leader' and Jonestown as a mini-totalitarian state ruled by a delusional and paranoid person. Of course the Jonestown story ended in mass suicide - or murder. The links between paranoia and violence are partially exemplified in the proceeding section - many of those political leaders termed paranoid were despots. However paranoid delusions also seem to lead to more conventional violent acts.

The case of James Hadfield (attempted assassin of George III) was mentioned in Chapter 2. Hadfield, labouring under a variety of delusional beliefs, was found not guilty of treason by virtue of insanity in 1800. His insanity, and his paranoid delusions, are apparent in contemporaneous accounts. The Times on 17th May 1880 reported almost verbatim the Privy Council's investigation of his attempt on the life of the King. Under questioning Hadfield claimed that he has tried to kill the King because he had been persecuted in France, without trial. As a result he felt he was a great martyr and was to be persecuted like Jesus Christ. His attempt on the life of the

King was an attempt to further his progression to martyrdom (Anonymous, 1800a). This case is particularly interesting in that Hadfield's acquittal left the establishment wondering what to do with him. He was, in fact, incarcerated in Newgate Prison overnight. The following day, spurred on by the Lord Chief Justice's call that "Something must be done" (Anonymous, 1800b), Parliament passed the Criminal Lunatics Act of 1800, which enabled the detention of disturbed offenders (including Hadfield) for indeterminate periods (Moran, 1985b). Soon afterwards, a Glaswegian tanner called Daniel M'Naughten attempted to kill the Prime Minister, Sir Robert Peel in 1843. Not having the benefit of newspaper photographs, M'Naughten mistook the Prime Minister's aide, Edward Drummond for Sir Robert, and shot him instead. (M'Naughten is, for some reason, occasionally referred to as a carpenter, and M'Naghten (for example in Prins, 1986), but these errors appear to reflect a reliance on secondary sources). Again M'Naughten was clearly and delusionally paranoid. Believing that he was being persecuted for not voting for the Conservatives, M'Naughten decided to kill his chief persecutor, the Prime Minister. At his trial he argued that: "The Tories in my native city have compelled me to do this; they follow and persecute me wherever I go, and have destroyed my peace of mind. They followed me to France, into Scotland, and all over England; in fact they follow me wherever I go; I can get no rest for [sic] them night or day. ... They have accused me of crimes of which I am not guilty; they have done everything in their power to harass and persecute me, in fact they wish to murder me. It can be proved by evidence. That's all I wish to say at present.... I am a very different man to what I was before they commenced this system of persecution." (Anonymous, 1843). M'Naughten was found not guilty on the grounds of insanity, a verdict which met with the displeasure of Queen Victoria. As a result, the Law Lords formulated the M'Naughten rules for the establishment of an insanity defence (Moran, 1985a). M'Naughten was reported to still be delusional two years later; "He still imagines that he is pursued by secret

enemies, and has gone to the extent of throwing any missiles at supposed foes... M'Naughten, in consequence of the violence he has displayed, has since been constrained by a straight-jacket or "muffle" as it is now called." (Anonymous, 1845). As this reporter commented, it is "not a little singular" that the victims of M'Naughten's attacks were two men also confined to Bedlam - The Bethlem Hospital - for criminal insanity. For myself this raises an interesting question of the Victorian treatment approaches to forensic psychiatry.

On an anecdotal level, Prins (1986) notes that many assassins, rapists and murderers seem have been suffering from paranoid delusions. This view is supported by case studies (Benezech, Bourgeois, Boukhabza, & Yesavage, 1981), reviews (Buchanan, 1993; Silva, Leong, Weinstock, Sharma, & Klein, 1994) and suggestions as to how to manage such potentially violent individuals (di Bella, 1979; Strentz, 1986).

It appears, however, that violence in paranoid subjects is not a necessary concomitant of delusional beliefs. Shore, Filson, Johnson and Rae (1989) comment upon the so-called "White House cases", individuals hospitalized because of their psychotic preoccupation with American politicians. Although paranoid delusions were common, the best predictor of arrest for violent crime after hospital discharge was an arrest for violent crime before hospitalization. On a more empirical level, in a sample of psychiatric patients with a general range of delusions (Buchanan, Reed, Wessely, Garety, Taylor, Grubin, et al., 1993), about half of the individuals reported that they had acted at least once in accordance with their delusions, although violence was uncommon. In a subsequent study, Buchanan and colleagues (1993), found that the likelihood of acting on delusions was unrelated to phenomenology of the delusion, but was predicted by the perceived evidential support for the belief, and negative emotions. Similarly, Calcedo-Barba and Calcedo-Ordonez (1994) found that the violence found in paranoid schizophrenics living in the community was much more

related to social abilities than psychopathology.

3.4.3 Querulent Paranoia

A final group of social problems identified in association with paranoia is so-called querulent paranoia. This has been described in terms that imply illness (i.e. "persons suffering from querulent paranoia") and is characterised by the making of vexatious, false allegations (Freckelton, 1988). Astrup (1984) reports on a series of admissions to a Norwegian hospital with a diagnosis of querulent paranoia. The common trait of these patients was described as an assertion of their rights and their strong feelings of being unjustly treated. Despite this rather scanty basis for a diagnosis of illness, biological treatments such as ECT and psychotropic drugs were employed. The use of neuroleptic medication for 'querulous-litigious delusional disorder' has been reported elsewhere (Ungvari & Hollokoi, 1993). Stalstrom (1980), for one, has commented upon the problems and dangers of the possible misuse of psychiatric diagnosis for political and social purposes not originally intended as coming within the domain of diagnostic categories.

3.4.4 Normal 'Paranoia'

If the above analyses are correct, one conclusion would be that paranoid ideation (falling short of delusions, or even over-valued ideas) is widespread in the general population. This issue will be partially dealt with below, when discussing scales for the measurement of paranoia in 'normal' subjects. It is worth, however, mentioning one area where quasi-paranoid ideas seem widespread - the feeling that

someone is watching you.

John Coover, Professor of Psychology at Stanford University, and Fellow of Psychical Research investigated the "feeling of being stared at" as part of a series of studies of telepathy, clairvoyance etc. (Coover, 1917). In an initial survey, he found that 77% of 1286 university students believed that they were generally aware when they were being watched. Coover proceeded to examine whether this widespread belief had any basis in fact. In single-subject experiments with ten different subjects, a subject sat with her or his eyes closed and covered while the experimenter, seated behind her or him, shook a die. On odd numbers he stared at the back of the subject's head, on even numbers he closed his eyes and thought of a favourite landscape. Clearly, by chance, the subjects should have correctly guessed whether or not they were being watched 50% of the time. Of 1000 guesses, 502 were correct. As Coover (1917) concludes: "the belief in 'the feeling of being stared at is quite common...[but]..shown to be groundless" (p.152).

3.5 Measurement

The measurement of the symptoms of schizophrenia and delusions has been discussed in previous Chapters. The area of paranoia and delusions of persecution is perhaps unique in that the problems with the delusional / non-delusional distinction have led to the wide-spread use of dimensional paranoia scales.

3.5.1 Paranoia scales

A number of contrasting questionnaires and scales have been developed. The vast majority of these seem to owe their origins to the MMPI - the Minnesota Multiphasic Personality Inventory. Dahlstrom, Welsh and Dahlstrom (1975), report a number of paranoia scales using MMPI items. I do not intend to cover all of these as they are discussed in Fenigstein and Vanable's (1992) development of a paranoia scale I shall discuss in detail below. On a general level, Poreh, Chapin, Rosen and Youssef (1994) found that the Delusions scale on the Scale for the Assessment of Positive Symptoms (Andreasen, 1984) and Scale 6 (Paranoia) on the MMPI (Dahlstrom, et al., 1975) correlated significantly in people with a formal diagnosis of schizophrenia. In fact the MMPI is so ubiquitous a measure that many researchers use its items as comparisons for assessing the construct validity of new measures of paranoid ideation (for example; Costa, Zonderman, McCrae, & Williams, 1986).

Fenigstein and Vanable (1992) examined several paranoia scales derived from the MMPI, extracting relevant items corresponding to a definition of paranoia along the lines of paranoid personality disorder (taken from Magaro (1980) and Millon (1981)). They developed a 20-item Paranoia Scale aimed at assessing paranoid ideation in normal subjects. This scale has acceptable internal consistency (alpha = 0.84) and test-retest reliability ($r = 0.71$) (Fenigstein & Vanable, 1992). Paranoia Scale was designed for non-patient participants, and construct validity was assessed by its correlation with other relevant questionnaires. Fenigstein and Vanable (1992) suggest that the correlation of their Paranoia Scale with a measure of self-consciousness (Fenigstein, Scheier, & Buss, 1975) is particularly important, indicating a causal role for self-consciousness. This point will be referred to later in this thesis.

The Paranoia Scale has been used by other researchers, with correlations

between scores on that measure and related items such as Machiavellianism (Christoffersen & Stamp, 1995). Smari, Stefansson and Thorgilsson (1994) found that scores on the Paranoia Scale correlated with clinical ratings of paranoia. However, paranoid and non-paranoid patients with a diagnosis of schizophrenia both scored highly on the Paranoia Scale, with no differences between these groups.

Despite doubts about the bases of these scales, the fact that they seem incestuous in their development, and their reliance on doubtful theories of personality structure, the findings of Poreh and colleagues (1994) are important. Clinically diagnosed patients with delusions of persecution score highly on these measures, while non-patient samples show predictable associations with other measures of cognitive functioning. However, the findings of Smari and colleagues (1994) also suggest that paranoid ideation is complex. It may be the case that measures applicable for normal and clinical populations are different.

3.6 Psychological theories of aetiology

Theories of delusion-formation in general have been discussed in Chapter 2. This section is more specific in its aim - to discuss psychological theories of the genesis of delusions of persecution and paranoia.

3.6.1 Freud

Sigmund Freud's theory of the causes of paranoia has been extremely influential. In essence it suggests that paranoia develops as a defensive support for a

denial of homosexual love. As mentioned above, broad acceptance of this model seems to have shaped American psychiatry's approach to paranoia. Strentz (, 1986), based at the FBI's Special Operations & Research Unit, suggested that "Male PS (paranoid schizophrenic) HTs (hostage takers) have problems in gender identity" (p.12). It is likely that this statement is an indirect legacy of Freud's (1915) theory.

Freud's understanding of paranoia developed over a number of years. The first case he reported (Freud, 1896/1950) was a young woman plagued by chronic paranoia. Freud suggested that her delusions were defences against unacceptable unconscious self-reproaches. In this original case, the reproaches were sexual in nature and apparently stemmed from half-remembered, half-repressed memories of sexual incidents in childhood. Although the role of psychological defense mechanisms in paranoia remained, Freud's general theory changed thereafter.

A senior German judge, Daniel Paul Schreber, suffered two episodes of paranoid psychosis between 1884 and 1885 and between 1893 and 1902 and wrote an autobiography detailing his experiences (Schreber, 1903/1955). Freud never met Schreber but, based entirely on Schreber's autobiography, developed a major theory of paranoid delusions. Although Freud is famous, in part, for developing the idea of close clinical observation as a basis for scientific psychology, he argued that Schreber's autobiography was appropriate material for psychoanalysis. In a startling undermining of his own approach, Freud suggested that an autobiography gave as much (and as honest) material as direct examination of a patient because; "...paranoiacs cannot be compelled to overcome their internal resistances and since in any case they only say what they choose to say" (Freud, 1911/1950; p.9).

On the basis of Schreber's autobiography, Freud (1911/1950) proposed a revised psychoanalytic theory of persecutory delusions. Paranoid delusions, Freud claimed, are parts of a process protecting the conscious ego from awareness of conflict with unacceptable homosexual impulses stemming from the id. Such homosexual

urges are, Freud argued, denied and/or contradicted and then countered by the defences of rationalization and projection. A male patient is essentially confronted with the idea that, "I (a man) love him (a man)". This is unacceptable, leading to reaction-formation and the idea: "I do not love him, I hate him". Such inhuman hatred is still unacceptable, and is rationalized as: "I hate him because he hates and persecutes me".

Freud's account is colourful, but has been widely challenged. Freud's model suggests that the delusional persecutor is a person for whom the patient expresses secret homosexual love. However, paranoid people frequently do not know their persecutors, or even have a specific idea of who they might be (this objection was first raised by Guirnaud (1950), cited in (Arthur, 1964)). Moreover, many patients fear persecution by members of the opposite sex, which are unlikely to be fuelled by repressed homosexual feelings. There exists also an interesting study by Busse (1989) using data from Schreber's physician, Paul Emil Flechsig (1847-1929). This suggests two things: First that Freud's clinical account account ignores important aspects of Schreber's illness. Second, suggests that Flechsig may have been conducting an affair with Schreber's wife. As the author suggests, this may have contributed to Schreber's condition.

Several tests of Freud's model have been conducted. Klaff and Davis (1960) and Moore and Selzer (1963) reported more overt homosexuality and more homosexual concerns in patients with paranoid schizophrenia than other groups, while Aronson (1952), Grauer (1954) and Meketon, Grifith, Taylor and Wiedman (1962) reported 'homosexual' responses by people with paranoid delusions. In experimental conditions, men with a diagnosis of paranoid schizophrenia pay greater attention to homosexual material than do other groups (Ericksen, 1951; Watson, 1965; Zamansky, 1956; Zamansky, 1958).

Such findings have not always been replicated (Lester, 1975) and are, in any case, difficult to interpret. It has been widely commented (Eysenck, 1985) that many

of Freud's theories are attractive but untestable. Although reporting of homosexual themes is consistent with Freud's model, so would an under-reporting (reflecting defensiveness). Finally, such findings are also consistent with a more general, more parsimonious, model invoking confusion in the self-concept. Such issues will be discussed later in this thesis.

It is notable that Freud's model of paranoia includes two elements, a defensive projection or externalisation of threatening material, and latent homosexuality. It is possible that the defensive component is more valid than the homosexual part. As I mentioned above, Freud's first case (Freud, 1896/1950) was a woman with sexual, but not homosexual, delusions. A third case (Freud, 1915/1956), reported after the analysis of Schreber's autobiography, was again of a woman with no homosexual ideation. In my opinion a prurient interest in sexuality (Nunberg (1938) for instance suggested paranoid individuals have a latent desire to fellate powerful men in order to absorb their power) has focussed attention away from more important aspects.

Psychoanalytic writers after Freud have suggested that persecutory delusions serve a defensive function, without necessarily stressing latent homosexuality. Bleuler (1911/1950), Henderson and Gillespie (1936), Kretschmer (1927) and Mayer-Gross, Slater and Ross (1954) all developed theories of paranoia which included attempts to attribute internal ideas to external agencies, but believed that these ideas were not necessarily homosexual in nature.

3.6.2 Hostility theory

In a theory that seems to owe a great deal to Freud, Swanson, Bohnert and Smith (1970) suggested that paranoid delusions reflect a projection of an individual's intense hostility onto others. To paraphrase Freud's linguistic or propositional

transformation; "I hate him" is changed to "He hates me".

It seems true that people with paranoid delusions tend to be hostile (Overall, Gorham, & Shawyer, 1961), and it might be true that the presentation of material with hostile content can exacerbate pathology (Silverman & Candall, 1970). However many of these studies are either invalid (using broad diagnostic categories) or unreplicable (Heilbrun, 1980). Secondly, the hostility expressed may be consequent upon perceived persecution, rather than cause it. Finally, it seems assumptive to view unconscious hatred towards the CIA as intrinsic. That is, the psychodynamic sources of the hatred are supposed to be founded in the individual's relationships, yet the paranoia is often focussed on strangers.

3.6.3 Cameron's stimulus generalization hypothesis

Cameron developed two theories of the aetiology of paranoia. In the first (Cameron, 1951), anxiety was seen as the key emotion. Cameron suggested that poor social skills lead to anxiety. As anxiety develops, a stimulus generalization gradient ensures that previously irrelevant stimuli now elicit anxiety. As the anxiety grows, perceptual ambiguity also grows, with delusions reflecting attempts to make sense of the disorganization.

Such an account has many aspects that accord with clinical experience. However, it does not successfully explain the onset of paranoia. In particular, the key question remains: Why do paranoid individuals respond to a stimulus generalization gradient with persecutory explanations?

3.6.4 Cameron's paranoid pseudo-community

Cameron's second hypothesis was less focussed on anxiety, and more on social skills deficits. He suggested (Cameron, 1955; 1959) that a person frustrated by a lack of social skills seeks refuge in fantasy and daydreams. However these primitive fantasies, born from frustration, also threaten the ego. Because the defences (fantasy) are immature, these threats are denied and repressed. Because the individual is not in contact with a normal social network, paranoia develops in a context of imagined and delusional others - the paranoid pseudo-community.

Although this model appears to me to be over-complex, with many assumptions, others have found it persuasive (Winters & Neale, 1983). Empirical data, however, undermine Cameron's central thesis, that paranoia is born from a lack of social skills. Zigler and Levine (1973) found that people diagnosed as having schizophrenia had a higher premorbid social adjustment if they had paranoid symptoms.

3.6.5 Cybernetic model

Melges and Freeman (1975) suggested that paranoia stems from a fear that one is losing control to others. They further suggest that these fears might be triggered by temporal disorganization (alterations in the rate, sequencing or goal-directedness of thinking). They argued that, as temporal disorganization increases, suspiciousness and the perception of threat increases. Evidence for this view was claimed from a number of studies (see Winters & Neale, 1983) where cannabis induced temporal disorganization and paranoia.

As with Cameron's theories, this model seems to have attractions. Again,

however, the weakness in the theory (why, given this sort of threat, paranoid subjects make paranoid interpretations) is papered over. The answer, according to Melges and Freeman (1975), is that paranoid individuals have an extreme need to maintain self-control and to control others. This appears a circular and tautological argument.

3.6.6 Heilbrun

Heilbrun developed a theory of paranoia that incorporates elements from the information-processing and learning theory approaches. Heilbrun (1973b, 1975) distinguishes between 'open' and 'closed' thinking styles in the response to aversive maternal behaviour. While both of these could lead to paranoid thinking, the routes to paranoia differ. In the 'open' approach people will search actively for information, but process information with a bias towards perception of negative self-evaluations and negative interpretations. In the 'closed' approach, evaluative information is avoided, and social situations shunned.

Heilbrun and colleagues conducted a number of empirical tests of this hypothesis (see Winters & Neale, 1983). However, the majority of these were analogue studies - conducted on non-deluded individuals. This approach can have weaknesses. Thus the majority of the evidential support for Heilbrun's model reported in Winters and Neale (1983) consists of research demonstrating that open-style and closed-style thinkers show similar patterns of information processing to reactive and process schizophrenics. But since the distinction between open and closed thinking styles is made on the basis of the same information processing measures, and people with both reactive and process schizophrenia can show paranoid delusions, it is difficult to make much of such findings.

3.6.7 Sullivan's humiliation theory

Harry Stack Sullivan (1965) suggested that self-blame was a central underlying theme in paranoia. Echoing the recurrent theme of externalization of unacceptable (but not necessarily homosexual) self-criticisms, Sullivan suggested that paranoia results from a tendency to blame others for personal inferiority. Sullivan also suggested that a suspicious outlook on life contributed to paranoid misinterpretations.

As is already clear, such theories are ubiquitous in paranoia. Sullivan's does not strike as particularly unusual.

3.6.8 Colby

Colby's model of persecutory delusions (Colby, Faught, & Parkinson, 1979) has clear associations with Freud's original formulation, and with Sullivan's model. Colby suggests that paranoia stems from a tendency to perceive or generate threats to their self-esteem combined with a protective mechanism of projection and externalization of the threat to others. In an interesting variant of this model, Colby (1981) described a computer simulation model of his theory. A Turing indistinguishability test was conducted, comparing his computer model with a real paranoid patient. Since clinical judges were reported as being unable to distinguish the two, the computer model was considered a success.

One of the main benefits of Colby's theory is that it is testable. People with delusions of persecution should readily perceive potential threats to self-esteem, and they should also locate the source of such threats as external. Many of the studies reported in this thesis are compatible with Colby's model.

3.6.9 Camouflaged depression

In a final summation of many of these psychological theories of persecutory delusions, Zigler and Glick (1988) suggested that paranoia is a form of camouflaged depression. This suggestion was originally formulated as a statement regarding classification, with paranoia being properly seen as a form of affective disorder. However the claim is also a causal one. Taking as sources the psychological theories of paranoia briefly reviewed above, Zigler and Glick (1988) claimed that paranoia consisted of a low self-esteem or feelings of inadequacy (the depression part) covered with defensive projections of these feelings onto others.

Although Zigler and Glick's (1988) model is perhaps as well-known as Freud's today, largely because they published in a widely read journal, it essentially adds little, and lacks important details of mediating cognitive processes.

3.7 Conclusions

3.7.1 Nature

Although delusions of persecution are seen as important symptoms of schizophrenia (and related disorders) in DSM-IV, paranoid ideation is evident in other contexts. The presence of paranoid personality disorders suggests paranoia is seen in non-psychotic conditions. The paranoid ideas behind a number of violent acts are too frequent to be coincidence. Finally, although scales measuring paranoia have problems, the existence of abnormalities and biases in the thinking of normal people expressing paranoid ideas suggest that paranoia is a concept best thought of in a dimensional way.

3.7.2 Causes

With few exceptions, authors have suggested that paranoia is self-blame projected outwards. Two questions ensue. First, is this true? Roger Bacon (1266/1928) identifies repeating the opinions of authority figures and following the majority view as two of the four causes of error. The fact that Freud and others have suggest such a cause of paranoia does not mean it is correct. Second, if this is a true cause of paranoia, how does it work?

One of the major criticisms of previous self-defensive or self-protective theories is that they propose such processes as 'projection' or 'externalization'. However, if paranoid individuals defend vulnerable self-concepts, specific processes achieving this end should be discoverable. I do not believe such processes are evident in the accounts reviewed above. One possible such process will be discussed later in this thesis.

Chapter 4.

The self-concept and self-discrepancy theory

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4.1 Introduction

This Chapter is not intended to give a comprehensive picture of the current psychological theory of the self-concept. Such an ambition would be misplaced, due to the vastness and complexity of the subject. Rather I intend to provide a brief overview of those aspects of the topic most pertinent to this thesis. In addition a particular approach to the study of the self-concept - self-discrepancy theory - will be introduced.

4.1.1 Self-concept in psychology

Descartes, Hume and Locke have been credited, as with much else, as initiating the sense of the 'self' in Western philosophy (Hayes, 1993). All important views of the self-concept are essentially cognitive in nature, as other psychological models place little emphasis on any active role. William James (1890) stressed the role of social comparison in the development of the self-concept, arguing that we develop an idea of what we are like by comparison with others.

4.2 Cognitive approach

Brewin (1996) reviewing the theoretical basis of Cognitive-Behaviour Therapy, suggested that mental representations of knowledge about the self (self-concepts) underlie disorders such as depression, social phobia and generalized anxiety disorder. One conclusion of this thesis is to suggest that the self-concept is central to paranoid ideation. However, the nature of such representations requires

some debate.

Generally, cognitive psychology assumes that the amount of information potentially available to an individual, and the number of ways in which elements may be combined, is so enormous that specific cognitive systems exist to aid efficient processing. Such systems or processes are not thought to be random, but are instead developed from past experience. These are termed frames (Minsky, 1975), scripts (Abelson, 1976), or schemata (Anderson, 1983). Schemata are 'mental models' (Johnson-Laird, 1985) of the world, hierarchically organised knowledge systems incorporating mental representations of objects, people, events and situations. I will use the term 'schemata' in this thesis.

4.2.1 Self-schemata

Although earlier evidence that individuals actively processed self-related information existed (Bem, 1967; Bem, 1972; Snyder, 1974), Hazel Markus was the first to conceptualize this as self schemata. These she defined as "cognitive generalizations about the self, derived from past experience, that organize and guide the processing of self-related information contained in the individual's social experience" (Markus, 1977; p.64).

Self-representations stem from many sources. Internal, physiological reactions (Bandura, 1977a) and emotions (Anderson & Ross, 1984) act as material from which inferences may be made. Reflecting William James (1890), people are also known to learn about themselves from others (see Markus & Wurf (1987) and Kihlstrom & Cantor (1984) for reviews). Harter (1983) suggested that people's self-descriptions become progressively more abstract (from "good at doing sums" to "intellectual") with age.

Although to cover all the evidence of information processing guided by self-schemata would be exhaustive, many different strands of research suggest that the self-concept is central. Markus (Markus & Wurf, 1987) termed the active involvement of the self-concept in information processing about the self and others the "dynamic self-concept".

4.2.2 Dynamic self-concept

4.2.2.1 Intrapersonal information processing

Markus (Markus & Wurf, 1987) and Kihlstrom and Cantor (1984) both outlined several lines of evidence suggesting that the self-concept directs or guides information-processing. Bargh (1982) found that people attend preferentially to self-relevant information such as trait descriptors pertinent to the self. That is, when self-descriptive words were played to experimental participants, performance was disrupted. Markus herself (Markus, 1977) found that information congruent to the self was more efficiently processed, with more information being produced more swiftly, than was incompatible information.

Similarly, self-congruent information is well-remembered compared with information about other people or situations (again see Markus & Wurf (1987) and Kihlstrom and Cantor (1984) for reviews). People also make confident judgements in areas related to one's own self-description (Anderson & Ross, 1984). Finally, information that is incongruent with one's self-description tends to be rejected (Markus, 1977; Swann & Hill, 1982).

Perhaps what is more important, the self concept has been credited with important roles in self-regulation (Markus & Wurf, 1987). On a simple level, it is assumed that self-representations are important in determining expectations or self-

efficacy (Bandura, 1977b; Bandura, 1986) but also seem to play a role in setting or selecting relevant self-goals (Markus & Nurius, 1986).

Cognitive schemata also act as guides for action. As such, schemata (or scripts) contain self-referent information, information about what actions are available to the individual, knowledge about the likelihood of success and about the consequences of previous actions (Anderson, 1982). Schemata are equally implicated in monitoring and evaluating action. Carver and Scheier (1981) suggest that self-regulation is enhanced when attention is self-focussed. Although others disagree, with Kuhl (1985) and Wicklund (1986) suggesting that self-focussed attention impairs self-regulation, clearly the self-concept is centrally implicated.

The self-concept is also given a central role in the regulation of affect. There have been many proposed cognitive mechanisms for regulating affect. The vast majority of these involve the self-concept. In general (see Markus & Wurf (1987) and Kihlstrom & Cantor (1984)) these mechanisms are presumed to use processes of self-consistency or self-enhancement, by which information supportive of a positive or self-consistent set of self-representations is deliberately incorporated into the working self-concept. One mechanism regulating the self-concept is believed to be that of selective interpretation of events. The next Chapter will discuss selective and self-maintaining biases in causal attributions.

4.2.2.2 Self and social perception

Beyond its central role in intrapersonal cognitive processes, the self-concept is implicated in a large number of interpersonal or social cognitive processes (for reviews see Markus, Smith, & Moreland (1985) and Higgins & Bargh (1987)).

The most general finding is that people use the same criteria to judge other

people as they use to evaluate themselves (Fong & Markus, 1982; Lewicki, 1984). More specifically, if information about other people is provided along dimensions for which the individual has a well-developed self-concept, then that information is swiftly and efficiently processed. Moreover, such information is processed more deeply (Kuiper, 1981) leading to more (and more confident) inferences (Fong & Markus, 1982; Markus, et al., 1985).

It is interesting that a number of studies have suggested that there are links between the self-concept and social perceptions that serve to enhance one's self-concept. Tesser (1986) suggested that people present those self-descriptions that reflect themselves in the best light. However, this is qualified by the claim that it is performance relative to others, as opposed to absolute performance, that is important. Moreover, the fact that people tend to evaluate others on personally salient dimensions enhances the likelihood that one will be seen as superior (Lewicki, 1983). Presumably this also leads depressed individuals (with a negative self-concept) to see themselves as inferior.

4.2.3 Accessibility of self-representations

Because the self-concept has so many roles, is implicated in so many processes, and is multidimensional, many theorists have suggested that it is inappropriate to refer to one self-concept. Rather (see Markus & Wurf (1987)) the idea has developed that a "shifting array of accessible self knowledge" (Markus & Wurf, 1987; p.306) is active at any one time. Markus and Wurf (1987) review a number of empirical studies that appear to show that the self-concept is malleable. Markus and Wurf (1987) interpret these as suggesting that the contents of the "working" self-concept have changed. As an example, Fazio, Effrein and Falender (1981)

reported that questions about extroversion lead experimental participants to view themselves as more extrovert than do questions about introversion.

Markus and Wurf (1987) concludes that there is a convergence of opinion towards the position that the self-concept contains a variety of representations. Moreover, these representations need not be solely verbal or propositional in nature. Self-representations, she claims, can be cognitive, affective, propositional or in other formats, and may reflect a number of different views of the self (including 'possible selves' (Markus & Nurius, 1986)).

Such a view is particularly powerful, and relevant to this thesis. Many social situations and experimental procedures may be envisaged which alter the reported self-concept. However it seems unsatisfactory to conclude that individual events substantially alter the self as a stable structure. If one accepts the premise of the working or accessible self concept, alterations in the reported self-representations may be explained as subtly shifting the focus of self-knowledge.

4.3 Self-concept in schizophrenia and paranoia

As seen in Chapters 1, 2, and 3, the theory that 'schizophrenia' in general, and paranoia in particular, may involve the self has a long history. Bleuler (1911/1950) suggested that "In schizophrenia the alteration of the ego and its attitude towards the world is more pronounced than in any other psychosis" (p.143) while Schneider (1959) regarded a weakening of ego boundaries as the cause of first rank symptoms. More recently Laing's (1965) view of the nature of mental illness has been influential. As seen in Chapter 3, classic psychodynamic (and other) accounts of persecutory delusions more centrally implicate the self-concept.

Evidence concerning a causal role of the self-concept in schizophrenia and

paranoid ideation will be discussed later. There is also considerable evidence that schizophrenia is a condition that profoundly affects the sense of self (Fabrega, 1989). Two processes seem to operate here.

Being diagnosed as having schizophrenia is clearly an extremely stigmatizing experience (McGrath, 1984), with the public perceiving mentally ill people as dangerous and to be avoided (Penn, Guynan, Daily, Spaulding, Garbin, & Sullivan, 1995). Schizophrenia also makes one question oneself (McGrath, 1984) and the bases of one's self-beliefs. In such a context, a negative effect on the self-concept is almost inevitable. Patients perceive hospitalization, in particular, as highly stigmatizing (Townsend & Rakfeldt, 1985) leading to potential worsening of the clinical picture.

Warner, Taylor, Powers and Hyman (1989) directly investigated the effects of accepting or rejecting the diagnosis of mental illness. They found that an internal locus of control was a positive indicator, as was acceptance of the diagnostic label. This was held by Warner and colleagues (1989) to be support for a psychodynamic model of schizophrenia, but could reflect a more prosaic reason. Townsend and Rakfeldt (1985) reported that self-esteem in psychiatric patients was facilitated by definitions of insanity that exclude the patient. Internal locus of control is commonly seen as positive (Davidson & Strauss, 1992). Patients therefore seem to benefit from a positive and collaborative role in their recovery from mental illness, but a definition of the illness that minimizes their involvement. An approach that suggests "this isn't your fault, but you can fight your way out of this situation" seems indicated.

In addition, of course, schizophrenia is a disorder that profoundly affects cognition. As can be seen in the results summarized in Chapters 1 and 2, many information-processing systems are disturbed in schizophrenia. However it is interesting that, as Bleuler (1911/1950) and Schneider (1959) suggested, such disruption appears to disproportionately affect the self-concept.

The close involvement of self-referent processes and schizophrenia have led

many to claim that schizophrenia is an illness intimately tied to the self-concept "Schizophrenia is an *I am* illness" (Estroff, 1989; p.189, italics in original). Empirical research has not disproved this argument, but has revealed complexities in the self-concepts of people with a diagnosis of schizophrenia.

4.3.1 Complexities within the self-concept in schizophrenia and paranoia

Despite the general finding that patients with diagnoses of psychotic illnesses report significantly higher self-esteem than patients with a diagnosis of depression (Silverstone, 1991), studies using direct measures of self-esteem show only moderate levels of disturbance in psychotic patients. In particular, patients with a diagnosis of schizophrenia make no more self-rejecting statements than normal subjects (Kaplan, 1975), and often give higher ratings of self-satisfaction (Wylie, 1979).

More elegant studies have examined differences between a person's self perceptions and their ideals. These studies have again found that such differences are no greater in groups of patients with a broadly defined diagnosis of schizophrenia than in normal subjects (Ibelle, 1961; Rogers, 1958) and smaller in patients diagnosed as paranoid in comparison with those with a diagnosis of schizophrenia (Havner & Izard, 1962). In the latter study, comparisons were also made between the acceptance of self and acceptance of others. This again revealed greatest discrepancies in the case of patients with a diagnosis of paranoid schizophrenia, with particularly low opinions of family members.

Such high levels of self-esteem or positive self-representations are not necessarily incompatible with the theories of delusion-formation described in previous Chapters. In fact, a preserved self-concept is entirely predicted. However (again not incompatible with predictions) there is evidence of more complex disturbances of the

self in people with psychotic symptoms. People with a diagnosis of schizophrenia have been described as possessing poorly elaborated (Gara, Rosenberg, & Baker, 1989; Robey, Cohen, & Gara, 1989) and contradictory (Gruba & Johnson, 1974) self-concepts. Garfield, Rogoff and Steinberg (1987) concluded that; "schizophrenic patients do not have uniformly lower self-esteem than normals, but, rather, specific domains of self-esteem are affected" (p.225). Gara and colleagues (1989) also commented that these effects apply to perception of others, with characteristically stereotypical and rigid conceptions.

Such a conclusion resonates with the concluding remarks in Chapter 3, suggesting that an important way to explain the nature of paranoia could be to examine specific domains of self-representation. Within the general model of a multiple self-concept (Markus & Wurf, 1987), E.Tory Higgins proposed a model of the self-concept termed Self-Discrepancy Theory (Higgins, 1987) which will be discussed in detail below.

4.3.2 Self-consciousness

A slightly different approach to the issue of the self-concept in paranoia has been taken by Alan Fenigstein and his colleagues. Fenigstein and Vanable's Paranoia Scale (Fenigstein & Vanable, 1992) was discussed in Chapter 3. As was briefly mentioned there, the same authors also developed a measure of self-consciousness (Fenigstein, Scheier, & Buss, 1975). The Self-Consciousness Scale is a 23-item questionnaire tapping three aspects of self-consciousness: private self-consciousness, public self-consciousness and social anxiety. Subjects are asked to rate the degree to which they agree or disagree with a set of statements reflecting their own thoughts and feelings (e.g., "I reflect about myself a lot"), their awareness of their impact on others'

perceptions (e.g., "I'm very concerned about the way I present myself") and discomfort in social situations (e.g., "I feel anxious when I speak in front of a group"). This questionnaire has acceptable reliability (test-retest reliability alpha = .80; (Fenigstein, et al., 1975)).

Fenigstein and Vanable (1992), in a study of non-patient participants, found that public self-consciousness was associated high scores on their measure of paranoid thought. An earlier study (Fenigstein, 1984) had suggested that perceiving the self as a target of other people's negative attentions (as measured by the authors' own questionnaire) was correlated with high levels of self-consciousness. From these observations, Fenigstein and Vanable (1992) suggested that self-consciousness (or self-directed attention) was a cause of paranoid thought.

However, the established empirical support, obtained in studies of non-patient participants, has not been replicated in clinical samples. Smari, Stefansson and Thorgilsson (1994) found that scores on Fenigstein and Vanable's Paranoia Scale (Fenigstein & Vanable, 1992) correlated with clinical ratings of paranoia. In addition to their finding reported above in Chapter 3 that paranoid and non-paranoid patients with a diagnosis of schizophrenia both scored highly on the Paranoia Scale, Smari and colleagues failed to replicate previous findings in other ways. Specifically, paranoia was related to private, but not to public self-consciousness. Moreover, although paranoia was associated with extreme evaluations of people, public self-consciousness was not. Similarly unsupportive findings from my own research will be mentioned later.

Overall, therefore, Fenigstein and Vanable's Self-consciousness theory of Paranoia (Fenigstein & Vanable, 1992) has some merits, but does not constitute a complete or acceptable theory. First, like many psychodynamic theories, it fails to provide clear linkage between the putative variable (self-consciousness) and paranoid ideation. Secondly, of course, the empirical support is poor. Finally, however, there

does seem to be some merit in the specific argument that self-focussed attention may be important in paranoia, and greater merit in the wider argument that self-conceptual cognitive processes are important.

4.4 Self-Discrepancy Theory

As its name suggests, in self-discrepancy theory the discrepancies between a person's perceptions of different aspects of themselves are considered particularly important. Markus (Markus & Nurius, 1986; Markus & Wurf, 1987) and Kihlstrom and Cantor (1984) suggested many possible domains of self-representation. Those considered particularly important by Higgins (1987) include the Actual self, the Ideal self and the Ought self. The Actual self-representation incorporates those attributes (self-descriptors) you think you actually possess. The Ideal self-representation incorporates those attributes you would ideally like to possess, your ultimate goals for yourself, while the Ought self comprises those attributes you believe you should or ought to possess.

Higgins and colleagues developed the Selves Questionnaire (Higgins, Bond, Klein, & Strauman, 1986) to assess self-discrepancies, and a description of the measure provides a good description of the general approach. When completing the Selves Questionnaire, participants are requested verbally to generate up to ten attributes (self-representations) for each domain of self-representation. That is, to write down ten adjectives that describe themselves as they actually are (self-actual), ten adjectives that describe how they would like to be (self-ideal), and ten adjectives that describe how they should be or ought to be (self-ought). These latter representations function as self-directive standards or self-guides. In addition, participants are requested to generate the attributes they believe significant others in their lives would

use to describe them in each of these domains. Finally, participants are asked to give a four-point numerical rating of the degree to which they possess (or would like to or feel they should possess) each attribute. To compute numerical self-discrepancies, comparisons are made between domains. This involves using a well-structured scoring system based on a combination of verbal comparisons of synonyms and antonyms used in the different domains (using computerized thesauruses) and comparisons of numerical ratings. On this basis the degree of similarity and dissimilarity (discrepancy) between the representations is calculated (see Scott & O'Hara, 1993, for further details of the methodology). Although this does not represent a major theoretical innovation, it does represent a clarification and operationalization of Markus and Kihlstrom & Cantor's multiple self-representations.

4.4.1 Self-discrepancies in anxiety and depression

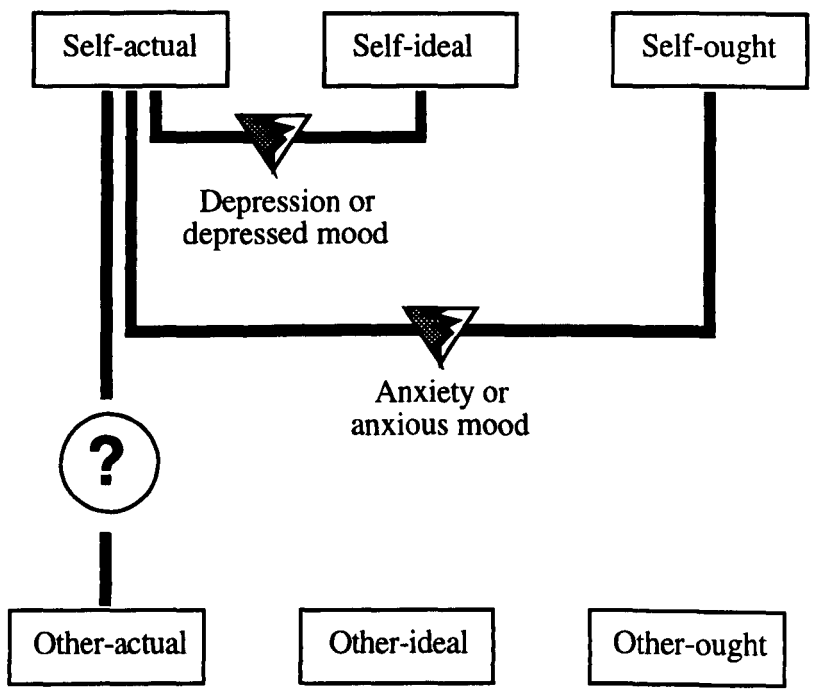
Self-discrepancy theory suggests that discrepancies between different representations (actual-self vs ideal-self or actual-self vs self-as-it-ought-to-be) are associated with particular affective states. Actual:ideal discrepancies are predicted to be associated with depression, while actual:ought discrepancies are associated with fear, worry and agitation. These predictions have been supported by empirical research on both clinical and analogue studies.

Dysthymic non-clinical participants (Strauman & Higgins, 1988), clinically anxious and depressed students (Scott & O'Hara, 1993) and depressed and socially phobic patients (Strauman, 1989) have all been shown to exhibit specific patterns of discrepancies predicted by the model. Such self-discrepancies also predict future emotional distress (Strauman & Higgins, 1988). Moreover, manipulations of these self-discrepancies in analogue participants have been shown to lead to predictable

changes in mood (Strauman & Higgins, 1987). In this latter study, participants were presented with self-discrepancies gathered in earlier administrations of the Selves Questionnaire. Priming self-actual:self-ideal mismatches induced momentary syndromes of dejection (sadness, decreased arousal) whereas priming self-actual:self-ought mismatches induced agitation (nervousness, increased arousal). Similar studies with patient samples (Strauman, 1989) also showed that priming of discrepancies has behavioural and physiological consequences. Patients required to think about actual:ideal discrepancies showed fluctuations in skin conductance together with a temporary slowing of their speech. Those required to think about actual:ought discrepancies, although exhibiting the same physiological changes, showed a temporary increase in their rate of speech. Similarly, autobiographical memory (Strauman, 1992) and even the physiological functioning of the immune system (Strauman, Lemieux, & Coe, 1993) have been shown to be affected by priming with information reflecting self-discrepancies.

A simple graphical representation of these results is presented in Figure 4.1.

Figure 4.1 Known and hypothesised relationships between self-discrepancies and emotional or psychological disorders



Note: The terms Self-actual etc. are described in the text, as are the empirical bases for the relationships claimed.

4.4.2 Self:Other discrepancies

One of the interesting aspects of the approach taken by the American self-discrepancy researchers is the summation of self-representations across viewpoints. That is, up to now I have discussed self-discrepancies within the self-as-I-perceive-myself point of view. In fact, although participants were asked to complete the Selves Questionnaire with the inclusion of sections asking what adjectives significant others would use for the actual, ideal and ought selves, the two main kinds of self discrepancy (actual:ought and actual:ideal) were summed across different viewpoints. In part this seems to have been an attempt to maximize discrepancies in analogue subjects, who tended to report internally consistent self-representations. However this also means that little attention was paid to discrepancies between different viewpoints.

As can be seen in Figure 4.1, other representations may be theoretically interesting. Potentially important domains of self-representation include the Other-actual (those attributes you believe other people think you actually possess), the Other-ideal (those attributes you believe other people would like you to possess) and the Other-ought (those attributes you believe other people think you should possess or you believe other people think you ought to possess). It is important to remember that these are still self-representations, as they do not reflect what others actually do believe, but individuals' beliefs concerning themselves.

Self-discrepancy theory provides a suitable framework for studying the self-concept of patients with paranoid delusions. It allows the independent measurement of the specific domains and viewpoints on the self. Evidence of the importance of such domain-specific abnormalities was outlined above. Moreover, an advantage of self-discrepancy theory is that it uses individuals' internal standards as reference points for the evaluation of the self. Conventional self-esteem measures, while possessing reasonable statistical reliability, may lack some validity by making comparisons with

external standards.

4.5 Latent self-representations

Squire, Knowlton and Musen (1993) discuss the possible differences between declarative and non-declarative memory systems. Brewin (1996) further discusses this distinction in terms of situationally accessible (or unconscious) knowledge and verbally accessible knowledge. He suggests that these different forms of knowledge may be stored separately in memory. Brewin (1989; 1996) also discusses the role of "deliberate, strategic" processes aimed at self regulation (Brewin, 1996; p.38). The implication is that processes such as causal attribution are related to the expression of verbally accessible knowledge. However the degree to which such strategic processes may be triggered by situationally accessible knowledge is open to debate. In most psychological disorders, but especially paranoia, latent (or situationally accessible) self representations may be particularly important. That is, discrepancies between different self-representations may remain latent until activated by an appropriate stimulus, situation or event.

4.6 Conclusions

At the end of Chapter 3, I posed two questions. The first asked what the self-concept of individuals with paranoid delusions might be. This question should be rephrased as asking what their self-representations are, and, more specifically, what self-discrepancies they display. Although I could discover no published research into self:other self-discrepancies, these are likely to be important in paranoia. The concept of paranoia implies a gulf between self and others, a feature likely to be reflected in the

self-concept. This issue will, I hope, be largely answered by the empirical studies reported in this thesis.

The second question asked what processes affect or moderate the self-representations of people with paranoid delusions. The most promising cognitive process in this regard is that of causal attributions. This issue will be discussed in the next Chapter.

Chapter 5.

Causal Attributions

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5.1 Introduction

In Chapter 4, I discussed a number of cognitive systems linked to the self-concept that serve to regulate and maintain positive self-representations. One important set of processes are causal explanations or causal attributions. Both Higgins and Bargh (1987) and Markus and Wurf (1987) discuss the role of attributions for events that pose potential threats to the self-concept in maintaining self-esteem. An example of this would be the finding of Kulik and her colleagues (Kulik, Sledge, & Mahler, 1986) that people make situational attributions for their own behaviour which is inconsistent with their self-view. That is, if you describe yourself as intelligent, and are then asked for your explanation for an episode of stupidity, you will give reasons that refer to situational influences - you will make excuses.

In general, causal attributions seem to play important and varied roles in human behaviour and cognition.

5.1.1 History

The story of causal attributions is generally assumed (Peterson, Buchanan, & Seligman, 1995) to have begun with Heider (Heider, 1958). His naive psychological approach is credited with triggering the ideas of both Kelley (1973) and Weiner (Weiner, 1986; Weiner, Russell, & Lerman, 1978) by suggesting that people make active and accessible attempts to understand their social world. Kelley, in particular, suggested that people act as 'lay scientists', attempting to find explanations for events in their social environment (Hewstone, 1989).

Causal explanations seem to be both common and important elements of cognition. Zullow, Oettingen, Peterson and Seligman (1988) estimated that one

explanation is found in every 100 words of ordinary speech. They also suggested that such explanations have important consequences, suggesting that dangerous actions taken by American President Lyndon Johnson during the Vietnam War were, at least in part, triggered by his over-optimistic explanations for past events. Studies of the consequences of causal attributions on a more ordinary level have tended to support this view of their importance.

Heider (1958) was one of the first to argue that the question of why events occurred was of great importance. He suggested that two broad classes of causes are given by individuals for the things that happen to them - personal and environmental. Personal, or internal, causes are construed as "relatively stable traits of the personality" (Heider, 1958; p.56) as opposed to environmental, or external, causes. This distinction in fact seems to be traceable to Aristotle (see White (1990), for a review of the philosophical bases of attributional research).

It is also interesting that, from a philosophical perspective, there exist distinctions between reasons, causes and explanations (White, 1990). This seems to shed some doubt on the bases of attributional theories I shall outline below, and suggests that the whole field is extremely complex. In a similar vein, McArthur (1972) has suggested that questions as to 'why' something occurred are different from questions as to 'how' they came about. Again, it is my impression that the complexities such distinctions suggest have not been adequately considered in traditional attributional theory.

Generally, the psychological response to understanding the issue of causal attribution seems poor. White (1990) discusses a range of philosophical approaches to cause and effect. Psychologists seem to assume that event B has cause A, and the interesting thing is what As people believe exist in different situations. The true picture is difficult to decide upon, but it seems that event B could have many causes (A, C, D, and E), could be its own cause, could be in a circular relationship whereby A causes

B, which causes A. Moreover there is a difference between causes, reasons and excuses. One conclusion might be that event B is likely to have, as potential causes, A, C, D and E. For a psychologist, which of these causes seems most salient for an individual may be an important issue.

Weiner (Weiner, 1986; Weiner, et al., 1978) is commonly credited with having developed a psychological theory of causal attributions. He was interested in the link between causal attributions and emotion, and particularly motivation. It has been argued that Weiner's research served to complete the dominance of cognitive models in psychology (Peterson, et al., 1995). As emotions and motivation were assigned to the status of consequences, dependent upon the explanations developed by individuals for the events in their lives, their role as primary psychodynamic forces declined. At the same time, because identical events could be interpreted or explained differently, with different consequences, purely behavioural approaches seemed inadequate.

Once developed as a theory, that attributional approach took two forms. The first investigated the rationality or appropriateness of individuals' explanations. Thus Nisbett and Ross (1980) suggested that social judgements are based on 'irrational' judgements and a use of information that is less than optimum. A second line of research, fundamental to this thesis, concerned Weiner's more central interests - emotions and motivation.

Most social-cognitive approaches stress the role of interpretations. For historical reasons the links between interpretative and attributional accounts of social cognition have been little understood. This seems to be a result of two forces. Abramson, Seligman and their colleagues proposed models of causal attribution in depression (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978) which have been extremely influential and will be outlined below. Further research involving attributions naturally followed the broad principles of these

accounts. Secondly, Seligman and colleagues developed an assessment measure which, although less than perfect, has dominated the field.

5.2 Measurement

Three approaches to the assessment and measurement of causal attributions can be seen. Theoretical principles tend to produce a limited number of dimensions on which causal attributions are believed to lie, or a number of causal loci to which events may be attributed. With these in mind, many researchers have developed specific measures to answer limited experimental hypotheses. More generally, these dimensions and loci have been generalized and converted into questionnaires designed for use in a range of situations, in particular Peterson, Semmel, von Baeyer, Abramson, Metalsky, and Seligman's (1982) Attributional Style Questionnaire. Finally, these dimensions and loci have been used in the rating of spontaneous speech and writing.

5.2.1 Specific measures employing theoretical dimensions

Both Heider (1958) and Weiner (Weiner, 1986; Weiner, et al., 1978) used a variety of specific measures to examine the causal attributions of the participants in their research. No simple set of measures resulted. A number of dimensions on which judgments of causality can be rated have been suggested.

As mentioned above, the distinction between internal and external causes has a long provenance. Heider (1958) believed that individuals could rate (using one method or another) the degree to which a particular event was believed to be caused by one's

own actions. This internality-externality dimension has continued to be influential, although other dimensions have been added. Weiner (1986) suggested three basic dimensions: loci of causality (internal vs external), stability (likelihood of recurrence), and controllability (the degree to which the individual can control the circumstances leading to the occurrence of the event).

On the basis of such theoretical dimensions researchers have investigated participants judgments of the causal determinants of events and circumstances. A literature search reveals a number of studies of the influence of causal attributions on a range of psychological phenomena that use such an approach.

5.2.2 The Attributional Style Questionnaire

The Attributional Style Questionnaire (ASQ: Peterson, et al., 1982) was developed primarily to test predictions made in the Reformulated Learned Helplessness Theory of Abramson, Seligman, and Teasdale (1978), which will be discussed at length below. Because this model uses dimensions of internality (the degree to which the events are attributed to self or external causes such as circumstances or other people), stability (the degree to which the causes are likely to be present in the future) and globality (the degree to which the causes are likely to influence a wide range of events), these formed the basis of the ASQ. Previous studies (see Reivich, 1995) had used measures in which potential causes were offered to participants in the form of choices. As is implicit in the previous section, however, these did not necessarily fall into the three dimensions considered important by Abramson, Seligman, and Teasdale. Moreover an idiographic measure, in which each participant is free to make their own choice of cause, was felt appropriate. To reduce the unreliability of a completely free-response approach, the ASQ requires participants

to generate reasons for the occurrence of twelve hypothetical events provided by the questionnaire; six positive and six negative. Participants are then required to rate the causal statements they have just generated on 7-point scales of internality, stability and globality.

A range of raw, derivative and composite scores result from the administration of the ASQ. Individual item scores of internality, stability and globality are summed across the six positive and six negative events. Reivich (1995) reviews a number of studies reporting reliability coefficients for these six subscale scores. Overall, the reliability of the ASQ subscales is reported as "unsatisfactory" (Reivich, 1995; p.27). The reported reliability coefficients (Chronbach's alpha) for the subscales of the ASQ ranged from .13 to .69. In meta-analyses (for instance, Sweeney, Anderson, & Bailey, 1986), the reliability of the internality subscale is generally generally given as being lower than the other two.

To improve the reliability (and consequent validity) of the ASQ, Peterson, Seligman and colleagues suggest the use of composite scores; summing scores for internality, stability and globality for the positive and for the negative items. Composite scores boost reliability. However, the dubious validity of collapsing subscale scores is recognized even by those who recommend it (Reivich, 1995).

There are a number of weaknesses in the use of the ASQ. The type, range and significance of the causal explanations used by an individual (even about a well-defined class of events or even a single event) are likely to be idiosyncratic and flexible. A fixed format questionnaire is unlikely to capture this subtlety. The ASQ was deliberately constructed in order to maximize the possibility of addressing this issue, as participants are asked to generate their own causes as opposed to selecting those suggested by the experimenters.

However such elegance does not mean that all subtleties are addressed. In particular only three possible dimensions are provided on which participants are

required to rate the causes generated; internality, stability and globality. Clearly, since Weiner (1986) used dimensions of internality, stability and controllability, potentially important aspects are necessarily ignored. This may be important, especially in the aspects of motivation and the self-concept. A large number of studies appear to show that external attributions for personal failure tend to lead to better future performance (see Schulman (1995) and Rettew & Reivich (1995) for reviews). However, some studies directly contradict these findings, suggesting that internal attributions for failure lead to better performance than internal attributions for success (Haisch, 1990). To my mind, the controllability dimension may be important. If you blame others for your failure, your motivation is unlikely to improve (it might even decline) and you are unlikely to attempt to correct inadequacies in your performance. Internal attributions for failure if, and only if, they are controllable, are likely to improve performance and motivation.

Finally, the internality-externality dimension of the ASQ has been criticised. I shall discuss this issue more comprehensively later.

5.2.3 Content analysis of speech

In contrast to the use of questionnaires, several groups of researchers have attempted to design systems for assessing causal attributions by analyzing the verbatim statements or written material of participants. These techniques will be discussed only briefly, as they were not employed in any of the studies described in this thesis.

Peterson, Luborsky, and Seligman (1983) reported the use of the CAVE technique - the Content Analysis of Verbatim Explanations - to analyze the explanations contained within individuals conversational speech. A good description of this technique was reported by Reivich (1995). Essentially the CAVE uses the same

theoretical taxonomy and structure of causal explanations as the ASQ - internality, stability and globality - and therefore displays the same weaknesses inherent in that measure. Reliability of the CAVE technique is probably quite high, although not all the relevant statistics have been reported. Inter-rater correlations are high (internality for negative items, .93; negative stability, .63; negative globality, .73; internality for positive items, .95; positive stability, .66; positive globality, .48; composite negative, .89 composite positive, .80 (Reivich, 1995)). Of course, the ASQ is a self-report measure, so inter-rater reliability is inappropriate. Importantly, alpha coefficients reflecting the consistency of participants' are not reported for the CAVEing technique. Reivich (1995) suggests that at least five negative items must be used to derive scores for an individual, because "consistency is less than 100%" (p.32). It is also recognised (Peterson & Seligman, 1984) that not all individuals show a consistent style, and reports of analyses of personal consistency would be interesting.

An extremely similar approach was reported by Stratton, Heard, Hanks, Munton, Brewin, and Davidson (1986). The Leeds Attributional Coding System (LACS: Stratton, Munton, Hanks, Heard, Brewin, & Davidson, 1988) was developed from the perspective of family therapy to analyze the explanations for personal or systemic dysfunction. This uses five dimensions; stable-unstable (as above), global-specific (as above), internal-external (as above), personal-universal (the degree to which the event is likely to affect the speaker specifically or a wider context), and controllable-uncontrollable (as above). This approach has been used primarily to investigate explanations in the process of family therapy (Stratton, et al., 1986), and explanations for psychological problems seen in family therapy (Stratton & Swaffer, 1988; Wright, Brownbridge, Fielding, & Stratton, 1990). It has also, like the ASQ and CAVE approaches, been advocated for wider use (Stratton, 1991).

5.2.4 Attributional complexity

Other aspects of causal attribution may be important in addition to the locus of blame. Kammer (1983) found that depressed individuals made more attributional statements than did other participants, and suggested that a focus on attributional style resulted in a lack of consideration of such factors. This view is supported by Wimer and Kelley's (1982) factor analysis of attributional ratings. They found that the dimensions apparent in these ratings included 'motivation', the degree to which finding explanations for events was considered important and 'simple-complex', the number of attributions generated for a single event. Fletcher, Danilovics, Fernandez, Peterson and Reeder (1986) explored these issues in the development of the Attributional Complexity Scale (ACS). Flett, Pliner and Blankstein (1989), Marsh and Weary (1989) and Flett and Hewitt (1990) all investigated the relationship between attributional complexity and depression. The results of these experiments suggest a complex, curvilinear, relationship between depression and attributional complexity, with depression serving to increase the motivation towards more complex attributions, except in the case of more severe depression, when motivation dropped once again.

Liu and Steele (1986) suggest that this may reflect a process which facilitates the maintenance of self-esteem. When faced with negative events a bias towards the generation of more and more complex explanations may serve to lessen the likelihood that self-blaming attributions are generated. Further, explaining events more completely may serve to assert the individuals' faith in their ability to understand their world. The role of attributional complexity in paranoia is untested, but may be important

5.3 Causal attributions in Depression and Anxiety

One particular line of research has examined the role of causal attributions in depression. Over time this train of investigation has become rather divorced from the approaches of Weiner and Heider, and has its own pedigree.

5.3.1 Helplessness and Hopelessness

5.3.1.1 Learned Helplessness

Building on animal models, Seligman (1975) suggested that learned helplessness provided a suitable model of depression. Thus, when exposed to inescapable aversive events, animals (usually dogs or rats) showed changes in apparent emotional behaviour and motivation which were believed to resemble the symptoms of human depression. Translating this to humans, Seligman (1975) suggested that depression was a form of 'learned helplessness' in which people have been led to believe that they have no ability to control or prevent the occurrence of aversive events or to bring about positive experiences.

The disparity between a behavioural model of learned helplessness and an acceptance of the cognitive elements of depression (beliefs, assumptions, or predictions) led Abramson and colleagues (1978) to develop the 'reformulated theory of learned helplessness'.

5.3.1.2 Reformulated Learned Helplessness Model

According to the reformulated learned helplessness model of depression (Abramson, Seligman, & Teasdale, 1978), individuals who attribute negative events to internal, global and stable causes are predisposed to depressive reactions following negative life events.

Such a model has broad empirical support. Although a few studies have failed to demonstrate the predicted attributional style (Hargreaves, 1985), researchers have generally found consistent differences between patients with psychiatric disorders and non-patient controls. Patients with depressed mood have been found to make excessively internal, global and stable attributions for the hypothetical negative items in the ASQ (Brewin, 1988; Robins & Hayes, 1995; Sweeney, et al., 1986). Sweeney and colleagues (1986), in particular, reported an analysis of 104 studies concluding that internal, global and stable attributions for negative events were related to depressive mood. This relationship was particularly strong for patients with clinical diagnoses of depression.

However, the precise relationship proposed in the Abramson, Seligman and Teasdale (1978) model has been doubted. In particular the causal link between internal, stable, global attributions for negative events at one point in time and later, consequent, depression has been criticised. Such an hypothesis would suggest that a pessimistic attributional style should be observed in people who have recovered from depression, leaving them vulnerable to future episodes (although logically this might only be the case for those individuals who do become depressed at a later date). Such a pattern has not always been found (Wilkinson & Blackburn, 1981). Prospective studies have also failed to find pessimistic (internal, global, stable) attributions preceding the onset of depression (Lewinsohn, Steinmetz, Larson, & Franklin, 1981; Miranda & Persons, 1988). Subsequent studies have similarly failed to identify clear

evidence that attributions play a causal role in the onset of depression (Robins & Hayes, 1995).

Brewin (1985), in a substantial and elegant review of such literature, concluded that a pessimistic attributional style for negative events is associated with the maintenance of depression, while a positive attributional style is associated with a resistance to future depression, but found no evidence that attributional abnormalities precede the onset of depression. A simple, linear, causal relationship between attributions and depression as envisaged in the reformulated learned helplessness model (Abramson, et al., 1978) is also weakened by the observation that a person's causal attributions do not seem to form a consistent trait. As Brewin (1985) observes, depressive attributional biases are not necessarily present either before or after clinical depression. Mood induction studies have also suggested that causal attributional biases can be artificially provoked. Forgas, Bower and Moylan (1990) found that attributions for real-life failure in exams were more internal and stable in a sad than in a happy mood. Peterson and colleagues (1983), analyzing transcripts from psychotherapy sessions, found that the degree of internality in a patient's attributions was extremely variable over time and predicted the patient's instantaneous mood.

5.3.1.3 Hopelessness Model

Such inconsistencies in the original model led Abramson, Metalsky and Alloy (1989) to develop the Hopelessness model of depression. In this model, there is a subtype of depression - 'hopelessness depression' - in which people feel that they cannot avoid aversive events or achieve positive events. Hopelessness is viewed as a proximal, sufficient cause for depression but attributional style is still seen as

functioning as a diathesis for depression because hopelessness is likely to occur when negative life events are attributed to global and stable causes. In this model, attributional style has a generally reduced role. Firstly causal attributions are no longer sufficient causes of depression, but are routes to hopelessness. Secondly, attributions are thought of as content-specific (i.e. specifically related to one's academic success) and flexible. Finally, the internality dimension of causal attributions is reduced in importance, contributing less to hopelessness, but having a residual role in low self-esteem.

5.3.2 Other psychological consequences of causal attributions

The range of psychological phenomena associated with attributional processes is enormous. I do not feel there is much to be gained from outlining all such strands of research, discussed in Antaki and Brewin (1982) and Hewstone (1989).

Causal attributions have been implicated in psychological explanations of behaviour (ranging from criminal violence to sales performance), social perception (from voting patterns to, as I will discuss below, paranoia) affect (in many forms) and beliefs (in depression and, as I shall also discuss below, paranoia). There seems no simple way to classify these findings - attributions are regarded as mediating cognitive processes in such a disparity of phenomena.

The philosophical background to the study of causation in psychology (White, 1990) may offer an explanation. Ideas of causation play an important role in people's lives. Events are perceived and remembered. In addition, the relationship between events is of great significance. This includes what has caused the events to occur and what, in turn, the events themselves cause. An example of the central importance of causal attributions comes from White (1988) and Poulin-Dubois and Heroux (1994),

who report that both children and adults spontaneously use "properties characteristic of animates" (Poulin-Dubois & Heroux, 1994; p.329) to describe animated films of geometric shapes.

5.4 Psychological genesis of causal attributions

Peter White (1988) comprehensively reviewed the psychological origins of causal attributions. Children seem to be aware of cause-effect relationships at around three months of age when they show surprise when collisions do not result in elastic trajectories. Links between such expectations of causal relationships and the processing of contiguity relationships in operant conditioning led White (1988) to suggest that the perception of a causal relationship was dependent on iconic processing (for instance the span of iconic memory) and therefore automatic, at least in young children.

Later, more subtle, information processing systems play a role in the development of causal attributions. White (1988) suggests that perception of generative relations, 'human intended action' and the observation of regularity and covariation are each important. Clearly these are each complex collections of processes, particularly the understanding of 'human intended action' (this issue will be referred to in Chapter 14). However, he also concludes that much more research is required concerning how different types of causal processing affect the perception of causal relationships.

From the discussion above, it is clear that causal attributional processes develop, but incorporate subtleties that potentially lead to individual differences. The possible origins of different attributional styles are important, but relatively unknown.

As in all aspects of psychology, genetic and environmental factors have both

been proposed as influences on individuals' characteristic patterns of explanations. Schulman, Keith and Seligman (1991) found that monozygotic, but not dizygotic twins showed a correlation between attributional style, indicative of a genetic origin. Gender differences are also seen in attributional style. Nolen-Hoeksema and Girgus (1995) report reliable gender differences in later adolescence and early adulthood, with women more pessimistic and self-blaming than men. They suggest that these differences are not seen in pre-pubertal children, however Burgner and Hewstone (1993) suggest that even five-year-old boys are 'self-enhancing' while five-year-old girls are 'self-derogating'. These differences are usually ascribed to environmental factors.

In children, the experience of trust may foster a positive (optimistic) attributional style whereas experience of distrust may foster a negative style (Eisner, 1995). More generally, experiences of uncontrollable unhappiness, such as parental divorce (Nolen-Hoeksema, Girgus, & Seligman, 1991) or criticism by teachers (see Eisner (1995) and Nolen-Hoeksema & Girgus (1995)) are likely to lead to pessimism or self-blame. However, despite findings such as these, a sense of complete understanding of the origins of explanatory or attributional style is absent. In my opinion this is due to the broad scope of causal attributions in our mental model of the world, and the multi-factorial nature of the explanations generated. More precise developmental models will probably require more focussed attention on specific aspects of explanations.

5.5 Causal attributions in Paranoia

In an initial study of attributional style in paranoia, Sue Kaney and Richard Bentall (1989) required paranoid patients, nondeluded psychiatric patients, and

non-patient participants to complete the Attributional Style Questionnaire (ASQ). Using the conventional scoring system of the ASQ, they found that deluded patients made excessively external, global and stable attributions for negative events. Depressed patients made excessively internal, global and stable attributions for negative events, in comparison to non-patient participants. The responses of the depressed participants are, of course, entirely consistent with Abramson and colleague's (1978) reformulated learned helplessness model. This 'externalizing attributional bias' in paranoid patients has been frequently replicated (Candido & Romney, 1990; Fear, Sharpe, & Healy, 1996; Kaney, Kinderman, & Bentall, submitted; Kinderman & Bentall, 1996; Lyon, Kaney, & Bentall, 1994) using the ASQ. In fact, one of the early investigations of attributional style in depressed patients (Raps, Peterson, Reinhard, & Seligman, 1982) used patients with a diagnosis of schizophrenia as a comparison group. This study found that such patients tended to make external attributions for negative events in comparison to their depressed peers, largely anticipating later findings.

Using a slightly different approach, Kaney and Bentall (1992) required participants to make repeated forced-choices on a computer game originally developed by Alloy and Abramson (1979). Two computer games were written in which participants were asked to make repeated forced-choices following instructions to discover the rules for making 'correct' judgements. They were informed that, from an initial position of 20 points, a point was lost following a 'wrong' choice and gained following a 'correct' choice. The games were, of course, programmed without participant control. One game was rigged as a winning game (with participants finishing with 33 points), the other as a losing game (with participants finishing with 7 points). After each game, participants were asked to estimate the percentage of trials in which they won points, and their degree of personal control over the outcomes.

The participants' estimates of the number of winning trials revealed that non-

patient participants estimated higher frequency of winning events than did the depressed patients, with deluded subjects estimating high frequency of wins on the 'win' game and a low frequency of winning on the 'lose' game. Participants in all three groups estimated that their degree of control over the games was greater in the 'win' game. Overall, the depressed patients made low estimates of control, a result that is consistent with the findings of Alloy and Abramson (1979). Non-patient participants demonstrated a self-serving bias, claiming little control over the 'lose' game, but significantly greater control in the 'win' game. This self-serving bias was significantly greater for paranoid patients.

Broadly consistent results have been found in experiments where people are asked to explain the actions of others. Richard Bentall and colleagues (Bentall, Kaney, & Dewey, 1991) used Kelley's attributional framework (Kelley, 1967) to examine how information pertaining to; consensus (the extent to which the observed action is also performed by other people), consistency (the degree to which the individual performing the action repeats it over time) and distinctiveness (the extent to which the action is distinct to, elicited by or directed towards a particular stimulus) affected attribution judgements about the actions of others towards a third party. Using a questionnaire based on one developed by McArthur (1972), participants were asked to judge the likely cause of positively valued (e.g. 'Sally said that she liked Kim') and negatively valued (e.g. 'Colin talked about Andrew behind his back') social events while distinctiveness, consistency and consensus (DCC) information was varied. High distinctiveness would be reflected in statements such as 'Colin talked about only Andrew and no-one else', high consistency in statements such as 'Colin talks about Andrew repeatedly' and high consensus in statements such as 'Everyone else talks about Andrew behind his back too'. After the presentation of each event, participants were asked to chose between three attributions: a person attribution (e.g. 'Something about Sally caused her to say that she liked Kim' or 'Something about Colin caused

him to talk about Andrew behind his back'), a stimulus attribution (e.g. 'Something about Kim made Sally say that she liked her' or 'Something about Andrew caused Colin to talk about him behind his back'), or a circumstance attribution (e.g. 'Something about the circumstances made Sally say that she liked Kim' or 'Something about the circumstancess caused Colin to talk about Andrew behind his back').

Overall, deluded patients, depressed patients and non-patient participants all utilised the DCC information in the manner expected, making fewer person attributions and more stimulus attributions in the high DCC conditions compared to the low DCC conditions. However, the deluded patients made more person attributions and fewer stimulus attributions than either of the control groups, and this effect was particularly evident for negatively valued actions. Deluded patients also tended to give higher certainty ratings than the other groups.

5.5.1 Self-serving biases in causal attribution in paranoia

A consistent pattern emerges from these findings. Whereas depressed individuals blame themselves for failure experiences, patients with paranoid delusions attribute failure to causes which are external to the self. These attributional biases can be seen as exaggerated versions of the biases which, in normal people, regulate self-esteem. This hypothesis is consistent with Colby's quasi-psychodynamic model of paranoia (Colby, 1981; Colby, Faught, & Parkinson, 1979), described in Chapter 3, and also with the suggestion that paranoia is a form of camouflaged depression (Zigler & Glick, 1988). It is not dissimilar from the psychoanalytical accounts of paranoia (Freud, 1896/1950; Freud, 1911/1950; Freud, 1914/1957; Freud, 1915/1956; Freud, 1924/1957) once the influence of homosexuality is dismissed.

5.6 The internal - external dimension of causal attributions

Both Hewstone (1989) and White (1991) critically evaluate the validity of the internal-external dimension in causal attributions. Despite its long history (dating back to Aristotle as I mentioned above), four main criticisms of this approach can be made.

Heider (1958) and others (see Hewstone (1989)) have made what Hewstone refers to as the "hydraulic assumption" (Hewstone, 1989; p.31). This means that, the more a person was seen as causing an event, the less it was due to the environment. Logically and psychologically, however, this assumption is flawed, as people do not tend to behave according to its principles (Hewstone, 1989), nor is it logically necessary (White, 1990). A second assumption is that there exist two categories, internal and external. This is again unnecessary, as there may be one category, many, or dimensional systems.

Hewstone (1989) also notes that, depending on the choice of words, internal attributions may be re-coded as external. For instance; "I want to be a Professor because I want to earn lots of money" seems an internal attribution, whereas "I want to be a Professor because Professorial salaries are highly attractive" seems external (or at least more external). As Hewstone reflects (1989; p.32) this linguistic subtlety might reflect real differences. That is, my choice of language in the example above may reflect the current state of self-representations and my beliefs about the world. Further elaboration may reveal other beliefs and assumptions concerning other elements and dimensions of causal attributions.

Finally, both Hewstone (1989) and White (1991) comment on the poor levels of validity and reliability of measures that attempt to assess the internality or externality of causal statements. This is reflected in the frequent observation that the internality dimension of the ASQ is less reliable than others (Reivich, 1995).

Hewstone (1989) and White (1991) both shed doubt on the entire concept of

internality in judgements of causal attributions. One response proposed by Hewstone (1989) is to address other aspects of attribution, such as controllability or stability. However, the results reviewed above also indicate the potential importance of this dimension. Chapter 9 of this thesis reports an empirical study that exemplifies the problems with the ASQ's internal-external dimension, and Chapter 10 describes the development of a novel measure of causal attributions.

Although a comprehensive taxonomy of causal attributions is probably untenable, one possible development is implicit in the ASQ itself. In this measure, internality judgements are made on a scale anchored from "Totally due to other people or circumstances" to "Totally due to me". Clearly, three distinct attributional loci are present: an internal focus (attributing the causes of events to self), a personal external focus (attributing the causes of events to the actions or omissions of identifiable others), and a situational external focus (attributing the causes of events in terms of circumstances or chance). Such a taxonomy is also consistent with the attributional framework employed by McArthur (1972) and used by Bentall and colleagues (1991), who believed that participants could distinguish between the roles of actors, subjects and circumstances.

It is therefore unsurprising that the internality scale has consistently been criticised for its poor reliability (Rehm, 1988; Reivich, 1995; Tennen & Herzberger, 1985).

5.7 Conclusions

Causal attributions play a central role in the development of our mental models of the world. This includes our understanding of our social surroundings. Causal attributions are also very important in the development and maintenance of our self-concept. Although this area is complex, and the measures used to assess causal attributions are crude in comparison to the subtlety of the issue, causal attributions seem central to our understanding of the cognitive processes in paranoia.

In particular paranoid patients appear to exhibit a pattern of attributions that serve to maintain a positive self-image. In combination with the abnormalities seen in the self-concept of paranoid individuals, a model of paranoid ideation can be developed.

Chapter 6.

A Detailed Model of Paranoid Ideation

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A paper presenting much of the material presented in this chapter has been published as: Bentall, R. P., Kinderman, P., & Kaney, S. (1994). The self, attributional processes and abnormal beliefs: Towards a model of persecutory delusions. *Behaviour Research and Therapy.* **32** : 331-341.

6.1 Introduction

In this chapter I will describe a model of paranoid ideation developed by myself and colleagues (Bentall, Kindeman, & Kaney, 1994) on the basis of available evidence on paranoid delusions. It was the hypotheses stemming from this model that were tested in the empirical studies that follow.

6.2 Conclusions from previous chapters

A number of conclusions can be drawn from the previous chapters. The validity of schizophrenia as a syndrome suggests that delusions be studied as phenomena in their own right. More precisely, paranoid delusions (delusions of persecution) appear to be valid and reliable psychological phenomena.

In psychological disorders generally, in schizophrenia and in paranoia in particular, the self-concept appears to play an important role. In the case of paranoid ideation, self-esteem appears largely preserved, but deeper abnormalities in the self-concept are likely. Given that there appear to be multiple domains of self-representation, Self-Discrepancy theory seems the best model for studying the self-concept in paranoia.

Causal attributions are important in psychology generally and in psychiatry. In the case of paranoia, investigations suggest the presence of defensive or self-esteem maintaining attributional processes. Moreover, further attributional investigations point to a deeper, implicit, negative self-concept.

Bentall (1993) suggested that persecutory delusions stem from exaggerated versions of the cognitive biases seen in normal individuals (Taylor, 1988). In particular, Taylor (1988) suggested that, in response to potential threats to self-esteem

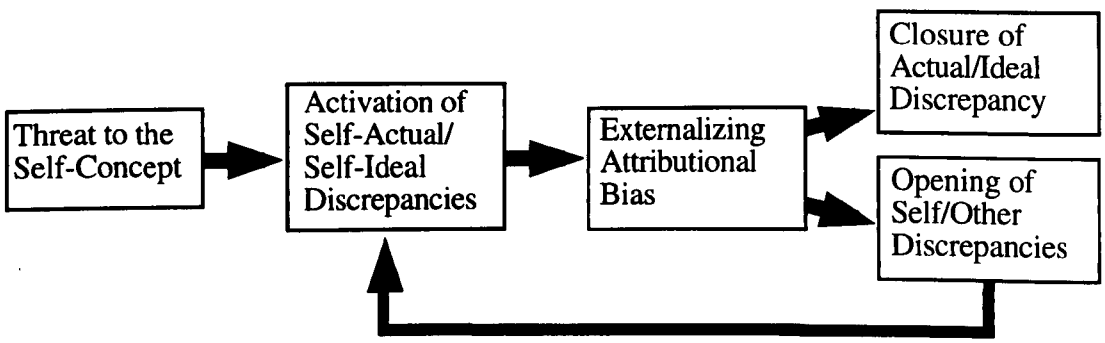
or the self-concept, negative events (failures etc.) are explained in terms of external factors. The data reviewed in Chapter 5 revealed that this externalizing bias is significantly greater for people with persecutory delusions than the general public, and contrasts with the attributional style of depressed individuals. Delusions might, therefore, be seen as products of an extreme system of self-esteem maintenance.

6.3 A detailed model of paranoid ideation

A detailed model of persecutory delusions was described by Richard Bentall, Sue Kaney and myself (Bentall, Kinderman, & Kaney, 1994). Within the framework of self-discrepancy theory, persecutory delusions are characterized as stemming from a process which reduces the gulf between self-perceptions and self-ideals to a minimum. More precisely, when possible self-ideal discrepancies are activated by negative life-events or stimuli, patients with delusions of persecution utilize externalizing causal attributions. In this model the self-actual:self-ideal discrepancies are assumed to lie latent until activated by stimuli or events.

Such external attributions are believed to minimise perceived differences between the actual self and the ideal-self, maintaining the self-concept. However, they also may have the consequence of opening discrepancies between the self and the perceived views of others about the self. If events are explained as stemming from the actions of other people, they are likely to be seen in a negative light. Moreover, if individuals believe that other people are behaving towards them in a negative manner, it seems likely that they will believe that such people also see them in a negative light. See figure 6.1 for a graphical representation of the model.

Figure 6.1 A diagrammatic representation of the model of paranoid ideation



This process is thought of as dynamic and responding to moment by moment changes of circumstances (Bentall, et al., 1994). It is also conceived of as an iterative process. That is, on the first occasion, an external attribution for a negative event will have three consequences. First, the consciously accessible self-representations (and consequent self-discrepancies) will be supported and maintained. This will mean that attributions for future negative events will be less likely. Second, the representations of other people's view of the self will be negative, leading to more marked self-actual:other-actual discrepancies. This will mean that other-blaming attributions for future negative events will be more likely. Finally, the involvement of such affect-laden processes in conjunction with the self-concept suggests that attention to potential threats to the self-concept will be heightened.

This model seems consistent with previous literature on self-esteem in schizophrenia and with the attributional style abnormalities observed in deluded patients. It also corresponds well with previous psychological models of paranoia, particularly that of Colby (Colby, Faught, & Parkinson, 1979). The links with self-discrepancy theory are also complementary. In particular, the comment by Higgins (1987) that people with high self-actual:self-ideal discrepancies are more likely to make internal attributions for negative or ambiguous events (because their most available explanatory representations are internal - "I failed because I'm not as good as I would like to be") is entirely consistent with this model.

Finally, this model is consistent with Fenigstein's model of self-consciousness in paranoia (Fenigstein & Vanable, 1992). Clearly, this model subsumes a self-consciousness model in its early stages. An individual who responds defensively with external attributions to easily perceived threats to the self-concept is, almost by definition, self-conscious.

6.4 Hypotheses stemming from this model

In each of the chapters that follow, concentrating on the empirical studies, detailed hypotheses which follow from the model of paranoid ideation will be presented and tested. In general terms, these hypotheses concern four areas.

6.4.1 Attentional biases for self-referent information

Two assumptions concerning possible attentional biases for self-referent information are explicit in our model of paranoid ideation.

First, it is assumed that paranoia is about the self-concept. Although this assumption has been made by many others (see Chapter 3), this assumption can be tested. This assumption would suggest that material related to the self-concept should be highly salient for people with paranoid delusions. As a consequence, it is predicted that people with delusions of persecution will show a bias towards attending to material relevant to the self-concept.

6.4.2 Self-discrepancies

The model of paranoid ideation outlined above predicts that specific patterns of self-discrepancies will be found in paranoid individuals. Self-actual:self-ideal (and self-actual:self-ought) discrepancies are hypothesized to be small - high consistency being predicted between these domains. At the same time self-actual:other-actual (and self-actual:other-ideal and self-actual:other-ought) discrepancies are predicted to be large in comparison with non-patient subjects.

6.4.3 Relationship between causal attributions and self-discrepancies

This model, although focussing on attributional processes in paranoia, makes wider assumptions. Specifically, it suggests a clear link between the making of particular causal attributions for negative events and consequent changes in self-discrepancies. That is, external attributions for negative events should lead to a closure of self-actual:self-ideal discrepancies, but an opening of self-actual:other-actual discrepancies when compared to internal attributions for such events.

6.4.4 Causes of attributional abnormalities

Finally, this model is built on the observations of certain attributional abnormalities in paranoia. These abnormalities are well-established (see Chapter 5). Nevertheless, if these abnormalities exist, they must have a cause. There must be some aetiological pathway for the genesis of these abnormalities.

6.5 Implications

The implications of this model, if substantiated, are two-fold. Our understanding of paranoid ideation and delusions of persecution (and hence, in part, psychosis) will be furthered.

In addition, and more generally, the investigation of such a model may help us develop our understanding of the nature of the links between self-representations, causal attributions, social and personal beliefs and emotions.

6.6 Conclusions

The second half of this thesis, the empirical studies, are tests of these assumptions. As such they act as empirical tests of the model.

Chapter 7.

Attentional Bias

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A paper based on the material presented in this chapter has been published as:

Kinderman, P. (1994). Attentional bias, persecutory delusions and the self concept. *British Journal of Medical Psychology*. **67** : 53-66.

7.1 Background

This chapter describes an empirical test of the hypothesis that people with persecutory delusions have a disturbed self-concept and consequently will show abnormalities in the processing of information related to the self. The abnormal attributional style of people experiencing delusions of persecutions has been described in detail above, as has the detailed model of paranoid ideation. The studies of attributional processes by Kinderman, Kaney, Morley and Bentall (1992) and Lyon, Kaney and Bentall (1994) both offer support for the hypothesis that the cognitive abnormalities seen in people with persecutory delusions reflect exaggerated forms of the self-serving biases seen in normal participants (Taylor, 1983; Taylor, 1988) and that these biases are absent when deluded patients are asked to make implicit rather than explicit judgements of causality.

The model of the self-protective nature of the attributional processes described above also implies that abnormalities in self-referent information-processing in paranoia would be most evident when automatic or non-effortful cognitive processes are assessed. Overt methods of accessing the self-concept of people with persecutory delusions, on the other hand, are more likely to be influenced by the self-serving or self-presentational biases.

7.1.1 Attention for self-referent material

Depressed patients, as well as exhibiting specific attributional biases (Sweeney, Anderson, & Bailey, 1986), have been shown to demonstrate increased attention to materials relevant to their concerns (for a general review see Brewin, 1988). Such biases of attention have most often been investigated using the Stroop test

(Stroop, 1935). On this test participants are required to name the colours of ink in which words of varying emotional salience are written. Attentional biases towards particular words or classes of words can be inferred from the degree of interference with performance as measured by speed of colour-naming. Williams and Broadbent (1986) showed that the performance of people who had attempted suicide was significantly slower for words related to the theme of overdosing, while Gotlib and McCann (1984) demonstrated interference for negatively-toned words only in the case of participants with high scores on a measure of depressed mood. The Stroop technique has been used with respect to persecutory delusions (Bentall & Kaney, 1989), where an abnormal attentional bias was show towards words of a threatening nature.

7.1.2 Hypotheses

The present study employed the Stroop task in a direct test of the hypothesis that, for people suffering from persecutory delusions, there exist abnormalities in the processing of information relevant to the self-concept similar to those present in people with depressed mood. It was further hypothesised that such abnormalities, when assessed using the Stroop task (which accesses automatic cognitive processes), would be inconsistent with the overt self-ratings of people with persecutory delusions. Specifically the experiment tested the hypothesis that people with persecutory delusions exhibit an attentional bias towards words related to low self-esteem while concurrently exhibiting a self-serving presentational bias.

7.2 Method

7.2.1 Participants

The participants in the experimental group (Group P) were 16 people, currently receiving treatment for psychiatric conditions, and suffering from persecutory delusions. 12 were men and 4 women. 13 met the DSM-III-R (American Psychiatric Association, 1987) criteria for schizophrenia and 3 the criteria for delusional disorder. All diagnostic classifications were made via a combination of interviews and examination of the case notes. In each case, mention was made in case-notes of the presence of delusional beliefs, and staff confirmed that the patient was currently experiencing delusions. The interviews carried out by the experimenter with the patients employed a series of questions selected from the Present State Examination (PSE 9th Edition; Wing, Cooper, & Sartorius, 1974) to ensure that the participants were currently experiencing persecutory delusions according to the PSE glossary, which also accords with the DSM-III(R) definition of delusion (see Appendix 3). Examples of the delusions experienced by people in this group included the belief that ward staff were part of conspiracy involving the Special Branch to poison the subject on the grounds of the special information he possessed referring to the Royal Family, the belief that the subject's parents had falsely informed the police that he was dangerous in an attempt to deprive him of his independence, and the belief that ward staff were part of conspiracy with the subject's parents to falsely accuse her of arson and ultimately to murder her. The presence or absence of auditory hallucinations was recorded. Nine patients were currently experiencing auditory hallucinations (in each case including content of a negative nature) while seven were not.

Six people in this group were currently in-patients and ten were out-patients. All were currently receiving neuroleptic medication. The mean age of this group was

34.3 years, SD = 12.5 years. In common with most other investigations in this area (eg. Kinderman, et al., 1992) a significant level of depressed mood was observed in this group. The mean BDI score (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) was 17.1, SD = 10.3. The mean elapsed time since the first psychiatric consultation mentioned in the case-notes was 7.35 years; SD = 8.2 years, range from 2 weeks to 25 years.

A psychiatric comparison group (Group D) was recruited from the same hospitals and clinics as the first group. The 16 patients were selected to match the experimental group in terms of age and gender. All patients in this group suffered a clinically significant level of depressed mood, and all had received a currently applicable diagnosis of depression. No participant experienced psychotic symptoms. This group consisted of 11 men and 5 women, with a mean age of 33.9 years, SD = 9.2 years. Eight people in this group were currently in patients and eight were out-patients. All were currently receiving anti-depressant medication. The mean BDI score was 27.3, SD = 9.3. The mean elapsed time since the first psychiatric consultation mentioned in the case-notes was 0.62 years; SD = 0.82 years, range from 2 months to 3 years, 2 months. The two patient groups clearly differed in terms of the length of their psychiatric difficulties, a one-way analysis of variance revealed that this difference reached statistical significance, $F(1,30) = 11.1$, $p < .005$.

A non-patient control group (Group C) was recruited from informal sources. Again participants were selected to match the previous groups in terms of age and gender. In order to control for levels of depressed mood, participants were only included in this group if their BDI scores were less than a cut-off of 10. This resulted in a group of 11 men and 5 women whose mean age was 31.3 years, SD = 11.0 years, with a mean BDI score of 5.3, SD = 2.1. None of the participants, when questioned after testing, indicated that they understood the hypothesis under examination.

Analyses of the ages and genders of the members of the three groups revealed that the groups were well matched. A one-way analysis of variance carried out on the ages of the group members revealed no significant differences between the groups, $F(2,45) = 0.3$, $p = .715$. The proportions of men and women in the three groups was not significantly different, $chi^2 = 0.2$, $p = .665$).

As expected the three groups differed in their levels of depressed mood as indicated by a one-way analysis of variance of BDI scores, $F(2,45) = 29.7$, $p < .001$. Planned pairwise comparisons (Tukey's HSD) revealed that both Groups D and P, $p < .01$ and $p < .01$ respectively, differed from the control group and from one another, $p < .01$, with Group D more depressed than Group P.

7.2.2 Materials

7.2.2.1 Beck Depression Inventory

The Beck Depression Inventory (BDI; Beck, et al., 1961), which was used to measure depressive symptomatology in the groups, is a 21-item scale which has been widely used in previous studies (see Appendix 4). In previously published research this scale has been shown to have good internal reliability and to be valid for use with both clinical (Williams, Barlow, & Agras, 1972) and non-patient (Blumberry, Oliver, & McClure, 1978) groups.

7.2.2.2 Stroop test

The method of administration of the Stroop test broadly resembled that of Bentall and Kaney (1989), which in turn was derived from that of Williams and

Broadbent (1986). Four separate cards were constructed from white A4 size card. On these were written ten rows of five words, in five colours of ink - red, blue, orange, brown and green. The first card consisted of strings of O's, each four, four, seven, eight and nine characters long. The second card consisted of five personally descriptive adjectives of positive content (CALM, WISE, CAPABLE, POSITIVE, and REALISTIC). The third card consisted of five personally descriptive adjectives of negative content (LAZY, WEAK, FOOLISH, CHILDISH and OBNOXIOUS)(see Appendix 5). The positive or negative nature of these words was assured by reference to Anderson's (1968) published list of value-judgements of 555 personal descriptors. As Anderson's study revealed a clear bipolar distribution of value-judgements of personal descriptors, with very few words being assessed as neutral, it was decided that the fourth card, consisting of neutral words, should employ words which could not normally be used as personal attributes outside the lyric arts (PALE, RIPE, RESIDENT, DOMESTIC and HYDRAULIC). The words were matched, across each of the three conditions, for character length and frequency of occurrence in the English language according to Thorndike and Lorge (1944). In each case the words were written in a quasi-random order such that all five words from each set appeared on each of ten lines and each of the five colours was used on each line.

7.2.2.3 Personal Profile Questionnaire

Participants also completed a questionnaire (the Personal Profile Questionnaire, PPQ) in which a total of sixty personally descriptive adjectives were presented (see Appendix 6). These included the target words for the Stroop task imbedded within the measure. Participants were required to rate each word according to the degree to which it described them on a five point Likert scale (from 'Describes

me very well' to 'Does not describe me at all'.) Thirty of the words were positive (fell into the upper half of Anderson's list) and thirty negative.

7.2.3 Procedure

After consent had been given the participants were asked to complete the BDI and PPQ. The participants' responses to the PPQ were scored by allocating numeric values to their choices, with 'Describes me very well' scoring five and 'Does not describe me at all' scoring one. Scores were derived separately for positive target words (those five positive adjectives subsequently appearing on the Stroop cards), negative target words, all positive words and all negative words.

Once these had been completed the participants were asked to name the colours to be employed in the Stroop task, presented on a separate sheet. This ensured correct discrimination and naming of the colours. Participants were then presented with each card and asked to name the colours as quickly as possible with out making mistakes. The cards were presented in random order and the time taken to colour-name each card recorded with a stop-watch.

7.3 Results

7.3.1 Stroop results

Table 7.1 shows the mean times for completion of the Stroop task. As can be seen both psychiatric groups took longer to complete the colour-naming than the normal control group under each condition. A two-way analysis of variance was

conducted with one between participants factor (the groups) and one within-subject factor (the various Stroop materials). Significant main effects were found for both content of Stroop material, $F(3,135) = 46.6$, $p < .001$, and group, $F(2,45) = 14.1$, $p < .001$. The interaction term was significant, $F(6,135) = 6.0$, $p < .001$. Tests of simple effect revealed that times for completion of the task differed across conditions for groups P, $p < .001$, and D, $p < .001$, but not for group C, $p = .094$. Planned pairwise comparisons (Tukey's T) revealed significant differences between groups P and D, $p < .01$, between groups P and C, $p < .01$ and between groups C and D, $p < .05$. Thus, both psychiatric groups took longer than the normal control group to complete the colour-naming of positive, negative and neutral words, with the group of people experiencing persecutory delusions taking longer than the depressed group.

In order further to investigate the effect of the nature of the stimulus on performance an 'interference index' was calculated for both positive and negative words by subtracting the time to complete the neutral words from the times taken to complete the affectively-laden words for each subject. These data are shown in Figure 7.1, and presented in Table 7.2.

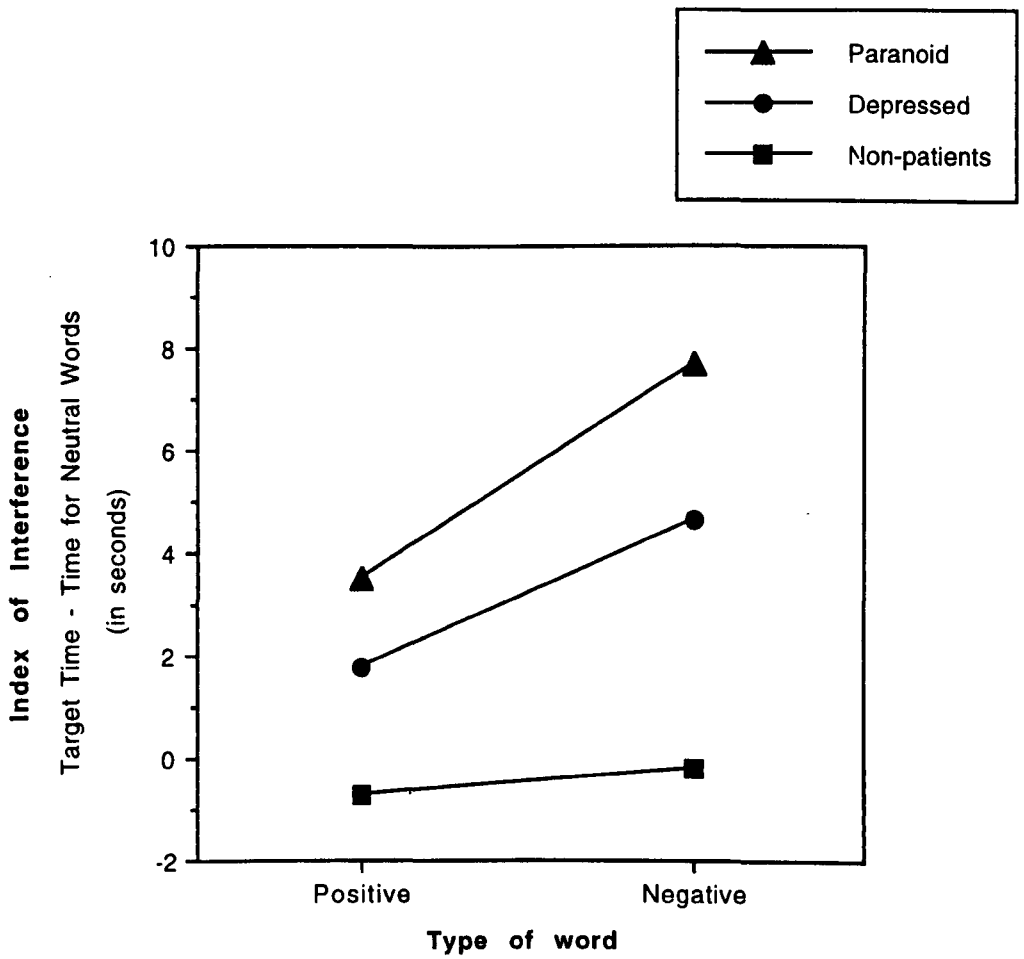
Table 7.1 Mean times in seconds (with standard deviations) taken to complete each Stroop card.

Group	O's	Neutral	Positive	Negative
Non-patients	34.38 (8.59)	37.88 (6.51)	37.13 (6.47)	37.69 (6.62)
Depressed	37.75 (9.15)	45.56 (11.78)	47.31 (9.15)	50.19 (11.68)
Paranoid	48.31 (13.30)	56.31 (14.61)	59.81 (13.73)	64.00 (14.56)

Table 7.2 Mean indices of interference (with standard deviations) of positive and negative words on the Stroop task. Calculated by subtracting time to complete neutral words from the time taken to complete positive words and negative words.

Group	Positive Interference	Negative Interference
Non-patients	-0.75 (6.24)	-0.19 (8.50)
Depressed	1.75 (3.28)	4.63 (5.25)
Paranoid	3.50 (5.14)	7.69 (8.50)

Figure 7.1. Indices of interference for the colour-naming of the emotionally-laden words for the three groups.



Inspection of Figure 7.1 suggests that the two patient groups demonstrated an interference with colour-naming for both positive and negative words, with Group P (the group suffering persecutory delusions) demonstrating the greater level of interference. For the normal control group, the absolute values of the indices of interference suggest that the nature of the words has no specific effect on attention.

A two-way analysis of variance was conducted on the indices of interference with one between-subjects factor (group) and one within-subject factor (positive or negative interference). The main effects of group membership, $F(2,45) = 8.9$, $p < .001$, and type of interference, $F(1,45) = 10.1$, $p < .05$, were significant. The interaction term was non-significant, $F(2,45) = 1.9$, $p = .167$. Tests of simple effect showed differences between the indices for groups P, $p < .005$, and D $p < .05$, but not group C, $p = .696$.

Planned pairwise comparisons (Tukey's HSD) showed that, for the relative interference by positive words, Group P was significantly different from Group C, $p < .05$, but not from Group D, $p > .05$. The difference between Groups C and D was not significant, $p > .05$. For the relative interference by negative words, planned pairwise comparisons (Tukey's HSD) revealed that Groups P, $p < .005$, and D, $p < .05$, both differed significantly from Group C but not from one another, $p > .05$.

Thus the two patient groups demonstrated an interference with the colour-naming of negative words when compared to neutral words, with those experiencing persecutory delusions demonstrating the greater level of interference. The group of people experiencing persecutory delusions, but not the depressed group, also showed an interference with the colour-naming of positive words. For the normal control group the nature of the words had no such specific effect on attention.

This pattern of interference was reflected on an individual as well as a group basis. Thus, in the deluded group 15 of the 16 individuals were 1 or more seconds slower to colour name negative words than neutral words, and 12 of the 16 were 1 or

more seconds slower to name positive words than neutral. For depressed participants 13 of the 16 individuals were 1 or more seconds slower to colour-name negative words than neutral words, and 10 of the 16 were 1 or more seconds slower to name positive words than neutral. For the normal control group, 6 of the 16 individuals were 1 or more seconds slower to colour-name negative words than neutral words, and 5 of the 16 were 1 or more seconds slower to name positive words than neutral.

7.3.2 Endorsement of personal adjectives

The levels of endorsement of the personal adjectives for the three groups are presented in Figure 7.2a for those words subsequently used in the Stroop test, and Figure 7.2b for all words (both target and non-target words) and in Table 7.3. As can be seen, the relative rates of endorsement of the target and non-target words are similar, and differences can be observed between the experimental groups.

Figure 7.2a. Levels of endorsement of positive and negative target words (those used subsequently in the Stroop test) for the three groups.

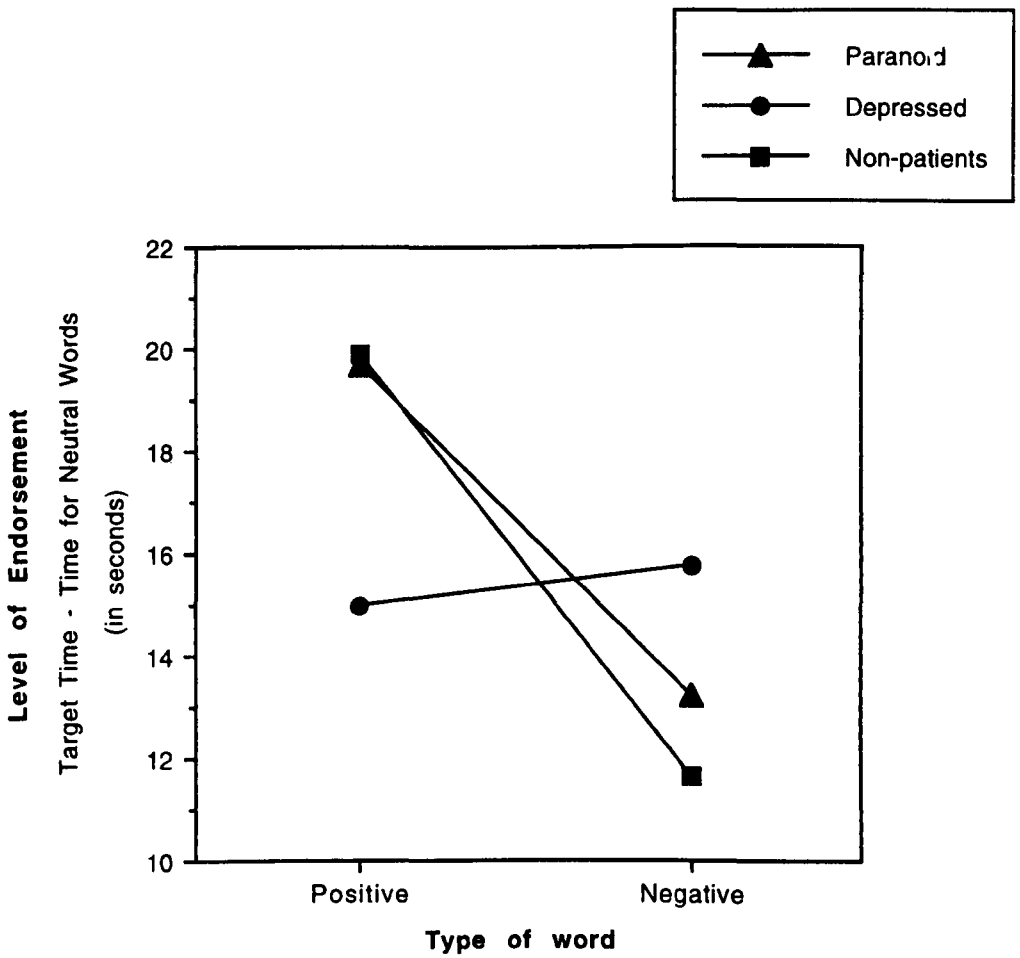


Figure 7.2b. Levels of endorsement of all positive and negative words for the three groups.

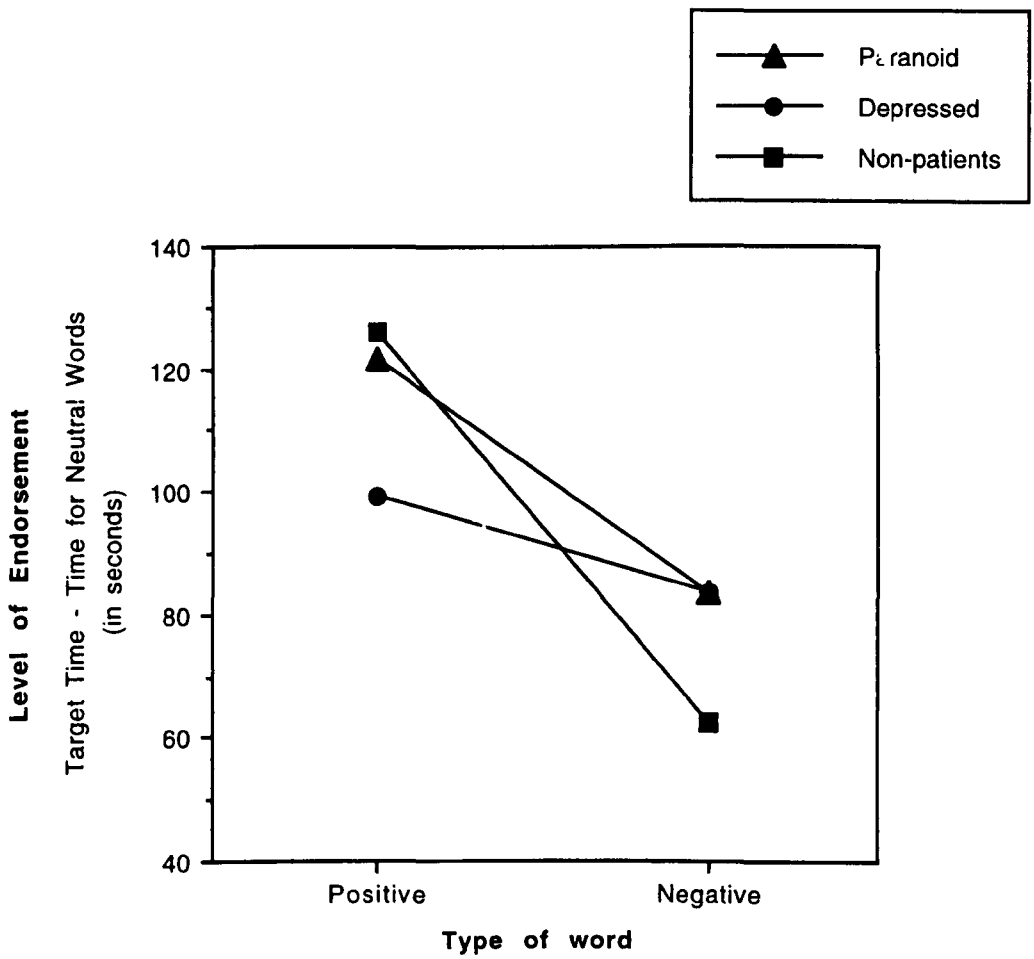


Table 7.3 Mean levels of endorsement as self-descriptive (with standard deviations) of positive and negative target words (words subsequently used on the Stroop task) and all positive and negative adjectives.

Target Adjectives

Group	Positive	Negative
Non-patients	19.88 (2.47)	11.63 (3.65)
Depressed	14.94 (4.03)	15.75 (3.80)
Paranoid	19.69 (3.84)	13.19 (5.00)

Total Adjectives

Group	Positive	Negative
Non-patients	106.00 (8.16)	50.81 (14.67)
Depressed	84.31 (17.02)	67.88 (15.95)
Paranoid	101.88 (13.03)	70.44 (18.19)

Two-way analyses of variance were conducted on the data for target and all words separately with experimental group as a between-subjects factor and type of word (positive or negative) as a within-subject factor. For the target words a significant effect was observed between the type of word, $F(1,45) = 24.4$, $p < .001$, but not between groups, $F(2,45) = 0.9$, $p = .412$. An interaction between group and type was significant, $F(2,45) = 10.6$, $p < .001$. Tests of simple effect revealed that, for both positive, $p < .001$, and negative, $p < .01$, words, the groups differed significantly. In addition tests of simple effect revealed that the relative rates of endorsement of positive and negative target words differed significantly for groups C, $p < .001$, and P, $p < .001$, but not group D, $p = .409$. Thus both the group of people with persecutory delusions and the normal control group endorsed more positive words than negative words as self-descriptive, but this pattern was not shared by the group of depressed people.

Planned pair-wise comparisons (Tukey's HSD) revealed that, for the positive target words, the rates of endorsement of groups P and C did not differ, $p > .05$, but the rate of endorsement of group D differed significantly from both groups P, $p < .01$, and C, $p < .01$. For the negative target words, the rates of endorsement of groups P and C did not differ significantly, $p > .05$, nor did the rates of endorsement of groups P and D, $p > .05$, but the rate of endorsement of group D differed significantly from group C, $p < .05$. The patients with persecutory delusions demonstrated a relatively low rate of endorsement of low self-esteem adjectives when compared to the depressed patients and a much higher rate of endorsement for positive adjectives (about equivalent to that of the normal participants but much higher than that of the depressives).

For the total group of words a significant effect was observed between the type of word, $F(1,45) = 77.1$, $p < .001$, and between groups, $F(2,45) = 37.4$, $p < .005$. The interaction between group and type was significant, $F(2,45) = 9.3$, $p < .001$.

Tests of simple effect revealed that for both the positive words, $p < .001$, and negative words, $p < .01$, there were significant differences between the groups. In addition tests of simple effect revealed that the relative rates of endorsement of positive and negative words in total differed significantly for groups C, $p < .001$, P, $p < .001$, and D, $p < .05$.

Planned pairwise comparisons (Tukey's HSD) revealed that, for the positive words, the rates of endorsement of groups P and C did not differ, $p > .05$, but the rate of endorsement of group D differed significantly from both groups P, $p < .01$, and C, $p < .01$. For the negative words, the rates of endorsement of groups P and D did not differ, $p > .05$, but the rate of endorsement of group C differed significantly from those of both groups P, $p < .05$, and D, $p < .05$. For the total group of words therefore, the patients with persecutory delusions demonstrated a relatively high rate of endorsement of low self-esteem adjectives (about the same as the depressed controls) but also a much higher rate of endorsement for positive adjectives (about equivalent to that of the normal participants but much higher than that of the depressives).

In order to examine the possible confounding effect of hallucinations, results were calculated separately for the nine hallucinating and seven non-hallucinating patients in group P. The levels of endorsement of target and non-target adjectives and indices of interference on the Stroop test are presented for these sub-groups in Table 7.4.

Table 7.4 Mean indices of interference (with standard deviations) with completion of positive and negative Stroop cards, and mean levels of endorsement of positive and negative target and non-target words. Presented for hallucinating and non-hallucinating deluded subjects separately, and including ANOVA results.

	Hallucinating	Non-hallucinating	F(1,14)	p
Indices of Interference				
Positive	3.55 (6.19)	3.14 (3.43)	0.002	0.963
Negative	6.44 (8.43)	9.86 (7.38)	0.717	0.411
Levels of Endorsement				
Positive Target	20.77 (3.96)	18.29 (3.45)	1.738	0.159
Negative Target	11.55 (4.00)	15.29 (5.27)	2.385	0.125
Positive Non-target	100.67 (11.82)	103.43 (15.27)	0.385	0.689
Negative Non-target	68.33 (18.91)	73.14 (18.32)	0.262	0.617

As can clearly be seen, the presence or absence of hallucinations has no effect on the relevant data, with hallucinating and non-hallucinating patients scoring similarly on all measures. One-way analyses of variance revealed that on no variable did these two sub groups differ. The results of these analyses are summarised in Table 7.4.

7.4 Discussion

This study demonstrated that, for people suffering from persecutory delusions, there exists a specific attentional bias for information of relevance to the self-concept. Specifically, people suffering from persecutory delusions showed a pattern of interference with colour-naming of personally descriptive words of both positive and negative content similar to that of people with depressed mood. At the same time the pattern of endorsement of these and other positive and negative words as being self-descriptive was significantly different between these two groups.

The time taken by participants to name the colours differed between groups, with the patient groups taking significantly longer than controls. This is in keeping with previous studies (eg. Bentall & Kaney, 1989) and may be interpreted as a general effect of psychiatric disturbance. The specific effect of content on processing may be seen in the indices of interference. Here both negative and positive words interfered with colour-naming for both patient groups (ie. both depressed and deluded) but not for the normal control group. This indicates that, for people with persecutory delusions, at least as much as for people with depressed mood, information related to the self-concept is highly salient. More specifically, for both the patient groups, negative (low self-esteem) words resulted in greater interference with colour-naming than did positive words. This is support for the hypothesis that information relating to potential threats to self-esteem is salient to both these groups

A significant number of the deluded participants were also experiencing auditory hallucinations, these were usually of a negative nature. None of the depressed participants experienced psychotic symptoms. This fact seems to provide a possible alternative explanation for the findings, in that the presence of negative self-referent auditory hallucinations could possibly be associated with increased salience of negative self-referent words. However, inspection of the data clearly revealed that the sub-group of deluded patients in the present study who did not experience hallucinations did not show different patterns of colour-naming interference or levels of endorsement of the various categories of words when compared to the hallucinating patients. The presence of hallucinations cannot, therefore, provide an adequate explanation of the findings.

The groups also differed significantly in important demographic respects. The length of diagnosed psychiatric problems was significantly greater in the case of the deluded group than for the depressed group. This is potentially important in that cognitive or intellectual functioning is implicated in the Stroop task, and such functioning can be assumed to be affected by both an extended period of psychiatric illness and psychiatric treatment. Such differences in psycho social functioning could be interpreted as possible explanations (in addition to the general effect of psychiatric disturbance) for the difference in raw response times to the Stroop tasks as found in this study. However, as mentioned above, the interesting conclusions are drawn from the calculation of specific indices of interference for particular classes of words. Possible generalised deficits in cognitive functioning cannot alone explain such specific differences within an individual's performance.

In the case of depressed mood it is widely accepted that there exists a 'negative cognitive schema' (Beck, Rush, Shaw, & Emery, 1979). This includes negative beliefs about ones-self (Brewin, 1988; Kuiper, Olinger, & Martin, 1988). The salience of negative descriptive words related to the self-concept for depressed people

has been widely demonstrated (Gotlib & McCann, 1984; Teasdale & Dent, 1987). The present study, in which the salience of negative self-descriptive words has been demonstrated for people experiencing persecutory delusions, provides support for the hypothesis that processing of information related to the self-concept is also important in paranoia.

Given that depressed mood is common to people with persecutory delusions, it might be expected that processing of information associated with depression would be expected to be similar in the two patient groups. What is important, however, is that the rates of endorsement of the adjectives as being self-descriptive differed between the two patient groups. For both the set of words subsequently used in the Stroop test and for the other words, the patterns of endorsement were different for depressed people and those suffering from persecutory delusions. Whereas the people with depressed mood endorsed relatively few of the positive words subsequently used in the Stroop test as self descriptive, the deluded patients endorsed as many of these positive words as did the normal control group. For the negatively-toned words subsequently used in the Stroop test, the group of people suffering from persecutory delusions endorsed fewer words as self-descriptive than did the depressed group, although both the patient groups endorsed relatively more than did the normal control group. For the total set of words the patterns of endorsement were similar, and again the patterns of endorsement of the two patient groups were significantly different.

To summarize these results, the depressed patients endorsed as self-descriptive those low self-esteem adjectives shown to be salient for them. The deluded patients, however, rated these words as being significantly less self-descriptive despite the fact that they were demonstrated to be highly salient. This discrepancy between interference and endorsement can be seen as being consistent with the hypothesis that defensive attributional processes play a role in persecutory delusions.

Chapter 8.

Self-Discrepancies and Paranoid Delusions

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A paper based on the material presented in this chapter has been published as: Kinderman, P., & Bentall, R. P. (1996). Self-discrepancies and persecutory delusions: Evidence for a defensive model of paranoid ideation. *Journal of Abnormal Psychology*. **106** : 106-114.

8.1 Background

The most central hypothesis stemming from the model of paranoid ideation outlined in Chapter 6 concerns the self-discrepancies of paranoid individuals: that the deluded individual's attribution of negative events to external factors serves to maintain consistency between self-perceptions and self-ideals at the expense of contributing to negative perceptions of the intentions of others. This chapter comprises an empirical test of this hypothesis.

8.1.1 Hypotheses

The self-discrepancies of paranoid, depressed and non-patient participants were assessed. It was predicted that paranoid delusions would be associated with positive self-ratings in the actual-self domain, and a high degree of consistency between the actual-self concept and both the ideal-self and ought-self concepts. At the same time, because it was hypothesised that paranoid attributional biases lead to the belief that other people hold negative views about the self, it was predicted that persecutory delusions would be associated with large discrepancies between self-perceptions and deluded patients' beliefs about how they are perceived by others.

8.2 Method

8.2.1 Participants

None of the participants in the present study had participated in the study reported in Chapter 7. The participants in the experimental group (Group P) were 22

people, currently receiving treatment for psychiatric conditions, and suffering from persecutory delusions. Eighteen were men and 4 women. Twenty patients met the DSM-III-R (American Psychiatric Association, 1987) criteria for schizophrenia and 2 for delusional disorder. In each case, mention was made in case-notes of the presence of delusional beliefs, and staff confirmed that the patient was currently experiencing delusions. Interviews were conducted by the first author to ensure that patients were currently experiencing persecutory delusions. Each interview employed questions from the delusions and hallucinations section of the 9th edition of the Present State Examination (PSE; Wing, Cooper, & Sartorius, 1974), which provides operational criteria for twelve different commonly-reported delusional systems (p.170), which accord with the DSM III-R definition of delusions (pp.395-396). The presence or absence of auditory hallucinations was also recorded. Six patients were currently experiencing auditory hallucinations (in 3 cases these included at least some content of a negative nature, the other 3 cases reported neutral or positive content) while 16 were not.

Fourteen people in this group were currently in-patients and 8 were out-patients. All were currently receiving neuroleptic medication. The mean age of this group was 33.68 years; SD = 10.29 years, range 19 to 51 years. In common with most other investigations in this area (eg. Candido & Romney, 1990; Kaney & Bentall, 1989; Kaney & Bentall, 1992; Kaney, Wolfenden, Dewey, & Bentall, 1992; Kinderman, 1994) a significant level of depressed mood was observed in this group. The mean Beck Depression Inventory score (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) of this group was 17.09; SD = 17.09, range 0 to 61. The mean elapsed time since the first psychiatric consultation mentioned in the case-notes was 5.71 years; SD = 6.91 years, range from 2 days to 21 years.

A psychiatric comparison group (Group D) was recruited from the same hospitals and clinics as the first group. The 22 patients were selected to match

approximately the experimental group in terms of age and gender. All patients in this group suffered a clinically significant level of depressed mood, and all met the DSM-III-R criteria for Major Depressive Episode. No subject reported experiencing psychotic symptoms and case notes were examined to ensure no differential diagnoses involving psychosis had been suggested. This group consisted of 15 men and 7 women, with a mean age of 32.68 years; SD = 8.64 years, range 20 to 57. Nine people in this group were currently in-patients, 13 were out-patients. All were currently receiving antidepressant medication. The mean BDI score of the group was 28.68; SD = 10.60, range 8 to 45. The mean elapsed time since the first psychiatric consultation mentioned in the case-notes was 4.22 years; SD = 7.70 years, range from 4 weeks to 13 years.

A non-patient control group (Group C) was recruited via informal contacts. Again participants were selected to match the previous groups in terms of age and gender. In order to control for levels of depressed mood, participants were only included in this group if their BDI scores were less than a cut-off of 10 (Rehm, 1988). This resulted in a group of 18 men and 4 women whose mean age was 28.64 years; SD = 6.50 years, range 18 to 44 years, with a mean BDI score of 4.22; SD = 3.75, range 0 to 9. None of the participants, when debriefed after the study, indicated that they had prior understanding of the hypotheses under test.

8.2.2 Measures

8.2.2.1 Beck Depression Inventory

The Beck Depression Inventory (BDI; Beck, et al., 1961), which was used to measure depressive symptomatology in the groups, was described in Chapter 7.

8.2.2.2 Personal Qualities Questionnaire

The Personal Qualities Questionnaire (PQQ), which was used to measure self-discrepancies in the groups, was modified from the Selves Questionnaire (Higgins, Bond, Klein, & Strauman, 1986; see Appendix 7). The Selves Questionnaire requires the subject verbally to generate up to ten attributes they would use to describe the person they actually are (self-actual), would ideally like to be (self-ideal) and feel they should be or ought to be (self-ought). In addition, participants are asked to generate the attributes they believe other people would use to describe them in each of these domains. This questionnaire and its scoring system were modified for the current study. In its original form, the Selves Questionnaire requires participants to make numerical ratings of the degree to which each word describes them. For simplicity participants were not required to make such ratings in the current study. In addition, changes were made to the directions given to participants concerning the 'other' perspective. In pilot investigations with psychotic patients, a variety of directions were given. Patients frequently reported having no friends nor people to whom they were close. Directions to consider the believed opinions of 'other people' in general tended to result in the subject asking which specific person's opinions were required, as different people have different views. As this would have made comparisons impossible (since each subject would be making a different judgement) it was decided to select an 'other' which was common to all participants. Participants were therefore required to generate words they believed reflected the perceptions of their parents. Thus, nine self-concepts were elicited from participants (self-actual, mother-actual, father-actual, self-ideal, mother-ideal, father ideal, self-ought, mother-ought and father-ought). However, although all those included in the study generated words in the self-actual, self-ideal and parent-actual domains, some participants did not generate words for the remaining domains (see Table 1 for the number of participants in each

group supplying adjectives in each domain).

The scoring system for the original questionnaire (Higgins, et al., 1986) was also simplified. Adapting the method employed by Scott and O'Hara (1993), the WordPerfect™ word-processing thesaurus and a supplementary thesaurus were used to calculate simple categorical discrepancies between domains of the self-concept. Discrepancies were calculated between the self-actual and self-ideal concepts (self-actual:self-ideal discrepancies), and for self-actual:self-ought, self-actual:parent-actual, self-actual:parent-ideal and self-actual:parent ought. Matches between domains were recorded when a word (or its synonym) was used in both domains, mismatches when a word was used in one domain and its antonym in another.

Inter-rater reliability calculated for the PQQ using matches and mismatches between self-actual and self-ideal concepts and also between self-ideal and parent-actual concepts, with two raters blind to each other's ratings, revealed a very high level of agreement (mean alpha = .985). Such a high reliability coefficient is unsurprising given that the coding of matches and mismatches is strictly governed by the use of thesauruses.

Analyses were conducted on matches and mismatches separately, but also on consistency scores. These were calculated by subtracting the number of mismatches from the number of matches for each subject for each type of discrepancy. Positive scores therefore indicated consistency between domains. In the case of consistency with parental domains, agreement with each parent was calculated, and the mean of the two used for analysis. It was decided to use the mean of the two parental perceptions for three reasons. First, many participants had one parent who had died. Analyses of mean parental ratings were therefore possible on a larger body of data. Second, I was interested in examining self:other discrepancies rather than specific relationship difficulties. Use of a mean rating would tend to minimise particular difficulties with specific individuals. Finally, I aimed to minimise the number of possible analyses in

order to avoid Type I errors.

8.2.2.3 Attributional Style Questionnaire

In addition to these measures, the Attributional Style Questionnaire (Peterson, Semmel, Von Baeyer, Abramson, Metalsky, & Seligman, 1982) and the Fenigstein & Vanable Self-Consciousness Scale (Fenigstein & Vanable, 1992) were also administered (see Appendices 8 and 9).

The Attributional Style Questionnaire (ASQ: Peterson, et al., 1982), which was used to measure the attributional style of the groups, is a 12-item scale which asks participants to generate likely causes for 6 positive and 6 negative hypothetical events. These causal attributions are then rated by the participants on three scales - internality (that is, the degree to which the events are attributed to self or to external causes such as circumstances or other people), stability (that is, the degree to which the causes are likely to be present in the future) and globalness (that is, the degree to which the causes are likely to influence a wide range of events in addition to the specific events mentioned in the questionnaire). One depressed and one paranoid subject failed to complete the ASQ.

8.2.2.4 Self-Consciousness Scale

A final scale employed in this study was Fenigstein, Scheier and Buss's (1975) Self-Consciousness Scale (see Appendix 9). Fenigstein and Vanable (1992), in a study of non-patient participants, observed that public self-consciousness was associated with their participants' scores on a measure of paranoid thought. Therefore

the measure of self-consciousness employed in that study was also employed in the present investigation. The Self-Consciousness Scale is a 23-item questionnaire tapping three aspects of self-consciousness - private self-consciousness, public self-consciousness and social anxiety. Participants rate the degree to which they agree with a set of statements reflecting inner thoughts and feelings (e.g. "I reflect about myself a lot"), their awareness of their impact on others' perceptions (e.g. "I'm very concerned about the way I present myself") and discomfort in social situations (e.g. "I feel anxious when I speak in front of a group"). This questionnaire has acceptable reliability (test-retest reliability alpha = .80; Fenigstein, et al., 1975).

8.3 Results

8.3.1 Demographic Variables

The three groups did not differ significantly in age, $F(2,63) = 2.11$, $p = .13$, or gender, $Chi^2 = 1.55$, $p = .46$. Analysis of the clinical details of the two patient groups revealed no significant differences for the length of time since first diagnosis, $F(1,42) = 2.55$, $p = .10$, or for the relative proportion of participants who were in-patients, $Chi^2 = 2.28$, $p = .13$.

8.3.2 Self-discrepancies

Table 8.1 shows the mean number of self-descriptive words employed in each domain, and Table 8.2 the mean consistency scores for self-actual:self-ideal, self-actual:self-ought, self actual:parent-actual, self-actual:parent-ideal and self-actual:parent-ought comparisons for the three subject groups.

Table 8.1 Number of self-descriptive words used

	Group								
	Non-patient			Depressed			Paranoid		
Domain	M	SD	n	M	SD	n	M	SD	n
Self-Actual	9.63	1.14	22	7.71	1.71	22	8.10	2.63	22
Self-Ideal	9.82	0.66	22	6.43	2.48	22	6.86	2.56	22
Self-Ought	9.91	0.43	22	4.95	2.63	20	5.40	2.85	18
Parent-Actual	9.91	0.43	22	5.14	2.10	22	6.14	2.46	22
Parent-Ideal	9.77	0.87	22	3.26	1.45	17	3.76	1.68	16
Parent-Ought	9.86	0.47	21	3.13	1.68	15	3.40	2.03	15

Table 8.2 Unadjusted inter-domain consistency scores

	Group					
	Non-patient		Depressed		Paranoid	
Discrepancy	M	SD	M	SD	M	SD
Self-Actual:						
Self-Ideal	+2.23	2.02	-0.36	1.65	+1.73	2.51
Self-Actual:						
Self-Ought	+1.91	2.97	-0.45	1.91	+2.50	1.42
Self-Actual:						
Parent-Actual	+2.52	1.99	+0.98	1.78	+0.45	1.36
Self-Actual:						
Parent-Ideal	+2.02	1.28	-0.06	1.59	+0.12	0.97
Self-Actual:						
Parent-Ought	+2.02	1.10	-0.27	1.78	+0.17	0.98

Note. Consistency scores represent mean number of matches between the domains minus mismatches. Positive scores represent greater consistency.

A full factorial multivariate analysis of variance (MANOVA) was conducted on the five consistency scores, with group membership as a between-subjects variable and type of self-discrepancy as a within-subject variable. This revealed significant differences between the groups in terms of overall level of consistency scores, $F(2,45) = 13.48$, $p < .0005$. There was no overall difference in consistency scores between the domains, $F(4,180) = 1.62$, $p = .172$, but, most importantly, the interaction term was highly significant, $F(8,180) = 4.13$, $p < .0005$, indicating that the pattern of self-discrepancies, as indicated by consistency scores in the different domains, differed between the groups. This analysis, however, is compromised by the observation that both patient groups used fewer self-descriptive words than did the non-patient controls, $F(2,63) = 5.66$, $p < .01$. This would mean that consistency scores between domains (matches minus mismatches) are likely to tend to zero in these groups.

Analyses were therefore conducted which controlled for this effect. Adjusted consistency scores were generated for all domains by dividing the original consistency scores by the maximum possible given the number of words generated. These adjusted consistency scores therefore ranged from -1 to +1, with a score of +1 indicating that all possible words generated were matched across domains. Table 8.3 shows mean adjusted consistency scores for self-actual:self-ideal, self-actual:self-ought, self-actual:parent-actual self-actual:parent-ideal and self-actual:parent-ought domains for the three subject groups.

Table 8.3 Adjusted inter-domain consistency scores

	Group							
	Non-patient		Depressed		Paranoid			
Discrepancy	M	SD	M	SD	M	SD	df	F
Self-Actual:								
Self-Ideal	$+0.25_a$	0.26	-0.02_c	0.21	$+0.18_a$	0.32	(2,63)	6.35†
Self-Actual:								
Self-Ought	$+0.20_a$	0.30	-0.06_b	0.25	$+0.33_a$	0.22	(2,57)	11.27‡
Self-Actual:								
Parent-Actual	$+0.30_a$	0.17	+0.17	0.19	$+0.10_b$	0.12	(2,63)	5.522¤
Self-Actual:								
Parent-Ideal	$+0.21_a$	0.13	$+0.0_b$	0.21	$+0.02_b$	0.14	(2,52)	9.095‡
Self-Actual:								
Parent-Ought	$+0.21_a$	0.12	-0.03_b	0.24	$+0.02_b$	0.14	(2,52)	9.43‡

Note. Scores represent raw consistency scores divided by the maximum possible number of matches and mismatches. ¤ represents $p < .01$, † $p < .005$, ‡ $p < .0005$. Means having different subscripts differ at $p < .01$ (subscripts a and b) and $p < .05$ (subscripts a and c) in Tukey's HSD pairwise comparisons.

A full factorial MANOVA was conducted on the five adjusted consistency scores, with group membership as a between-subjects variable and type of self-discrepancy as a within subject variable. This revealed significant differences between the groups in terms of overall level of adjusted consistency scores, $F(2,45) = 11.20$, $p < .0005$, and between the domains, $F(4,180) = 3.51$, $p < .001$. The interaction term was significant, $F(8,180) = 5.21$, $p < .0005$, indicating that the pattern of self-discrepancies, as indicated by adjusted consistency scores in the different domains, differed between the groups. These domains were then investigated using individual univariate analyses.

Analysis of the adjusted consistency scores in the self-actual:self-ideal domain revealed that the three groups differed significantly, $F(2,63) = 6.35$, $p < .005$. Planned pairwise comparisons (Tukey's HSD) revealed that the adjusted consistency scores of groups C (control participants) and P (deluded patients) did not differ significantly. Both group C, $p < .01$, and group P, $p < .05$, had higher adjusted self-actual:self-ideal consistency scores than group D.

Analysis of the adjusted consistency scores in the self-actual:self-ought domain revealed that the three groups again differed significantly, $F(2,57) = 11.27$, $p < .0005$. Planned pairwise comparisons (Tukey's HSD) revealed again that groups C and P did not differ significantly, while both had higher adjusted self-actual:self-ought consistency scores than group D, $p < .01$ for each comparison.

Analysis of the adjusted consistency scores in the self-actual:parent-actual domain revealed that the three groups again differed significantly, $F(2,63) = 5.52$, $p < .01$. Planned pairwise comparisons (Tukey's HSD) revealed that group C had higher adjusted self actual:parent-actual consistency scores than group P, $p < .01$, with group D in an intermediate position, significantly different from neither of the other two groups.

Analysis of the adjusted consistency scores in the self-actual:parent-ideal

domain again revealed that the three groups differed significantly, $F(2,52) = 9.09$, $p < .0005$, with planned pairwise comparisons (Tukey's HSD) revealing higher scores for group C than groups D, $p < .01$, and P, $p < .01$. Groups P and D did not differ.

Finally, analysis of the adjusted consistency scores in the self-actual:parent-ought domain revealed that the three groups again differed significantly, $F(2,52) = 9.43$, $p < .0005$. Planned pairwise comparisons (Tukey's HSD) revealed that group C had greater adjusted consistency scores than groups D, $p < .01$, and P, $p < .01$, but that groups P and D did not differ.

8.3.3 Valence of self-descriptive words

Both patient groups showed a low level of agreement between self-actual and parent-actual concepts. This finding is open to a number of interpretations. A discrepancy between self and parents could be viewed as either positive or negative, depending on whether it is believed that parents have positive views of the self when the self-actual description is negative ("My parents like me, but I think I'm unpleasant"), or have an unwarranted negative view of self when the self-actual description is positive ("I'm a pleasant person, but my parents don't like me").

Since paranoid patients have a high consistency in self-actual:self-ideal domains, while depressed patients do not, the implication of self-actual: parent-actual discrepancies for the paranoid group seems reasonably clear. Nevertheless detailed analysis appears desirable. Therefore, the words used by participants were coded as either positive, negative, or neutral or uncodable using Anderson's (1968) list of the 'likeableness' of self-descriptive words. Similar analyses of valence were not conducted for the ideal and ought domains since words used in these domains are personal goals and must, therefore, by definition, be considered positive in valence (as

already indicated, it is a strength of self-discrepancy theory that such self-evaluative comparisons are made to internal rather than external criteria). Table 8.4 shows the mean number of positive and negative words used in the self-actual and parent-actual domains for the three subject groups.

Table 8.4. Numbers of positive and negative words used

	Group					
	Non-patient		Depressed		Paranoid	
Domain	M	SD	M	SD	M	SD
Self-Actual						
Positive words	5.73	1.72	3.09	2.43	4.55	2.91
Negative words	2.09	1.54	3.14	2.49	1.14	1.55
Parent-Actual						
Positive words	5.68	1.61	2.20	1.70	1.55	1.35
Negative words	2.80	1.56	1.43	1.23	2.84	2.62

Note. Words were classified as positive or negative according to Anderson's (1968) criteria. Positive and negative words used in the self-actual and parent-actual domains are presented separately.

A full factorial MANOVA was conducted, examining the number of positive and negative words employed, with group-membership as a between-subjects variable, and perspective (self and parent) and valence (positive and negative) as within-subjects factors. This revealed predictable differences, with the groups differing in terms of the overall number of words employed, $F(2,63) = 26.37$, $p < .00005$, and with more positive than negative words used overall , $F(1,63) = 25.38$, $p < .00005$. The combined interaction term was highly significant , $F(2,63) = 8.51$, $p < .0005$, indicating that the groups differed in the relative proportion of positive and negative words used in the self-actual and parent-actual domains. As the interaction term was significant, individual two-way analyses of variance were conducted, examining number of positive and negative words used in both self-actual and parent-actual domains. In each case group membership was a between subjects variable, with type of word (positive vs negative) as a within-subjects variable.

Overall, participants used more positive than negative words in the self-actual domain, $F(1,63) = 25.63$, $p < .00005$. Again the groups differed in the total number of words (both positive and negative) employed, $F(2,63) = 5.58$, $p < .01$, and the interaction term was significant, $F(3,63) = 6.68$, $p < .005$. Tests of simple effect revealed that non-patient participants, $F(1,63) = 20.75$, $p < .0005$, and paranoid patients, $F(1,63) = 18.24$, $p < .0005$, both used significantly more positive than negative words. Depressed patients used no more positive words than negative words, $F(1,63) = .003$, $p = .96$.

Overall, the groups also differed in the total number of words produced in the parent-actual domain, $F(2,63)=50.10$, $p < .00005$, and participants as a whole reported more positive than negative words, $F(1,63) = 4.49$, $p < .05$. The interaction term was also significant, $F(2,63) = 10.55$, $p < .0001$. Tests of simple effect revealed that non-patient participants used significantly more positive than negative words, $F(1,63) = 20.09$, $p < .0005$. Depressed patients did not, $F(1,63) = 1.44$, $p = .24$,

while the paranoid patients used more negative words than positive words, $\underline{F}(1,63) = 4.05$, $\underline{p} < .05$.

8.3.4 Attributional style

For analysis of attributional style, internality scores were collated for positive and negative events separately. Participants' ratings of internality for positive and negative events are presented in Table 8.5.

Table 8.5 Mean subjects' ratings of internality on the ASQ for the three groups. Score for positive and negative events are presented separately.

	Internality			
	Positive Events		Negative Events	
Group	M	SD	M	SD
Non-patient	27.86	7.11	23.23	6.47
Depressed	27.30	5.26	32.30	5.97
Paranoid	31.19	8.26	24.71	8.46

A two-way analysis of variance was conducted on these scores, with group membership (C, D and P) as a between-subjects variable and type of event (positive vs negative) as a within-subjects variable. The group differences in internality approached significance, $F(2,56) = 4.28$, $p = .06$. Planned pairwise comparisons (Tukey's HSD) indicated that group C differed from group D, $p < .05$, but that no other differences were significant. Overall, for the three groups together, positive events were not attributed more to internal causes than were negative events, $F(1,56) = 1.70$, $p = .20$. The interaction term was significant, $F(2,56)= 7.45$, $p < .005$. Tests of simple effect revealed that the difference in attribution for positive and negative events was not significant for non-patient participants, $F(1,56) = 2.30$, $p = .14$. Depressed patients made significantly more internal attributions for negative than positive events, $F(1,56) = 5.37$, $p < .05$, while paranoid patients made significantly more internal attributions for positive than negative events, $F(1,56) = 9.47$, $p < .005$.

8.3.5 Self-consciousness

Participants' ratings of the three components of self-consciousness: public and private self-consciousness and social anxiety, are presented in Table 8.6.

Table 8.6 Mean scores (and standard deviations) for the three groups (and sub groups of the paranoid subjects) for the three aspects of self consciousness on the SCS.

	Aspect of Self-consciousness					
	Public		Private		Social Anxiety	
Group	M	SD	M	SD	M	SD
Non-patient	16.14	5.06	20.67	7.71	9.71	6.95
Depressed	20.39	5.45	24.33	7.55	15.50	4.37
Paranoid	21.29	5.91	25.19	8.04	14.71	5.54
Paranoid - Depressed	24.50	3.66	28.58	4.94	17.33	4.54
Paranoid - Non-depressed	17.00	5.72	20.67	9.37	11.22	4.94

A two-way analysis of variance, with the three aspects of self consciousness as within-subject factors and group membership as a between-subjects factor was conducted. This revealed significant differences between the groups in overall self-consciousness, $F(2,57) = 5.97$, $p < .005$. Planned pairwise comparisons (Tukey's HSD) revealed that both patient groups differed from control participants, with depressed patients, $p < .01$, and paranoid patients, $p < .05$, both recording a higher general level of self-consciousness than the control participants. The scores for the three aspects of self-consciousness of course differed since they reflected differing scoring systems, $F(2,114) = 62.20$, $p < .0001$. The interaction term was, however, non-significant, $F(4,114) = 0.23$, $p = .88$.

Post hoc analyses, comparing depressed and non-depressed paranoid patients (patients scoring above versus below 10 on the BDI) revealed that these differences could be ascribed to the effect of depressed mood alone. Depressed paranoid patients revealed higher levels of self-consciousness (in all three aspects) than the non-depressed paranoid patients, $F(1,19) = 12.26$, $p < .005$. A non-significant interaction term indicated that the scores of the three different aspects of self-consciousness did not vary independently, $F(4,114) = 0.30$, $p = .88$. No specific relationship to paranoia alone was therefore found.

8.4 Discussion

The findings in this study were consistent with the model of persecutory delusions presented in Chapter 6. As predicted, both patients suffering from delusions of persecution and non-patient participants showed very small discrepancies between their perceptions of their actual selves and their ideal and ought self-representations, in contrast to the depressed patients. At the same time there were large discrepancies

between the self-perceptions of paranoid patients and the views they believed their parents had of them. In this respect they differed from non-patient participants, but were similar to the depressed patients. Thus, only the deluded patients in the present study showed a pattern of high self-actual:self ideal and self-actual:self-ought consistency accompanied by high self:parent discrepancies. Content analyses revealed that paranoid patients used predominately positive words to describe themselves, but felt that their parents would use more negative than positive words. Depressed patients used as many negative words as positive words to describe themselves, and felt that their parents would do likewise. Non-patient participants used predominately positive words to describe themselves, and believed their parents would too. These group differences are consistent with the suggestion that paranoia results from a set of externalizing attributions for negative events which tend to prevent the explicit activation of discrepancies between actual-self representations and self-guides at the expense of activating discrepancies between the self and others.

Analysis of the attributional style of the deluded patients replicated findings reported in Chapter 5, and further support a model of paranoid ideation implicating defensive attributional processes. Consistent with the findings of Smari, Stefansson, and Thorgilsson (1994), no group differences were observed on the Fenigstein & Vanable Scale once depressed mood had been controlled for. This does not necessarily mean that self-consciousness is unimportant in paranoia. That levels of self-consciousness were high in both depression and paranoia suggests that both conditions are associated with a high degree of salience of the self-concept and, hence, that the self-concept is aetiologically important. However, it does sugget that self-consciousness is not a sufficient nor a specific causal agent in paranoia.

8.4.1 Possible limitations

Although consistent with the model of paranoid ideation outlined in Chapter 6, some alternative interpretations of the data must be considered. It may be argued that these results reflect paranoid patients' lack of awareness about their illness, with consistency between actual self representations and self-guides reflecting patients' beliefs that they are well, but discrepancies between the parents' view of the self and actual-self representations reflecting the experience of being told that they are ill. However inspection of the words used by paranoid patients in the self-actual domain of the Personal Profile Questionnaire revealed that only 9 out of 177 words referred to psychiatric health, and that all of these were negative. In the parent actual domain, 8 out of 165 words related to psychiatric ill-health. Therefore, the majority of discrepancies between self-actual and parent-actual representations did not reflect denial of illness.

A second alternative interpretation of these results is that discrepancies between self and parents for the paranoid group may reflect particular, long-standing, relationship difficulties with family members. It would be unwise at present to assume a general pattern of social cognition from the observations, given that some theorists have suggested that pathological relationships with parents are an aetiological feature of schizophrenic disorders in general (Jortner, 1990; Laing & Esterson, 1968) and paranoid disorders in particular (Heilbrun & Norbert, 1971; Heilbrun & Norbert, 1972; Kaffman, 1983; Schatzman, 1973), and given evidence that parental attributions and criticism may play a role in the maintenance of severe psychiatric disorders (Barrowclough, Johnston, & Tarrier, 1994; Vaughn & Leff, 1976).

An account of paranoid ideation which places emphasis on attributions and self discrepancies is not, however, inconsistent with an account which focusses on parent-child relationships. As mentioned in Chapter 4, Higgins (1989) has argued that

parental involvement with the child, and consistent, clear and responsive parenting are important in the development of appropriate self guides. Moreover, he has suggested that discrepancies between different self-guides (for instance between parental and personal guides) may be associated with uncertainty, identity confusion and rebelliousness in adolescence. In this context it is worth noting that family processes have been implicated in the cognitive biases found in depressed patients (Hammen, 1991; Rose, Abramson, Hodulik, Halberstadt, & Leff, 1994). The difference between an account of the present data which exclusively emphasizes family relationships and the broader account I have offered therefore concerns the generalizability and origins of the pattern of self-discrepancies observed. In the present study self-actual:parent-ideal and self-actual:parent ought discrepancies were marked in the case of the deluded patients. This suggests that the aspirations as well as perceptions of the paranoid individuals may be orthogonal to those of their parents, and such features deserve further study. Further research using methods of assessing patients' perceptions of wider groups of individuals, together with studies that may link family processes with self-discrepancies is likely to advance significantly our understanding of the etiology of paranoid disorders.

It might also be suggested that the group differences observed could be consequences of psychiatric disorder rather than intrinsic to the disorders themselves. Psychiatric conditions are powerfully depressing experiences which are associated with powerlessness and stigmatization (Angermeyer, Link, & Majcher-Angermeyer, 1987; Barham, 1984; Leete, 1987) and which might be expected to have a profound impact on the self-concept. That the observed group differences cannot be explained in this way is evident from two facts. First, despite suffering from a condition with a high suicide rate (Black & Fisher, 1992; Caldwell & Gottesman, 1992; Roy, 1992) and having measurably low mood, the paranoid patients had a high consistency between self and ideals. Second, the pattern of self-discrepancies was different for the

two patient groups. This would not be expected if the observed self discrepancies were simply the effect of the social consequences of becoming a psychiatric patient. Taken together, these observations indicate that the abnormalities of the self-concept seen here are intrinsic to paranoid ideation.

The important question of whether or not the pattern of self-discrepancies observed predates the onset of paranoid beliefs may be hard to resolve by direct empirical research, as prospective, longitudinal investigations of the precursors of psychiatric disorders are difficult and expensive to carry out. However, the model of paranoid ideation proposed by ourselves suggests that the repeated use of externalising attributions in response to activation of implicit negative self-representations leads to discrepancies between self-perceptions and beliefs about the perceptions of others which, in extreme form, are manifest as persecutory delusions. Although the developmental origins of the abnormal attributional style of paranoid patients are not understood, it is possible that such a style develops in the context of chronic threats to the self-concept which predate the appearance of florid psychosis. Indeed, it seems likely that both an abnormal attributional style, and an implicit negative actual-self representation, develop symbiotically over a period of time.

The observation by Lyon, Kaney and Bentall, (1994) that paranoid patients make internal attributions for negative events on an implicit measure of attributional style is consistent with the hypothesis that deluded patients have implicit negative self-representations. Although a distinction between implicit and explicit self-representations appears to depart from conventional self-discrepancy theory, it is consistent with experiments which have shown that self-discrepancies only become available to individuals when primed by appropriate stimuli (Strauman, 1989; Strauman, 1992; Strauman, Lemieux, & Coe, 1993). On our account, the negative actual-self representations of deluded patients remain implicit precisely because attributional processes prevent them from becoming explicit.

Previous studies of self-discrepancies have indicated that discrepancies between self perceptions and the believed perception of the self by others are associated with anger and agitation. In particular, "actual-own:ought-other discrepancy (AOO) was uniquely related to agitation and to anger at others and resentment" (Strauman & Higgins, 1988; p. 685). In the present study, large self-actual:parent-ought discrepancies were observed in paranoid patients. It is possible that the externalizing attributions of paranoid patients have important emotional consequences, leading to agitation and hostility towards others. Paranoid delusions have been associated with anger and aggression (Kennedy, Kemp, & Dyer, 1992) and also with violence towards others (Taylor, 1985; Wessely, Buchanan, Reed, Cutting, Garety, & Taylor, 1993).

Chapter 9.

An Analogue Study of the relationship between Attributional Style and Self-Discrepancies

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A paper based on the material presented in this chapter and chapter 11 has been submitted for publication as: Kinderman, P., & Bentall, R. P. Self-discrepancies and causal attributions: Analogue studies of hypothesised relationships.

9.1 Background

Having produced evidence supportive of the model of paranoid ideation outlined in Chapter 6 in terms of the salience of the self-concept (Chapter 7) and explicit self-discrepancies (Chapter 8), evidence for the dynamic validity of the model was sought. In particular the causal links between causal attributions for negative events at one point in time and subsequent self-discrepancies were the focus of investigation. Secondly, the role of self-discrepancies in facilitating particular causal attributions were of interest.

To investigate these points, an experiment was designed in which normal participants were asked to make attributions about negative events after they had reported their self-representations, allowing the examination of the relationship between self-discrepancies at time 1 and subsequent attributions. The participants then reported their self-representations a second time, allowing the assessment of the impact of their attributions on self-discrepancies at time 2.

9.1.1 Hypotheses

It was hypothesized that high self-actual:self-ideal discrepancies at time 1 would be predictive of internal attributions for negative events. It was also hypothesized that excessively internal attributions for negative events would be associated with increased levels of both self-actual:self-ideal discrepancies and self-actual:other-actual discrepancies at time 2. Following the model of paranoid ideation (Chapter 6), it was also hypothesized that excessively external attributions would be associated with reduced levels of self-actual:self-ideal discrepancies at time 2, but also increased self-actual:self-other discrepancies at that time. Attributions

which are neither particularly internal or particularly external were hypothesized to result in no change in either self-actual:self-ideal or self-actual:other-actual discrepancies.

9.2 Method

9.2.1 Participants

Participants were 120 undergraduate students of occupational therapy and radiography. (A further 4 participants were excluded from the study as they failed correctly to complete the questionnaires.) Participants were approached for their cooperation, which was voluntary, in class-groups. No incentive was offered for participation. None of the participants, when questioned after the experiment, indicated that they had understood the hypothesis under test. The mean age of the group was 25.47 years; SD = 7.96, range 18 to 51, 110 were women and 10 were men.

9.2.2 Materials

9.2.2.1 Beck Depression Inventory:

The BDI has been described in previous chapters.

9.2.2.2 Self-Concept Checklist

The Self-Concept Checklist (SCC) was designed specifically for this experiment and measured self-discrepancies in the domain of personal achievement following the general principles of Self-Discrepancy Theory (Higgins, 1987) (see Appendix 10). It consists of three sections. The first assesses participants' opinions of their actual level of achievement, the second, their ideal level of achievement and the third, the level of achievement they believe their peer group ascribes to them. 30 positive and 30 negative achievement-related words were presented three times on separate pieces of paper headed by the instructions, "Please read the following list of words and select those which best describe you as you think YOU ACTUALLY ARE" for the first section, "...as you think YOU WOULD IDEALLY LIKE TO BE" for the second section and "...as you think THE OTHER PEOPLE IN YOUR YEAR THINK YOU ACTUALLY ARE" for the third. Participants were asked to place a tick beside those words they felt described them in each domain/perspective and a cross beside those words that they felt definitely did not describe them. This resulted in three overall measures of self-concept in the specific field of achievement (scored in each case by the addition of positive words endorsed positively and negative words endorsed negatively, and subtracting both positive words endorsed negatively and negative words endorsed positively). Finally, self-discrepancy scores (self-actual:self-ideal and self-actual:other actual) were calculated by arithmetic subtraction of the scores in one domain from the scores in the other. When reliability checks were conducted on the three scales within the self-concept check-list separately using data from all 120 participants when first completing the questionnaire, Cronbach's a was found to be 0.977 for the self-actual representation, 0.991 for the self-ideal representation, and 0.966 for the other-actual representation.

It is worth mentioning, at this point, that the SCC is a nomothetic measure, as

opposed to the idiographic Selves Questionnaire developed by Higgins (1987), which is an idiographic measure. Whereas the Selves Questionnaire is designed to measure discrepancies between self-reported self representations and self-reported self-guides, the SCC uses experimenter generated descriptions. In this respect it can be seen as analogous to a test of recognition as opposed to free-recall memory. This simplification was undertaken because of the need to assess self-discrepancies in a large group of individuals, and because I believed that the changes in between self discrepancies would be sufficiently robust to be detected by this kind of method. Items in the domain of achievement were chosen in the expectation that these would be particularly salient to undergraduate students.

Two versions of the SCC were constructed, allowing for administration before and after the administration of the Attributional Style Inventory. The only difference between the two versions was that the order of the words was reversed in the second version.

9.2.2.3 Attributional Style Inventory

The Attributional Style Inventory (ASI) was designed to prime schemata involving personal failure in achievement-related areas and also to measure the attributions made by participants for such events (see Appendix 11). It was constructed following the model of the Attributional Style Questionnaire (Peterson, Semmel, Von Baeyer, Abramson, Metalsky, & Seligman, 1982) and consisted of 18 negative items related to personal achievement (failure to achieve promotion, having one's advice ignored, etc). Each item required the subject to generate a cause for the given event, and then to rate that cause on a 7-point scale according to the degree to which that cause was internal ("totally due to me") as opposed to external ("totally due

to other people or circumstances"). Unlike previous measures of attributional style, the globalness and stability of attributions were not measured.

The possible range of this scale is from 18 (scoring wholly external for all 18 questions) to 128 (scoring a maximum 7 on all questions), with a mid point of 74. Scores on the ASI revealed a near-normal distribution, with a mean of 74.46; SD = 11.37, range 33 to 105. The reliability of the ASI (Cronbach's alpha = 0.668), although less than ideal, compares well with that of the original Attributional Style Questionnaire, which has been criticised for its low internal consistency (Tennen & Herzberger, 1985).

9.2.3 Procedure

Participants were approached in class-groups and asked for their consent. After consent had been given the participants were asked to work through a booklet incorporating the questionnaires in the following order: BDI, first SCC, ASI, and second SCC.

9.3 Results

9.3.1 Demographic details

The mean level of depressed mood was 4.59; SD = 4.34, range 0 to 24. All participants, including 14 participants scoring over 10 on the BDI (the conventional cut-off point for mild depression; (Rehm, 1988)), were included in the analysis in order to maximize heterogeneity of attributional styles and self-discrepancies. For the purpose of some analyses, participants were allocated into three groups on the basis of

ASI scores. Cut-off points were chosen to give three groups as equal as possible in numbers. Group 1, internal attributors (mean ASI score = 86.26, SD = 6.81, range 80 to 105), had 42 participants, Group 2, median attributors, (mean ASI score = 73.86, SD = 2.76, range 70 to 79) had 37 participants, and Group 3, external attributors, (mean ASI score = 62.93, SD = 6.85, range 33 to 69) had 41 participants. One-way analysis of variance (ANOVA) confirmed that, as intended, these groups differed significantly in terms of the internality of their attributional style, $F(2,117) = 163.26$, $p < .0005$.

In keeping with predictions drawn from Self-Discrepancy Theory, discrepancy scores were calculated from SCC scores both before and after administration of the ASI. Self-actual:self-ideal discrepancy scores were calculated by subtracting self ideal scores from self-actual scores, and self-actual:other-actual discrepancy scores by subtracting other-actual scores from self-actual scores. Summary data for the self-concept checklist at both times are presented in Table 9.1. Although the variance was large, the majority of participants revealed themselves to have ideals well in excess of their perceived actual selves, and felt that others saw them in a slightly worse light than they saw themselves. This is reflected in a mean negative self-actual:self-ideal discrepancy and a mean positive self-actual:other-actual discrepancy.

Table 9.1. Summary statistics for Self-Concept Checklist for the three groups divided on the basis of scores on Attributional Style Inventory. Figures represent discrepancies between achievement scores in the relevant domains before and after completion of the ASI. Also presented are change scores (arithmetic subtraction of discrepancy scores at time 1 from time 2) and adjusted scores following anaysis of covariance with scores at time 1.

	Groups					
	Group 1 Internal Attributors		Group 2		Group 3 External Attributors	
Discrepancy	M	SD	M	SD	M	SD
Actual-Ideal (time 1)	-22.73	17.16	-19.81	14.07	-15.71	11.74
Actual-Ideal (time 2)	-23.50	17.17	-16.00	12.13	-13.66	12.19
Change	-0.71	9.16	+3.81	7.01	+2.05	5.80
Adjusted*	-1.28		+2.90		+2.38	
Self-Other (time 1)	+6.33	11.26	+2.32	12.34	+3.73	8.03
Self-Other (time 2)	+6.14	8.60	+2.47	8.60	+1.05	7.52
Change	-0.19	9.17	+0.16	7.56	-2.68	5.85
Adjusted*	+0.83		-0.67		-2.86	

Note * refers to adjusted change scores following analyses of covariance

9.3.2 The relationship between self-actual:self-ideal discrepancies, depressed mood and subsequent attributions

Consistent with the previous findings of Strauman and colleagues (e.g. Strauman & Higgins, 1988), levels of depressed mood, as measured by BDI scores, were associated with large self-actual:self-ideal discrepancies at time 1, Spearman $r = 0.360$, $p < .005$, and were associated, although less strongly, with self-actual:other-actual discrepancies, Spearman $r = 0.203$, $p < .05$. Regression analysis was conducted on ASI scores, with BDI scores and self-actual:self-ideal discrepancy scores at time 1 as independent variables. This revealed a significant negative association for self-discrepancies, beta = -0.156, $t(2,117) = -2.05$, $p < .05$, indicating that high self-actual:self-ideal discrepancies at time 1 were predictive of internal attributions for negative events on the ASI. However, BDI scores at time 1 did not predict ASI scores, beta = 0.197, $t(2,117) = 0.76$, $p = .446$.

9.3.3 The relationship between causal attributions and subsequent self-discrepancies

In order to investigate changes in the accessibility of self-discrepancies following administration of the ASI, difference scores were calculated. For each subject, discrepancies calculated in the manner described above at time 1 (before administration of the ASI) were subtracted from the same scores calculated at time 2. This resulted in measures of change in self-actual:self ideal and in self-actual:other-actual discrepancies. A negative self-actual:self ideal discrepancy change score (larger negative scores at time 2) reflects increased accessibility of this type of discrepancy. In contrast, a positive self actual:other-actual discrepancy change score reflects increased accessibility of this type of discrepancy. Summary statistics for these difference scores

are presented in Table 9.1.

Because the predictions for the effects of attributions on the accessibility of self-discrepancies were nonlinear (it was predicted that self-actual:self-ideal discrepancies would increase only for Group 1, the internal attributors, but also that self-actual:other-actual discrepancies would be increased in Group 1 and in Group 3, the external attributors, but not in Group 2) group comparisons were carried out to test for these effects. In keeping with statistical recommendations for the analysis of change scores (Cook & Campbell, 1979), analyses of covariance (ANCOVA) were used to assess the impact of attributions on changes in self-discrepancies, with group membership as a between-subjects variable and self-discrepancy scores at time 1 as covariates. These analyses allowed us to examine the relationship between attributions and subsequent changes in self-discrepancies while controlling for the influence of self-discrepancies at time 1 on attributions.

The group effect for changes in self-actual:self-ideal discrepancies was statistically significant, $F(2,116) = 4.03$, $p < .05$. Planned pair-wise comparisons revealed that both the external attributors in Group 3, $t(117) = 2.29$, $p < .05$, and the participants in Group 2, $t(117) = 2.62$, $p < .01$, differed from the internal attributors in Group 1, but that Groups 2 and 3 did not differ from each other, $t(117) = 0.33$, $p = .371$. Examination of the adjusted means revealed that the internal attributors had significantly increased their self-actual:self-ideal discrepancies compared to Groups 2 and 3 (external attributors).

A similar ANCOVA on self-actual:other-actual change scores revealed a statistically significant difference between the groups, $F(2,116) = 3.96$, $p < .05$. Planned pair-wise comparisons revealed that the internal attributors in Group 1 differed significantly from the external attributors in Group 3, $t(117) = 2.78$, $p < .005$. Groups 2 and 3 were marginally different from each other, $t(117) = 1.65$, $p = .051$, but Groups 1 and 2 did not differ significantly , $t(117) = 1.13$, $p = .130$.

Inspection of the adjusted group means reveals a near-linear relationship between attributions and change in self-actual:other actual discrepancies, with participants making internal attributions showing increased self-actual:other-actual discrepancies while participants making external attributions showed reduced discrepancies.

9.3.4 Post-hoc analyses of types of external attributions

In the above analyses, internal attributions were associated with greater self-actual:self-ideal discrepancies and also with greater self-actual:self-other discrepancies. Both of these findings are consistent with the predictions made at the outset of the experiment. However the predictions made for the impact of excessively external attributions on self-discrepancies were not completely supported by the data. While such external attributions were associated with reduced accessibility of self-actual:self-ideal discrepancies, they were also associated with reduced accessibility of self-actual:other-actual discrepancies. The second of these latter findings appears to contradict the relationship predicted by the model of paranoid ideation outlined in Chapter 6.

A further set of post-hoc analyses were therefore conducted to attempt to explain these results, with particular emphasis on why external attributions appeared to result in reduced accessibility of self-other discrepancies. One explanation of the present findings may concern the nature of internality judgements. Researchers in the learned helplessness tradition have treated internality-externality as a bipolar dimension (see Chapter 5) and this is reflected in the format and scoring of the ASQ (Peterson, et al., 1982) from which the ASI was developed. However, several authors have noted that participants' responses on simple internality versus externality scales are inconsistent and have proposed alternative ways of categorising attributions for

internality (Stratton, Heard, Hanks, Munton, Brewin, & Davidson, 1986; White, 1991). In fact, three distinct attributional foci can be identified in the attributional classification developed by Seligman and his colleagues: an internal focus (attributing the causes of events to self), a personal external focus (attributing the causes of events to the actions or omissions of identifiable others), and a situational external focus (attributing the causes of events in terms of circumstances or chance). It is therefore unsurprising that the internality scale in Peterson and colleague's (1982) Attributional Style Questionnaire has consistently been criticised for its poor reliability (Rehm, 1988; Reivich, 1995; Tennen & Herzberger, 1985).

The difference between personal as opposed to situational external attributions may be theoretically important. Attributing the cause of negative events to the actions of "other people" may have substantially different effects compared with attributions to situational factors ("circumstances"). In particular, it might be expected that only personal external attributions would be associated with increased self-actual:other-actual discrepancies and (at a clinical level) paranoid ideation.

In a post-hoc analysis, individual causal statements given by participants on the ASI were given to two independent raters who were asked to classify all judgements which were coded by the subject as external (i.e. scores of 1 or 2 on the internality scale) as either personal (relating to the actions, omissions, behaviour or attitudes of individuals or groups) or situational (relating to circumstances, luck or chance). The two raters demonstrated a high degree of inter-rater agreement (exact inter-rater agreement for 89.2% of attributions, Cronbach's alpha = 0.846). This exercise generated a further variable, the proportion of external attribution which were personal, which was unrelated to overall ASI internality scores (Spearman r = .073, p = .652). A one-way ANOVA for the entire group of participants (with number of personal vs situational external attributions as a within-subjects comparison) revealed a highly significant tendency to attribute externally to situational as opposed to personal

causes, $F(1,119) = 77.4$, $p < .0005$.

Group 3 (the external attributors) was divided into two sub-groups on the basis of the proportion of external attributions made to personal causes. An unequal division (due to the generally greater proportion of situational attributions) resulted in 8 individuals who made more personal than situational attributions and 33 others. A one-way analysis of variance revealed a significant difference between these two sub-groups in terms of change in self-actual:other-actual discrepancies, $F(1,39) = 4.92$, $p < .05$. Those making situational attributions showed reduced self-actual:other-actual discrepancies (mean change = -3.39, SD = 5.94) while those making personal attributions demonstrated increased discrepancies (mean change = +1.375, SD = 2.13).

9.4 Discussion

This study revealed evidence of the dynamic relationship between self-representations and explanatory style hypothesized at the outset. As predicted, high self-actual:self-ideal discrepancies were associated with a tendency to make internal (self-blaming) causal attributions for negative events. This is consistent with Higgins' (1987) argument that internal attributions will be made when self-representations can be accessed which involve likely explanatory factors (e.g. "I am not as competent as I would like to be"). This effect was independent of depressed mood. Indeed, although correlating with self-actual:self-ideal discrepancies, depressed mood had no independent impact on attributions.

The failure to find an association between internality and depressed mood might be thought partially consistent with more recent attributional accounts of depression (Abramson, Metalsky, & Alloy, 1989) which have de-emphasized the role

of internality judgements in depression in comparison with previous accounts (Abramson, Seligman, & Teasdale, 1978). However, the observation that self discrepancies were predictive of internality also suggests that at least this aspect of attributional style may not be completely trait-like. Indeed, on the basis of the current findings we can predict that changes in self-representations will result in changes in the kinds of attributions offered for negative events. This prediction is consistent with Forgas and colleague's (Forgas, Bower, & Moylan, 1990) observation that people make more internal and stable attributions following failure experiences.

As predicted, internal attributions were associated with increased self-actual:self-ideal discrepancies, in comparison with external attributions, which were associated with reduced discrepancies. The observation that external attributions for negative events are associated with reduced self-actual:self-ideal discrepancies suggests that such attributions may function in a self-protective manner, and is consistent with the model of paranoid ideation (Chapter 6). However, internal attributions were also associated with opening of self-actual:other-actual discrepancies, which was not predicted. Taken at face value, this observation would predict the occurrence of paranoid ideas in people who make excessively internal attributions. Consistent with this hypothesis, it has been observed that psychotically depressed patients sometimes harbour delusional ideas about the opinions that other people have about them (e.g. "The whole world hates me") (Gelder, Gath, & Mayou, 1983).

The post hoc analysis I conducted on the participants' external attributions helps to clarify the present findings. This analysis indicated that there may be important differences in the impact of different types of external attributions on social perception. Specifically, when participants made personal external attributions (where the believed causes of negative events are held to be the actions or omissions of other people) self-actual:other-actual discrepancies became more accessible. On the other hand, situational external attributions (where the causes of negative events are

explained in terms of circumstantial or chance factors) were associated with reduced self-actual:other-actual discrepancies. This should not be surprising. The delusions of paranoid patient usually involve complex explanations of observed events which clearly implicate the intentions of other people. On the other hand, ordinary excuse making, which usually involves the generation of situational external attributions (e.g. "I'm late because the traffic was heavy") is clearly psychologically benign.

9.4.1 Implications

These observations have two implications. First, the role of the internality dimension in psychopathology may have been undervalued by some authors because it has been ill-defined in most studies. The present findings provide support for those theorists who have been critical of current conceptions of internality (White, 1991), and help to explain why reliability estimates for the ASQ internality subscales have been so poor (Rehm, 1988; Reivich, 1995; Tennen & Herzberger, 1985). Although I have suggested that the internality dimension should be reconstrued in terms of three attributional foci, other authors have suggested other methods of subdividing the internality dimension (Stratton, et al., 1986).

Second, although many aspects of the model of paranoid ideation were supported by the present findings, the findings indicate that the model may need to be revised to take into account these three attributional foci. Predictions based on a simple internal-external dimension were not supported, although the original model of paranoid ideation (Chapter 6) conceptualised persecutory delusions as stemming from purely external attributions for negative events. Reformulating the model with the incorporation of the distinction between personal and situational external attributions may help explain aspects of the presentation of persecutory delusions. On this view,

the paranoid patients' tendency to make defensive attributions, together with the failure to identify situational as opposed to personal external causes, contribute together to generate paranoid delusions. The fact that internality scores did not correlate with scores along the situational-personal dimension suggests that these two processes are independent. Indeed, it can be predicted that it will be possible to make a distinction between well-adjusted externalizers (elegant excuse-makers) and poorly adjusted externalizers (paranoid patients) on the basis of individuals' abilities to identify potential situational causes.

9.4.2 Limitations

A number of limitations of the present study must be mentioned. The study involved only normal participants, although it was designed to inform understanding of psychological disorders, particularly depression and paranoia. Clearly, extrapolation of these findings to clinical populations must only be done with caution. Similarly, the experimental intervention resulting in changing accessibility of self discrepancies (the completion of the ASI, a simple questionnaire) may have only limited ecological validity. Extrapolations of the present findings to real-life social situations in which individuals make attributions for negative experiences must therefore be made with caution. Most importantly, although the self-concept is of central importance in most psychological problems (see Chapter 4), the attributional and self-concept abnormalities uncovered in this investigation are likely to be accompanied by, and interact with, other abnormalities of cognition and affect.

The present study was not conducted using strict experimental methodology, which may have required random allocation to three groups, each of which would be required to make specific attributions. It is not possible, therefore, to conclude with

certainty that the attributional manipulation caused the changes in self-representation. It is possible, indeed highly likely, that other variables were related to both choice of attributions and self-representations. However, although it is possible to construct hypotheses concerning the consequences of natural attributions, it may not be possible to make such predictions concerning the consequences of attributions supplied by an external agent. In fact, the known psychological processes of reactance (Watts, Powell, & Austin, 1973) and compensatory self-enhancement (Greenberg & Pyszczynski, 1985) may confound studies in which individuals are required to make particular types of attributions.

Additionally, the sample was predominantly female. Given that paranoia is a phenomenon known to predominantly affect men (Chapter 3) whereas depression tends to affect women, it is conceivable that men and women might differ in their typical responses, either to threats to the self concept or to attributional tasks. Against this, however, it may be argued that the present study examined the effects of both internal and external attributions for negative events on the self-concept within this sample. It may be the case that women might make more self-blaming attributions than men, but it would be highly surprising that the effect of such attributions would have different consequences in the different genders.

A final limitation of the present study concerns the use of a nomothetic, rather than idiographic measure of self-discrepancies in the present study. By examining the reported endorsement of participants on a checklist of words supplied by the experimenters, it may be argued that self-esteem (comparisons with a global rather than individual idea) was studied rather than self discrepancies. However, although the questionnaires used in the present study may have limited the range of self-representations reported by the participants, the participants were free to specify, within these parameters, their ideals and their percieved actual selves with a high degree of reliability. The present findings should, perhaps be subjected to replication

using a more idiographic measure. This was the goal of the study reported in Chapter 11. Nevertheless, the findings reported here seem to provide important evidence concerning the dynamic relationships between self-representations and causal attributions.

Chapter 10.

The Internal, Personal, and Situational Attributions Questionnaire

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A paper based on the material presented in this chapter has been published as : Kinderman, P., & Bentall, R. P. (1996). A new measure of causal locus: The Internal, Personal and Situational Attributions Questionnaire. *Personality and Individual Differences*. **20** : 261-264.

10.1 Background

The data presented in Chapter 9 strongly suggest two important lines of further research. First, distinctions within the 'external' pole of causal attributions seem important for theoretical reasons. Second, such distinctions necessitate a revision of the model of paranoid ideation presented in Chapter 6. This chapter will describe the development and validation of a new measure of causal attributions.

10.1.1 Hypotheses

The Internal, Personal, and Situational Attributions Questionnaire (IPSAQ) was developed for pragmatic reasons. Many of the specific hypotheses stemming from its development will be outlined in subsequent chapters. In the development phase, it was hypothesized that a reliable measure of causal locus could be developed using three loci; to internal (self-blaming), personal-external (other-blaming) and situational-external causes. It was hypothesized that such a measure would have higher levels of reliability than previous dichotomous measures such as the ASQ.

It was further hypothesized that the validity of such an instrument would be demonstrated by correlating participants' scores on this new measure with measures of paranoid ideation and depressed mood.

10.2 Method

10.2.1 Design of the IPSAQ

Items in an earlier questionnaire (Bentall, Kaney, & Dewey, 1991) were modified for use in the current measure. The IPSAQ has 32 items which describe 16 positive and 16 negative social situations in the second person (e.g. "A friend tells you that she respects you" and "A friend thinks you are interesting"). For each item the respondent is required to write down a single, most likely, causal explanation for the situation described. The respondent is then required to categorize this cause as being either internal (something to do with the respondent), personal (something to do with another person or persons) or situational (something to do with circumstances or chance) by circling the appropriate choice (see Appendix 11). Three positive and three negative subscale scores are then generated by summing the number of internal attributions, the number of personal attributions and the number of situational attributions chosen for both the positive and negative items.

Two cognitive bias scores are derived from these six subscale scores. Externalizing Bias (EB) is calculated by subtracting the number of internal attributions for negative events from the number of internal attributions for positive events. A positive EB score therefore indicates strong self-serving biases (blaming oneself less for negative events than for positive events). Personalizing Bias (PB) indicates the proportion of external attributions for negative events which are personal as opposed to situational and is calculated by dividing the number of personal attributions by the sum of both personal and situational attributions for negative events. A PB score of greater than 0.5 therefore represents a greater tendency to use personal rather than situational external attributions for negative events.

10.2.2 Additional Materials

10.2.2.1 Beck Depression Inventory

The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) has been described in previous chapters.

10.2.2.2 Paranoia Scale

The Paranoia Scale (PS; Fenigstein & Vanable, 1992) is a 20-item self-report measure of paranoid ideation derived from MMPI items such as "People have said insulting and unkind things about me" and "I am bothered by people outside, in cars, in stores, etc. watching me" (see Appendix 12). It is designed to be administered to normal participants and has been shown to have good internal consistency, test-retest reliability and validity as indicated by its relationship with a number of theoretically interesting measures (Fenigstein & Vanable, 1992).

10.2.2.2 Attributional Style Questionnaire

The Attributional Style Questionnaire (ASQ; Peterson, Semmel, Von Baeyer, Abramson, Metalsky, & Seligman, 1982) has been described in previous chapters.

10.2.3 Participants and Procedure

Participants were 88 undergraduate students taking introductory courses in psychology. Three participants returned incomplete questionnaires and were excluded from further analyses. No incentive was offered for completion of the questionnaires. Participants were recruited in two groups. One group (N = 24) completed only the BDI and IPSAQ, the other participants (N = 64) also completed the PS and the ASQ. The mean age of the combined group was 22.44 years, SD = 6.86 (range 18 to 52). 58 were women and 30 were men. The mean BDI score was 6.24, SD = 5.24 (range 0 to 29), the mean PS score was 39.64, SD = 12.60 (range 20 to 76).

10.3 Results

10.3.1 Reliability

Mean numbers of the three different types of attributions made by participants on the IPSAQ were, for positive events: Internal 8.19, SD = 3.04, Personal 3.75, SD = 2.47, and Situational 4.06, SD = 2.51, and for negative events: Internal 5.88, SD = 3.24, Personal 5.15, SD = 2.77, and Situational 4.98, SD = 3.37. Reliability statistics (Cronbach's alpha), revealed acceptable levels of internal reliability for all six subscales (Positive-Internal alpha = .717, Positive-Personal alpha = .611, and Positive-Situational alpha = .605; Negative-Internal alpha = .732, Negative-Personal alpha = .629, and Negative-Situational alpha = .755; mean alpha = .675). These reliability statistics are substantially superior to those reported for the internality subscales of the ASQ (Cronbach's alphas for negative internality between .44 and .52, for positive internality between .39 and .40; Reivich, 1995; p.27).

The two cognitive bias scores, Externalizing Bias (EB) and Personalizing Bias

(PB), were calculated for these participants. Mean EB was 2.32, SD = 4.56, and mean PB was .54, SD = .26. Reliability statistics (Cronbach's alpha), revealed acceptable levels of internal reliability for these two composite scores: EB alpha = .719, PB alpha = .761. EB and PB were unrelated to one another, Spearman's r = -.136, p = .208.

10.3.2 Validity

The validity of the IPSAQ was assessed by examining its relationship with the three other questionnaires employed. EB correlated significantly with the negative internality subscale of the ASQ, Spearman's r = .387, p < .002, and also with the bias scores calculated by subtracting ASQ internality scores for positive events from those for negative events, Spearman's r = .427, p < .0005. The ASQ internality subscales and IPSAQ EB scores therefore appear to be tapping similar constructs of bias in self-blame.

Participants were allocated into three groups on the basis of their scores on the IPSAQ. Group 1 (internal attributors) comprised 27 participants who had EB scores less than or equal to 0, Group 2 (personal attributors) comprised 33 participants who had EB scores greater than 0 and PB scores greater than .5, while Group 3 (situational attributors) comprised 28 participants who had EB scores greater than 0 and PB scores less than or equal to .5. A group-comparison analysis was used to examine the relationship between IPSAQ scores and other measures, rather than a correlational approach, because qualitatively different predictions are made for individuals whose predominant choice of attributions for negative events implicates each of the three attributional loci identified in the questionnaire.

A one-way analysis of variance (ANOVA) conducted on BDI scores with

group-membership as a between-subjects factor, revealed significant differences between the groups, $F(2,85) = 3.98$, $p < .05$. Planned pairwise comparisons (Tukey's HSD) revealed that participants in Group 2, (personal attributors) did not differ significantly from participants in either Group 1, (internal attributors) or Group 3, (situational attributors), but that participants in Group 1 (internal attributors) had significantly higher BDI scores than participants in Group 3, (situational attributors), $p < .05$.

Regression analysis revealed that scores on the PS were strongly predicted by BDI scores, beta = .847, $t = 3.32$, $p < .002$. In order to investigate the possible relationship between attributional style and paranoid ideation, the residual PS scores after controlling for the influence of the BDI were examined. A one-way ANOVA revealed highly significant differences between the groups, $F(2,85) = 33.00$, $p < .0005$, in the levels of residual PS scores. Planned pairwise comparisons (Tukey's HSD) revealed that participants in all three groups differed significantly from each other. Group 3, (situational attributors), had the lowest residual PS scores, significantly lower than either of the other groups, $p < .01$. Participants in Group 2, (personal attributors), had the highest residual PS scores, significantly higher than the other two groups, $p < .01$. Participants in Group 1, (internal attributors), had intermediate residual PS scores, significantly lower than participants in Group 2 (the personal attributors), $p < .01$, but significantly higher than participants in Group 3 (the situational attributors), $p < .01$.

10.4 Discussion

Attributional style remains a central concept in cognitive models of psychopathology (Brewin, 1988; Buchanan & Seligman, 1995). However, the most

appropriate approach to the classification of causal attributions remains a matter of controversy (Reivich, 1995). In particular the nature of the internality dimension as identified and objectively defined in the ASQ (Peterson, et al., 1982) has been criticized for its low level of internal reliability (Rehm, 1988; Reivich, 1995; Tennen & Herzberger, 1985) and alternative classificatory systems have been suggested (Stratton, Heard, Hanks, Munton, Brewin, & Davidson, 1986; White, 1991). The IPSAQ was designed to measure the degree to which individuals generate internal, personal or situational causal attributions for both positive and negative events.

The IPSAQ subscales had acceptable levels of reliability which were superior to those of the internality subscales of the widely used ASQ. This finding indicates that a three-way discrimination of causal judgements into internal, personal and situational may be more appropriate than a single internal/external dimension.

The validity of this attributional classification is also supported by the differential associations between the three predominant types of attributions, and other significant variables. Specifically, internal attributions for negative events seemed to be more closely associated with low mood than were personal or situational external attributions. Perhaps more interestingly (once correlations with low mood had been controlled for) paranoid ideation, as measured by the Paranoia Scale (Fenigstein & Vanable, 1992) was associated primarily with external-personal attributions for negative events. Thus internal attributions for negative events may be associated with depressive reactions, personal external attributions for negative events with paranoid ideation, while situational external attributions appear to be psychologically benign.

Chapter 11.

A Second Analogue Study

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A paper based on the material presented in this chapter and Chapter 9 has been submitted for publication as: Kinderman, P., & Bentall, R. P. Self-discrepancies and causal attributions: Analogue studies of hypothesised relationships.

11.1 Background

The IPSAQ was developed (Chapter 10) primarily in response to the observations made about the effects of different types of external causal attributions reported in Chapter 9. External attributions appeared to have different psychological effects according to whether they involved other individuals or purely situational or circumstantial factors.

Criticisms of the attributional models that have informed the reformulated learned helplessness (Abramson, Seligman, & Teasdale, 1978) and hopelessness (Abramson, Metalsky, & Alloy, 1989) theories of depression were included in earlier chapters. Similarly I have already discussed the poor reliability statistics for the internality, stability and globalness subscales of the ASQ (Rehm, 1988; Reivich, 1995; Tennen & Herzberger, 1985).

11.1.1 Replication and extension of the study reported in Chapter 9

This chapter describes a replication and extension of the study reported in Chapter 9, using a modified measure of self-discrepancies and the IPSAQ as a measure of causal attributions. The present study was designed to test directly the hypothesized associations between causal attributions and the self-concept, taking account of the amendments to the conceptualization of causal loci outlined above, and using an idiographic measure of self-discrepancies. As in the study described in Chapter 9, participants' self-discrepancies were elicited both before and after the completion of a measure of causal attributions.

11.1.2 Hypotheses

It was hypothesised that, as before, excessively internal attributions for negative events would be associated with high levels of both self-actual: self-ideal discrepancies and self-actual:other-actual discrepancies. As in Chapter 9, it was predicted that internal attributions would be associated with increased levels of self-actual:self-ideal discrepancies at time 2, and increased self-actual:self-other discrepancies at that time. Personal-external attributions (identifying others as the source of blame) were predicted to result in increased self-actual:other-actual discrepancies at time 2, but reduced self-actual:self-ideal discrepancies. Causal attributions to situational-external loci were predicted to result in no change to either self-actual:self-ideal or self-actual:other-actual discrepancies.

11.2 Method

11.2.1 Participants

Participants were 81 undergraduate students of the professions allied to medicine (N=38) and engineering (N=43). Participants were approached for their cooperation, which was voluntary, in class-groups. No incentive was offered for participation. None of the participants, when questioned after the experiment, indicated that they had understood the hypothesis under test. The mean age of the group was 20.83 years; SD = 3.81, range 18 to 45, 45 were men and 36 were women.

11.2.2 Materials

11.2.2.1 Selves Questionnaire (modified)

The Selves Questionnaire (modified) (SQm) was designed specifically for this experiment to measure self-discrepancies with the flexibility necessary to investigate the experimental hypotheses. It closely followed the original Selves Questionnaire (Higgins, 1987), and consisted of three sections. The first required participants to generate up to ten words that described them as they actually are, the second required participants to generate ten words that described their ideal selves (the kind of person they would ideally like to be like). The third section required participants to generate ten words that they believed other people would use to describe them. Following the generation of each list of words, participants were required to provide a numerical rating from 1 to 10 depending on to what degree they felt they actually possessed, or would like to possess, or other people would say they possessed, each characteristic. These ratings were labelled as; 1 = slightly or a very little, 5 = moderately or quite a lot and 10 = extremely or very much; intermediate ratings were listed, but not labelled. These numerical ratings are the only difference between the format of the original Selves Questionnaire and the SQm, as the original version used a four-point numerical scale. The numerical ratings were adapted to provide the participants with a wider range of choices, with the expectation that this would allow for more change in ratings between administrations (see Appendix 14).

The SQm results in two numerical scores; Self-actual:self-ideal consistency / discrepancy and Self-actual:other-actual consistency / discrepancy. The scoring of the SQm again very closely matched the scoring of the original Selves Questionnaire (Scott & O'Hara, 1993). Modifications in the scoring system were related to the increased range of numerical ratings. Synonymous matches (scoring+2) were recorded where a word (or its synonym) was used in two domains, and the numerical

ratings were identical. Partial matches (scoring+1) were recorded where a word (or its synonym) was used in two domains, and the numerical ratings differed by only one point (i.e. intelligent rated 8 in one domain and rated 9 in the other). When a word (or its synonym) was used in two domains, but the numerical ratings differed by two points, a score of 0 was recorded. Synonymous mismatches (scoring -1) were recorded where a word (or its synonym) was used in two domains, but the numerical ratings differed by three or more points (i.e. intelligent rated 3 in one domain and rated 9 in the other). Finally, mismatches (scoring -2) were recorded where a word was used in one domain, but the antonym was used in the other (i.e. stupid in one domain and intelligent in the other). Total consistency / discrepancy scores were calculated by summing these numerical scores for each comparison. Positive scores therefore reflect consistency, whereas negative scores reflect discrepancy between the domains. Reliability of the Selves Questionnaire (in its original and modified forms) is very high, with reliability coefficients above .90 (Kinderman & Bentall, 1996; Scott & O'Hara, 1993). Clearly, the separate analysis of self-actual:self-ideal and self-actual:other-actual discrepancies is a departure from previous use of the Selves Questionnaire.

Two versions of the SQm were constructed, allowing for administration before and after the completion of a measure of causal attribution. The only difference between the two versions lay in the instructions on the cover sheet, where the second version stated that it was the "...second part of the questionnaire seen earlier". Participants were reminded that the questionnaires should be completed in the order in which they were presented.

11.2.2.2 Beck Depression Inventory

The BDI has been comprehensively described in earlier chapters.

11.2.2.3 Internal, Personal and Situational Attributions Questionnaire (Negative items)

The Internal, Personal and Situational Attributions Questionnaire (Negative items) (IPSAQn) was designed to prime schemata involving negative interpersonal scenarios and also to measure the causal attributions made by participants for such events. It was constructed simply by extracting the 16 negative items from the Internal Personal and Situational Attributions Questionnaire (see Chapter 10).

11.2.3 Procedure

It has been suggested (Tourangeau & Rasinski, 1988) that the presentation of depression-related material in questionnaires may prime depressive cognitions. Consequently, it was decided to administer the BDI after the first administration of the SQm, to ensure unbiased completion of this measure, but before the completion of the measure of causal attribution. This represents a modification of the procedure deployed in the study reported in Chapter 9.

Participants were approached in class-groups and asked for their consent. After consent had been given the participants were asked to work through a booklet incorporating the questionnaires in the following order: first SQm, BDI, IPSAQn, and second SQm. I was present at all times to ensure correct completion.

11.3 Results

The mean score on the BDI was 8.23 (SD = 5.11, range 1 to 25). All participants, including 26 participants scoring over 10 on the BDI (the conventional cut-off point for mild depression; Rehm, 1988), were included in the analysis to maximize heterogeneity of attributional styles and self-discrepancies.

Participants' scores on the IPSAQn were used to classify individuals into three groups. Participants who made 8 or more (of the 16 items) attributions to internal causes were allocated to Group I (internal attributors). Participants who made 8 or more attributions to external causes were allocated to one of two other groups. If the majority of these external attributions were made to personal causes, individuals were allocated to Group P (personal-external attributors), while the remainder were allocated to Group S (situational-external attributors). All subsequent statistical analyses were conducted on a group basis. Group I consisted of 24 individuals (15 men and 9 women), Group P consisted of 32 individuals (14 men and 18 women) and Group S consisted of 25 individuals (16 men and 9 women).

A one-way analysis of variance (ANOVA) revealed that the three groups did not differ significantly in age, $F(2,57) = 0.96$, $p = .388$, and the distribution of gender was equitable, Chi squared = 3.00, $p = .224$. ANOVA analysis of the scores on the BDI revealed that the three groups did not differ on this measure, $F(2,78) = 0.04$, $p = .958$.

Self-actual:Self-ideal and Self-actual:Other-actual discrepancy scores were calculated as described above for both administrations of the SQm. Summary SQm scores for the three groups at both times are presented in Table 11.1. Although these scores will be referred to in the context of the term 'self-discrepancy', it is important to note that positive SQm scores indicate consistency between domains.

Table 11.1 Scores on the Selves Questionnaire (modified)

	Group						
	I Internal		P Personal		S Situational		
Discrepancy	M	SD	M	SD	M	SD	F(2,78)
Actual-Ideal (time 1)	1.38	(3.59)	0.38	(3.95)	0.96	(4.33)	
Actual-Ideal (time 2)	0.13	(4.52)	1.13	(3.41)	2.08	(3.59)	
Change	-1.25a	(2.89)	0.75b	(2.90)	1.12b	(3.24)	4.79**
Adjusted change scores following analysis of covariance	-1.10		+0.58		+1.14		4.45*
Self-Other (time 1)	4.17	(4.15)	4.28	(3.80)	3.40	(3.51)	
Self-Other (time 2)	4.17	(4.89)	3.63	(3.56)	5.40	(3.94)	
Change	0.00a	(3.59)	-0.66a	(3.23)	+2.00b	(3.79)	4.12**
Adjusted change scores following analysis of covariance	+0.08		-0.54		+1.81		3.71*

Note: Positive SQm discrepancy scores represent consistency between domains. Also presented are change scores (arithmetic subtraction of discrepancy scores at time 1 from time 2) and adjusted scores following analysis of covariance with scores at time 1. F-ratios relate to analyses of variance and covariance for the change scores; * $p < .05$, ** $p < .0005$. Means having different subscripts differ at $p < .05$ (subscripts a and b), at $p < .01$ (subscripts a and c) and $p < .001$ (subscripts a and d) in planned pairwise comparisons (Tukey's HSD).

At the initial administration of the SQm, there were no significant differences among the three groups in either self-actual:self-ideal, $F(2,78) = 0.45$, $p = .640$, or self-actual:other-actual, $F(2,78) = 0.42$, $p = .661$, discrepancy.

Contrary to previous findings by Strauman and colleagues (Strauman & Higgins, 1988), and the findings reported in Chapter 9, levels of depressed mood, as measured by BDI scores, were not associated either with larger self-actual:self-ideal discrepancies, Spearman $r = 0.096$, $p = .396$, or with larger self-actual:other-actual discrepancies, Spearman $r = 0.122$, $p = .276$.

Because the predictions for the effects of attributions on self-discrepancies were nonlinear (it was predicted that self-actual:self-ideal discrepancies would increase only for Group I, the internal attributors, but also that self-actual:other-actual discrepancies would be increased in Group I and in Group P, the personal-external attributors, but not in Group S, the situational-external) group comparisons were carried out to test for these effects.

A repeated-measures MANOVA was conducted on the two self-discrepancy consistency scores, with group membership (I, P and S) as a between-subjects variable and type of self-discrepancy and time of assessment (before or after administration of the IPSAQn) as within-subject variables. This revealed no significant differences among the groups in terms of overall level of consistency scores, $F(2,78) = 0.30$, $p = .743$. Self-actual:other-actual consistency scores were systematically higher than self-actual:self-ideal scores.

The difference between first and second administrations of the IPSAQn on combined consistency scores was non-significant, $F(1,78) = 1.25$, $p = .267$, but the interaction between domain and time was significant, $F(2,78) = 4.87$, $p < .05$, indicating a differential effect on self-actual:self-ideal and self-actual:other-actual discrepancies. Most important, the overall interaction term was significant, $F(2,78) = 3.57$, $p < .05$, indicating that the consistency scores in the different domains altered

with time differently in the three groups. These effects were therefore investigated using individual repeated-measures analyses of variance.

Analysis of self-actual:self-ideal consistency scores confirmed that there were no overall differences between the groups, $F(2,78) = 0.40$, $p = .675$. For the combined group, there was no overall difference between the two assessment times, $F(1,78) = 0.38$, $p = .541$. Importantly, however, there was a significant interaction term, $F(2,78) = 4.49$, $p < .05$, indicating that the three groups experienced significantly different changes in self-actual:self-ideal consistency scores following administration of the IPSAQn. Tests of simple effect indicated that Group I (internal attributors) experienced a statistically significant increase in the accessibility of self-actual:self-ideal discrepancies, $F(1,78) = 4.16$, $p < .05$. Group P (personal-external attributors) showed no significant change in self-actual:self-ideal discrepancies, $F(1,78) = 2.00$, $p = .162$, while Group S (situational-external attributors) experienced a trend towards reduced accessibility of self-actual:self-ideal discrepancies, $F(1,78) = 3.48$, $p = .066$.

Similar analysis of self-actual:other-actual consistency scores found no overall differences between the groups, $F(2,78) = 0.11$, $p = .895$, and there was no overall difference between the two assessment times, $F(1,78) = 1.29$, $p = .259$. Again, there was a significant interaction term, $F(2,78) = 4.19$, $p < .05$, indicating that the three groups experienced significantly different changes in self-actual:other-actual consistency scores following administration of the IPSAQn. Tests of simple effect indicated that Group I (internal attributors) experienced no change in self-actual:other-actual discrepancies, mean scores were identical at the two times of administration. Group P (personal-external attributors) showed no significant change in self-actual:other-actual discrepancies, $F(1,78) = 1.12$, $p = .294$, while Group S (situational-external attributors) experienced a significant reduction in the accessibility of self-actual:other-actual discrepancies, $F(1,78) = 8.10$, $p = .006$.

In keeping with statistical recommendations for the analysis of change scores (Cook & Campbell, 1979) and to ensure compatibility with the study reported in Chapter 9, analyses of covariance (ANCOVA) were also used to assess the impact of attributions on changes in self-discrepancies, with group membership as a between-subjects variable and self-discrepancies at time 1 as covariates. For each subject, consistency scores were calculated in the manner described above at time 1 (before administration of the IPSAQn) and subtracted from the same scores calculated at time 2. Summary statistics for these difference scores are also presented in Table 1.

Analysis of covariance (ANCOVA), with self-actual:self-ideal consistency score at time 1 as a covariate, revealed trivial differences from the preceding analysis. The self-actual:self-ideal change scores differed between the groups, $F(2,77) = 4.45$, $p < .05$, with Group S showing a decreased accessibility of self-actual:self-ideal discrepancies, Group I an increased accessibility of self-actual:self-ideal discrepancies, with Group P in an intermediate position. A similar ANCOVA on self-actual:other-actual change scores also revealed a statistically significant difference between the groups, $F(2,77) = 3.71$, $p < .05$, with Group P showing an increased accessibility of self-actual:other-actual discrepancies relative to Groups I and S.

11.4 Discussion

This study was designed to examine further a proposed relationship between self-representations and explanatory style suggested in the model of paranoid ideation presented in Chapter 6, and further supports the idea advanced in Chapters 9 and 10 that different external attributional loci have importantly different psychosocial consequences. As predicted, it was found that internal attributions for hypothetical negative social events were associated with increases in self-actual:self-ideal

discrepancies, in comparison with personal-external and situational-external attributions. This finding replicates similar findings presented in Chapter 9.

Again replicating previous findings (see Chapter 9), internal attributions were also associated with no changes in self-actual:other-actual discrepancies. In the present study, attributions made to personal-external loci (identifying other individuals as the cause of your problems) were associated with increases in the accessibility of self-actual:other-actual discrepancies, in comparison with situational-external attributions. In simple terms this suggests that internal (self-blaming) attributions serve to open self-actual:self-ideal discrepancies; personal-external (other-blaming) attributions preserve self-actual:self-ideal discrepancies, but open self-actual:other-actual discrepancies; while situational-external attributions appear relatively benign, limiting both forms of discrepancy. These findings are, therefore, consistent with attributional accounts of depression (Brewin, 1986) and paranoia (Chapter 6). The observation (also made in Chapter 9) that internal attributions were associated with increases in self-actual:other-actual discrepancies, is consistent with the observation that psychotically depressed patients often delusionally believe that others view them in a negative light (Gelder, Gath, & Mayou, 1983). These findings, therefore, provide considerable support for models of psychopathology that link the self-concept, specifically self-discrepancies, with attributional processes.

Besides clarifying the role of causal attributions in psychopathology, the present findings also help to clarify the nature of causal loci in attributional processes, and may help explain previous measurement difficulties. In the present study, internal attributions, personal-external attributions and situational-external attributions exhibited different psychological effects. This is consistent with preliminary research using the measures employed here and reported elsewhere in this thesis, and may explain difficulties in interpreting data gathered using measures of attributional style which fail to discriminate between the two forms of external attribution. If these

differences prove robust, considerable progress may be made in understanding the role of attributional processes in self and social perception.

In the present study there was no significant association between self-actual:self-ideal discrepancies at time 1 and future internal attributions. This is an important difference to the study reported in Chapter 9. One possible explanation for this difference lies in the different methodologies employed. In the previous study initial self-discrepancies were recorded after administration of the BDI. As mentioned above, it was decided in the present study to administer the BDI after the first assessment of self-discrepancies. It is consistent with the suggestions of (Tourangeau & Rasinski, 1988) that assessment of self-discrepancies were assessed unbiased by the priming of depressive cognitions. As such, the heightened accessibility of self-actual:self-ideal discrepancies following internal attributions and self-actual:other-actual discrepancies following external-personal attributions is more striking.

These findings support a slight modification of the model of paranoid ideation presented in Chapter 6, but one that strengthens the model. Specifically, we must now hypothesize that when patients experiencing delusions of persecution are faced with events that threaten to activate discrepancies between self-representations and self-guides, they tend to attribute such negative events to the actions of other people. This is subtly, but importantly different from the reaction of non-deluded individuals, who appear also to make external attributions in such circumstances, but to more benign, situational, factors. Personal external attributions, one must assume, serve to maintain consistency between self-perceptions and self-guides. The effect of such attributions in contributing to negative perceptions of the intentions of others becomes, therefore, clearer.

Such findings have several consequences worthy of discussion. In its original form, the model of paranoid ideation (Chapter 6) hypothesized links between internal

(self-blaming) attributions and self-actual:self-ideal discrepancies were clear. It was less obvious how external attributions would open discrepancies with other people's representations of the self. The present findings clarify this issue, as they suggest that external attributions that implicate other people are prevalent in paranoia. Clearly such attributions for negative social interactions should be hypothesised to be likely to open self-other discrepancies, the idea that other people see one in a negative light.

In addition, the results of this study have implications for our understanding of causal attributions more generally. In particular, certain weaknesses in the internal-external dimension as suggested in Peterson et al's (1982) Attributional Style Questionnaire and currently employed widely (Buchanan & Seligman, 1995) have been highlighted. The differences observed between clinical groups illustrates the validity of a new categorization of the locus of causal attribution. The deployment of such a categorization may have important consequences. One can speculate, for instance, about the attributions for negative events made by persons with a history of violent crime or assaultative behaviour, or the attributions made by persons suffering from social anxiety.

When compared to the ASQ, the IPSAQ, and therefore the IPSAQn is relatively untested. Although it appears to measure something valid in clinical groups, it has been much less frequently used than the ASQ. However, it does seem to have high levels of psychometric reliability, and behaves, in non-patient samples, in a predictable manner (see Chapter 10). The concept behind the development of the IPSAQ, moreover, has been supported both in general reviews of the field of causal attributions (White, 1991) and in experimental studies (see Chapter 12).

A number of limitations of the present study must be mentioned. This study involved only non-patient participants, undergraduate students. Clearly, extrapolating these findings to inform understanding of psychological disorders, particularly depression and paranoia, should be done only with caution. Similarly, the

experimental intervention, the completion of the SQm - a simple questionnaire - may be only marginally related to real-life social situations in which individuals make attributions for negative experiences and alter the accessibility of self discrepancies. Again, extrapolations of the present findings to must be made only with caution. Most importantly, although the self-concept is believed to be of central importance in many psychological problems (Wylie, 1979), the attributional and self-concept processes studied here are likely to be accompanied by, and interact with, other cognitive and affective processes.

In the present study subjects were not allocated randomly to experimental groups in a strict experimental design. The determination of causality in the relationship between attributional processes and self-representation is, therefore, difficult. It is highly likely that other variables were related to both choice of attributions and self-representations. However, the design of controlled experiments into the consequences of attributions may prove difficult. Psychological reactance (Watts, Powell, & Austin, 1973) and compensatory self-enhancement (Greenberg & Pyszczynski, 1985) may confound experimental designs in which individuals are required to make particular types of attributions. Many psychological processes are hypothesized to influence self-representations. Many of these are thought of as active strategies preserving the self concept. The actions of these processes may result in unpredicted, and uncontrolled, changes in the self-concept, or unexplained absences of effect.

Chapter 12.

Internal, Personal, and Situational Attributions in Paranoia

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A paper based on the material presented in this chapter has been accepted for publication as: Kinderman, P. & Bentall, R. P. (in press) Causal attributions in paranoia: Internal, personal and situational attributions for negative events. *Journal of Abnormal Psychology.*

12.1 Introduction

Previous research with psychiatric patients has implicated abnormal attributional processes in a range of disorders, particularly depression and paranoia (see Chapter 5). This chapter reports a study of causal attributions in depressed and paranoid patients, using the IPSAQ.

Attributional abnormalities in depression and paranoia were comprehensively described in previous chapters, particularly Chapter 5. That discussion also addressed many limitations of the current attributional typology, and Chapter 10 described the development of the IPSAQ. In that measure the difficulty inherent in the ASQ's internal-external dimension is addressed. The IPSAQ provides separate measures of Externalizing Bias (the relative tendency to attribute positive and negative events to causes which are not internal) and Personalizing Bias (the tendency to make external attributions for negative events that implicate persons as opposed to situations). As reported in Chapter 10, BDI scores of non-patient participants correlate negatively with Externalizing Bias (that is, that more depressed subjects made more internal attributions for negative events than for positive events) and non-patient participants' scores on Fenigstein and Vanable's (Fenigstein & Vanable, 1992) Paranoia Scale correlated positively with Personalizing Bias scores.

12.1.1 Hypotheses

Clinical paranoia is characterized by abnormal concerns about the perceived malevolent intentions of others. Paranoid patients' abnormally external attributions for negative events on the ASQ have been described in previous chapters. It seems likely that the external attributions made by paranoid patients for negative events will be

predominantly personal and not situational.

It was hypothesized that both paranoid and non-patient participants would show positive externalizing bias scores, reflecting strong self-protective attributional biases. It was further hypothesised that both depressed and non-patient participants would show low personalizing bias scores, reflecting a small proportion of personal-external attributions for negative events. Only paranoid patients were hypothesised to possess a combination of high externalizing and personalizing bias scores.

These predictions were tested in the present study.

12.2 Method

12.2.1 Participants

The experimental group (Group P) was composed of 20 people currently receiving treatment for psychiatric conditions, and suffering from persecutory delusions. Thirteen were men and seven women. Eighteen patients met the DSM-IV diagnostic criteria for schizophrenia and two for delusional disorder. In each case, mention was made in medical records of the presence of delusional beliefs, and staff confirmed that the patient was currently experiencing delusions. Interviews were conducted by the first author to ensure that patients were currently experiencing persecutory delusions using the structured clinical interview of the Positive and Negative Syndrome Scale (PANSS: Kay & Opler, 1987). This is a 30-item rating instrument developed to evaluate the positive, negative and general symptoms of schizophrenia, yielding scores for individual symptoms and for three subscales - positive symptomatology, negative symptomatology and general psychopathology. The assessment is based on a semi-structured clinical interview in addition to other sources of information such as data obtained from case notes and consultations with

care staff. The PANSS is a reliable and valid means of assessing psychotic symptomatology (Kay, Opler, & Fiszbein, 1988; Kay, Opler, & Lindenmayer, 1989).

The mean positive symptomatology score of Group P in the present study was 19.20 (SD= 4.76, range 12 to 27), mean negative symptomatology score was 16.60 (SD= 6.13, range 7 to 30) and mean general psychopathology score was 31.35 (SD= 9.17, range 18 to 50). These scores are comparable with those obtained by Kay, Opler and Fiszbein (1986) from a sample of 240 medicated, hospitalized, patients with a diagnosis of schizophrenia.

The presence or absence of auditory hallucinations was elicited as part of the PANSS interviewing method and recorded. Four patients were currently experiencing auditory hallucinations (in each case these included at least some content of a negative nature) while 16 were not.

Fourteen people in this group were currently in-patients (with a mean length of hospital stay of 7.78 weeks; SD= 8.38 weeks, range 1 to 24 weeks) and six were out-patients. All except one patient in this group had been prescribed neuroleptic medication. One patient (with a diagnosis of delusional disorder) had not been prescribed medication, in addition one patient, with a diagnosis of schizophrenia, had been prescribed neuroleptic medication, but was not taking it.

The mean age of this group was 35.25 years (SD= 8.97 years, range 19 to 58 years). In common with most other investigations in this area (discussed throughout this thesis) a significant level of depressed mood was observed in this group. Nineteen of the 20 participants in Group P completed the BDI, with a mean score of 18.89 (SD= 11.39, range 0 to 34). The mean elapsed time since the first psychiatric consultation mentioned in the medical records was 4.08 years (SD= 3.76 years; range from 1 week to 12 years).

To control for the levels of depressed mood, hospitalization status and other concomitants of psychiatric care observed for Group P, a psychiatric comparison

group (Group D) was recruited. The 20 patients in this group were selected from the same clinics and hospitals to match approximately the experimental group in terms of age and gender. All patients in this group suffered a clinically significant level of depressed mood, and all met the DSM-IV criteria for Major Depressive Episode. No participant reported experiencing psychotic symptoms and case notes were examined to ensure no differential diagnoses involving psychosis had been suggested. This group consisted of 15 men and five women, with a mean age of 35.70 years (SD= 14.29 years, range 18 to 64). Twelve people in this group were currently in-patients (with a mean length of hospital stay of 4.67 weeks; SD= 6.37 weeks, range 1 to 24 weeks), eight were out-patients. Seventeen patients in this group were currently receiving antidepressant medication. All participants in Group D completed the BDI, with a mean score of 29.30 (SD= 13.50, range 11 to 49). The mean elapsed time since the first psychiatric consultation mentioned in the medical records was 4.13 years (SD= 7.57 years, range from 1 week to 25 years).

A non-patient control group (Group C) was recruited via informal contacts. Again participants were selected to match the previous groups in terms of age and gender. To control for levels of depressed mood, participants were only included in this group if their BDI scores were less than a cut-off of 10 (Rehm, 1988). This resulted in a group of 15 men and five women whose mean age was 30.50 years (SD= 15.94 years, range 18 to 63 years). All participants completed the BDI, with a mean score of 3.70 (SD= 2.43, range 0 to 9). None of the participants, when debriefed after the study, indicated that they had prior understanding of the hypotheses under test.

12.2.2 Materials

12.2.2.1 The Beck Depression Inventory

The BDI has been extensively described in previous chapters.

12.2.2.2 Internal, Personal and Situational Attributions Questionnaire

The IPSAQ has been extensively described in previous chapters.

12.2.2.3 Paranoia Scale

The Paranoia Scale (Fenigstein & Vanable, 1992) has been described in Chapter 10. In the present study all participants in Group C, 18 of the 20 participants in Group D and all participants in Group P completed the PS.

12.3 Results

12.3.1 Demographic Variables

A one-way analysis of variance (ANOVA) revealed that the three groups did not differ significantly in age, $F(2,57) = 0.92$, $p = .403$, and the distribution of gender was equitable, Chi squared = 0.66, $p = .720$. Comparison of the clinical data for the two patient groups revealed no significant differences for the length of time since first diagnosis, $F(1,38) = 0.0006$, $p = .980$, for the length of in-patient stay for the hospitalized patients, $F(1,25) = 1.11$, $p = .113$, or for the relative proportion of

participants who were in-patients, Chi squared = 1.10, $\underline{p}$ = .294.

12.3.2 Internal, Personal and Situational Attributions Questionnaire

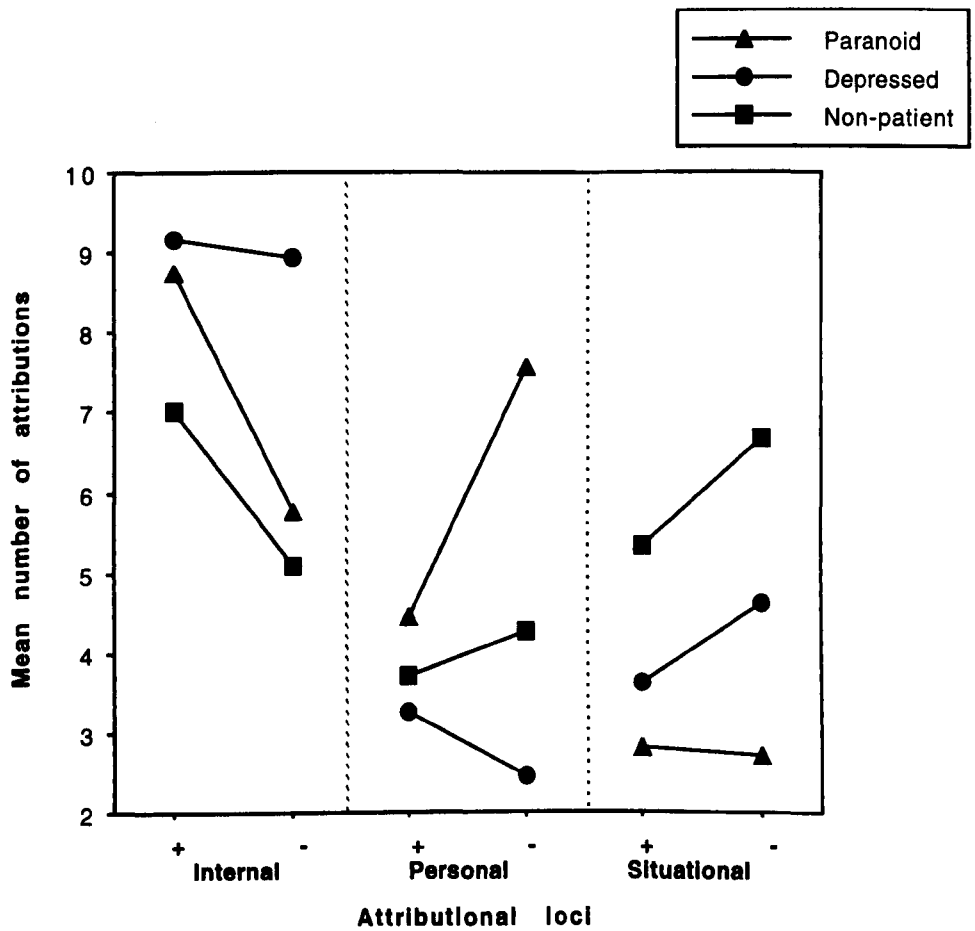
Table 12.1 shows the mean number of attributions made to internal, external-personal and external-situational causes for both positive and negative events for the three participant groups; these data are also shown graphically in Figure 12.1.

Table 12.1 Scores on the IPSAQ

	Group						
	Control		Depressed		Paranoid		
IPSAQ subscale scores	M	SD	M	SD	M	SD	F(2,57)
Positive Events							
Internal	7.00	2.51	9.15	3.23	8.75	2.81	
Personal	3.70	1.98	3.25	2.51	4.45	2.19	
Situational	5.30	2.25	3.60	2.62	2.80	2.24	
Negative Events							
Internal	5.10_c	2.15	8.95_a	3.90	5.75_b	2.63	
Personal	4.25_a	2.73	2.45_a	2.42	7.55_{bd}	2.93	
Situational	6.65	3.31	4.60	3.22	2.70	2.77	
Externalizing Bias	1.90	2.85	0.20_a	3.86	3.00_b	3.58	3.50*
Personalizing Bias	0.40_c	0.26	0.32_c	0.26	0.75_a	0.24	16.55**

Note. IPSAQ subscale scores represent the mean number of each type of attribution made for positive and negative events, calculation of externalizing bias and personalizing bias is explained in the text. F-ratios relate to analyses of variance for the derivative scores; * $p < .05$, ** $p < .0005$. Means having different subscripts differ at $p < .05$ (subscripts a and b), at $p < .01$ (subscripts a and c) and $p < .001$ (subscripts a and d) in post hoc Scheffé tests (subscale scores) and planned pairwise comparisons (derivative scores).

Figure 12.1 IPSAQ subscale scores represent the mean number of internal, external-personal and external-situational attributions made for both positive and negative events by the three participant groups.



A full factorial multivariate analysis of variance (MANOVA) was conducted on these data, with group membership as a between-subjects variable and type of attribution (internal, personal and situational) and type of event (positive and negative) as within-subject variables. This revealed significant differences in the overall proportions of internal, external-personal and external-situational attributions made for all events, $F(2,114) = 26.67$, $p < .0005$. The group x choice of attribution interaction term was significant, $F(4,114) = 9.86$, $p < .0005$, indicating that the three groups used the attributional loci differently. A significant interaction term for choice of attribution x type of event, $F(4,114) = 9.00$, $p < .0005$, indicated there was a significant difference between the attributions made for positive and negative events. Finally, and most important, the overall interaction term was significant, $F(4,114) = 4.47$, $p < .005$, indicating that the pattern of attributions for the different type of events differed between the groups.

Following the significant main interaction term, separate analyses of variance were conducted for the three attributional loci. A two-way analysis of variance was conducted on the internal attributions made by participants in the three groups (between-subjects factor) for positive and negative events (within-subject factor). This revealed a significant difference between the groups in the total number of attributions made, for both positive and negative events, to internal loci, $F(2,57) = 8.17$, $p < .001$. Planned pairwise comparisons (Tukey's Honestly Significant Difference; HSD) revealed that Group D made more internal attributions than both groups P, $p < .01$, and C, $p < .01$, while Group P made more internal attributions than Group C, $p < .01$. Overall, for all three groups, more internal attributions were made for positive events than for negative events, $F(1,57) = 14.51$, $p < .0005$. Finally, the interaction term was significant, $F(2,57) = 3.33$, $p < .05$, indicating that the relative number of internal attributions made for positive and negative events differed for the three groups. Tests of simple effect revealed that the groups differed in the number of

attributions made to internal loci for both positive, $F(2,57) = 3.18$, $p < .05$, and negative events, $F(2,57) = 9.52$, $p < .0005$. Post-hoc Scheffé tests did not reach statistical significance for internal attributions for positive events but revealed that Group D made more internal attributions for negative events than groups P, $F(2,57) = 3.83$, $p < .05$, and C, $F(2,57) = 5.54$, $p < .01$, while groups P and C did not differ, $F(2,57) = 0.16$, $p = .854$. Tests of simple effect also revealed that both Group C, $F(1,57) = 6.04$, $p < .05$, and Group P, $F(1,57) = 15.07$, $p < .0005$, made more internal attributions for positive than negative events, but that Group D did not, $F(1,57) = 0.067$, $p = .797$.

A similar two-way analysis of variance revealed a significant difference between the groups in the total number of attributions made, for both positive and negative events, to personal loci, $F(2,57) = 12.89$, $p < .0005$. Planned pairwise comparisons (Tukey's HSD) revealed that Group P made more personal attributions than both groups C, $p < .01$, and D, $p < .01$, while groups D and C did not differ significantly. Overall, for all three groups, more personal attributions were made for negative events than for positive events, $F(1,57) = 6.17$, $p < .05$. Finally, the interaction term was significant, $F(2,57) = 8.94$, $p < .0005$, indicating that the relative number of personal attributions made for positive and negative events differed for the three groups. Tests of simple effect revealed that the groups differed in the number of attributions made to personal loci for negative events, $F(2,57) = 18.35$, $p < .005$, but did not differ in the number of attributions made to personal loci for positive events, $F(2,57) = 1.47$, $p = .238$. Post-hoc Scheffé tests were not, therefore, conducted on the attributions made for positive events but revealed that Group P made more external-personal attributions for negative events than groups C, $F(2,57) = 4.98$, $p < .05$, and D, $F(2,57) = 11.89$, $p < .00005$, while groups C and D did not differ, $F(2,57) = 1.48$, $p = .236$. Tests of simple effect also revealed that Group P, $F(1,57) = 21.91$, $p < .005$, made more personal attributions for negative events than for

positive events, while groups C, $F(1,57) = 0.69$, $p = .410$, and D, $F(1,57) = 1.46$, $p = .232$, did not.

Finally, a two-way analysis of variance was conducted on situational attributions for positive and negative events across the groups. This revealed a significant difference between the groups in the total number of attributions made, for both positive and negative events, to situational loci, $F(2,57) = 9.40$, $p < .0005$. Planned pairwise comparisons (Tukey's HSD) revealed that Group C made more situational attributions than both groups P, $p < .01$, and D, $p < .05$, while groups P and D did not differ significantly. Overall, for all three groups, more situational attributions were made for negative events than for positive events, $F(1,57) = 4.06$, $p < .05$. Finally, the interaction term was not significant, $F(2,57) = 1.38$, $p = .261$, indicating that the relative number of situational attributions did not vary across the groups.

Further clarity to these findings was sought by using the derivative scores; Externalizing Bias (EB) and Personalizing Bias (PB). These data are also presented in Table 1. A one-way analysis of variance conducted on EB scores revealed a significant difference between the groups, $F(2,57) = 3.50$, $p < .05$. Planned pairwise comparisons (Tukey's HSD) indicated that Group P had higher EB scores than Group D, $p < .05$, with Group C in an intermediate position, significantly different from neither of the other two groups. A one-way analysis of variance conducted on PB scores also revealed a significant difference between the groups, $F(2,57) = 16.55$, $p < .0005$. Planned pairwise comparisons (Tukey's HSD) indicated that Group P had higher PB scores than both Group C, $p < .01$ and Group D, $p < .01$. Groups C and D did not differ significantly.

12.3.3 Measures of Depressed mood and Paranoid ideation

A one-way ANOVA on subjects' BDI scores revealed that all three groups differed on this measure, $F(2,56) = 30.86$, $p < .0005$. Planned pairwise comparisons (Tukey's HSD) revealed that both groups D, $p < .01$, and P, $p < .01$, had significantly higher mean levels of depressed mood than Group C. Group D also a higher mean level of depressed mood than Group P, $p < .01$. Regression analysis revealed that BDI scores were significant predictors of the number of internal attributions for negative events, $\beta = 0.105$, $t=3.81$, $p < .0006$, and EB scores, $\beta = -0.076$, $t = -2.43$, $p < .05$, but were unrelated to PB scores, $\beta = 0.001$, $t = -0.51$, $p = 0.616$. EB and PB scores were unrelated to one another $\beta = 0.581$, $t = 0.38$, $p = 0.707$.

The mean score on the Paranoia Scale for Group C was 34.75 ($SD = 8.87$, range 24 to 49), for Group D was 57.72 ($SD = 19.37$, range 24 to 81), and for Group P was 60.40 ($SD = 20.59$, range 18 to 100). A one-way ANOVA conducted on these scores revealed a significant difference between the groups, $F(2,57) = 13.24$, $p < .0005$. Planned pairwise comparisons (Tukey's HSD) revealed that Groups P, $p < .01$, and D, $p < .01$, both had higher scores than Group C, but that the scores of groups P and D did not differ significantly.

Regression analysis revealed that scores on the BDI were strongly predictive of PS scores, $\beta = 0.685$, $t = 4.16$, $p < .0005$. This is consistent with previous research outlined in Chapter 10. When the residuals of this regression, the components of Paranoia Scale scores not attributable to the BDI scores, were analyzed, there was a non-significantly higher mean residual score for Group P over group D, $F(1,35) = 2.78$, $p = .09$. Regression analysis revealed that PS scores did not significantly predict either EB scores, $\beta = -0.033$, $t = -1.38$, $p = .174$, or PB scores, $\beta = 0.002$, $t = 1.10$, $p = .277$.

12.4 Discussion

The results obtained from depressed patients are broadly consistent with previous investigations carried out by investigators who have tested the revised learned helplessness model using Peterson and Seligman's bipolar internality construct (see Chapter 5). The depressed patients made a greater number of internal causal attributions for negative events than both the paranoid and normal groups and, consistent with some previous studies with the ASQ, were in fact even-handed, blaming themselves equally for negative and positive outcomes. In contrast, both the paranoid and normal subjects showed evidence of a robust self-serving bias, attributing positive events more often to internal causes than negative events.

This self-serving bias in the paranoid patients is consistent with previous studies employing the ASQ or experimental measures, and with the model of the cognitive processes involved in paranoid ideation outlined in Chapter 6. However, it is notable that in this study employing the IPSAQ, the self-serving bias evidenced by the normal subjects, as judged only from their internality subscale scores, was comparable to that of the paranoid patients. In previous studies with the ASQ, a greater self-serving bias has been consistently found with paranoid in comparison with normal subjects (see Chapter 5). It seems likely that this difference reflects the availability to subjects of three attributional loci in the present study.

Consistent with this account, subjects' choices of non-internal attributions revealed important differences between the paranoid and normal groups. Whereas the self-serving bias in the normal subjects was accounted for by their preference for situational attributions for negative events, the paranoid patients showed a strong preference for making personal attributions for such events. In fact, the paranoid patients showed a marked reluctance to make situational attributions for either positive or negative events. These findings indicate that some important modifications are

required to the model of persecutory delusions proposed in Chapter 6. Specifically, we must now hypothesize that when patients experiencing delusions of persecution are faced with events that threaten to activate discrepancies between self-representations and self-guides, they tend to attribute such negative events to the actions of other people and that it is only when such attributions are made that discrepancies between self-representations and the believed views of others about the self are activated. In the case of the normal subjects, discrepancies between self-representations and self-guides are avoided without activation of discrepancies between self-representations and the believed views of others about the self because the attributions made for negative events do not implicate other people. This employment of situational attributions for negative events (e.g. "I was late because the traffic was terrible") is the essence of effective excuse making, and is psychologically benign.

12.4.1 Origins of paranoid attributions

Earlier chapters included speculation about possible aetiological routes that might lead to the existence of a simple externalizing attributional bias in patients suffering from paranoia. There is known to be a significant correlation between the attributional style of children and that of their parents (Hammen, 1991; Hoffart & Torgesen, 1991; Seligman, Peterson, Kaslow, Tanenbaum, Alloy, & Abramson, 1984). It also appears that general familial emotionality (Valone, Norton, Goldstein, & Doane, 1983), parental criticism (Brown, Birley, & Wing, 1972; Leff & Vaughn, 1980) and in particular patient-directed blame (Barrowclough, Johnston, & Tarrier, 1994; Brewin, MacCarthy, Duda, & Vaughn, 1991) may play a role, as exposure to such stimuli is likely to reinforce an attributional defence against self-discrepancies, and is known to be predictive of relapse in psychotic patients. With regard more

specifically to the personal nature of the paranoid attributions observed in the present study, speculation about aetiological pathways must be very tentative. However, it is possible that the present findings can be accounted for by assuming that individuals vulnerable to paranoia have a specific difficulty in generating situational attributions. In order to generate such attributions for the negative behaviour of another individual towards the self (e.g. "Tom is rude to me because he has had a bad day") it is necessary to attribute intentional states to that individual (that is, it is necessary to attempt to see the world from Tom's point of view). In the field of autism it is widely accepted that there exist neurological deficits specific to the processing of information concerning the thoughts of other people (Leslie, 1991), and Frith (1994) has recently suggested that paranoid patients may also have difficulty understanding the intentional states of other people. This hypothesis is tested in the study reported in Chapter 14.

The observations made in this study have implications for our understanding of causal attributions in general. Certain weaknesses in the internal-external dimension assessed by the Attributional Style Questionnaire, which continues to be widely employed in psychopathology research (Buchanan & Seligman, 1995) have been highlighted and an alternative typology proposed. It is possible that this typology will have implications for other kinds of abnormal states and behaviours in which attributions may play a causal role, for example social anxiety (Mineka, Pury, & Luten, 1995) or assaultative behaviour (Betancourt & Blair, 1992; Holtzworth & Hutchinson, 1993; Holtzworth, Jacobson, Fehrenbach, & Fruzzetti, 1992; Hunter, Stringer, & Watson, 1991; Overholser & Moll, 1990). In addition, attributional processes are likely to be related to other cognitive processes, particularly those involved in self-representation and the representation of the mental states of others.

14.4.2 Limitations

Some limitations of the present study must be acknowledged. In particular, when compared to the ASQ, the IPSAQ is relatively untested. However, it does seem to have high levels of psychometric reliability (see Chapter 10) and the present findings indicate that it discriminates effectively between different clinical groups. A more telling criticism of the analysis of paranoia offered in this paper is that it is descriptive rather than explanatory. However, a descriptive account of a psychiatric disorder may be of value if it is empirically testable, if it makes theoretical links between psychopathology and research into normal human functioning, if it points to aetiological factors, and if it suggests novel psychological interventions. The account of paranoia offered in this paper clearly meets the first three of these criteria.

Chapter 13.

Attributional Therapy for Paranoia: A case study

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A paper based on the material presented in this chapter has been accepted for publication in Behavioural and Cognitive Psychotherapy as: Kinderman, P., & Bentall, R. P. (in press). Attributional Therapy for Paranoia: A case study. *Behavioural and Cognitive Psychotherapy.*

13.1 Background

The material presented in previous chapters suggests that defensive, personal-external attributions for negative or ambiguous events serve to protect the self-concept but contribute to paranoia.

The case study reported in this chapter serves two aims. The role of such attributions are described in detail for one individual. Secondly, a therapeutic strategy designed to allow a patient suffering from persecutory delusions to re-attribute negative life experiences to situational causes rather than to a conspiracy directed towards himself is described. It was hypothesized that no resistance was encountered from the patient because no attempt was made to re-attribute negative events to internal causes. A reduction in paranoid ideation, which was maintained at follow-up, was accompanied by changes on formal measures of attributions.

13.1.1 Dangers of addressing defensive attributional biases

The abnormal attributions of paranoid patients present a dilemma for the cognitive-behaviour therapist. If these attributions are defensive as hypothesized, the reattribution of negative events to the self, which would seem necessary to bring about a reduction in paranoid ideation, is likely to lead to the activation of discrepancies between self-perceptions and self-ideals. This in turn may lead to depression or, more likely, to intense resistance by the patient. A possible way of resolving this dilemma would be to encourage paranoid patients to attribute negative experiences to causes which are not self-blaming but which do not serve to open discrepancies between the patients' view of self and the believed views of other people.

In fact, the research outlined above, de-constructing the external pole of the

traditional internal-external dimension on the ASQ, serves to free the therapist from this dilemma. Whereas attribution of negative events to the personal focus is likely to lead to discrepancies between self-perceptions and beliefs about others' perception of the self, the attribution of negative events to situational factors (as in everyday excuse-making) seems unlikely to do so. Thus encouraging paranoid patients to make situational attributions should allow them to avoid the paranoid ideation associated with the personal focus but at the same time allow them to avoid the negative feelings about the self associated with the internal focus.

On the basis of this theoretical analysis, a therapeutic strategy was developed for individuals with paranoid ideation. This is compatible with other reality testing (Chadwick & Lowe, 1990) and cognitive restructuring methods (Garety, Kuipers, Fowler, Chamberlain, & Dunn, 1994; Kingdon, Turkington, & John, 1994; Tarrier, Beckett, Harwood, Baker, Yusupoff, & Ugarteburu, 1993) developed by other authors.

This chapter describes the use of this strategy with a patient experiencing severe persecutory delusions.

13.2 Method

13.2.1 Patient

The patient, BI, was a 33 year old single white male. At the time of referral to me, he was an inpatient on a psychiatric ward of a large general hospital. His inpatient stay had begun 35 days earlier, after a referral from a local accident and emergency department. Prior to his admission, BI had been a university student studying modern languages. Before this he had travelled widely, usually supporting himself by teaching English as a foreign language. BI was the youngest of three children. Both his parents

were dead, his father having died of natural causes when he was 24, his mother having committed suicide when he was 13 years old.

BI presented to the accident and emergency department after swallowing a large number of aspirin tablets. He reported a two-week history of low mood, with suicidal thoughts, loss of appetite, loss of weight and low self-esteem, but with a normal sleep pattern. He stated that these depressed feelings stemmed from his conviction that the members of a large, internationally-organized group of drug barons were conspiring to pressure him into joining their organization. He was unshakably convinced of the reality of this conspiracy, but had doubts as to who was involved, harbouring suspicions about both the ward staff and the first author. He reported that he had known of the existence of the plot for seven years, but that it had only recently begun to depress him significantly. Staff on the ward reported these delusional ideas to be held with full delusional conviction, with BI refusing to accept assurances by staff-members that they were innocent. As this was BI's first formal contact with psychiatric services, it is impossible to assess the true chronology of the delusional ideas, and neuroleptic medication had never before been prescribed.

On admission to the ward, BI had received a provisional diagnosis of paranoid schizophrenia (as he was not known to meet the duration of symptoms criterion for confirmed diagnoses). Although clearly depressed, the opinion of the clinical staff, and the reports of BI himself, indicated that his low mood and hopelessness was a consequence of a long-standing and seemingly inescapable persecutory delusion. Delusional disorder was considered as a possible alternative diagnosis, given that BI's low mood was brief compared to the duration of his reported delusional beliefs, however BI's social isolation and his inability to work appeared to constitute a significant impairment of social functioning. The use of soft drugs had been listed in the medical notes as a possible complicating factor as BI reported that he was a regular user of cannabis, smoking between a quarter and half of an ounce (7 - 14 g) of

cannabis resin a week. The use of cannabis is, however, common, and, as will be outlined below, it seems unlikely that this factor can adequately explain BI's psychotic symptoms and his subsequent recovery. No drug screens were performed while BI was on the ward, so it was impossible to verify his testimony, however he gave assurances that he had taken no illicit drugs since his admission. From the first day of admission he was prescribed 50 mg chlorpromazine at nighttime, 5 mg trifluoperazine three times a day and 5 mg procyclidine twice daily. This regime did not change through his admission and was continued on discharge.

BI's main symptoms were his complex and persistent persecutory delusions. He did not report hallucinatory experiences and, when contacted by the first author 35 days after his admission, appeared only moderately depressed. However, he reported that he experienced anxiety associated with his delusional beliefs which fluctuated according to circumstances. He agreed to psychological therapy aimed at reducing his anxiety, raising his mood, and rationalizing who, if anyone, was part of the conspiracy. BI had also clearly been depressed as a consequence of his delusional state. His admission to hospital had been precipitated by a suicide attempt. BI reported that this had been a desperate response to the unrelenting nature of his perceived persecution. BI reported a number of decisions that appeared to be related to his delusional ideas, including frequent changes in residence. Apart from these decisions, however, BI had not acted upon his delusions. His conviction in the delusional ideas appeared total. BI told the first author that his only doubts concerned who exactly was a member of the conspiracy; he had no doubts as to the reality of such a plot.

13.2.2 Initial Assessment

BI's mental state was assessed using the Positive and Negative Syndrome Scale (PANSS; Kay & Opler, 1987). This scale gives scores for individual symptoms and also for three subscales - positive symptomatology, negative symptomatology and general psychopathology - based on ratings of data obtained from case notes, consultations with care staff, and a short semi-structured interview with the patient and has been shown to be a reliable and valid means of assessing psychotic symptomatology (Kay, Opler, & Fszbein, 1988). BI recorded a positive syndrome score of 33 (98th percentile compared to 'medicated schizophrenics'; Kay, Opler, & Fiszbein, 1986), a negative syndrome score of 22 (50th percentile), and a general psychopathology score of 46 (75th percentile).

An initial psychological assessment was conducted using the Beck Depression Inventory (BDI), Fenigstein and Vanable's Paranoia Scale, and the Internal, Personal and Situational Attributions Questionnaire (IPSAQ). These measures have been described in previous chapters.

At initial assessment, BI recorded a BDI score of 30, indicating moderate to high levels of depressed mood. His Fenigstein Paranoia Scale score was 78, consistent with a high level of paranoid ideation. His responses on the IPSAQ were, for positive items, 12 internal, 3 personal and 1 situational attributions. For negative items his responses were 7 internal, 6 personal and 3 situational attributions. These scores can be accounted for by two attributional processes. First, BI had a tendency to make self-protective attributions, with a significantly greater number of internal attributions for positive as opposed to negative events than normal undergraduate subjects (BI's positive minus negative difference score = +5; 99 percent confidence limits for normal subjects = 0.105 to 2.677). Second, the proportion of external attributions made by BI for negative events which were personal as opposed to

situational was greater for BI than for normal undergraduate subjects (proportion for BI = 0.667, 99 percent confidence limits for normal subjects = 0.587 to 0.427; see Chapter 10).

Throughout therapy, BI kept daily records of salient psychological variables using a personal record booklet containing a record sheet for each day of the week. Each record sheet contained three 10 cm visual analogue scales. (See Appendix 15) In this way BI was asked each evening to record his feelings in terms of depressed mood (scale labelled from "Very happy" to "Very sad or depressed"), self-esteem (scale labelled from "Very good about myself as a person" to "Very bad about myself as a person"), and paranoid anxiety (scale labelled from "Not at all worried about any conspiracy" to "Extremely worried about a conspiracy"). Anxiety, rather than simple conviction, was selected as the target variable for two pragmatic reasons. First, it was felt that daily questions of conviction might trigger psychological reactance (Watts, Powell, & Austin, 1973), thus consolidating paranoid ideation. Second, BI had targeted his anxiety about a conspiracy as a therapeutic focus, rather than conviction.

13.3 Intervention

The cognitive-behavioural intervention, which was carried out by myself, centred around the attributional model of paranoid ideation outlined earlier. A structured therapy based on attributional style was conducted during approximately weekly sessions. Although the therapeutic target was specific paranoid ideation, therapy initially focused on less emotional topics. At the outset the importance of a cognitive approach to emotional disturbance was established by pointing out the links between feelings and the understanding of the events. With reference to the IPSAQ data collected from BI, discussions then centred on the importance of explanation as a

means of understanding and the likely effects of the three types of attribution. It was agreed that BI had a tendency to pay less attention to situational attributions than he might. Using conventional diary sheets, BI was encouraged to practice generating multiple explanations for everyday events, paying special attention to the consideration of situational attributions. This therapeutic strategy was then brought to bear on the paranoid ideation by encouraging BI to examine those events that triggered his paranoid ideation. He was encouraged to generate possible situational attributions for these events. One event discussed in detail in this phase was seeing police officers. BI initially concluded that they intended to arrest or assault him, but then consciously generated the situational attribution that they were probably going about normal police patrols (that is, that their presence was due to circumstantial, situational factors). BI also became paranoid about the disappearance of a book from his locker on the ward, concluding initially that this was part of a plot to destabilize him mentally, but then attributing the loss to other patients thinking that the book was general ward property and borrowing it. The non-arrival of a social security benefit payment also triggered thoughts that BI's payments were being interrupted by the drugs barons, but was then reattributed to postal delays. In each of these cases, the situational attributions were subsequently accepted as correct.

The therapeutic strategy employed in this case is entirely compatible with conventional cognitive-behaviour therapy based on engagement, elicitation of cognitive schemas and reality testing. Engagement and elicitation of cognitions were important elements of therapy. However the specific intervention was directed at eliciting the client's explanations for negative or ambiguous events, and helping the client, through the generation of multiple possible alternatives, develop a pattern of attributions for such events that avoided the biases in attribution hypothesised to lie behind paranoid ideation.

In order to interpret the data from psychological measures, the treatment period

was divided into two. Prior to meeting the first author, BI had been an inpatient for 35 days, no quantitative data are available for this period. Phase one of treatment refers to the period from initial referral (this will be subsequently referred to as day 1) until the point where active reattribution was prescribed (day 28). Phase two refers to the time from the beginning of active reattribution until the end of psychological treatment. BI was discharged from inpatient care on day 37, but continued with psychological therapy. Psychological treatment was ended 13 days later after a total of 9 appointments, 48 days after initial referral and 83 days after admission. BI remained under the care of a psychiatric aftercare team.

13.4 Results

At the final session of psychological therapy the formal assessment was repeated. BI's BDI score was 8, within the normal range (Rehm, 1988), and his score on Fenigstein's Paranoia Scale was 40. His responses on the IPSAQ had changed so that they now included 7 internal, 4 personal and 5 situational attributions for positive items, and 5 internal, 5 personal and 6 situational attributions for negative items. These scores lie within the normal confidence limits for undergraduate subjects (see Chapter 10), both in terms of the relative number of internal attributions for positive and negative events (a difference of 2) and the proportion of external attributions that were personal (0.455). This represents a shift from excessively self-protective attributions identifying other individuals as the source of his problems to a more evenhanded position in which BI was more aware of possible circumstantial causes for negative events. Summary statistics for these data are presented in Table 13.1

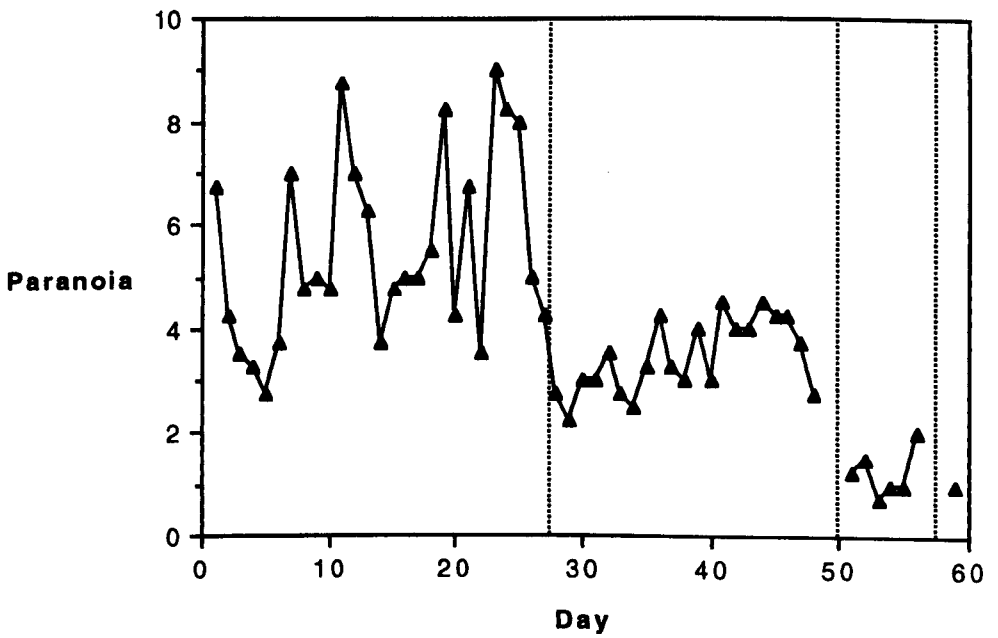
The daily record sheets completed by BI also reflected a substantial reduction in his paranoid ideation.

Table 13.1 Summary statistics for formal psychological assessments at initial and final sessions. Raw and percentile scores, calculated from Kay, Opler & Fiszbein's (1986) sample of psychotic inpatients, are presented for the PANSS positive syndrome, negative syndrome and general psychopathology scales. Mean scores on the idiographic visual analogue scales are presented for the two phases of treatment, before and after active reattribution therapy, and at one and five month follow-up. Data were not collected are indicated by ‡.

Formal measures	Pre-treatment	Post-treatment
BDI score	30	8
FPS score	78	40
PANSS +ve syndrome	33 (98 %ile)	‡
-ve syndrome	22 (50 %ile)	‡
General Psychopathology	46 (75 %ile)	‡
IPSAQ Externalizing bias	+5	+2
Personalizing bias	0.67	0.46

	Phase 1 (27 records)		Phase 2 (21 records)		1month (5 records)		5 month (1 record)	
Visual analogue scales	M	SD	M	SD	M	SD	M	SD
Paranoid anxiety	5.52	(1.82)	3.45	(0.70)	1.25	(0.45)	1	(-)
Low mood	5.30	(1.73)	3.90	(1.42)	‡		‡	
Self-esteem	4.59	(0.83)	3.37	(0.79)	‡		‡	

Figure 13.1. Daily recordings of paranoid ideation made on a 10cm visual analogue scale labelled ranging from "Not at all worried about any conspiracy" to "Extremely worried about a conspiracy". Data are presented in two phases of treatment, with phase 1 being before and phase 2 after instructions to develop alternative situational explanations for ambiguous events. Follow-up records occurred on days 82 to 87 (the 12th week after commencement of treatment) and on day 147.



Data from the paranoid anxiety analogue scale are illustrated in Figure 13.1. As can be seen from inspection of this figure, BI's paranoid concerns, as measured by the visual analogue scales, dropped sharply at the point in therapy at which BI began actively to generate alternative, situational, attributions for worrying events. Examination of medical case-notes revealed that no change had occurred in BI's medical care around this time. One-way analysis of variance (comparing the two phases of treatment) revealed that BI's paranoid anxiety dropped by a statistically significant degree, $F(1,47) = 24.08$, $p < .0005$. At the same time, his ratings of low mood, $F(1,47)=10.11$, $p < .005$, and poor self-esteem, $F(1,47) = 12.04$, $p < .005$, dropped significantly. As can be seen in Table 13.1, these ratings were maintained at both one month and five month follow-up.

Anecdotal accounts support these changes. Following therapy, BI made a number of statements that strongly suggest that the changes in paranoid ideation noted were associated with cognitive and attributional changes. Two examples illustrate this. On one occasion BI reported that he had smoked "a large amount" of cannabis. He then saw a woman with two "nasty-looking" dogs. He reported that his initial reaction was that, "this was part of a plan to frighten me". He then actively reminded himself that cannabis can exacerbate paranoid feelings, and that this was probably why he was frightened. On another occasion the key did not fit in the door of his new flat. "Ordinarily this would have made me very paranoid, but this time not at all, I thought the estate agents had given me the wrong keys, went back and got the right ones." These examples provide evidence that cognitive restructuring had occurred, and that this involved the accessing of situational attributions.

The success of the therapeutic approach is further supported by statements made at five months follow-up. When asked directly about his paranoid fears, BI stated that "I've really forgotten about that. I mean, I'm sure there was something going on back in Malaysia, but it's over now. I just let things go now. I reckon that if

there was a conspiracy, they've stopped now. Maybe there never was one, but I think there was."

13.5 Discussion

This case study illustrates a number of important theoretical and practical aspects of the model of paranoid ideation outlined in Chapter 6 and revised in the following chapters. The systematic measurement of paranoid anxiety and attributional processes over time allowed an examination of the relationship between these variables within a single individual, indicating that different levels of symptomatology are associated with different kinds of attributions as predicted by the model. BI, when paranoid, exhibited an externalizing, defensive attributional pattern, blaming other individuals for negative events. However, it was also noted that BI's paranoia seemed to be related to a specific difficulty in generating situational attributions for negative events (such as those employed in normal excuse-making). It might be hypothesized that it is the combination of a defensive attributional style, together with the failure to recognize situational determinants of the behaviour of others, that leads to paranoid accounts of negative experiences.

This case study also illustrates the potential applicability of the cognitive model of paranoid ideation. When therapy was specifically targeted at altering the pattern of attributions generated by BI, his level of expressed paranoia, as assessed by a number of measures, dropped. This observation, along with those of other authors (Chadwick & Lowe, 1990; Garety, et al., 1994; Hartman & Cashman, 1983; Kingdon, et al., 1994; Tarrier, et al., 1993; Watts, et al., 1973), suggests that relatively straightforward cognitive therapy strategies may be beneficial to deluded patients. A common feature of the cognitive-behavioural strategies which have been advocated for

use with deluded patients, including the strategy advocated here, is the avoidance of direct confrontation of the patient.

It is possible that several factors in addition to cognitive-behaviour therapy may have contributed to BI's improvement. Neuroleptic medication is, of course, known to be effective in psychotic illness (see Chapter 1) and BI had been prescribed a regime of medication within the normal therapeutic range. However, this regime remained stable throughout the period of BI's hospital stay, including the 35 days prior to first contact with the therapist, and during follow-up. BI's cannabis use was noted as a possible complicating factor at admission. However, this had stopped on admission to hospital and the time between admission and first contact with the therapist was sufficient for cannabanoids to clear the body (Mathers & Ghodse, 1992). Furthermore, BI reported that he returned to normal cannabis use after discharge from hospital, while paranoid ideation continued to be absent. Finally, examination of the medical and nursing notes revealed no significant change in medical, nursing or social circumstances until after the observed therapeutic changes. BI's paranoid ideation altered at the time expected according to the cognitive model, when paranoia-inducing attributions were actively challenged.

It is notable that the client's paranoid anxiety fluctuated significantly. There seem two possible explanations for this. It is possible that BI was using a successful approach to reality-testing, but on an episodic or occasional basis. This is an unlikely explanation, as the variation in anxiety was greatest before active reattributional strategies were developed. When questioned during clinical sessions about the marked differences in anxiety, BI reported that the peaks in anxiety were associated with specific events, occurring on an occasional basis, which triggered paranoid explanations. This account is consistent with an attributional model of paranoid ideation, and is also consistent with both the reduced level of overall anxiety and the reduced variability following the development of reattributional strategies.

Further research is needed to explore the relationship between attributional processes and symptom changes in deluded patients. In contrast to the trait-view of attributional style put forwards by depression researchers (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978) it is clear from this case study, from the research outlined in Chapter 5, and also from the other experimental studies reported in this thesis, that attributional processes are complex, may involve the interaction of multiple cognitive processes (particularly self-representations), and may vary both in the short and long term. It seems likely that cognitive-behavioural interventions for psychotic patients are most likely to be effective if informed by an adequate understanding of the cognitive processes implicated in the specific symptoms which are the target of therapy.

Chapter 14.

Theory of Mind Deficits, Causal Attributions, and Paranoia

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A paper based on the material presented in this chapter has been submitted for publication as: Kinderman, P., Dunbar, R., & Bentall, R. P. Theory-of-mind deficits and causal attributions.

14.1 Background

The ability to understand and conceptualize the mental processes of other people is considered to play a vital role in social interactions. Deficits in this area, sometimes called Theory-of-Mind (ToM) deficits, have been identified as playing a possible causal role in autism and Asberger's syndrome. Difficulties in representing the mental states of others have also been suggested as characteristic of schizophrenic disorders, particularly paranoia. Paranoia has also been associated with an abnormal attributional style. Paranoia may thus serve as a natural experiment illustrating the role of causal attributions as mediating factors between ToM and social perception. This chapter describes a study examining the relationship between attributional processes and ToM deficits in non-patient participants. Such a relationship might help shed some light on possible aetiological factors leading to the attributional abnormalities, and disturbance in the self-concept outlined in previous chapters.

14.1.1 Theory-of-mind

We do not, as a rule, appraise other people's actions and conversation as simple behavioural or verbal patterns. Rather, behaviour is used as a clue to discover wishes, hopes, beliefs, intentions etc. Such an understanding is termed Theory of Mind (ToM), and is believed to be vital for successful social intercourse (Premack & Woodruff, 1978). There have been four areas in which ToM has been studied in depth. ToM was first invoked to understand the limits of cognition in chimpanzees by Premack and Woodruff (1978). In this context ToM is a particularly human ability. Although primates appear able to deceive each another (Byrne, 1995), little evidence of a systematic ability to comprehend the mental processes of others has emerged. In

humans, the developmental and social consequences of understanding the intentions behind the actions of others have been widely investigated (Astington & Gopnik, 1991; Astington & Jenkins, 1995; Davis & Pratt, 1995; Hall, Frank, & Ellison, 1995; Lalonde & Chandler, 1995; Raver & Leadbeater, 1993; Sullivan, Winner, & Hopfield, 1995).

In the clinical field, empirical associations between ToM deficits and psychological problems were first demonstrated in autism, and have been held to explain the difficulties in communication, imagination and the formation of relationships that are characteristic of that disorder (Baron-Cohen, Leslie, & Frith, 1985; Baron-Cohen, Tager-Flusberg, & Cohen, 1993; Frith, 1989; Frith, Morton, & Leslie, 1991; Happé & Frith, 1994; Leslie, 1991; Loveland & Tunali, 1991). For example, autistic children, in comparison with appropriate controls, tend to suggest that actors would look for hidden objects in places where they (the children) know them to be despite the fact that the actors cannot have this knowledge (Baron-Cohen, et al., 1985).

Many recent advances in understanding of ToM processes have stemmed from such investigations. In particular Simon Baron-Cohen, a principal researcher of ToM influences in autism, has recently suggested a modular neurocognitive ToM system (Baron-Cohen, 1994). This comprises four sub-systems: an intentionality detector (ID) which represents behaviour in terms of volitional states; an eye direction detector (EDD) which not only detects eye-direction, but also appraises what the eyes are looking at; a shared attention mechanism (SAM) which represents whether the individual is attending to the same object or event as others; and a theory of mind mechanism. The theory of mind mechanism integrates input from the other components and develops a coherent mental model.

14.1.2 Theory-of-mind and paranoia

A number of researchers, in particular Chris Frith (see Frith, 1994) have linked ToM deficits with both the positive and negative symptoms of schizophrenia. The links between ToM deficits and the symptoms of schizophrenia have been tested empirically. Corcoran, Mercer and Frith (1995) found that patients with schizophrenia performed poorly on a task requiring participants to draw inferences from indirect but clear hints. Similarly, Frith and Corcoran (1996) found that patients with schizophrenia displayed significant problems with the recall of material that required the ability to mentalize, while Corcoran, Cahill and Frith (submitted) found that such patients were less able to understand the point of jokes dependent on ToM.

With respect to paranoia, Frith (1994) has suggested that patients may misunderstand the intentions of others partly because they suffer from a mentalizing deficit. The ToM deficits believed to be important in paranoia are thought of as specific to paranoia but not absolute - meaningthat the skills remain present to a limited degree (Corcoran, et al., submitted). Although Frith's position is succinct, and has the appeal of common sense explanations, specific hypotheses about how such deficits might relate to other psychological abnormalities displayed in paranoid patients are not specified. ToM deficits may well contribute to paranoid and other conditions, but presumably by influencing other cognitive processes. The nature of the cognitive abnormalities may be illuminated by the observation of links between paranoia and ToM deficits on the one hand, and paranoia and attributional style on the other. The links between attributional style and paranoia is discussed at length earlier in this thesis.

14.1.3 Theory-of-mind deficits, causal attributions and paranoia

It is not difficult to imagine how ToM deficits might contribute to the tendency to make external-personal attributions of the sort associated with paranoid ideation. When involved in a negative social interaction (for example, say a friend is unusually curt) it is possible to explain the behaviour of the other person either in terms of a trait (the friend is hostile) or in terms of the situation (the friend has had a bad day, is feeling unwell, etc.). The former involves making an external-personal attribution whereas the latter involves making an external-situational attribution. However, in order to make the external-situational attribution in this example, it is necessary to be able to appreciate the other person's point of view (we must be able to imagine the world from their perspective, and the way that their experiences may affect their disposition towards us today). It would be impossible to do this without an adequate ToM. If this account is correct, ToM deficits should be associated with external-personal attributions for negative events. This chapter describes a preliminary test of this formulation carried out with normal participants.

14.1.4 Hypotheses

It was predicted that considerable variation in the ability to process information concerning the mental states of other individuals would be observed in normal individuals. It was further predicted that, when asked to account for hypothetical negative social interactions, those individuals less able to understand the mental states of others would make a higher proportion of external-personal attributions as opposed to external-situational attributions. Individuals who did not show ToM deficits, on the other hand, were expected to make more external-situational attributions than

external-personal attributions for negative events.

14.2 Method

14.2.1 Participants

Participants were 77 undergraduate students of Occupational Therapy and Physiotherapy at Liverpool University. Participants were approached for their cooperation, which was voluntary, in three class-groups. No incentive was offered for participation. None of the participants, when questioned after the experiment, indicated that they had understood the hypotheses under test.

14.2.2 Materials

14.2.2.1 Internal, Personal and Situational Attributions Questionnaire

The development of the IPSAQ was described in Chapter 10. Subsequent investigations using it and its derivatives were described in chapters 11 to 13.

14.2.2.2 Imposing Memory Task

The Imposing Memory Task (IMT) was a measure designed to assess mentalizing (ToM) ability. A series of five stories were read out to participants at the same time as being presented on an over-head projector. Four of these stories involved complex social situations which required listeners to understand the perspective and intentions of the actors (e.g. Employees attempting to persuade their employer to

increase wages). The fifth story involved only one participant, an unfortunate man who fell asleep while smoking a cigarette. Participants were presented with a booklet containing questions tapping memory for information contained in the stories. Each question comprised a forced-choice of two alternatives, one correct and one incorrect. Questions either concerned ToM elements of the stories (the expectations or beliefs of the participants) or were memory questions. In the latter case the actions of participants were the subject of the questions, but no demand was made on participants' ability to mentalize.

Both ToM and memory questions were asked at a number of levels of complexity. First order questions required the correct recall of one piece of information, either factual (memory) or concerning actors' intentional state (ToM). More complex memory questions required recall of information where two, and more, pieces of information were logically related. Higher order ToM questions required correct recall of the mental states of actors where those mental states concerned the mental states of others. Thus, first order ToM questions related to what an actor thought, second order ToM questions related to what an actor thought another actor thought, third order ToM questions related to what an actor thought a second actor thought that the first actor thought, and so on. The highest order ToM question was fifth order, the most complex memory question involved the appraisal of six related aspects. Not all stories included questions tapping the highest orders of complexity. (see Appendix 16).

In previous studies of ToM reported in the literature, ability to mentalize has sometimes been assessed using questions which required the generation of attributions (ie. participants are required to make inferences about the causes of the behaviour of others; Happé & Frith, 1994). Such a confound between ToM questions and attributions would clearly undermine the test of hypotheses being conducted in this study. For this reason, none of the ToM or memory questions included in the present

assessment required the generation of causal attributions.

14.2.3 Procedure

Participants were presented with the IPSAQ and IMT answer booklet. The first story was presented and read out, then hidden from view. Participants were asked to open their IMT booklet and answer questions on the first story. The second story was then read and presented, participants turned the page to reveal the second set of questions and these were answered. This procedure continued for all five stories. Participants were then asked to take away the IPSAQ and return it. 46 of the 77 participants (59.7 %) returned completed IPSAQ's.

14.3 Results

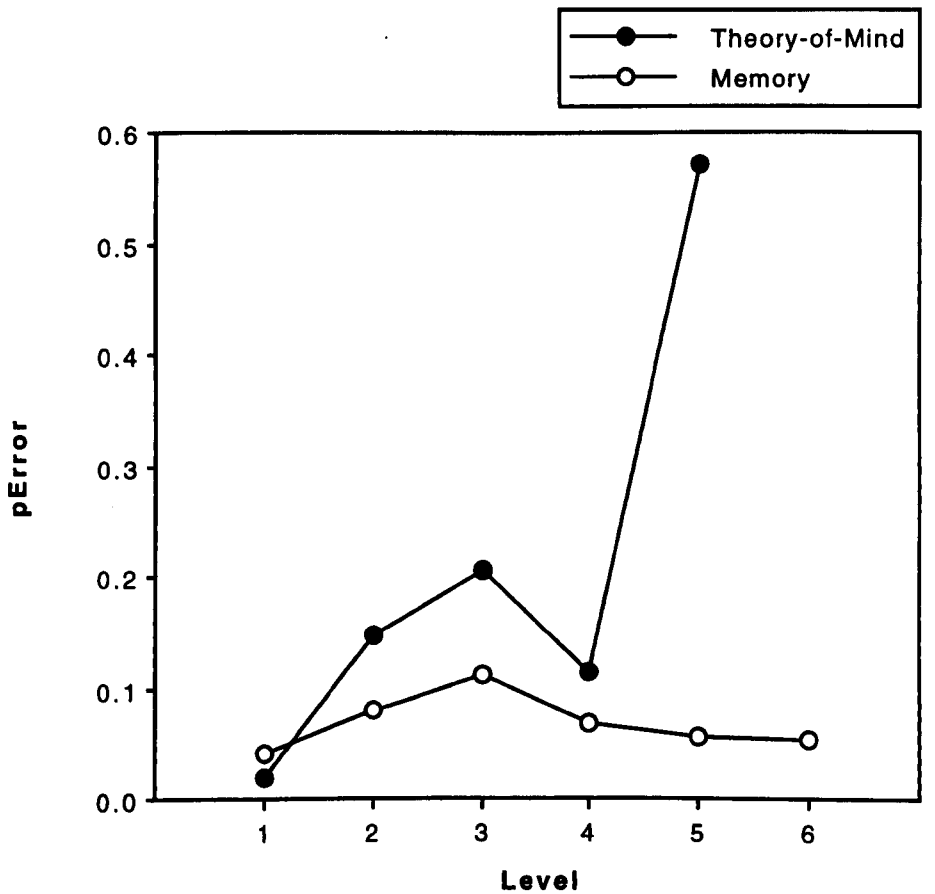
Participants' scores on the IPSAQ revealed a general pattern of attributions similar to that commonly seen in normal participants and outlined in previous chapters. Mean Externalizing Bias (EB; the number of internal attributions for positive events minus internal attributions for negative events) was 2.13 (SD = 3.38, range -7 to +10; 99 percent confidence limits for normal participants = 0.105 to 2.677). Mean Personalizing Bias (PB; the proportion of external attributions for negative events which were personal as opposed to situational) was .49 (SD = 0.27, range .00 to 1.00; 99 percent confidence limits for normal participants = .587 to .427).

Scores for all 77 participants on the Imposing Memory Task revealed a mean of 2.59 Theory-of-Mind (ToM) errors for the 16 ToM questions (SD = 1.63 range 0 to 8). The mean number of memory errors was 1.54 (SD = 1.77, range 0 to 9) for 24

questions. The number of ToM and memory errors correlated significantly, Spearman's $r = .3713$, $p < .001$, with participants who made larger number of ToM errors also making more memory errors.

For each level of complexity, the mean number of errors on the IMT was calculated for each participant by dividing the total number of errors by the number of questions at that level of complexity. The mean ToM and memory error rates for each level of complexity are presented in Figure 14.1.

Figure 14.1. Proportion of incorrect answers to Theory-of-Mind and Memory questions for all 77 participants. Proportions are presented separately for the two types of questions at each level of complexity.



It can be seen that error rates for memory items do not increase with the complexity of the questions. However, whereas error rates for ToM items at level 1 is similar to that for level 1 memory items, error rates for subsequent ToM items exceed those for the comparable memory items, especially at level 5. An analysis of variance (ANOVA) was conduced on the ToM and memory error scores for the first five levels of complexity, with type of error (ToM and memory) and order of complexity as within-subjects factors. This revealed that overall there was a significantly higher proportion of ToM than memory errors, $F(1,76) = 105.94$, $p < .00005$, and that the order of complexity had a significant effect on error rate, $F(4,304) = 44.51$, $p < .00005$. The interaction term was significant, $F(4,304) = 44.00$, $p < .00005$, indicating that the two types of error rates varied differently with increasing complexity.

Tests of simple effect revealed that there were no more ToM errors than memory errors at the first level of complexity, $F(1,76) = 2.23$, $p = .139$. There were significantly greater numbers of ToM errors at levels two, $F(1,76) = 8.44$, $p < .005$, and three, $F(1,76) = 15.73$, $p < .0005$. Fourth-order questions produced a non-significant trend towards the generation of more ToM errors than memory errors, $F(1,76) = 3.56$, $p = .063$, while there were significantly more ToM than memory errors at the fifth level of complexity, $F(1,76) = 81.70$, $p < .0005$.

The 46 participants who returned competed IPSAQ's were divided medially into two groups on basis of ToM scores. Those scoring three or more errors were allocated to the 'Deficit' group, all others were allocated to the 'Non-Deficit' group.

A full factorial MANOVA was conducted on the scores for the six sub-scales on the IPSAQ, with group membership (deficit or non-deficit group) as a between-subjects variable and locus of attribution (internal, personal or situational) and valence of item (positive or negative) as within-subject variables. This revealed predictable and significant differences between the number of attributions made to

internal, personal and situational loci, $F(2,88) = 6.10$, $p < .005$. Planned pairwise comparisons (Tukey's HSD) revealed that there were more internal attributions than personal, $p < .01$, and situational, $p < .05$. The number of personal and situational attributions did not differ significantly. A significant interaction term between attributional locus and valence, $F(2,88) = 11.77$, $p < .00005$, revealed that different attributional loci were chosen for positive and negative items. Tests of simple effect revealed that more internal loci were chosen for positive than for negative items, $F(1,44) = 16.70$, $p < .0005$. More personal, $F(1,44) = 10.21$, $p < .005$, and situational, $F(1,44) = 4.313$, $p < .05$, loci were chosen for negative events than for positive. A significant interaction term between attributional locus and group membership, $F(2,88) = 6.55$, $p < .005$, revealed that the groups differed in their choice of locus. Tests of simple effect revealed that the deficit group used more internal, $F(1,88) = 4.94$, $p < .05$, and fewer situational attributions, $F(1,88) = 12.85$, $p < .01$, than the non-deficit group. The difference in the number of attributions made to personal loci was not significant, $F(1,88) = 1.87$, $p = .177$. The overall interaction term was non-significant, $F(2,88) = 0.84$, $p = .4368$, indicating that, although the groups differed in their choices of attributional loci, these choices were not differentially affected by the valence of the items.

To investigate these findings more precisely, Memory error scores, Externalizing Bias (EB) and Personalizing Bias (PB) were calculated for the Deficit and Non-Deficit group separately. These scores are presented in Table 14.1.

Table 14.1. Mean numbers of attributions made to each attributional locus, mean Externalizing Bias (EB) and Personalizing Bias (PB) scores and memory question error rates for the Theory-of-Mind Deficit and Non-Deficit groups.

	Group			
	Non-Deficit n = 27		Deficit n = 19	
	M	SD	M	SD
Positive items				
Internal	6.93	3.61	8.53	2.22
Personal	3.26	2.86	4.05	1.87
Situational	5.78	3.73	3.42	3.08
Negative items				
Internal	4.63	3.12	6.68	2.65
Personal	4.26	4.26	5.68	2.26
Situational	7.11	3.81	3.63	2.87
EB	2.33	4.00	1.84	2.32
PB	0.38	0.25	0.63	0.22
Memory Errors	1.11	0.80	2.16	2.50

One-way analyses of variance (ANOVA) revealed no differences between the groups in terms of Externalizing Bias (EB), $F(1,44) = 0.23$, $p = .633$. However Personalizing Bias (PB) was significantly higher for the Deficit group than for the Non-Deficit group, $F(1,44) = 12.41$, $p < .0001$. There was a trend for the ToM Deficit group to record higher levels of memory errors which almost reached statistical significance, $F(1,44) = 4.16$, $p = 0.051$.

14.4 Discussion

As predicted, those individuals with a relative inability to process information concerning the mental states of others were shown to have a pattern of causal attributions that was significantly different from those individuals relatively skilled at this kind of social cognition. Specifically, those individuals making relatively many errors on questions measuring ToM ability attributed significantly more negative events to persons as opposed to situational factors. The implications of these observations will be discussed for, first, normal social cognition and, second, theories of psychosis. Necessarily, much of this discussion must be quite speculative.

With regard to normal social cognition, the discovery of significant differences in mentalizing ability in normal participants, and the observed pattern of increasing rates of errors as the complexity of the task increased, suggests that such ability is not either present or absent. Rather, the ability to process information about the mental states of others appears to be exhibited to a greater or lesser degree in different participants. It is interesting to note that, despite the higher error rates for ToM questions than for memory questions at high levels of complexity, a significant correlation was observed between memory errors and ToM errors and there was a tendency for the ToM deficit group to fail more memory questions. This observation is

consistent with the hypothesis that ToM performance depends on more general cognitive resources, in particular executive function or working memory (Corcoran, et al., 1995; Davis & Pratt, 1995). This raises the possibility that the association between ToM and causal attributions reported here are caused by a third variable such as IQ or memory ability.

Memory functioning and other executive cognitive processes are important factors to examine in order to understand the bases of ToM ability. Such cognitive capacities are likely to be important factors in determinng ToM ability. In turn, causal attributions are likely to be mediating factors between ToM deficits and social behaviour. In the present study, however, it is unlikely that differences in intelligence are responsible, given that all participants were undergraduate university students. In future studies intellectual funcioning, executive memory functioning and other variables may be important topics of investigation.

The pattern of causal attribution associated with ToM deficits in this study is similar to that known to be associated with analogue measures of paranoid ideation (Chapter 9), and is implicated in clinical delusions of persecution (Chapter 12). The present findings therefore suggest one way in which ToM deficits may contribute to an abnormal attributional style which, in turn, may lead to paranoid symptomatology. The question remains whether ToM deficits are alone sufficient to generate persecutory delusions. There are a number of reasons for supposing that they are not. First, many autistic and Asberger patients, who have demonstrable ToM deficits, do not show paranoid symptomatology. Frith and Corcoran (Frith & Corcoran, submitted) suggest that many of the differences between autistic and paranoid individuals stems from the developmental time-course of their difficulties. There is an important difference, it is argued, between never understanding other people's minds and losing that ability.

Second, paranoid patients have been shown to exhibit a wide range of cognitive biases affecting attention, memory, attributions and hypothesis-testing (as

outlined earlier in this thesis). Third, the reasoning of paranoid patients is different for positive than for negative information affecting the self, suggesting that the cognitive biases observed in these patients have a motivational and defensive function. Corcoran, Mercer and Frith (Corcoran, et al., 1995) suggest that many of the abnormalities outlined above may be caused by ToM deficits. ToM deficits may play an important role in the abnormalities seen in paranoid patients, but do not form a complete explanation. For example, paranoid individuals have a tendency to make fewer self-blaming causal attributions, but fewer benign excuses. A ToM deficit may explain why more of these self-exonerating explanations are directed at other individuals, but does not alone provide an explanation for the self-protective biases. Clearly further research is needed to tease out the complicated inter-relationships in this area.

An example of the complexity of these issues concerns the difference between memory and ToM questions. Although attempts were made to ensure equivalence of ToM and memory items on the IMT, it is clear that these two types of questions are qualitatively different. Specifically, memory questions, no matter how complex, essentially refer to one comparison (Is it the case that X?). Events described in the question are compared with the individual's model of reality, a single comparison. ToM questions involve a number of simultaneous comparisions (Did X think that Y thought that Z thought ... ?). This complexity, although presenting methodological difficulties, may be theoretically significant.

Finally, of course, none of the participants in the present study were clinically paranoid (although it is possible that more severe ToM deficits than those observed in the present study are required for full-blown clinical paranoia). Taken together, these observations suggest that ToM deficits are but part of a complex causal pathway which leads to paranoid thinking. Most likely, ToM deficits only contribute towards paranoid ideation when there is also strong motivation to avoid internal explanations for

negative experiences. Further research exploring ToM and other abnormalities in paranoid patients may help to clarify these causal pathways.

These observations raise the question of whether or not the ToM deficits implicated in paranoid thinking are enduring or episodic. The fact that paranoid disorders generally occur in adulthood, and that sometimes they resolve in time or following treatment, suggests that at least some of the cognitive abnormalities implicated must change over time. It is possible that this is the case for ToM deficits. It may be the case that ToM tasks require additional cognitive capacity in comparison with equally complex memory tasks. Psychotic breakdowns in general seem to be associated with general impairments of attention and memory which may be indicative of such reduced capacity (Neuchterlein, Buchsbaum, & Dawson, 1994). Thus, it is possible that ToM deficits are but one point on a causal pathway that leads from general cognitive dysfunction to psychotic symptoms.

One important limitation of this study must be acknowledged. The study has not involved participants suffering from clinical paranoia. Rather, it has studied in normal participants the interaction between two types of cognitive processes which may play a role in paranoia. At this stage it is important to be cautious when generalizing from normal participants to patients suffering from a psychiatric disorder. Clearly, further studies are required in which ToM and attributional processes are studied in clinical populations. Despite this observation, the present findings suggest ways in which attributional and other cognitive processes interact in the mental economy of both normal and clinical participants. Whereas previous research has tended to assume that attributional style is an enduring trait which may have important consequences for the emotional well-being of the individual (Buchanan & Seligman, 1995) the present study, together with the previous investigation of the interaction between attributions and self-representations in normal individuals (outlined in chapters 9 and 11), suggests that complex cognitive processes may be involved in the

generation of attributions and, in particular, that the kind of attribution generated by an individual in a particular situation will depend on the accessibility of different kinds of relevant information.

Chapter 15.

Conclusions

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15.1 Overview

This thesis comprises a series of studies designed to test the predictions inherent in the attributional model of paranoia outlined in Chapter 6. This model links paranoid ideation to the individuals' self-concept through the mechanism of causal attribution. The findings of the eight investigations in this thesis will be briefly outlined below.

15.2 Overview of Investigations

15.2.1 Attentional Bias

One prediction stemming from the attributional model of paranoia is that persons with delusions of persecution would show evidence of an implicit (and defended) negative self-concept. This prediction was tested using an emotional Stroop test.

Attentional bias towards positive and negative trait words was studied in participants suffering from persecutory delusions, matched participants with depressed mood and non-patient participants using an emotional Stroop task. Participants were required to name the ink-colours of; i) meaningless strings of O's, ii) low self esteem personal adjectives, iii) high self-esteem adjectives and iv) neutral adjectives. Participants were also asked to rate the degree to which they endorsed as self-descriptive these and other personally-descriptive adjectives. Individuals with persecutory delusions showed a significantly higher rate of endorsement for positive adjectives than negative adjectives, but showed a marked degree of interference when colour-naming both positive and negative words.

The findings of this study have three implications. One prediction of the model

of paranoid ideation described in Chapter 6 was tested and supported. The central relevance of stimuli related to the self-concept in individuals with delusions of persecution was established. Finally, the notion of an implicit or unacknowledged negative self-concept in paranoia was strengthened.

15.2.2 Self-discrepancies

The second empirical study addressed the hypothesised proximal consequences of externalizing causal attributions in paranoia. The attributional model of paranoid ideation suggests that external causal attributions for negative events lead to reduced self-actual:self-ideal discrepancies, but increased self-actual:other-actual discrepancies.

The self-discrepancies of paranoid patients, depressed patients and normal subjects were examined using a modified version of Higgins' Selves Questionnaire (Higgins, 1987). Normal subjects showed high consistencies between all domains of the self-concept, while depressed subjects showed marked self-discrepancies. Paranoid patients alone displayed a high degree of consistency between self-perceptions and self-guides together with discrepancies between self-perceptions and the believed perceptions of parents about the self. Paranoid patients also believed that their parents had more negative views of them than did other subjects.

These findings are clearly consistent with the theoretical model of paranoid ideation.

15.2.3 An Analogue Study of the relationship between Attributional Style and Self-Discrepancies

The central aspect of the model (externalizing causal attributions for negative events) has been supported in several independent empirical studies (Candido & Romney, 1990; Fear, Sharpe, & Healy, 1996; Kaney & Bentall, 1992; Kinderman & Bentall, 1996; Lyon, Kaney, & Bentall, 1994), and were largely antiplicated by Raps, Peterson, Reinhard and Seligman (1982). Similar results were also found in the study described in Chapter 8. The study described in Chapter 8 also revealed a pattern of self-discrepancies consistent with the attributional model. However, these studies are persuasive but not conclusive. Chapter 9 described an experimental investigation of the dynamic processes inherent in the model, using nonpatient participants.

One hundred and twenty undergraduate students completed a questionnaire measure of the actual-self, ideal-self and self-as-perceived-by- others derived from Higgins' (1987) Self-Discrepancy Theory. After completing this questionnaire, participants were required to make causal attributions for negative events before completing the measure of self-representations for a second time. Internal attributions for negative events were associated with larger initial discrepancies between self-perceptions and self-ideals. Discrepancies between self-perceptions and ideals increased following internal, but not external, attributions for failure experiences. These findings offer partial support for some aspects of the attributional model of paranoia, in that external attributions for negative events appeared self-protective.

The impact of external attributions on self-actual:other-actual discrepancies was initially less clear. External attributions appeared to reduce self-actual:other-actual discrepancies. This is counter to predictions. However, post hoc analyses revealed differences between types of external attributions with potentially important theoretical, methodological and clinical consequences. External attributions, if made to

personal factors, resulted in increases in discrepancies between self- perceptions and the perceived views of others. Attributions to situational factors were found to result in reduced discrepancies in both domains.

This study supported the general principles of the attributional model of paranoia outlined in Chapter 6, but suggested important modifications. Most importantly the differences between internal, external-personal and external-situational attributions needed addressing.

15.2.4 The Internal, Personal and Situational Attributions Questionnaire

Attributional style is commonly assessed using Peterson and colleague's (1982) Attributional Style Questionnaire (ASQ), the principal measure used in previous investigations of paranoid thought. The ASQ has been criticised for its low internal reliability, particulary regarding the internality subscales. The study described above, and in Chapter 9, has pointed to the potential utility of taxonomies of causal locus not included in the ASQ. Chapter 10 described the development of a new method of assessing causal locus, the Internal, Personal and Situational Attributions Questionnaire (IPSAQ).

Two measures of internality are derived from responses on the questionnaire, a measure of self-blame and a measure of the extent to which external attributions implicate other persons as opposed to situations. In a group of non-patient participants, the IPSAQ subscales were adequately reliable. Self-blame was significantly associated with ASQ internality scores and with depressed mood. Scores representing the proportion of personal as opposed to situational external attributions were significantly associated with an analogue measure of paranoia. The IPSAQ was therefore ratified for use in subsequent studies.

15.2.5 A Second Analogue study

The IPSAQ was first employed in a replication of the analogue investigation of the dynamic predictions of the attributional model as outlined in Section 15.2.3 above.

As in the first analogue study, non-patient participants completed a questionnaire derived from Higgins' (1987) Self-Discrepancy Theory before and after completing the IPSAQ. This revealed significant differences between three loci of causal attributions. Internal attributions led to increased self-actual:self-ideal and self actual:other-actual discrepancies; personal-external attributions led to decreased self-actual:self ideal discrepancies and increased self-actual:other-actual discrepancies; situational-external attributions led to decreased self-actual:self-ideal and self-actual:other-actual discrepancies.

15.2.6 Internal, Personal and Situational Attributions in Paranoia

The use of the IPSAQ revealed important differences between internal, personal and situational loci of attribution. The role of these loci in paranoia could be readily predicted on the basis of the second analogue study, but required direct investigation.

Causal attributions for positive and negative hypothetical social events made by paranoid patients, depressed patients and non-patient participants were therefore examined using the IPSAQ. Depressed patients tended to attribute negative social events to internal (self-blaming) causes. Non-patient participants and patients with delusions of persecution tended to avoid such self-blame. However, whereas non-patient participants tended to choose situational or circumstantial external attributions, paranoid patients tended to choose external attributions that located blame in other

15.2.8 Theory of Mind Deficits, Causal Attributions and Paranoia

The final empirical study in this thesis comprised an attempt to understand more of the aetiological bases of the attributional and self-concept abnormalities clarified in previous studies.

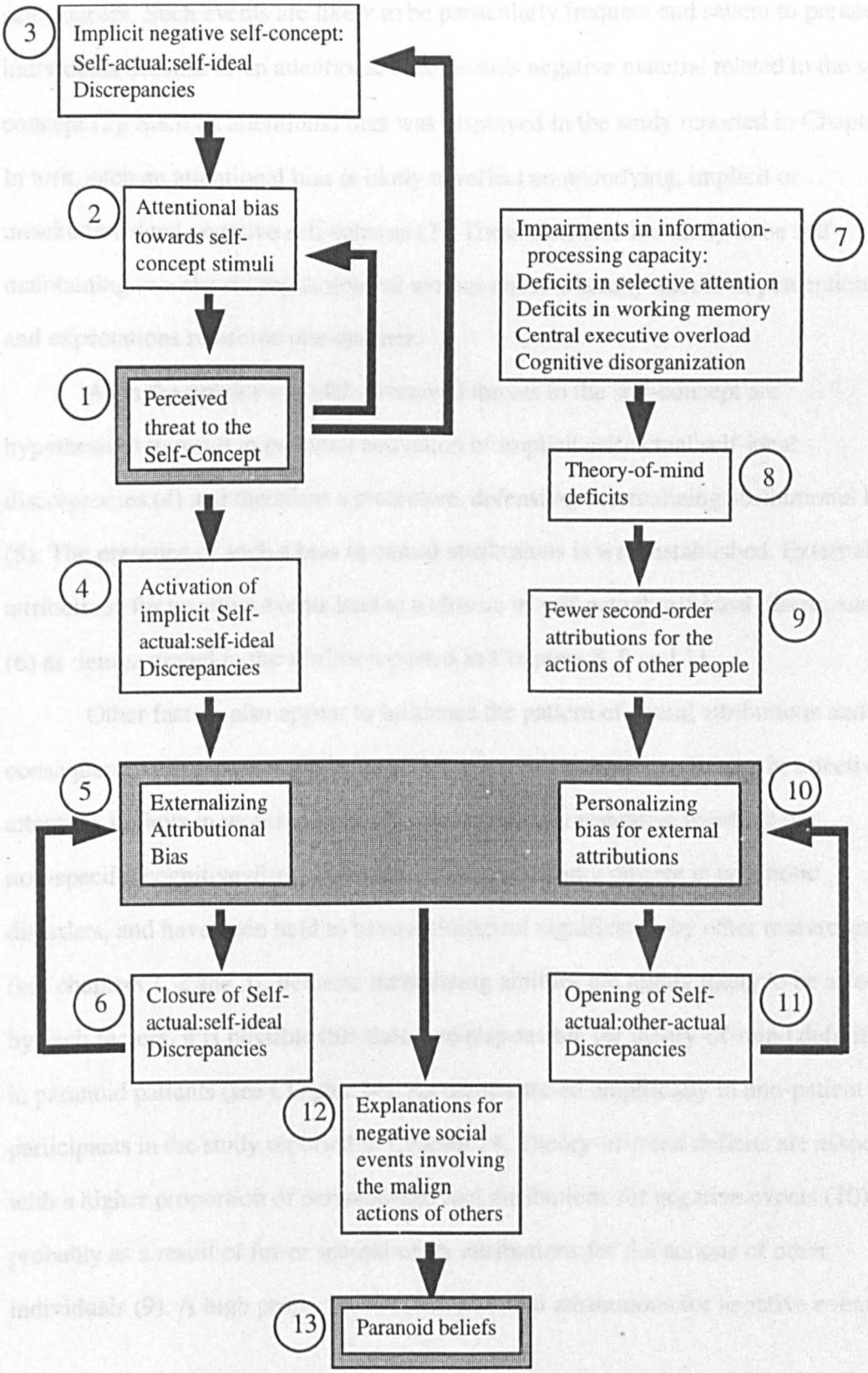
The ability to understand and conceptualize the mental processes of other people is considered to play a vital role in social interactions. Deficits in this area or Theory-of-Mind (ToM) deficits have been identified as playing a possible causal role in autism and Asberger's syndrome. Difficulties in representing the mental states of others have also been suggested as characteristic of schizophrenic disorders, particularly paranoia. Paranoia thus serves as a natural experiment illustrating the role of causal attributions as mediating factors between ToM and social perception. Chapter 14 described a study examining the relationship between attributional processes and ToM deficits. Seventy-seven undergraduate participants completed a ToM task and forty-six also completed the IPSAQ. ToM deficits were associated with an increased tendency to identify other individuals as responsible for negative social situations.

This study may be significant in understanding the roots of a pattern of causal attributions in paranoia that now appears reasonably well-established.

15.3 A Revised Model of Paranoid Ideation

On the basis of the empirical investigations outlined above, together with the research reviewed in the first section of this thesis, a revised model of paranoid ideation may be proposed.

Figure 15.1 A diagrammatic representation of a revised model of paranoid ideation



This model requires some discussion. As in the previous model (Chapter 6), a triggering event is likely to be an event (1) which is perceived as threatening to the self-concept. Such events are likely to be particularly frequent and salient to paranoid individuals because of an attentional bias towards negative material related to the self-concept (2). Such an attentional bias was displayed in the study reported in Chapter 7. In turn, such an attentional bias is likely to reflect an underlying, implicit or unacknowledged negative self-schema (3). These elements are likely to be self-maintaining in a classic psychological vicious circle whereby attention, perceptions and expectations reinforce one-another.

As in the previous model, perceived threats to the self-concept are hypothesised to result in potential activation of implicit self-actual:self-ideal discrepancies (4) and therefore a protective, defensive, externalizing attributional bias (5). The presence of such a bias in causal attributions is well-established. External attributions for negative events lead to a closure of self-actual:self-ideal discrepancies (6) as demonstrated in the studies reported in Chapters 8, 9 and 11.

Other factors also appear to influence the pattern of causal attributions and their consequences. Impairments in information-processing capacity, deficits in selective attention, deficits in working memory, general central executive overload or non-specific cognitive disorganization (7) are commonly present in psychotic disorders, and have been held to have aetiological significance by other researchers (see chapters 1, 2 and 3). Because mentalizing abilities are highly likely to be affected by such factors, it is possible that these are responsible for theory-of-mind deficits (8) in paranoid patients (see Chapter 14). As demonstrated empirically in non-patient participants in the study reported in Chapter 14, Theory-of-mind deficits are associated with a higher proportion of personal-external attributions for negative events (10), probably as a result of fewer second-order attributions for the actions of other individuals (9). A high proportion of such personal attributions for negative events

(10) are characteristic of paranoid patients, as revealed in Chapter 12.

As revealed in the second analogue study, described in Chapter 11, a pattern of external attributions for negative events (5), of which a high proportion are personal (10) leads to an opening of self-actual:other-actual discrepancies (11). This pattern may now be considered characteristic of paranoid individuals. This pattern is likely to be reinforced in a circular reaction by the consequential changes in self-discrepancies. Small self-actual:self-ideal discrepancies are likely to be associated with fewer internal attributions for negative events. Large self-actual:other-actual discrepancies are likely to result in blame being attached to other individuals.

Because of this pattern of attributions, individuals are likely to generate explanations for negative social events that involve the malign actions of other people (12). Such a pattern is likely to result ultimately in paranoid beliefs (13).

15.4 General Limitations of the Studies

Specific limitations in the individual empirical studies have been discussed in previous chapters. There remain, however, some general limitations to the work carried out in this thesis that are worth discussing.

15.4.1 Malevolence of Paranoid Attributions

One possible criticism of the general attributional model of paranoia proposed here is that is essentially self-evident. That is, paranoia is characterised by a belief that other people are responsible for one's misfortunes. To suggest that paranoid individuals believe that the bad things that happen are the responsibility of other people

is hardly surprising.

This criticism is, however, worthy of little attention. The variety and scope of the attributional and self-concept abnormalities revealed in the studies comprising this thesis are such that a comprehensive theoretical model of paranoid ideation can be constructed. The utility of this model is evidenced in two ways. As Karl Popper (Popper, 1968) suggested, scientific knowledge proceeds not towards truth, but away from falsehood. One consequence of this is that an increasing number of questions are posed as one appears to clarify the situation. Comparison of figures 6.1 with 15.1 appears to suggest that our understanding of paranoia is becoming increasingly detailed, with an increasing number of potential avenues of investigation. More significantly, the utility of the theoretical model is supported by its apparent utility in developing clinical formulations.

However, out of this criticism stems another. Clinical experience suggests that paranoid attributions for negative events do not simply implicate other individuals. Rather, malevolent intent directed at the individual is suggested. Such malevolent, directed, intent is not fully incorporated in the design of the IPSAQ. Although it could be argued that the IPSAQ contains a more thorough taxonomy of attributional loci than the ASQ, it is severely limited. Heider (1958) discussed a hierarchy of personal responsibility in causal attributions, depending on the degree to which a person intended an outcome to happen. This hierarchy is outlined below:

Level I - Global Association	The individual is merely associated with the outcome
Level II - Causality	The individual caused the outcome, but accidentally and in such a way that it could not have been foreseen
Level III - Foreseeability	The individual caused the outcome accidentally, but it could have been foreseen that such an accident could occur
Level IV - Intentionality	The outcome was caused deliberately, but without justification
Level V - Justification	The individual caused the outcome intentionally, and with justification

Such a hierarchy does not apply directly to paranoia, as the actions of malevolent others cannot be said to be justified, but it seems likely that paranoid attributions lie at an equivalent to Level V. That is, paranoid individuals appear to believe that a variety of negative events are attributable to the deliberate, intentional actions of other individuals, done with the knowledge of that individual and performed with the aim of harming the patient.

Moreover, clinical experience suggests that paranoid individuals believe that these actions are initiated partially as a response to the patient themselves. That is, paranoid individuals may typically believe that changes in the taste of foods are due to other individuals attempting to poison them. Moreover, this campaign of poisoning may be believed to be conducted because of the patients' special knowledge or information concerning politicians or royalty.

Clearly the IPSAQ, as currently designed, fails to address these issues. Both issues, moreover, have yet to be investigated empirically. Further research may clarify these issues.

15.4.2 Aetiology of Attributional Biases

Although I hope the revised model of paranoid attributions outlined above appears comprehensive, large gaps remain. The most striking of these is aetiological. In Figure 15.1, cognitive disorganization and an implicit negative self-concept are factors extrinsic to the model. However, these factors are entirely inadequate as causes of paranoia. As I argued in chapters 1, 2 and 3, many competitor models of paranoia can be criticised because the factors implicated are neither sufficient nor necessary. Neither cognitive disorganization nor an implicit negative self-concept appear sufficient for the development of paranoia. Many patients suffer from severe cognitive impairment and have a negative self-concept without becoming paranoid.

There are many directions in which research may develop. First, the role of these two elements may be clarified. They may, for instance, be necessary for the development of paranoia if not sufficient. Other routes to paranoid attributions also require investigation.

15.4.2.1 Family Dynamics

One possibility is that the pattern of causal attributions observed here reflects pathological relationships with family members. Family relationships have frequently been suggested as aetiologically important in schizophrenic disorders in general (Jortner, 1990; Laing & Esterson, 1968) and paranoid disorders in particular (Heilbrun & Norbert, 1972; Kaffman, 1983; Schatzman, 1973). Moreover, parental attributions and criticism may play a role in the maintenance of severe psychiatric disorders (Barrowclough, Johnston, & Tarrier, 1994; Vaughn & Leff, 1976). Brewin (Brewin, MacCarthy, Duda, & Vaughn, 1991) in particular, has suggested that the

attributions of family members concerning the behaviour of sufferers are particularly important. Patient-blaming attributions are believed to be particularly damaging and are, of course, personal, other-blaming attributions.

There are two possible sub-theories here. First, it is possible that attributional style is learned directly. There seems sufficient evidence to conclude that general cognitive patterns are familially transmissible (Hammen, 1991; Hammen, Dyck, & Micklovitch, 1986; Hammen, Marks, Mayall, & De Mayo, 1985). In particular causal attributions may be learned from parents (Hammen, et al., 1986). It is quite simple to imagine how paranoid, other-blaming, attributions may be learned. First, observing one's parents exhibiting a pattern of personal attributions may simply lead to the occurrence of this pattern in the child. With young children, issues of fault and blame are frequently raised. It is relatively simple to imagine the difference between methods of addressing this issue.

These issues have not, to my knowledge, been examined. From personal experience, however, children accept and reject "fault" for accidents. To respond by telling a child that incidents are "your fault", "his fault" or "my fault" may be very frequent. Parents who take trouble to address the importance of "no-one's fault, sometimes things just happen and it's no-body's fault" may create a different attributional schema than those who allocate blame.

More pathologically, it is possible to imagine self-perpetuating familial relationships that may reinforce paranoid attributions. Children may occasionally be the targets of inappropriate blame from their parents, echoing Brewin's (Brewin, et al., 1991) observation of patient-blaming attributions. In this issue several dynamics are set up. Self-esteem is threatened. The child observes that external attributions are used for psychological purposes, and learns that these attributions target others. The child also learns that other people, their parents, think ill of them, and feels under threat. A powerful mix results. Such a pattern of behaviour and thought is entirely

speculative. The model of paranoid thought outlined here, however, provides the framework for renewed interest in family dynamics in psychotic illnesses.

Secondly, attributions and self-discrepancies may both be related to parent-child relationships. In this context Higgins (1989) argued that parental involvement with the child, in particular consistent, clear and responsive parenting is important in the development of appropriate self guides. Discrepancies between different self-guides (for instance between parental and personal guides) may be associated with uncertainty, identity confusion and rebelliousness in adolescence. In this context, family processes have been implicated in the cognitive biases found in depressed patients (Hammen, 1991; Rose, Abramson, Hodulik, Halberstadt, & Leff, 1994). The difference between an account of the present data that exclusively emphasizes family relationships and the broader account we have offered therefore concerns the generalizability and origins of the pattern of self-discrepancies observed. In the present study self-actual:parent-ideal and self-actual:parent-ought discrepancies were marked for the deluded patients. This suggests that the aspirations and perceptions of the paranoid individuals may be orthogonal to those of their parents, and such features deserve further study. Further research using methods of assessing patients' perceptions of wider groups of individuals, together with studies that may link family processes with self-discrepancies is likely to advance significantly our understanding of the etiology of paranoid disorders.

15.4.2.2 Theory-of-Mind

The association between ToM deficits and paranoia seems reasonably well-established. This may have limited aetiological significance (as outlined above) by suggesting that a personalizing bias in causal attributions may be due to a paucity in

mentalizing ability.

This idea might, in turn, have other aetiological significance. Frith and Corcoran (submitted) found that mentalizing ability was impaired, as predicted, in people with active symptoms of schizophrenia, but not in people whose illness had remitted. This finding is particularly important, as it suggests that the abnormalities in Theory-of-Mind ability are dependent, in turn, on abnormalities in other processes. In Figure 15.1 I suggested that these may involve impairments in information-processing capacity such as deficits in selective attention, working memory, central executive overload or general cognitive disorganization. Such hypotheses are, of course, highly speculative, but easily testable.

15.4.2.3 Other Dynamic Processes

As reviewed in chapters 1, 2, and 3, many cognitive, generally psychological and psychodynamic processes have been thought to be aetiologically significant in paranoia. It would serve no useful purpose to address these again. However it is worth suggesting that the empirical studies outlined in this thesis strongly indicate that such putative processes are likely to involve the self-concept, and specifically defensive, self-enhancing, interpretations of social situations. It is therefore in such areas that paranoia research should focus. To quote Higgins and Bargh (1987) "salient stimuli attract attention, but relevant schemas guide their processing." (p 376) This thesis has suggested that stimuli salient to paranoid individuals involve the self-concept, and the relevant schemas involve external-personal attributions for negative events.

15.5 The Accidental

Goldwert (1993) commented on the teleological and religious quality of many delusions that "a patient's recovery is heralded by the acceptance of the accidental in life." (p 778). I would echo this. If the situational-external locus of causal attribution is meaningful, it is essentially an acceptance of the accidental. Such an acceptance may be important in many areas of life. As I suggested earlier, it may be the case that children need to learn that some things are no-body's fault. Perhaps patients need to learn that sometimes strange things just happen. Scientists and lay-people both might do well to accept that the reasons for certain phenomena may be discoverable, but are simply at present unknown. Litigants (perhaps especially in the United States of America or in forensic medicine) may do well to be aware that tragedies occasionally occur without blame. It may even be argued that some perceived personal benefits of eastern philosophies lie in their acceptance of the inexorable, but unattributable, patterns of happenstance. An acceptance of the accidental in life may be psychologically important.

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Appendix 1: Diagnostic criteria of ICD-10 Schizophrenia

Paranoid schizophrenia (F20.0) : relatively stable, often persecutory, delusions usually accompanied by auditory hallucinations. Few disturbances of affect, volition or speech.

Hebephrenic schizophrenia (F20.1) : prominent affective changes. Fleeting and fragmentary delusions and hallucinations. Behaviour frequently irresponsible and mannerisms common. Affective disturbance is common, as are negative symptoms. Recommended to be applied only to adolescents and young adults.

Catatonic schizophrenia (F20.2) : prominent psychomotor disturbances that often alternate between extremes such as hyperkinesia and stupor, with odd postures often maintained for hours, and periods of violent agitation common. Vivid dream-like hallucinations sometimes present.

Undifferentiated schizophrenia (F20.3) : general diagnostic criteria for schizophrenia are met but the diagnostic specifications for any one subtype is not met.

Post-schizophrenic depression (F20.4) : a depressive episode, possibly prolonged, following an episode of schizophrenia. Some schizophrenic symptoms must still be present (otherwise the diagnosis would be depression alone), but they should not be florid or prominent (otherwise diagnosis is another subtype).

Residual Schizophrenia (F20.5) : chronic form of schizophrenia in which negative symptoms (psychomotor slowing, underactivity, affective blunting, passivity and amotivation, poverty of speech and nonverbal communication, and poor self-care and social performance) are predominant.

Simple schizophrenia (F20.6) : insidious development of the negative symptoms of residual schizophrenia without previous episode of acute psychotic symptoms. Rather, a "progressive development of oddities of conduct, inability to meet the demands of society and decline in total performance" (p327) is present.

In addition, codes F20.8 (Other schizophrenia) and F20.9 (Schizophrenia, unspecified) are included. Other disorders also classified under the heading of Schizophrenia are the following:

Schizotypal disorder (F21) : eccentric and anomalous thinking resembling schizophrenia but with no characteristic or definite symptoms. Odd ideas may be present, but will not amount to delusions, a tendency to social withdrawal may be observed, as might quasi-psychotic phenomena such as illusions or hallucinations. It is described as similar in course to a personality disorder.

In addition, the subcategory Persistent delusional disorders covers several psychotic conditions highly pertinent to this thesis:

Delusional disorder (F22.0) : one or more delusion is the single observed psychotic phenomenon. Other phenomena (hallucinations, disturbance of affect or volition etc.) are not present.

Other persistent delusional disorders (F22.8) : similar to simple delusional disorder, except for the presence of hallucinations. These must not meet the criteria for other schizophrenic disorders.

Persistent delusional disorder, unspecified (F22.9).

Other possible related ICD-10 diagnoses include: F23.0 Acute polymorphic psychotic disorder without symptoms of schizophrenia, F23.1 Acute polymorphic psychotic disorder with symptoms of schizophrenia, F23.2 Acute schizophrenia-like psychotic disorder, F23.3 Other acute predominately delusional psychotic disorders, F23.8 Other acute and transient psychotic disorders, F23.9 Acute and transient psychotic disorder, unspecified. F234 Induced delusional disorder is described simply as a delusional disorder shared by two or more people. It is claimed that "only one of the people suffers from a genuine psychotic disorder; the delusions are induced in the other(s)" (p. 331). This, of course, poses the question of what the difference between "genuine" and non-genuine delusions are.

ICD-10 also includes schizoaffective disorders, where both schizophrenic and affective symptoms are present, but a diagnosis of neither schizophrenic nor affective disorder is justified. These include: F25.0 Schizoaffective disorder, manic type, F25.1 Schizoaffective disorder, depressive type, F25.2 Schizoaffective disorder, mixed type, F25.8 Other schizoaffective disorders, F25.9 Schizoaffective disorder, unspecified.

As always in such classificatory systems, ICD-10 includes catch-all categories; F28 Other nonorganic psychotic disorders and F29 Unspecified nonorganic psychosis.

Appendix 2: Diagnostic criteria of DSM-IV Schizophrenia

Paranoid Type (295.30) : prominent delusions or frequent auditory hallucinations. Not diagnosed in the presence of prominent: disorganized speech, disorganized or catatonic behaviour or flat or inappropriate affect.

Disorganized type (295.10) : disorganized speech, disorganized behaviour and flat or inappropriate affect.

Catatonic type (295.20) : at least two of the following: motor immobility or stupor, excessive motor activity, extreme negativism (motiveless resistance or the motionlessness), peculiar voluntary movements (posturing or mannerisms) and echolalia or echopraxia (repetition of the words or movements of others).

Undifferentiated type (295.90) : criteria for schizophrenia are met but the criteria for paranoid, disorganized or catatonic sub-types are not met.

Residual type (295.60) : at least one previous diagnosis of schizophrenia but there is an absence of symptoms such as delusions, hallucinations, disorganized speech and grossly disorganized or catatonic behaviour. Evidence of continuing illness. This could be negative symptoms or the positive symptoms to a lesser degree (such as continuing odd beliefs).

Schizophreniform disorder (295.40) : All the symptoms necessary for a diagnosis of schizophrenia are met, including the fact that full symptoms have been seen for one month, but the duration of the disturbance is less than the six months required for schizophrenia.

There are also a number of related conditions not technically termed schizophrenia in DSM-IV. Schizoaffective disorder (295.70), is essentially a mixture of schizophrenic and affective symptoms. Patients with this diagnosis will have an episode of depression or mania and will meet the main criteria of schizophrenia (with the obvious omission of the exclusion criteria). They will experience delusions or hallucinations for at least two weeks without major mood symptoms, but again the condition cannot be diagnosed if drugs or medical conditions might be a cause of the symptoms. Delusional disorder (297.1), is characterized by the presence of non-bizarre delusions in the absence of other criteria for schizophrenia or mood disturbance. Brief psychotic disorder (298.8) comprises the positive symptoms of schizophrenia (delusions, hallucinations, disorganized speech and grossly disorganized or catatonic behaviour) lasting for less than one month. Shared psychotic disorder (297.3) is also called folie à deux and is diagnosed if an individual develops a delusion that shares the characteristic of a delusion already possessed by another person in a close relationship with the patient. DSM-IV also includes: Psychotic disorder due to a general medical condition (293.81 with delusions 293.82 with hallucinations), where it is clear that a physiological, medical, condition causes prominent hallucinations or delusions, Substance-induced psychotic disorder (which is numerically classified according to the substance responsible) and Psychotic disorder not otherwise specified (298.9).

Appendix 3: Diagnostic interview questions

These are questions that I'm going to be asking of everyone I see, so it's likely that some don't apply to you. I'm just going to go through all questions with everyone to be consistent.

To begin with, I should like to get an idea of the sort of problems that led you to come into hospital.
What were the main difficulties?

Have you worried a lot over the past month?
[What do you worry about?]

What is it like when you worry?
Do unpleasant thoughts constantly go round and round in your mind?

Have you had the feeling that something terrible might happen?

Do you ever get the feeling that something odd is going on that you can't explain?

Do you blame anybody else for your troubles?

Is anyone deliberately trying to harm you, for instance trying to poison you or kill you?
[How, is there an organization like the Mafia behind it?]
[Is there any other kind of persecution?]
[How do you explain this?]

Appendix 4: Beck Depression Inventory: BDI

BECK DEPRESSION INVENTORY

NAME . . . . . DATE . . . . . .

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 over the PAST WEEK, INCLUDING TODAY! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

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 anymore
	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 in myself
	3	I hate myself
8.	0	I don't feel I am any worse 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 chance
10.	0	I don't cry anymore 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 usual
	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 the 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 about 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 that make me look unattractive
	3	I believe I look ugly
15.	0	I can work about as well as before
	1	It takes 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 before
	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 more tired 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 5 pounds I am trying to lose weight
2 I have lost more than 10 pounds by eating less YES __ NO ___
3 I have lost more than 15 pounds

20. 0 I am no more worried about my health than usual
1 I am worried about physical problems such as aches and pains, upset stomach, or constipation
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

21. 0 I have not noticed any recent change in my interest in sex
1 I am less interested in sex than I used to be
2 I am much less interested in sex now
3 I have lost interest in sex completely

Appendix 5: Stroop stimulus cards

Blue

Red

Green

Orange

Brown

Note: In the process of duplication, some colour reproduction suffered. The original colours (red, blue, orange, brown and green) were chosen to be maximally distinguishable.

Calm Wise Capable Positive Realistic

Positive Realistic Calm Wise Capable

Realistic Capable Positive Calm Wise

Wise Positive Realistic Calm Capable

Capable Calm Realistic Positive Wise

Positive Calm Wise Capable Realistic

Capable Wise Positive Realistic Calm

Calm Realistic Capable Wise Positive

Realistic Positive Calm Wise Capable

Wise Capable Positive Realistic Calm

Lazy Weak Foolish Childish Obnoxious

Foolish Obnoxious Childish Lazy Weak

Childish Foolish Lazy Weak Obnoxious

Weak Childish Obnoxious Foolish Lazy

Obnoxious Lazy Childish Weak Foolish

Childish Obnoxious Weak Lazy Foolish

Weak Lazy Foolish Childish Obnoxious

Obnoxious Foolish Lazy Childish Weak

Lazy Weak Obnoxious Foolish Childish

Foolish Childish Weak Obnoxious Lazy

Pale Ripe Resident Domestic Hydraulic

Resident Hydraulic Pale Domestic Ripe

Domestic Pale Ripe Hydraulic Resident

Hydraulic Ripe Domestic Resident Pale

Ripe Domestic Hydraulic Pale Resident

Hydraulic Resident Pale Ripe Domestic

Pale Hydraulic Resident Ripe Domestic

Domestic Pale Ripe Hydraulic Resident

Resident Domestic Hydraulic Pale Ripe

Ripe Resident Domestic Hydraulic Pale

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Appendix 6: Personal Profile Questionnaire: PPQ

Name : . . . . . Date : . . . . . .

Personality Profile Questionnaire

Please read through the list of words below, and rate each one on the scale provided according to how much it accurately describes you now.

Please tick one box for **each** word.

	Describes me very well	Describes me a little	Neutral	Does not describe me	Does not describes me at all
1. Sincere					
2. Honest					
3. Understanding					
4. Timid					
5. Loyal					
6. Unpopular					
7. Trustworthy					
8. Intelligent					
9. Dull					
10. Dependable					
11. Bad					

	Describes me very well	Describes me a little	Neutral	Does not describe me	Does not describes me at all
12. Cowardly					
13. Thoughtful					
14. Wise					
15. Crazy					
16. Considerate					
17. Feeble					
18. Pathetic					
19. Reliable					
20. Mature					
21. Untrustworthy					
22. Kind					
23. Foolish					
24. Friendly					
25. Obnoxious					
26. Happy					

	Describes me very well	Describes me a little	Neutral	Does not describe me	Does not describes me at all
27. Insincere					
28. Interesting					
29. Lazy					
30. Cheerful					
31. Helpless					
32. Clumsy					
33. Realistic					
34. Insecure					
35. Optimistic					
36. Mediocre					
37. Nervous					
38. Entertaining					
39. Depressed					
40. Relaxed					
41. Weak					

	Describes me very well	Describes me a little	Neutral	Does not describe me	Does not describes me at all
42. Inefficient					
43. Capable					
44. Nice					
45. Vain					
46. Childish					
47. Skilled					
48. Unfair					
49. Superficial					
50. Popular					
51. Calm					
52. Unpleasant					
53. Boring					
54. Positive					
55. Offensive					
56. Annoying					
57. Normal					

	Describes me very well	Describes me a little	Neutral	Does not describe me	Does not describes me at all
58. Dislikeable					
59. Outstanding					
60. Successful					

Appendix 7: Personal Qualities Questionnaire: PQQ

Personal Qualities Questionnaire

Name : . . . . . Date : . . . . . .

This questionnaire asks questions about what you think you are like - what your personal attributes or characteristics are. Then it asks how you would really <u>like</u> to be, and then how you think you <u>should be.</u>

ie: Actual self : Those attributes you think you *actually* possess.

Ideal self : Those attributes you would *ideally* like to possess, your ultimate goals for yourself.

Ought self : Those attributes you believe you *should* or *ought to* possess.

Other questions ask you how you think people close to you would describe you.

In each case you can put down up to ten different words. If you think it appropriate then you can use the same word in different parts of the questionnaire (For instance you may think that 'amusing' describes you both as you *actually are* <u>and</u> as you would *ideally* like to be).

Please be as honest as you can.

1. HOW YOU ARE NOW

Please write down ten words (qualities or characteristics) which you would use to describe yourself as you ***actually are.*** It might be hard to think of so many, but remember that you can include things you do not like about yourself as well as those you do like. (eg : honest, selfish, caring, demanding etc.)

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

2. HOW YOU WOULD LIKE TO BE

Please write down ten words which describe you as you ***would ideally like to be.*** Some of these qualities might be the same as those you have written in other questions.

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

3. HOW YOU THINK YOU SHOULD BE OR OUGHT TO BE

Please write down ten words which describe you as you think you ***should be or ought to be.*** Some of these qualities might be the same as those you have written in other questions.

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

4. HOW YOUR MOTHER THINKS YOU ARE

Please write down ten words which ***your mother*** would use to describe you ***as she thinks you actually are.*** Some of these qualities might be the same as those you have written in other questions.

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

5. HOW YOUR MOTHER WOULD LIKE YOU TO BE.

Please write down ten words which ***your mother*** would use to describe you ***as she would like you to be.*** Some of these qualities might be the same as those you have written in other questions.

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

6. HOW YOUR MOTHER THINKS YOU SHOULD BE.

Please write down ten words which ***your mother*** would use to describe you ***as she thinks you should be.*** Some of these qualities might be the same as those you have written in other questions.

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

7. HOW YOUR FATHER THINKS YOU ARE

Please write down ten words which ***your father*** would use to describe you ***as he thinks you actually are.*** Some of these qualities might be the same as those you have written in other questions.

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

8. HOW YOUR FATHER WOULD LIKE YOU TO BE.

Please write down ten words which ***your father*** would use to describe you ***as he would like you to be.*** Some of these qualities might be the same as those you have written in other questions.

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

9. HOW YOUR FATHER THINKS YOU SHOULD BE.

Please write down ten words which ***your father*** would use to describe you ***as he thinks you should be.*** Some of these qualities might be the same as those you have written in other questions.

1) . . . . . .

2) . . . . . .

3) . . . . . .

4) . . . . . .

5) . . . . . .

6) . . . . . .

7) . . . . . .

8) . . . . . .

9) . . . . . .

10) . . . . . .

Appendix 8: Attributional Style Questionnaire: ASQ

Attributional Style Questionnaire

Name : . . . . . Date : . . . . . .

Instructions

Please vividly imagine yourself in each of the situations that follow. If such a situation happened to you, what would you feel would have caused it ? While events may have many causes, we want you to pick only one :

THE MAJOR CAUSE IF THIS EVENT HAPPENED TO YOU

Please write the cause in the blank space provided after each event. Next we want you to answer three questions about the cause you provided. First, is the cause of this event something about you or something about other people or circumstances ? Second, is the cause of this event something which will persist across time or something which will never again be present ? Third, is the cause of this event something which affects all situations in your life or just affects this type of event ?

To summarize, we want you to :

1. Read each situation and vividly imagine it happening to you.
2. Decide what you feel would be the one major cause of the situation if it happened to you.
3. Write the cause in the blank space provided.
4. Answer three questions about the cause.
5. Go on to the next situation.

1. **You meet a friend who compliments you on your appearance**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

2. **You have been looking for a job unsuccessfully for some time**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

3. **You become very rich**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

4. **A friend comes to you with a problem and you don't try to help them.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

5. **You give an important talk in front of a group and the audience reacts negatively**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

6. **You do a project which is highly praised**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

7. **You meet a friend who acts hostilely towards you**

Write down the <u>one</u> major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

8. **You can't get all the work done that others expect of you**

Write down the <u>one</u> major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

9. **Your spouse (boyfriend/girlfriend) has been treating you more lovingly**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

10. **You apply for a position that you want very badly (eg: important job, university admission, etc) and you get it**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

11. **You go out on a date and it goes badly**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

12. **You get a raise**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

In the future, in similar situations, will this cause again be present ? (circle one number)

Will never again be present	1 2 3 4 5 6 7	Will always be present

Is this cause something that affects only this type of situation, or does it also influence other areas of your life ? (circle one number)

Influences just this particular situation	1 2 3 4 5 6 7	Influences all situations in my life

Appendix 9: Self-Consciousness Scale: SCS

Self-Consciousness Scale

Name : . . . . . Date : . . . . .

.

Please read the statements listed below and decide how much they reflect how you feel.

Then, please circle the number which best represents how you feel.

		Not at all like me				Very much like me
1.	I'm always trying to figure myself out	0	1	2	3	4
2.	I'm concerned about my style of doing things	0	1	2	3	4
3.	Generally, I'm not very aware of myself	0	1	2	3	4
4.	It takes me time to overcome my shyness in new situations	0	1	2	3	4
5.	I reflect about myself a lot	0	1	2	3	4
6.	I'm concerned about the way I present myself	0	1	2	3	4
7.	I'm often the subject of my own fantasies	0	1	2	3	4
8.	I have trouble working when someone is watching me	0	1	2	3	4
9.	I never scrutinize myself	0	1	2	3	4
10.	I get embarrassed very easily	0	1	2	3	4

		Not at all like me				Very much like me
11.	I'm self-conscious about the way I look	0	1	2	3	4
12.	I don't find it hard to talk to strangers	0	1	2	3	4
13.	I'm generally attentive to my inner feelings	0	1	2	3	4
14.	I usually worry about making a good impression	0	1	2	3	4
15.	I'm constantly examining my motives	0	1	2	3	4
16.	I feel anxious when I have to talk in front of a group	0	1	2	3	4
17.	One of the last things I do before I leave the house is look in the mirror	0	1	2	3	4
18.	I sometimes have the feeling that I'm off somewhere watching myself	0	1	2	3	4
19.	I'm concerned about what other people think of me	0	1	2	3	4
20.	I'm alert to changes in my mood	0	1	2	3	4
21.	I'm usually aware of my appearance	0	1	2	3	4
22.	I'm aware of the way my mind works when I work through a problem	0	1	2	3	4
23.	Large groups make me nervous	0	1	2	3	4

Appendix 10: Self-Concept Checklist: SCC

Self-Concept Checklist 1

This questionnaire is designed to measure what you think you are like as a person. Please read the following list of words and select those which best describe you as you think **YOU ACTUALLY ARE.**

Thus, if you think that any of the words listed below **describes you very well** as you actually are, please put a tick in the box next to that word. If, on the other hand, you think that any of the words listed below **definitely does not describe you** as you actually are, please put a cross in the box. If a word does not apply to you, and neither does its opposite, leave it blank.

ie : 1: Male ☐
2: Female ☐

Would you say you were ...

Childish	☐	Qualified	☐	Bright	☐
Ambitious	☐	Untrustworthy	☐	Competent	☐
Incapable	☐	Assured	☐	Listless	☐
Skilled	☐	Authoritative	☐	Unqualified	☐
Achieving	☐	Dull-witted	☐	Confident	☐
Determined	☐	Helpless	☐	Decisive	☐
Incompetent	☐	Hesitant	☐	Disappointing	☐
Able	☐	Lazy	☐	Failed	☐
Accomplished	☐	Unprofessional	☐	Inept	☐
Amateur	☐	Unreliable	☐	Productive	☐
Capable	☐	Unskilled	☐	Intelligent	☐
Dull	☐	Proficient	☐	Unproductive	☐
Blundering	☐	Mediocre	☐	Outstanding	☐
Uninspiring	☐	Gifted	☐	Aimless	☐
Brilliant	☐	Creative	☐	Awkward	☐
Independent	☐	Reliable	☐	Efficient	☐
Inefficient	☐	Talented	☐	Expert	☐
Masterful	☐	Inefficient	☐	Petty	☐
Purposeful	☐	Resourceful	☐	Successful	☐
Inexperienced	☐	Unintelligent	☐	Weak	☐

Please read the following list of words and select those which best describe you as you think **YOU WOULD IDEALLY LIKE TO BE.**

Thus, if you think that any of the words listed below **describes very well** how you would ideally like to be, please put a tick in the box next to that word. If, on the other hand, you think that any of the words listed below **definitely does not describe you** as you would ideally like to be, please put a cross in the box. If a word does not apply to you, and neither does its opposite, leave it blank.

ie : 1: Male ☐
2: Female ☐

Would you say you were ...

Childish	☐	Qualified	☐	Bright	☐
Ambitious	☐	Untrustworthy	☐	Competent	☐
Incapable	☐	Assured	☐	Listless	☐
Skilled	☐	Authoritative	☐	Unqualified	☐
Achieving	☐	Dull-witted	☐	Confident	☐
Determined	☐	Helpless	☐	Decisive	☐
Incompetent	☐	Hesitant	☐	Disappointing	☐
Able	☐	Lazy	☐	Failed	☐
Accomplished	☐	Unprofessional	☐	Inept	☐
Amateur	☐	Unreliable	☐	Productive	☐
Capable	☐	Unskilled	☐	Intelligent	☐
Dull	☐	Proficient	☐	Unproductive	☐
Blundering	☐	Mediocre	☐	Outstanding	☐
Uninspiring	☐	Gifted	☐	Aimless	☐
Brilliant	☐	Creative	☐	Awkward	☐
Independent	☐	Reliable	☐	Efficient	☐
Inefficient	☐	Talented	☐	Expert	☐
Masterful	☐	Inefficient	☐	Petty	☐
Purposeful	☐	Resourceful	☐	Successful	☐
Inexperienced	☐	Unintelligent	☐	Weak	☐

Please read the following list of words and select those which best describe you as you think **THE OTHER PEOPLE IN YOUR YEAR THINK YOU ACTUALLY ARE.**

Thus, if you think that any of the words listed below **describes you very well** as the other people in your year think you actually are, please put a tick in the box next to that word. If, on the other hand, you think that any of the words listed below **definitely does not describe you** as they think you actually are, please put a cross in the box.

ie : 1: Male ☐
2: Female ☐

Would you say you were ...

Childish	☐	Qualified	☐	Bright	☐
Ambitious	☐	Untrustworthy	☐	Competent	☐
Incapable	☐	Assured	☐	Listless	☐
Skilled	☐	Authoritative	☐	Unqualified	☐
Achieving	☐	Dull-witted	☐	Confident	☐
Determined	☐	Helpless	☐	Decisive	☐
Incompetent	☐	Hesitant	☐	Disappointing	☐
Able	☐	Lazy	☐	Failed	☐
Accomplished	☐	Unprofessional	☐	Inept	☐
Amateur	☐	Unreliable	☐	Productive	☐
Capable	☐	Unskilled	☐	Intelligent	☐
Dull	☐	Proficient	☐	Unproductive	☐
Blundering	☐	Mediocre	☐	Outstanding	☐
Uninspiring	☐	Gifted	☐	Aimless	☐
Brilliant	☐	Creative	☐	Awkward	☐
Independent	☐	Reliable	☐	Efficient	☐
Inefficient	☐	Talented	☐	Expert	☐
Masterful	☐	Inefficient	☐	Petty	☐
Purposeful	☐	Resourceful	☐	Successful	☐
Inexperienced	☐	Unintelligent	☐	Weak	☐

Appendix 11: Attributional Style Inventory: ASI

Attributional Style Inventory

Name : . . . . . Date : . . . . . .

Instructions

Please vividly imagine yourself in each of the situations that follow. If such a situation happened to you, what would you feel would have caused it ? While events may have many causes, we want you to pick only one :

THE MAJOR CAUSE IF THIS EVENT HAPPENED TO YOU

Please write the cause in the blank space provided after each event. Then we want you to answer whether the cause of this event is something about you or something about other people or circumstances.

To summarize, we want you to :

1. Read each situation and vividly imagine it happening to you.
2. Decide what you feel would be the one major cause of the situation if it happened to you.
3. Write the cause in the blank space provided.
4. Answer the question <u>about the cause.</u>
5. Go on to the next situation.

1. **You give an important talk in front of a group and the audience reacts negatively**

   Write down the one major cause _____

   Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

   Totally due to the other person or circumstances 1 2 3 4 5 6 7 Totally due to me

2. **You do a project which is criticised.**

   Write down the one major cause _____

   Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

   Totally due to the other person or circumstances 1 2 3 4 5 6 7 Totally due to me

3. **You have been looking for a job unsuccessfully for some time**

   Write down the one major cause _____

   Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

   Totally due to the other person or circumstances 1 2 3 4 5 6 7 Totally due to me

4. **You become very poor**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

5. **You can't get all the work done that others expect of you**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

6. **You get a pay cut**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

7. **You lose money in a game of cards**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

8. **You perform worse than expected in an examination.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

9. **A colleague, more junior than yourself, is promoted to a job that you also applied for.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

10. **A colleague ignores your advice.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

11. **You are fired from your job.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

12. **After your first term at university, your tutor tells you that your grades are not up to standard.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

13. **You have a lot of trouble understanding what your new employer requires of you.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

14. **You have trouble with one of your tutors.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

15. **You repeatedly fail to pass your driving test.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

16. **You are unable to solve a problem at work.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

17. **An employer describes you as unreliable.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

18. **You have not been given a pay rise for several years.**

Write down the one major cause _____

Is the cause of this due to something about you or something about other people or circumstances ? (circle one number)

Totally due to the other person or circumstances	1 2 3 4 5 6 7	Totally due to me

Appendix 12: Internal, Personal and Situational Attributions Questionnaire

I.P.S.A.Q.

Name: _____ Sex: _____

Age: _____ Occupation: _____

Date Completed: _____

INSTRUCTIONS

Please read the statements on the following pages. For each statement please try to vividly imagine that event happening to you. Then try to decide what was the main cause of the event described in each statement. Please write the cause you have thought of in the space provided. Then tick the appropriate letter (a,b or c) according to whether the cause is :

a) Something about you
b) Something about another person (or a group of people)
c) Something about the situation (circumstances or chance)

It might be quite difficult to decide which of these options is exactly right. In this case, please pick **one option**, the option which **best** represents your opinion. Please pick **only one** letter in each case.

Thank you for your time and co-operation.

Note For Users

This scale was designed by Peter Kinderman and Prof. Richard P. Bentall, of the Department of Clinical Psychology, Whelan Building, P.O. Box 147, Liverpool, L69 3BX, based on previous work by McArthur (1972) and Bentall, Kaney and Dewey (1991). The scale is a research tool and should not be used for routine clinical assessment. Permission is granted for its use in research protocols on condition that the authors are first notified.

References

Bentall, R.P., Kaney, S., & Dewey, M.E. (1991) Paranoia and social reasoning: An attribution theory analysis. British Journal of Clinical Psychology, 30, 13-23.
McArthur, L.A. (1972) The how and what of why: Some determinants and consequences of causal attribution. Journal of Personality and Social Psychology, 22, 171-193.

1. **A friend gave you a lift home.**

What caused your friend to give you a lift home?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

2. **A friend talked about you behind your back.**

What caused your friend to talk about you behind your back?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

3. **A friend said that he(she) has no respect for you.**

What caused your friend to say that he(she) has no respect for you ?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

4. **A friend helped you with the gardening.**

What caused your friend to help you with the gardening?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

5. A friend thinks you are trustworthy.

What caused your friend to think you are trustworthy?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

6. A friend refused to talk to you.

What caused your friend to refuse to talk to you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

7. A friend thinks you are interesting.

What caused your friend to think you are interesting?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

8. A friend sent you a postcard.

What caused your friend to send you a postcard?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

9. A friend thinks you are unfriendly.

What caused your friend to think that you are unfriendly?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

10. A friend made an insulting remark to you.

What caused your friend to insult you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

11. A friend bought you a present.

What caused your friend to buy you a present .
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

12. A friend picked a fight with you.

What caused your friend to fight with you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

13. A friend thinks you are dishonest.

What caused your friend to think you are dishonest?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

14. A friend spent some time talking to you.

What caused your friend to spend time talking with you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

15. A friend thinks you are clever.

What caused your friend to think you are clever?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

16. A friend thinks you are sensible.

What caused your friend to think that you were sensible?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

17. A friend refused to help you with a job.

What caused your friend to refuse to help you with the job?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

18. A friend thinks you are unfair.

What caused your friend to think that you are unfair?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

19. A friend said that he(she) dislikes you.

What caused your friend to say that he(she) dislikes you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

20. A friend rang to enquire about you.

What caused your friend to ring to enquire about you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

21. A friend ignored you

What caused your friend to ignore you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

22. A friend said that she(he) admires you.

What caused your friend to say that she(he) admired you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

23. A friend said that he(she) finds you boring.

What caused your friend to say that he(she) finds you boring?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

24. A friend said that she(he) resents you.

What caused your friend to say that she(he) resents you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

25. A friend visited you for a friendly chat.

What caused your friend to visit you for a chat?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

26. A friend believes that you are honest

What caused your friend to believe that you are honest?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

27. A friend betrayed the trust you had in her.

What caused your friend to betray your trust?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

28. A friend ordered you to leave.

What caused your friend to order you to leave?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

29. A friend said that she(he) respects you.

What caused your friend to say that she(he) respects you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

30. A friend thinks you are stupid.

What caused your friend to think that you are stupid?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

31. A friend said that he(she) liked you.

What caused your friend to say that he(she) liked you?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

32. A neighbour invited you in for a drink.

What caused your friend to invite you in for a drink?
(Please write down the one major cause)

.....

Is this :

a. Something about you ?
b. Something about the other person or other people ?
c. Something about the situation (circumstances or chance) ?

Appendix 13: Paranoia Scale: PS

Please read each of the following statements and indicate how far it applies to you by circling a number on the scale.

	Not at all applicable to me				Extremely applicable to me
1. Someone has it in for me	1	2	3	4	5
2. I sometimes feel as if I'm being followed	1	2	3	4	5
3. I believe that I have often been punished without cause	1	2	3	4	5
4. Some people have tried to steal my ideas and take credit for them	1	2	3	4	5
5. My parents and family find more fault with me than they should	1	2	3	4	5
6. No one really cares much what happens to you	1	2	3	4	5
7. I am sure I get a raw deal from life	1	2	3	4	5
8. Most people will use somewhat unfair means to gain profit or advantage, rather than lose it	1	2	3	4	5
9. I often wonder what hidden reason another person may have for doing something nice for you	1	2	3	4	5
10. It is safer to trust no one	1	2	3	4	5

	Not at all applicable to me				Extremely applicable to me
11. I have often felt that strangers were looking at me critically	1	2	3	4	5
12. Most people make friends because friends are likely to be useful to them	1	2	3	4	5
13. Someone has been trying to influence my mind	1	2	3	4	5
14. I am sure I have been talked about behind my back	1	2	3	4	5
15. Most people inwardly dislike putting themselves out to help other people	1	2	3	4	5
16. I tend to be on my guard with people who are somewhat more friendly than I expected	1	2	3	4	5
17. People have said insulting and unkind things about me	1	2	3	4	5
18. People often disappoint me	1	2	3	4	5
19. I am bothered by people outside, in cars, in stores, etc. watching me	1	2	3	4	5
20. I have often found people jealous of my good ideas just because they had not thought of them first	1	2	3	4	5

Appendix 14: Selves Questionnaire (modified)

Personal Qualities Questionnaire

We are interested in how you see yourself, and especially in how this self-perception changes. We know that self-perceptions vary on a moment-to-moment basis.

This questionnaire asks questions about what you think you are like - what your personal attributes or characteristics are. Then it asks how you would really like to be, and then how you think other people see you.

Actual self : Those attributes you think you *actually* possess.

Ideal self : Those attributes you would *ideally* like to possess, your ultimate goals for yourself.

Others' views: Those attributes you believe *other people* think you possess.

In each case you can put down up to ten different words. If you think it appropriate then you can use the same word in different parts of the questionnaire (For instance you may think that 'amusing' describes you both as you *actually are* and as you would *ideally* like to be and as you think *other people* see you).

After you have written down your list of words,
please give each word a rating from **1** to **10.**

That is, in the *Actual-Self* domain, give a rating of **1** if you possess that characteristic only slightly or a very little, up to **10** if you possess that characteristic very much or extremely.
In the *Ideal-Self* domain, give a rating of **1** if you would like to possess that characteristic only slightly or a very little, up to **10** if you would wish to possess that characteristic very much or extremely.
And, of course, in the *Other people's views* domain, give a rating of **1** if you feel that other people think you possess that characteristic only slightly or a very little and **10** if you feel that other people think you possess that characteristic very much or extremely

It is very important that you answer this questionnaire as you feel right now. As I mentioned, we are interesting in moment-to-moment changes in self-perception.

Please be as honest as you can.

1. HOW YOU ARE NOW

Please write down ten words (qualities or characteristics) which you would use to describe yourself as you **actually are.** It might be hard to think of so many, but remember that you can include things you do not like about yourself as well as those you do like. (eg : honest, selfish, caring, demanding etc.)

Now, please rate the degree to which you possess each characteristic on a scale from **1** to **10**.

1 = slightly or a very little
2
3
4
5 = moderately or quite a lot
6
7
8
9
10 = extremely or very much

1) . . . . . ☐

2) . . . . . ☐

3) . . . . . ☐

4) . . . . . ☐

5) . . . . . ☐

6) . . . . . ☐

7) . . . . . ☐

8) . . . . . ☐

9) . . . . . ☐

10) . . . . . ☐

2. HOW YOU WOULD LIKE TO BE

Please write down ten words which describe you as you **would ideally like to be.** Some of these qualities might be the same as those you have written in other questions.

Now, please rate the degree to which you would like to possess each characteristic on a scale from **1** to **10**.

1 = slightly or a very little
2
3
4
5 = moderately or quite a lot
6
7
8
9
10 = extremely or very much

1) . . . . . ☐

2) . . . . . ☐

3) . . . . . ☐

4) . . . . . ☐

5) . . . . . ☐

6) . . . . . ☐

7) . . . . . ☐

8) . . . . . ☐

9) . . . . . ☐

10) . . . . . ☐

3. WHAT OTHER PEOPLE THINK YOU ARE LIKE.

Please write down ten words which **other people** would use to describe you **as they think you actually are.** Some of these qualities might be the same as those you have written in other questions.

Now, please rate the degree to which you believe that other people would say that you possess each characteristic on a scale from **1** to **10**.

1 = slightly or a very little
2
3
4
5 = moderately or quite a lot
6
7
8
9
10 = extremely or very much

1) . . . . . ☐

2) . . . . . ☐

3) . . . . . ☐

4) . . . . . ☐

5) . . . . . ☐

6) . . . . . ☐

7) . . . . . ☐

8) . . . . . ☐

9) . . . . . ☐

10) . . . . . ☐

Personal Qualities Questionnaire

Version Two

This is a repetition of the questionnaire you filled in earlier. As we said before, we are interested in how the way you see yourself varies on a moment-to-moment basis.

To assess these changes, we would like you to complete this questionnaire a second time. As before, we are interested in your :-

Actual self :	Those attributes you think you *actually* possess.
Ideal self :	Those attributes you would *ideally* like to possess, your ultimate goals for yourself.
Others' views:	Those attributes you believe *other people* think you possess.

ANSWER THIS QUESTIONNAIRE AS YOU FEEL RIGHT NOW.

The scoring is the same as before :-

In each case you can put down up to ten different words. If you think it appropriate then you can use the same word in different parts of the questionnaire (For instance you may think that 'amusing' describes you both as you *actually are* and as you would *ideally* like to be and as you think *other people* see you).

After you have written down your list of words,
please give each word a rating from **1** to **10**.

That is, in the Actual-Self domain, give a rating of **1** if you possess that characteristic only slightly or a very little, up to **10** if you possess that characteristic very much or extremely.
In the Ideal-Self domain, give a rating of **1** if you would like to possess that characteristic only slightly or a very little, up to **10** if you would wish to possess that characteristic very much or extremely.
And, of course, in the Other people's views domain, give a rating of **1** if you feel that other people think you possess that characteristic only slightly or a very little and **10** if you feel that other people think you possess that characteristic very much or extremely

IT IS VERY IMPORTANT THAT YOU ANSWER THIS QUESTIONNAIRE AS YOU FEEL RIGHT NOW. DO NOT LOOK BACK TO PREVIOUS ANSWERS, OR EVEN TRY TOO HARD TO REMEMBER THEM, JUST TRY TO DESCRIBE YOURSELF AS YOU FEEL **RIGHT NOW**.

Please be as honest as you can.

Appendix 15: Idiographic Patient Record Booklet

Personal Record Booklet

It is important that I get a good idea about the sorts of problems which you are experiencing at the moment. Discussing things that have already happened is fine, but a better way of getting to know how you feel is to record it on a day-to-day basis. I would like you, every day, to record how you feel in this booklet.

Please take a few moments, each evening, to complete a record of how you have been feeling that day.

I would like to know :

a) How sad or depressed you have been feeling
b) How you have felt about yourself as a person
c) How worried you have felt about other people

Please record how you have felt each day by putting a mark [√] on the appropriate line at the position that you feel best represents your mood. Please fill in the form in the evening and think of how you have been feeling that day.

There is a form for each day of the week. Please try to complete one each day. Keep this booklet private, and remember to fill in a page every day.

Date :

Today I have felt :

Very Happy	Very Sad or Depressed
Very good about myself as a person	Very bad about myself as a person
Not at all worried about any conspiracy	Extremely worried about a conspiracy

Appendix 16: Imposing Memory Task

An Imposing Memory Task

This study is part of on-going research into how people see their world. We are particularly interested in what people remember of social situations, and how they explain some of the things that happen to them. There are two parts to the study, each part has a different questionnaire. It is VERY IMPORTANT that we keep both the questionnaires together.To do this we would like you to think of any three-digit number (137, 735) and write it on the top of BOTH questionnaires. That way, if the questionnaires get separated, we can correlate the answers.

This booklet is the first questionnaire, and relates to the first task.

You will be read a set of five short stories, one at a time.
Please listen very carefully to each one as it is read out, because you will be asked to answer questions about it later.
In this booklet are the questions about the stories. DO NOT LOOK AT THE BOOKLET UNTIL ASKED TO DO SO.

What we would like you to do is : Listen to the stories as they are being read out

When the first story is finished, turn over to the first page
Answer the questions about the first story

Each question is composed of two statements
What you have to do is chose the correct statement of the two

When you have finished the questions about the first story,

WAIT!!

When you have listened to the second story, turn over to the second set of questions
This pattern will continue.

DON'T turn over the pages until AFTER the next story has been read out

Thank you for your interest

WHERE'S THE POST OFFICE ?

Sam wanted to find a Post Office so he could buy a Tax Disc for his car. He asked Henry if he could tell him where to get one. Henry told him that he thought there was a Post Office in Elm Street. When Sam got to Elm street, he found it was closed. A notice on the door said that it had moved to new premises in Bold Street. So Sam went to Bold Street and found the new Post Office. When he got to the counter, he discovered that he had left his MOT certificate at home. He realized that without an MOT certificate, he could not get a Tax Disc, so he went home empty-handed.

Where's the Post Office ?

Please tick the correct answer to each question:

1. (a) Sam wanted to go to the Post Office to buy a stamp
   (b) Sam wanted to go to the Post Office to buy a Tax Disc

2. (a) Henry thought Sam would find the Post Office in Elm Street
   (b) Henry thought Sam would find the Post Office in Bold Street

3. (a) The Post Office had moved from Bold Street to Elm Street
   (b) The Post Office had moved from Elm Street to Bold Street

4. (a) Sam thought that Henry knew the Post Office was in Bold Street
   (b) Sam thought that Henry knew the Post Office was in Elm Street

5. (a) The Post Office in Elm Street had a notice in the window saying it had moved to Bold Street
   (b) The Post Office in Elm Street had a notice on the door saying it had moved to Bold Street

6. (a) Sam thought that Henry believed that Sam wanted to buy a Tax Disc
   (b) Sam thought that Henry did not know that Sam wanted to buy a Tax Disc

7 (a) When Sam got to Bold Street to buy his Tax Disc, he realised that he wouldn't be able to buy it because he had forgotten his MOT certificate.
   (b) When Sam got to Bold Street to buy his Tax Disc, he realised that he wouldn't be able to buy it because he had forgotten his insurance certificate.

JOHN'S PROBLEM

It was nearly the end of the day, John thought it might be nice to go to the pub for a drink after work. At first, he wasn't sure whom he should ask to go with him. He very much wanted to ask Sheila, whom he fancied, but he thought that she didn't like him enough to want to give up her aerobics class to go drinking with him. He could, of course ask Pete, his usual drinking companion. Pete was always happy to spend an hour or two in the pub before going home. Then he happened to see Penny. He knew that Penny was one of Sheila's friends. Penny might be able to help him out. She would know whether Sheila would be willing to go out for a drink rather than go to her aerobics class. "Listen Penny," he said, "I thought I might go for a drink after work. I was going to ask you and Sheila if you wanted to come. Would you ask Sheila whether she would like to come for a drink with us ?" Penny looked surprised. John had never asked her to go out with him before, but she thought that he was very keen on Sheila. She began to suspect that John wanted to find out whether she knew what Sheila might want to do.

John's Problem - Please tick the correct answer to each question :

1. (a) The story was set in the morning
   (b) The story was set in the afternoon

2. (a) John wanted to go home after work
   (b) John wanted to go to the pub after work

3 (a) After work, Sheila was going to an aerobics class
   (b) After work, Sheila was going home

4. (a) John thought Sheila would not like to go to the pub with him
   (b) John thought Sheila would like to go to the pub with him

5. (a) John and Pete often went for a drink together
   (b) John and Pete only rarely went for a drink together

6. (a) John thought that Penny knew what Sheila wanted to do
   (b) John thought that Penny did not know what Sheila wanted to do

7. (a) John's friend, Pete, occasionally went for a drink, but never after work, always going home immediately
   (b) John's friend, Pete, occasionally went for a drink in the evening, after work

8. (a) Penny believed that John thought she would not know what Sheila would want to do
   (b) Penny believed that John was hoping she would know what Sheila would want to do

9. (a) John spoke to Penny, but neither Sheila or Pete, about going for a drink after work
   (b) John spoke to Penny and Pete, but not Sheila, about going for a drink after work

10. (a) John thought that Penny thought that John wanted Penny to find out what Sheila wanted to do because John wanted to go out with Sheila on her own
   (b) John thought that Penny thought that John wanted Penny to find out what Sheila wanted to do because John wanted to go out with them both

11 (a) Penny, the woman that John spoke to about asking Sheila about going for a drink after work, after he had thought of asking Pete, was a friend of Sheila
   (b) Penny, the woman that John spoke to about asking Sheila about going for a drink after work, after he had thought of asking Pete, did not know Sheila

EMMA'S DILEMMA

Emma worked in a greengrocer's. She wanted to persuade her boss to give her an increase in wages. So she asked her friend Jenny, who was still at school, what she should say to the boss. "Tell him that the chemist near where you live want's you to work in his shop." Jenny suggested. "The boss won't want to lose you, so he will give you more money" she said. So when Emma went to see her boss, that is what she told him. Her boss thought that Emma might be telling a lie, so he said he would think about it. Later, he went to the chemist's shop near Emma's house and asked the chemist whether he had offered a job to Emma. The chemist said he hadn't offered Emma a job. The next day the boss told Emma that he wouldn't give her an increase in wages, and she could take the job at the chemist's instead.

Emma's Dilemma

Please tick the correct answer to each question :

1. (a) Emma worked for a greengrocer
   (b) Emma worked in a chemist's

2. (a) Emma wanted more money
   (b) Emma wanted a different job

3 (a) Emma's friend, Jenny, was still at school
   (b) Emma's friend, Jenny, worked in a bank

4. (a) Jenny thought the boss would believe Emma's story
   (b) Jenny knew the boss would not believe Emma's story

5. (a) Emma told her boss, the greengrocer, that she had been offered a job in an bank
   (b) Emma told her boss, the greengrocer, that she had been offered a job in an chemist's

6. (a) Emma thought the boss believed that the chemist wanted her to work for him
   (b) Emma thought the boss knew that the chemist had not offered her a job

7. (a) Emma's boss, the greengrocer, asked the chemist if he had offered Emma a job
   (b) Emma's boss, the greengrocer, asked Jenny if Emma had been offered a job

8. (a) Jenny thought that Emma hoped that the boss would believe that the chemist wanted Emma to work for him
   (b) Jenny thought that Emma believed that the boss knew that the chemist did not want Emma to work for him

9. (a) The chemist's shop, where Jenny had suggested that Emma tell her boss that she had been offered a job, was near where Emma lived
   (b) The chemist's shop, where Jenny had suggested that Emma tell her boss that she had been offered a job, was in a different town

SIMON THINKS

Simon was 19 years old and worked as a mechanic. His cousin, Jim, was quite a lot older, and worked as a milkman. Because he got up early in the morning, he seldom went out in the evening. Jim's friend, Edward, worked in a bank, and therefore had more opportunity to go out in the evenings. Simon knew that Jim wanted to marry Susan. Simon also knew that Jim believed that Susan wanted to marry Edward. So he thought that if he could convince Jim that Susan thought that Edward wanted to marry Betty, Jim might be persuaded that Susan would say "Yes" if he asked her to marry him.

Simon Thinks

Please tick the correct answer to each question :

1. (a) Simon worked as a mechanic
   (b) Simon worked in a greengrocers

2. (a) Jim wanted to marry Susan
   (b) Jim did not want to marry Susan

3. (a) Jim's friend, Edward, worked in a bank
   (b) Jim's friend, Edward, worked as a mechanic

4. (a) Simon believed that Jim was convinced that Susan would not marry him
   (b) Simon thought that Jim though that Susan would marry him

5. (a) Simon, who was 19 years old, was Jim's cousin
   (b) Simon, who was 19 years old, was Jim's brother

6. (a) Jim believed that Susan thought that Edward would like to marry Betty
   (b) Jim thought that Susan knew that Edward did not want to marry Betty

7. (a) Because Jim worked as a milkman, and Edward worked in a bank, neither went out very often
   (b) Because Jim worked as a milkman, but Edward worked in a bank, Edward went out more often than Jim

8. (a) Simon hoped that Jim would believe that Susan thought that Edward wanted to marry Betty
   (b) Simon thought that Jim would believe that Susan thought that Edward did not want to marry Betty

9. (a) Edward's friend, Jim, who was Simon's cousin, was older than Simon, who was 19
   (b) Edward's friend, Jim, who was Simon's cousin, was younger than Simon, who was 19

A MACABRE TALE

It was late and the old man felt sleepy. Still, he thought, he would have one last cigarette before going to bed. He lit up and puffed quietly in the half light as he watched the flickering screen of the TV. It wasn't long before he fell asleep in the chair. The cigarette he was holding fell down the side of his chair. There it smouldered until the material caught light. This in turn set fire to the foam padding. As the foam burned, it gave off poisonous fumes which killed the old man. When the police discovered the tragedy in the morning, they called the fire-brigade fire investigation team. They found that the fire had spread to the carpet and its rubber underlay, which had burned fiercely. Examining the scene, they quickly realized where the fire had started, and what had killed the old man.

A Macabre Tale

Please tick the correct answer to each question :

1. (a) The old man fell asleep in his chair
   (b) The old man fell asleep in bed

2. (a) The fumes from the foam padding killed the old man
   (b) The fumes from the material of the chair killed the old man

3. (a) When the old man fell asleep, his cigarette fell into his lap and burned a hole in his trousers
   (b) When the old man fell asleep, his cigarette fell down the side of the chair and set light to the material

4. (a) When the old man fell asleep, his cigarette fell down the side of the chair and set light to the material. The foam in the chair gave of poisonous fumes which killed him
   (b) When the old man fell asleep, his cigarette fell onto the floor. It set light to the rubber underlay of the carpet which gave off poisonous fumes that killed him