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Precursors for Schizophrenia: Are Schizotaxia and Schizotypy Related?

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A thesis submitted for the degree of Doctor of Philosophy at the University of Otago, Dunedin, New Zealand.

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ABSTRACT

Meehl's (1962, 1989, 1990b) schizotypy and Tsuang et al.'s (1999b, 2000a, 2000b) schizotaxia are fundamentally different notions of the schizophrenia precursor. Both represent a categorical precursor but differ in the nature of their relationships to schizophrenia. Specifically, schizotypy is dimensional, unchanging despite the presence or remission of schizophrenia. In contrast, schizotaxia is a transitional precursor; the presence of schizophrenia signals the end of schizotaxia. There are also differences in the way in which risk is determined. Schizotypy is reflected in a variety of information processing and experiential aberrations, is typically assessed using self-report measures, and is best identified using taxometric analyses. In contrast, schizotaxia is characterised by negative symptoms of schizophrenia and neurocognitive impairment, can be assessed using standardised clinical measures, and is diagnosed at the individual case level.

The aim of Phase 1 of this study was to investigate the manifest structure of Meehl’s schizotypy in a sample of psychiatric patients. The aims of Phase 2 were to determine if schizotypy group membership was associated with poorer functioning and to determine the nature of the relationship between Meehl’s (1962, 1989, 1990b) schizotypy and Tsuang et al.’s (1999b, 2000a, 2000b) schizotaxia. Participants in Phase 1 were 109 psychiatric patients and all completed a self-report measure of schizotypy, the Thinking and Perceptual Style Questionnaire (TPSQ; Linscott & Knight, 2004). Multivariate taxometric analyses of TPSQ subscales yielded evidence of a manifest group structure within the sample. The prevalence of the latent group, presumed to reflect schizotypy, was estimated to be 32% (SD = 8%), as yielded by MAXCOV analyses. MAXCOV analyses also yielded a mean indicator validity of 1.02; variance of 7; base rate estimates
of .08; and a goodness of fit index of .98. MAMBAC analyses yielded a mean base rate of 56\% (SD = 18\%).

Twenty-nine participants from Phase 1 took part in Phase 2. Fourteen were from the schizotypy group (had a \( p \) value of .85 or higher of schizotypy group membership) and 15 from the nonschizotypy group (had a \( p \) value of .03 or lower of schizotypy group membership). Participants completed tests of attention, verbal memory, and executive functioning. Negative symptoms of schizophrenia were also rated and diagnosis was determined using a diagnostic interview. The schizotypy group was significantly impaired relative to the nonschizotypy group on neuropsychological test scores spanning domains of attention, verbal memory, and executive functioning. A current DSM-IV diagnosis was made for 71\% of the schizotypy group and 43\% of the nonschizotypy group. Individuals were classified as having met criteria for schizotaxia if they had a negative symptom impairment and a neuropsychological impairment in two domains. A total of 7 people of 29 met criteria for schizotaxia, 6 of these people were from the schizotypy group. There was statistical evidence that Meehl's (1962, 1989, 1990b) schizotypy and Tsuang et al.'s (1999b, 2000a, 2000b) schizotaxia are not independent. The proposed precursors for schizophrenia may reflect the same construct, not separate entities. Limitations and implications of these results are considered.
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Schizotaxia and schizotypy are thought to be states that precede schizophrenia. The overall aim of this thesis was to investigate the nature of the relationship between schizotaxia and schizotypy. This was undertaken by comparing and contrasting two notions of risk for schizophrenia. One of these theories views the schizophrenia-spectrum disorders as quantitatively different from normality while the other views the schizophrenia-spectrum disorders as qualitatively different from normality. It is not clear from the research how the two conceptualisations are related to each other. This thesis attempts to address this problem and clarify the relationship between the schizophrenia-spectrum disorders.

In Chapter 2, the concept of schizophrenia is examined. This begins with a review of the historical origins of schizophrenia dating back to Bleuler (1911/1950) and Kraepelin (1919/2002). The current conceptualisations of schizophrenia, including the features of schizophrenia, are also appraised. Diagnostic systems of classification, such as the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994), and the International Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10; World Health Organisation, 1992) will be briefly described. Recent research has identified a number of problems with
these diagnostic systems and these problems have many implications for how schizophrenia is clinically evaluated. These criticisms will be introduced and discussed.

Over the past few decades the area of research into predisposition and risk for schizophrenia has expanded. One notion of risk for schizophrenia is Meehl’s (1962, 1989, 1990b) quasi-dimensional theory. This will be introduced in Chapter 3. Meehl (1962, 1990b) proposed a theory of predisposition for schizophrenia involving a genetic liability that he called schizotaxia. He further hypothesised that virtually all people who have schizotaxia develop a personality organisation that he labeled schizotypy. Risk for schizophrenia has been investigated using two main strategies. One of these strategies involves participants who are recruited on the basis of their genetic relationships to individuals with schizophrenia and the other strategy involves participants who are identified as having high scores on psychometric indicators of risk. This type of research has contributed to developing an understanding of the genetic and environmental factors involved in risk for schizophrenia. In relation to Meehl’s (1962, 1989, 1990b) theory, the focus has been on psychometric research involving the construct of schizotypy. This has considered the relationship between schizotypy and correlates such as neuropsychological, psychophysiological and psychopathological functioning, the later development of schizophrenia, and schizotypal personality disorder. The structure of Meehl’s schizotypy has also been considered.

Research in the area of risk for schizophrenia has tended to focus on the measurement of Meehl’s schizotypy rather than schizotaxia. Recently, however, Tsuang and colleagues (Tsuang et al., 1999b; Tsuang, Stone, & Faraone, 2000a, 2000b) developed a number of research criteria for measuring schizotaxia. They have based their conceptualisation of schizotaxia on a modification of Meehl’s original theory. Tsuang et al.’s (1999b, 2000a, 2000b) categorical theory of risk for schizophrenia will be discussed
in Chapter 4. Their conceptualisation of schizotypia is similar to Meehl's schizotypy yet Tsuang and colleagues maintain that it is very different. The differences and similarities between Meehl's theory and Tsuang and colleagues theory will be discussed. Tsuang and colleagues have developed a set of research criteria for schizotypia based on research of target features for schizophrenia. The small number of empirical studies that have been carried out looking at the criteria for schizotypia and the effectiveness of medications at alleviating the symptoms of schizotypia will be reviewed. Tsuang and colleagues have advocated for their conceptualisation of schizotypia to be incorporated into diagnostic systems and for more treatment studies to be conducted. Before these steps can be taken it needs to be clearly established that their view of schizotypia is valid and reliable. As their conceptualisation is based on Meehl's (1962, 1990b) theory of risk, it is obvious that the relationship between the constructs, as conceptualised by both groups of researchers, needs to be determined. More specifically, the degree of similarity or overlap between Meehl's schizotypy and Tsuang and colleagues' schizotypia needs to be investigated before further research can take place.

Chapter 5 introduces taxometric analysis (Golden, 1982; Golden & Meehl, 1979; Meehl 1973; Meehl & Yonce, 1994, 1996; Waller & Meehl, 1998), a type of empirical statistical analysis. These procedures can be used to identify the underlying structure of a construct (such as schizotypy, psychopathy, or depression) in terms of whether it is taxonic or dimensional. The theory, application, and interpretation of these procedures will be discussed. Then the empirical evidence, implications, and limitations of the procedures will be reviewed with particular reference to schizotypy.

In Chapter 6, the key conceptual points made in the previous five chapters are brought together to emphasise the rationale of the current study. The first empirical study (Phase 1) is described in Chapter 7. The aim of Phase 1 was to investigate the latent
structure of Meehl’s (1962, 1989, 1990b) schizotypy in a psychiatric sample. A self-report measure of schizotypy was administered to a group of psychiatric inpatients and outpatients. Taxometric analyses were then applied to the responses to determine if two groups exist: a schizotypy group and a nonschizotypy group. Taxometric analyses identified a qualitative boundary in the psychiatric sample. This confirms that the manifest structure of schizotypy in the psychiatric sample is taxonic, as opposed to dimensional.

The aim of Phase 2 (described in Chapter 8) was to determine the nature of the relationship between Meehl’s (1962, 1989, 1990b) schizotypy and Tsuang et al.’s (1999b, 2000a, 2000b) schizotaxia. Sub-samples of participants from Phase 1 who had extreme probabilities of belonging to the schizotypy group took part in Phase 2. Participants were administered a number of standardised neuropsychological measures that assessed the domains of attention, verbal memory, and executive functioning. In addition, the presence of negative symptoms was also assessed and a semi-structured clinical interview was administered. Statistical analyses were conducted to attempt to answer 2 questions: firstly, is schizotypy group membership associated with poorer functioning; and secondly, are schizotaxia and schizotypy independent. It was found that people in the schizotypy had impaired performance on the measures of functioning relative to the nonschizotypy group. To answer the second question, it was first determined who in the Phase 2 sample met Tsuang et al.’s (1999b, 2000a, 2000b) criteria for schizotaxia. Then the degree of overlap between Meehl’s (1962, 1989, 1990b) schizotypy and Tsuang et al.’s (1999b, 2000a, 2000b) schizotaxia was evaluated using a chi-square analysis. A statistical result was observed indicating that schizotypy and schizotaxia are dependent, as opposed to independent, of each other.
The implications of the findings of this thesis for the understanding of risk of schizophrenia are discussed in Chapter 9. There are a number of potential consequences for the conceptualisation of the constructs of schizotaxia and schizotypy, as viewed by both Meehl (1962, 1989, 1990b) and Tsuang and colleagues (1999b, 2000a, 2000b). In particular, these findings raise questions as to the way in which the Tsuang and colleagues’ construct of schizotaxia is assessed, diagnosed, and treated.
CHAPTER 2

The Identification of Schizophrenia

The conceptualisation of schizophrenia has been through many transformations and developments over the past century yet there are still problems with the classification systems that are used today. The problems with classification systems and the definition of schizophrenia have implications for many facets of psychopathology. These include the areas of incidence, the role of features of schizophrenia, diagnosis, comorbidity, and aetiology.

Historical Origins of Schizophrenia

The conceptualisation of mental illness, including psychosis and schizophrenia, has varied greatly across time and dates back to before the first century (Palha & Esteves, 1997). Some of the early explanations for madness include supernatural causes, witchcraft, and possession by the devil (Palha & Esteves, 1997). In the 18th and 19th centuries, models of psychosis involving anatomy and brain pathology began to emerge, along with new terminology. The term *dementia praecox* was first used by Benedict Morel (1809-1873), a French physician, and was defined as an early or premature loss of mind with onset frequently occurring in adolescence (Morel, 1852, cited in Gottesman, 1991). He described the case of an adolescent boy who had initially appeared to be intelligent and outgoing but experienced a gradual mental deterioration which resulted in his case being viewed as hopeless (cited in Gottesman, 1991). Morel proposed that the
cause of dementia praecox was entirely hereditary (Gottesman, 1991). Around the mid-1800s, two German psychiatrists, Kahlbaum and Hecker (cited in Adityanjee et al., 1999), described different forms of psychosis. They were catatonia, which referred to the symptoms of patients who remained physically immobile and did not react to any form of external stimuli; and hebephrenia (or hebetic paraphrenia), which referred to patients who experienced hallucinations, delusions, and odd behaviour (Shean, 2004). Kahlbaum was instrumental in establishing a focus on the importance of the course of psychosis (Adityanjee et al., 1999).

It was following this that Emil Kraepelin, a German psychiatrist, brought together the conceptualisations of schizophrenia of the time and organised his own classification structure to describe dementia praecox. Kraepelin (1919/2002) classified individuals on the basis of their symptoms and the course of their disorder. He described a number of symptoms that he believed were common to dementia praecox. These symptoms fell into two broad categories: (a) “a weakening of those emotional activities which permanently form the mainspring of volition” (Kraepelin, 1919/2002, p.74), currently considered to be negative symptoms (Andreasen, 1997); and (b) “the loss of the inner unity of the activities of intellect, emotion, and volition in themselves and among one another” (Kraepelin, 1919/2002, p.74-75), also known as bizarre or disorganised thought and behaviours. These symptoms included impairments of perception and attention, hallucinations, thought disorder, unusual sexual sensations, confabulation, impaired mental efficiency, delusions, flat affect, diminished volition, impulsive and repetitive behaviour, odd behaviour, autism or stupor, changes in personality, impaired work functioning, mutism, and disordered word-finding (Kraepelin, 1919/2002). Kraepelin also described a number of physical symptoms that he considered to be indicative of dementia praecox but acknowledged that these had not been fully researched.
Kraepelin (1919/2002) initially divided dementia praecox into 3 subtypes: hebephrenic, catatonic, and paranoid. Later, he described 9 subtypes of dementia praecox: dementia simplex, silly dementia, simple depressive dementia, delusional depressive dementia, circular dementia, the agitated dementias, catatonia, the paranoid dementias, and confusional speech dementia. All subtypes were thought to progress to an end-state of dementia (Kraepelin, 1919/2002).

Dementia simplex was proposed as a subtle and mild form of the dementia praecox symptoms. Kraepelin suggested that in some cases dementia simplex may precede other forms of dementia praecox and result in a progression towards severe dementia praecox while others may experience a partial recovery. Silly dementia, also called hebephrenia, was related to dementia simplex, and characterised by a period of mania (involving grandiose delusions) followed by depression as well as fluctuating emotions and an odd writing style (Kraepelin, 1919/2002).

Kraepelin proposed that simple depressive dementia began with a depressive period followed by a gradual decline in functioning. It was also characterised by hallucinations, delusions of sin or persecution, and impaired volition, with a minority of individuals expected to experience any recovery (Kraepelin, 1919/2002). Delusional depressive dementia was dominated by the presence of delusions as well as auditory hallucinations, which were thought to have a profound effect on behaviour (Kraepelin, 1919/2002). The group of agitated dementias covered circular dementia which was characterised by depression and delusions, with approximately half of individuals thought to experience improvement and then relapse; and agitated dementia which was characterised by a period of excitement, hallucinations, delusions, and fluctuating mood. Kraepelin (1919/2002) described catatonia as often beginning with a period of depression, followed by symptoms of excitement or mania, and then a state of stupor of varying severity. The paranoid
dementias included paranoid dementia gravis and paranoid dementia mitis, both characterised by paranoid delusions but the difference being in the severity of the symptoms. Kraepelin (1919/2002) included confusional speech dementia as a type of dementia praecox where an individual’s presentation was dominated by derailment of speech and nonsense language.

Although Kraepelin (1919/2002) described a number of subtypes of dementia praecox, he also emphasised the theory that dementia praecox is a single entity. Rieder (1974) pointed out that Kraepelin did this in 3 main ways. Firstly, he highlighted the symptoms that were common across the subtypes of dementia praecox; secondly, he pointed out that the symptoms began in adolescence and progressed to an end state; and thirdly, he conjectured that there was a common aetiology involving a single process in brain functioning fundamental to the progression of the dementia praecox. In addition, Kraepelin (1919/2002) held a categorical view of dementia praecox and made a clear distinction between dementia praecox, which he considered to be a progressive disorder, and manic-depressive psychosis, an episodic disorder.

Kraepelin was influential for the longitudinal approach that he took when observing the symptoms people experienced (Hoch, 1960). From this Kraepelin (1919/2002) reported that he observed periods of remission in 26% of his cases and that typically this did not last longer than 3 years before relapse occurred. He further maintained that levels of premorbid functioning would never be obtained by individuals who had experienced dementia praecox. In regards to the aetiology of dementia praecox, Kraepelin (1919/2002) considered young age to be a risk factor and more males than females to be affected. He observed that dementia praecox frequently occurred in siblings and placed emphasis on the role of hereditary predisposition in the development of the disorder. Kraepelin also suggested that obstetric complications, such as premature birth, may have a role in the
aetiology of dementia praecox and that this is expressed in the form of physical abnormalities. Furthermore, Kraepelin (1919/2002) thought that dementia praecox involved structural changes to the motor cortex, frontal and temporal lobes of the brain.

Kraepelin has been criticised to some extent because although he explored several psychosocial factors such as prison, prostitution, and physical illness, he disregarded any impact that these factors may have on dementia praecox (Shean, 2004). Questions have also been raised as to why Kraepelin used the category of manic-depressive psychosis to cover the disorders of mania, depression, and bipolar disorder, rather than considering bipolar and unipolar depression as distinct concepts (Angst, 2002). In addition, there have been debates about how to diagnose an individual who did not progress to an end-state of terminal dementia and whether this was still dementia praecox (Shean, 2004).

During the time that Kraepelin was organising his classification structure for dementia praecox, Eugen Bleuler, a Swiss psychiatrist, introduced the term *schizophrenia* (1911/1950). Bleuler used schizophrenia to describe what he considered to be one of the key features of the disorder: "the 'splitting' of the different psychic functions" (1911/1950, p. 9). He established the new term for two key reasons. Firstly, Bleuler (1911/1950) claimed that dementia praecox described the disease but not the individual; and secondly, *praecox* implied that the onset of the disorder was in adolescence followed by a progression to a terminal state. Bleuler (1911/1950) maintained that this was not always the outcome.

Bleuler (1911/1950) divided the symptoms of schizophrenia into 2 categories: fundamental symptoms and accessory symptoms. He conjectured that the fundamental symptoms were present in all individuals with schizophrenia and involved simple functions and compound functions. The simple functions that were impaired in schizophrenia included thought processes (association); emotional functioning (affect);
and contradictory emotions, will, and intellect (ambivalence) (Bleuler, 1911/1950). The compound functions that were impaired in schizophrenia included withdrawal from reality (autism), attention, and volition (Bleuler, 1911/1950). Bleuler (1911/1950) considered the accessory symptoms to occur in a range of disorders in addition to schizophrenia and proposed that they did not have a diagnostic function. The accessory symptoms included delusions, hallucinations, catatonia, pressured speech, and secondary physical symptoms. Bleuler’s consideration of fundamental and accessory symptoms is thought to correspond to the concepts of negative and positive symptoms used today (Andreasen, 1997). It has been suggested that although Bleuler aimed to narrow the diagnostic criteria for schizophrenia he ironically broadened the criteria because of the difficulty and ambiguity that arose in observing some of the symptoms he described (Shean, 2004).

In regards to the aetiology of schizophrenia, Bleuler (1911/1950) agreed with Kraepelin’s view of a biological role but he also incorporated the impact of psychological factors. Bleuler (1911/1950) acknowledged that stressful events do not cause schizophrenia but may exacerbate symptoms or trigger the manifestation of symptoms that arise as the result of a genetic predisposition. He described this as “an abnormal reaction of the already altered psyche” (Bleuler, 1911/1950, p.346). He also discussed the impact of internal and external factors on an individual’s prognosis or outcome and this was influenced by Freud’s psychodynamic philosophies. Bleuler (1911/1950) disagreed with Kraepelin’s view that schizophrenia was a progressive disorder where all individuals progressed to an end state with perhaps temporary recoveries. To demonstrate this, he highlighted a number of cases where individuals had experienced periods of long remission, both with and without relapse.

Bleuler’s perspective prevailed throughout much of the early 1900s. It became clear, however, by the middle of the 20th century, that an increase in the diagnostic
reliability of schizophrenia was required (Andreasen, 1997). Schneider (1959, cited in Shean, 2004) attempted this by creating an emphasis on the more dramatic symptoms of schizophrenia, such as hallucinations and delusions. As a consequence these positive symptoms became the most prominent part of the construct of schizophrenia at the time (Andreasen, 1997). Schneider believed that he had captured the symptoms that were essential to the disorder of schizophrenia (Shean, 2004). He placed emphasis on the presenting symptoms rather than on the course of the disorder as Kraepelin and Bleuler had. Schneider’s (1959, cited in Shean, 2004) 11 first-rank symptoms can be seen in Table 2.1, he proposed that any one of the symptoms indicated the presence of schizophrenia.

Table 2.1

Schneider's First-rank Symptoms

<table>
<thead>
<tr>
<th>Symptoms</th>
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<tr>
<td>1 The patient hears voices speaking his or her thoughts aloud.</td>
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<tr>
<td>2 The patient hears two or more voices talking about him or her.</td>
</tr>
<tr>
<td>3 Hallucinated voices describe the patient’s actions as they happen.</td>
</tr>
<tr>
<td>4 Bodily sensations are imposed by an external force.</td>
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<tr>
<td>5 Thought withdrawal (the patient feels that their thoughts are extracted by an external force).</td>
</tr>
<tr>
<td>6 Thought insertion (thoughts that are not the patient’s are inserted among his or her own thoughts).</td>
</tr>
<tr>
<td>7 Thought broadcast (the patient experiences his or her thoughts being transmitted to others).</td>
</tr>
<tr>
<td>8 Alien feelings are imposed by an external force.</td>
</tr>
<tr>
<td>9 Alien impulses are imposed by an external force.</td>
</tr>
<tr>
<td>10 “Volitional” actions are imposed by an external force.</td>
</tr>
<tr>
<td>11 Perceptions are delusional and not understandable.</td>
</tr>
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</table>
Questions have been raised as to the methods Schneider used when he chose the first-rank symptoms and why he considered these to be more central to the diagnosis of schizophrenia than other symptoms (Crichton, 1996). Schneider has also been criticised for failing to emphasise that many of the first-rank symptoms are not exclusive to schizophrenia and can be observed in a variety of other disorders (Varga & Kroll, 1977). In addition, research has shown that Schneider's first-rank symptoms may not be valid cross-culturally. In a study of people from Saudi Arabia with schizophrenia, Zarrouk (1978) found that 56% of 92 individuals endorsed first-rank symptoms. This is in contrast with a study by Mellor (1970) who found 71.7% of 166 individuals from England with schizophrenia had first-rank symptoms. Zarrouk (1978) suggested that the discrepancy was partly due to traditional beliefs held by Saudi Arabians that trusting in the supernatural is normal but the discrepancy could also be due to a diagnostic bias. Despite these flaws, Schneider's first-rank symptoms have been used widely. The symptoms have been incorporated into a number of structured interviews and diagnostic tools, including the *Diagnostic and Statistical Manual of Mental Disorders* and the *International Classification of Diseases and Related Health Problems*.

**Current Diagnostic and Classification Systems**

The aim of the first edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 1952) was to create diagnoses that were more standardised. However, schizophrenia was described in rather general terms in the DSM and was heavily influenced by Bleuler's conceptualisation. Bleuler's approach also prevailed in the DSM-II (APA, 1968). The definition of schizophrenia in both the DSM and DSM-II has been criticised for being too vague (Tsuang et al., 2000a). A key goal of DSM-III (APA, 1980) was to increase the reliability of schizophrenia diagnoses.
and this was achieved to some extent. Schneider's first-rank symptoms were influential in this edition and psychosis was essential to the definition of schizophrenia.

Currently, the DSM-IV (APA, 1994) describes a number of specific criteria that a person must meet in order to be diagnosed with schizophrenia and its various subtypes. The key symptoms involve the presence of 2 or more positive and/or negative symptoms that can involve the distortion or loss of a wide range of areas of functioning. The positive symptoms include delusions, hallucinations, disorganised speech, and disorganised or catatonic behaviour; while the negative symptoms include affective flattening, alogia, and avolition (APA, 1994). These symptoms can be manifested in a variety of forms. Only one of these symptoms is necessary if an individual experiences bizarre delusions or if their hallucinations are comprised of a voice that provides an ongoing commentary on the individual's behaviour or thoughts, or two or more voices talking with each other (APA, 1994). Furthermore, the symptoms must be associated with a significant impairment in social or occupational functioning. The symptoms must be present for the majority of a 1-month period, which is called the active phase (APA, 1994). In addition, there must be evidence of the presence of the disorder for at least 6 months, in either a prodromal, active, or residual form (APA, 1994). A diagnosis of schizophrenia is only given if the individual's symptoms are not better accounted for by another disorder, or the physiological effects of a substance or medical condition. The DSM-IV includes 5 subtypes of schizophrenia which are characterised by the main symptom a person presents with at the time they are assessed. The subtypes are: paranoid type (dominated by delusions and hallucinations), disorganised type (dominated by disorganised speech and behaviour and flat affect), catatonic type (dominated by catatonic symptoms), undifferentiated type (active-phase symptoms are present but do not meet criteria for the
previous 3 subtypes), and residual type (symptoms are present but criteria are not met for active-phase symptoms; APA, 1994).

The *Tenth Revision of the International Statistical Classification of Diseases and Related Health Problems* (ICD-10; World Health Organisation, 1992) is the other major classification system used for the diagnosis of schizophrenia. The DSM-IV and the ICD-10 criteria for schizophrenia are generally similar but have 2 main differences. The DSM-IV requires an impairment in social or occupational functioning which is not required in the ICD-10. In addition, the DSM-IV requires evidence of the presence of schizophrenia for a minimum of 6 months compared to 1 month in the ICD-10. The subtypes of schizophrenia in both the ICD-10 and DSM-IV are very similar except that the DSM-IV disorganised type is called hebephrenic schizophrenia in the ICD-10.

The DSM-IV and ICD-10 diagnostic systems are used widely around the world. The use of these systems has a direct impact on incidence and prevalence rates of schizophrenia. This will be considered next.

*Incidence of Schizophrenia*

With current conceptualisations of schizophrenia, general prevalence rates vary from 0.2% to 2.0% (APA, 1994) and the incidence rate is between 20 and 50 per 100,000 per year (Jablensky, 2000). Prevalence and incidence rates vary across countries and the methods by which the information is obtained (Jablensky, 1997). In 1994, 5% of all mental health first-admissions to hospitals in New Zealand were for a diagnosis of schizophrenia (New Zealand Health Information Service, 1998). For mental health first-admissions, the age-standardised rate of schizophrenia was 12.2 per 100,000 for males and 6.6 per 100,000 for females in 1994 (New Zealand Health Information Service, 1998). These rates are similar to that found in a large-scale study in Nottingham, England.
Brewin et al. (1997) assessed the occurrence of schizophrenia in Nottingham and found the standardised incidence rate to be 8.7 per 100,000 per year over the period 1992 to 1994. In the period of July 2000 to June 2001 in New Zealand, 2953 people, from a total population of 3.8 million (77.7 per 100,000), were admitted to a public hospital with an ICD-10 diagnosis of schizophrenia (New Zealand Health Information Service, 2004). It is important to note that these data also include readmissions. Patients had a mean in-patient stay of 44.4 days for treatment of schizophrenia in public hospitals (New Zealand Health Information Service, 2004).

The incidence and prevalence rates of schizophrenia are affected by the diagnostic systems that are used to classify people as having schizophrenia. This can vary with the way in which the construct is defined and which features are considered to be indicative of schizophrenia. These features will be considered next.

**Features of Schizophrenia**

Studies have considered the nature of the features that differentiate people with schizophrenia from people without schizophrenia. These studies have found that the features form three general types of symptoms: positive symptoms, negative symptoms, and cognitive or neuropsychological impairment. Positive symptoms include hallucinations, delusions, and thought disorder (Kay, Fiszbein, & Opler, 1987). Negative symptoms include blunted affect, asociality, poverty of speech, and a decrease in spontaneous movements (Minas, Klimidis, Stuart, Copolov, & Singh, 1994). Cognitive impairment includes deficits in executive functioning, attention, and memory.

The features of schizophrenia can contribute to impairments in a number of secondary areas of functioning such as occupational and social functioning. These include difficulties with work, school, parenting, personal self-care, relationships, independent
living, and recreational activities (Mueser & McGurk, 2004). These problems can appear long before the onset of florid positive symptoms such as hallucinations and delusions (Häfner, Löffler, Maurer, Hambrecht, & an der Heiden, 1999).

Positive symptoms have a tendency to be episodic while negative symptoms do not fluctuate to the extent that positive symptoms do (Fenton & McGlashan, 1991). In addition, negative symptoms have been shown to have a strong association with poor psychosocial functioning compared to positive symptoms (Sayers, Curran, & Mueser, 1996). The presence of many negative symptoms in the early stages of schizophrenia is associated with poorer outcome in the long-term (Fenton & McGlashan, 1991). Diagnostic criteria and research have traditionally been dominated by the positive symptoms of schizophrenia. Over the past few decades, attention has turned to the negative symptoms of schizophrenia in an attempt to understand the cognitive mechanisms underlying schizophrenia (Andreasen, 1997).

The classification systems that are currently used incorporate only some of the key features of schizophrenia, namely positive and negative symptoms. This means that the criteria have a focus on psychosis. Neuropsychological impairment is not included in diagnostic systems. This is despite the fact that research over the past decade has indicated that neuropsychological impairment is a central characteristic of schizophrenia (Lewis, 2004). There are a number of other features which researchers consider to be indicative of schizophrenia but that are not included in diagnostic systems. These include language impairments (Andreasen & Grove, 1986), neurological soft signs (e.g., Cuesta et al., 2002; Flashman, Flaum, Gupta, & Andreasen, 1996) and a range of subjective experiences (e.g., Maggini & Raballo, 2004; Myin-Germeys, Delespaup, & DeVries, 2000; Peralta & Cuesta, 1994, 1998). The following section will focus on neuropsychological impairments associated with schizophrenia.
Neuropsychological Features of Schizophrenia

Research has found that individuals with schizophrenia experience a wide range of deficits including impairments in the domains of neuropsychological functioning such as memory, attention, executive functioning, and IQ. Neuropsychological impairment has been shown to be associated with negative symptoms of schizophrenia (Herbener & Harrow, 2004). The impairments in neuropsychological functioning are typically more severe in the acute phase of schizophrenia (Spaulding, Reed, Poland, & Storzbach, 1996). The impairments are also present, however, in the prodromal and residual phases of schizophrenia, to varying degrees of severity (Spaulding et al., 1996).

Memory is an area of cognitive functioning that is often impaired in people with schizophrenia. Impairments in verbal memory in particular are a common feature of schizophrenia. Brébion, Smith, Amador, Malaspina, and Gorman (1997) investigated the link between depression, memory, and schizophrenia symptoms in a group of 31 people with schizophrenia. They observed a negative correlation between severity of symptoms of depression and performance on memory tasks requiring deep encoding where more severe symptoms were associated with poorer memory performance. In addition, a relationship between positive symptoms of schizophrenia and incorrect memory responses was observed where patients with positive symptoms tended to make more false alarms and perseverations (Brébion et al., 1997). Other research has demonstrated an association between impaired verbal memory involving deep encoding and high levels of emotional discomfort and an association between impaired superficial encoding and impaired cognitive symptoms such as poor attention and disorganised thinking (Lysaker, Bell, Greig, & Bryson, 2000). An association has also been observed between impaired verbal
memory and poor motor performance in people with schizophrenia (Manschreck et al., 2000).

The neuropsychological function of attention or vigilance (also called working memory) is frequently impaired in people with schizophrenia. Continuous Performance Tests are generally used to assess this domain and consistent results have been observed across a wide range of studies demonstrating attention impairments in individuals with schizophrenia (e.g., Cornblatt & Keilp, 1994; Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989; Cornblatt & Malhotra, 2001; Gooding & Tallent, 2002). In addition, a relationship has been observed between impaired attention and problems with executive functioning which is another key domain in which individuals with schizophrenia often experience impairments (Gooding & Tallent, 2002). Individuals with schizophrenia have been found to make more perseverative errors and take longer to achieve categories than control individuals without a psychiatric diagnosis (Gooding & Tallent, 2002).

Another neuropsychological domain impaired in individuals with schizophrenia is IQ. Research has shown that people with schizophrenia tend to have lower IQ than normal comparisons (Aylward, Walker, & Bettes, 1984). In addition, studies have found that people with schizophrenia have impaired performance IQ relative to their verbal IQ on standardised intelligence measures (Amminger et al., 2000; Aylward et al., 1984). It has been shown that these differences in IQ may be present prior to the onset of schizophrenia (Amminger et al., 2000).

Many studies have investigated only one or two domains of neuropsychological functioning; however, research has also considered impairments across a range of neuropsychological domains in individuals with schizophrenia. Heinrichs and Zakzanis (1998) conducted a meta-analysis of 22 neuropsychological test variables, the results of which were gathered from 204 studies published between 1980 and 1997. The analysis
covered the domains of memory, attention, intelligence, spatial functioning, executive functioning, language, and motor functioning. The studies involved comparisons of groups of people with schizophrenia with control groups. The meta-analysis yielded moderate to large raw effect sizes and Heinrichs and Zakzanis (1998) observed an impairment rate of 61% to 78% in their review, a relatively large deficit rate. Heinrichs and Zakzanis (1998) postulated that the large deficit rate could potentially be explained by: (i) the notion that neuropsychological functioning exists on a continuum; (ii) neuropsychological impairment is secondary to the primary pathology of schizophrenia; and (iii) a large proportion of people with schizophrenia have impairments in all areas of neuropsychological functioning (Heinrichs & Zakzanis, 1998).

There is clear evidence that a range of neuropsychological impairments are observed in individuals with schizophrenia. Furthermore, research has found that these impairments are present independent of age (Fucetola et al., 2000; Lewis, 2004) and intellectual functioning (Kremen, Seidman, Faraone, & Tsuang, 2001). As with other symptoms of schizophrenia, such as positive and negative symptoms, different individuals can experience different types of impairments (Galderisi et al., 2002; Kremen, Seidman, Faraone, Toomey, & Tsuang, 2004). There is also a noticeable degree of variability in the severity of neuropsychological impairments observed in individuals with schizophrenia. This ranges from severe impairments in a wide range of domains to very mild impairments. In addition, some individuals with schizophrenia may not experience any neuropsychological impairment (e.g., Palmer et al., 1997; Penadés, Gastó, Boget, Catalán, & Salamero, 2001). Palmer et al. (1997) evaluated the neuropsychological performance of a group of 171 people with schizophrenia and 63 normal controls. They assessed participants on the neuropsychological domains of verbal ability, psychomotor skill, abstraction and cognitive flexibility, attention, learning, retention, motor skills, and
sensory ability. Palmer et al. (1997) found that 47 (27.5%) of the 171 people in the schizophrenia group and 54 (85.7%) of the 63 normal controls were classified as having normal neuropsychological functioning. Neuropsychological impairment was based on global neuropsychological ratings. Similar results have been observed by other researchers (e.g., Kremen, Seidman, Faraone, Toomey, & Tsuang, 2000).

It is clear from research in this area that individuals with schizophrenia often, but not always, have neuropsychological impairments that occur across a wide range of domains. This raises the question as to how unique these impairments are to schizophrenia. Recent research has attempted to answer this question. Mojtabai et al. (2000) carried out comprehensive neuropsychological assessments with people with schizophrenia, bipolar disorder with psychotic features, and major depressive disorder with psychotic features. The schizophrenia group had significantly poorer performance than the bipolar and major depression groups on the variables of attention, concentration, visual memory, verbal fluency and non-semantic verbal short-term memory (Mojtabai et al., 2000). Other researchers have observed that schizophrenia and bipolar groups have similar types of impairments but that the schizophrenia group has impairments of greater severity (Seidman et al., 2002b). These results tend to suggest that the neuropsychological impairments observed in people with schizophrenia are not unique to schizophrenia. The difference between people with schizophrenia and people with other types of psychosis appears to be related to the severity of the neuropsychological impairment.

In summary, the main features of schizophrenia typically involve positive and negative symptoms and various impairments in neuropsychological functioning, yet only positive and negative symptoms are included in diagnostic systems. Research has shown that a large proportion of individuals with schizophrenia experience a wide range of neuropsychological impairments independent of age and intellectual functioning. As with
other symptoms of schizophrenia, there is a degree of variability in neuropsychological impairments in people with schizophrenia. This includes variations in the types of impairments and severity. Although neuropsychological impairments are not unique to schizophrenia, it appears that the severity of the impairment may distinguish schizophrenia from other types of disorders. Considering the role of neuropsychological impairment in the psychopathology of schizophrenia, it may be time for this characteristic to be included in diagnostic systems. Lewis (2004) has suggested the addition of a nonessential neuropsychological impairment criterion for schizophrenia in the DSM-IV. He has defined this as cognitive impairment in 2 of 3 neuropsychological domains including attention, memory, and executive functioning. Further research is required to determine the reliability and specificity of criteria such as this.

It is clear that neuropsychological impairment is a key feature of schizophrenia yet it is not included in diagnostic systems. This is one of the main criticisms of classification systems. Other problems associated with classification systems will be considered in the next section.

Additional Problems with Classification Systems

There are many advantages to having diagnostic criteria and classification systems: they improve reliability and clinical communication, they assist and enhance research, and they help with comparing and standardising research and treatment on a national and global level (Andreasen, 1997; Follette, 1996). Throughout the past century, however, diagnostic and classification systems have had various problems associated with them. Indeed, Kraepelin (1919/2002) acknowledged the difficulty that exists when attempting to classify people using diagnostic criteria. In regards to creating a classification framework for dementia praecox he wrote:
There is certainly a whole series of phases which frequently return, but between them there are such numerous transitions that in spite of all efforts it appears impossible at present to delimit them sharply and to assign each case without objection to a definite form (Kraepelin, 1919/2002, p. 89).

Some of the disadvantages of diagnostic criteria in general have been suggested to include clinicians relying on criteria at the cost of a comprehensive assessment, a partially complete clinical picture, a tendency to fail to be sensitive to individuals, turning a provisional conclusion into a definitive one, and preventing people from thinking about schizophrenia in a creative manner (Andreasen, 1997).

Early editions of the *Diagnostic and Statistical Manual of Mental Disorders* were heavily criticised for their conceptualisation of schizophrenia and associated criteria. Subsequent editions have attempted to address these criticisms and flaws. Despite this, the DSM-IV still receives many criticisms of the way in which it deals with schizophrenia, as well as other psychiatric disorders. Many criticisms of the DSM focus on the criteria themselves, namely the way in which they are set; the heterogeneous nature; and the inclusion and exclusion of particular symptoms. Other criticisms of the DSM include critiques of the overlap between constructs, a lack of evidence for the reliability and validity of constructs, problems with cross-cultural application, and the categorical approach that is used. In addition, a DSM-IV diagnosis is thought to provide limited information about the aetiology of a disorder, its course, and outcome and these factors combine with the above problems to impact on treatment (Follette, 1996; Tsuang & Faraone, 2002).

A number of problems in the way the criteria are set in the DSM-IV and used have been raised. In the DSM-IV individuals are required to meet a set number of criteria in order to be diagnosed with a particular disorder. The problem with this is that a large
proportion of people present with less than the required number of symptoms and therefore fall short of diagnostic criteria. In some cases these people have severe distress or impairment yet may not strictly qualify for treatment. In other cases they may fall into the not otherwise specified category (Malik & Beutler, 2002). Problems also occur with the opposite situation when an individual just meets criteria for a diagnosis. The DSM-IV has been criticised for this because so-called normal people may be classified as having psychopathology (Regier, Narrow, First, & Marshall, 2002). This has implications in terms of stigma and treatment. Research has shown that stigma may also be perpetuated by clinicians and how they apply the diagnostic criteria. A study by Clafferty, McCabe, and Brown (2001) of 246 clinicians found that only 59% reported that they would tell a patient with schizophrenia what their diagnosis was in the first episode of the disorder. In addition, 15% of the clinicians reported that they used terminology other than schizophrenia (Clafferty et al., 2001).

The heterogeneity of the schizophrenia criteria in the DSM-IV is another facet of the diagnostic system that has been criticised. An individual may present with bizarre delusions and mild social impairment while another may present with hallucinations, delusions, severely disorganised behaviour, and very poor social functioning; yet both are diagnosed with schizophrenia (Andreasen, 1987). In addition, it is argued that the tendency for criteria in the DSM to have a descriptive basis, reflecting the heterogeneous nature, rather than a theoretical basis is used to disguise the tendency of psychiatric disorders to be based on a medical model (Malik & Beutler, 2002). This factor is thought to impede research that attempts to understand the aetiology of disorders (Carson, 1991; Malik & Beutler, 2002). It has been suggested that if the tendency to ignore information about the aetiology for schizophrenia is continued in the DSM, then the classification of schizophrenia may suffer from a lack of development (Tsuang & Faraone, 2002). From a
behavioural perspective, the DSM-IV has also been criticised for only counting behaviours rather than considering them in terms of their function or context (Follette, 1996).

The DSM-IV has been criticised for focusing on psychosis in the criteria for schizophrenia. Psychosis has been essential to the definition of schizophrenia throughout various editions of the DSM (Tsuang & Faraone, 2002). Indeed, in the current DSM-IV four of the five key symptoms of schizophrenia are related to psychosis and people are required to meet criteria for psychosis to be diagnosed with schizophrenia, otherwise the disorder is not recognised (Tsuang et al., 2000a). This is despite the fact that psychosis is not specific to the disorder of schizophrenia and is observed in a variety of other disorders such as traumatic brain injury and mood disorders. It may therefore be more appropriate to view psychosis as an end-state symptom (Tsuang et al., 2000a). It has been suggested that there are many other indicators of schizophrenia that are more proximal to the aetiology and pathophysiology of schizophrenia that could be included in the diagnostic criteria (Tsuang & Faraone, 2002).

Related to this are criticisms of the DSM approach for not including features thought to be central to the nature of schizophrenia. Alpert (1985) criticised diagnostic systems of the time for failing to focus on the development of objective measures which he viewed as essential to the assessment of schizophrenia as well as practicable to construct. For example, rate of speech is considered to be a strong indicator of psychosis, yet is not usually measured (Alpert, 1985). Today, in the DSM-IV, disorganised speech is one of the criteria for a diagnosis of schizophrenia and is defined as occurring in a variety of forms, including changing topics, unrelated answers, and incoherent speech. However, the rate of a person's speech is not directly referred to in the DSM-IV (APA, 1994). The DSM has also been criticised because neuropsychological impairment has not been
incorporated into the criteria for schizophrenia (Lewis, 2004). As discussed in the previous section, research has shown that neuropsychological impairment is a deficit that is frequently observed in schizophrenia. In addition, it has been postulated that impairments of this kind may have a key role in understanding the aetiology of schizophrenia (Lewis, 2004). Despite this, the most commonly used diagnostic systems do not include neuropsychological impairments.

The appropriateness of a system where individuals can present with symptoms that have a high degree of overlap between multiple disorders has been questioned (Carson, 1991). Research has shown that comorbidity across all psychiatric diagnoses occurs in 50 to 60% of patients, which is a relatively high proportion (e.g., Cassano, Pini, Saettoni, Rucci, & Dell’Osso, 1998; Shear et al., 2000). More specifically, schizophrenia has been observed to have a high level of comorbidity with various psychiatric disorders. Cassano et al. (1998) studied a group of 31 people with a schizophrenia-spectrum disorder (schizophrenia, schizophreniform disorder, schizoaffective disorder, or delusional disorder) and found that 18 (58.1%) had a comorbid psychiatric disorder. Psychiatric diagnoses that have a high level of comorbidity with schizophrenia include autism (Konstantareas & Hewitt, 2001), panic disorder (Labbate, Young, & Arana, 1999), substance abuse (Soyka, 1996) and obsessive-compulsive disorder (Cassano et al., 1998).

In addition to problems with comorbidity, concerns have been raised about the reliability and validity of the DSM constructs. Prior to the publication of the DSM-IV, Nelson-Gray (1991) stressed the importance of the reliability and validity of diagnoses to be included in the manual. Carson (1991) has criticised the DSM in general for the lack of research given to the construct validity of disorders. Bentall, Jackson, and Pilgrim (1988) reviewed a number of studies looking at the reliability and validity of the diagnosis of schizophrenia. They concluded that a lack of evidence in support of the reliability and
absolute boundaries dividing it from other mental disorders or from no mental disorder” (APA, 1994, p. xxii). Despite this statement, the discrete category approach has prevailed in the DSM-IV. This means that an individual is not diagnosed with schizophrenia until they experience the symptoms currently defined in the DSM-IV (Tsuang & Faraone, 2002). This can affect the treatment an individual may or may not receive and how they are classified for research.

The problems that have been identified with the DSM potentially contribute to inconsistency in the use of the diagnostic system and this has implications for treatment. Many studies that investigate the efficacy of treatment for psychiatric disorders use DSM criteria to determine which participants are included in treatment groups. The implication of this is that if a treatment is found to be effective then it is effective in treating the symptoms that form the disorder under investigation. This would be beneficial for patients in clinical settings if there was high level of agreement between clinical diagnoses and diagnoses obtained on the basis of DSM criteria. This is not always the case. The results of a study by Shear et al. (2000) highlight the increased need for treatment studies that are based on factors other than diagnostic criteria. Shear et al. (2000) investigated the level of agreement between clinical diagnoses and diagnoses made on the basis of the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995). They observed a kappa value of .24 for agreement between primary diagnoses in individuals’ psychiatric files and diagnoses received from the SCID. This may also be a reflection of clinicians’ tendency to ignore the classification criteria of the DSM-IV which also highlights the need for an improved classification system that is accepted by clinicians.

The DSM-IV is one of the major diagnostic systems that are used today for diagnosing schizophrenia. There are many problems with the DSM approach and these
have a number of implications for the construct of schizophrenia, including how it is
defined, assessed, and treated. An alternative approach to classification systems will be
considered next.

An Alternative Approach to Classification Systems

It is clear that there are many problems with the DSM approach to schizophrenia. The question has been raised as to why, with a lack of evidence supporting the strength of the schizophrenia diagnosis, do people continue to use the categorical approach (Bentall et al., 1988)? There have been many criticisms of the DSM but fewer practical suggestions as to how to overcome these problems or better methods that would be appropriate substitutes. Kendell (2002) has suggested that a new classification system would be considered an improvement if it was more comprehensive and easier to use; defined clinical significance appropriately; had higher reliability and clear operational definitions; and greater validity gained from discriminant function analysis. Other improvements are expected as knowledge increases about the underlying aetiology and processes involved in numerous psychiatric disorders (Regier et al., 2002). However, a major criticism is that the categories or criteria of diagnostic systems are used in place of a comprehensive understanding of aetiology. A change in this trend would have to take place before the diagnostic systems could benefit from developments in the understanding of the aetiology of schizophrenia.

A more specific suggestion to improve diagnostic systems considers diagnosis from a dimensional approach. Carson (1996) has postulated that this would involve firstly, determining the nature of the underlying dimensions of psychopathology and secondly, developing measures that are reliable and valid to assess the dimensions. He claims that the benefits to patients would outweigh the magnitude of this task.
Consistent with a dimensional approach, it has been suggested that diagnostic systems would be improved if the focus changed from being on the symptoms of a disorder to criteria based on the underlying pathology of a disorder (Kihlstrom, 2002). In the case of schizophrenia, this would not only include physical pathology but also the disturbances of function, such as problems with cognitive and emotional functioning, that underlie many of the unusual and abnormal experiences that people may have (Kihlstrom, 2002).

In addition to underlying pathology, general functioning needs to be considered. This is because individuals' diagnoses are not always good predictors of the impairments that they experience. For example, Stordal et al. (2005) investigated the neuropsychological domain of executive functioning as well as level of general psychopathology in 43 patients with major depression and 47 patients with schizophrenia. They assessed four components of executive functioning with five neuropsychological tests. Stordal et al. (2005) found that compared to DSM-IV diagnosis, participants' level of general psychopathology was a better predictor of variance in executive functioning, however, diagnosis still made a smaller, independent contribution to the variance of executive functioning. This indicates that differences in executive functioning cannot be considered simply as a function of diagnosis and that research needs to incorporate measures of general psychopathology in addition to diagnosis. A dimensional approach would encompass these factors.

Various dimensional models have been proposed for schizophrenia and related disorders. Nicholson and Neufeld (1993) have suggested that instead of viewing the difference between paranoid schizophrenia and nonparanoid schizophrenia as a categorical difference, that it be viewed on a continuum. They have proposed a model where the severity of symptoms and severity of the disorder are considered. Another potential
solution to the classification debate is to consider how spectrum models of classification would complement current systems (Maser & Patterson, 2002). Maser and Patterson’s (2002) spectrum model incorporates both the categories of DSM-IV as well as the features considered to be sub-threshold which may also impact on a person’s functioning. A dimensional perspective such as this considers normal behaviour as well.

It is thought that a dimensional approach may provide a more accurate definition of schizophrenia. A dimensional approach to schizophrenia and related disorders will be considered next.

**Schizophrenia-spectrum Disorders and a Dimensional View of Schizophrenia**

Disorders related to schizophrenia are thought to exist on a psychotic continuum. This conceptualisation of schizophrenia is consistent with a dimensional approach. The term, *schizophrenia-spectrum disorders*, was initially introduced in adoption studies looking at schizophrenic probands (Kety, Rosenthal, Wender, & Schulsinger, 1968). The notion of a spectrum or dimensional relationship between schizophrenia and other disorders was alluded to by Meehl (1962). In his theory, Meehl (1962) proposed the existence of a dimensional relationship between schizotaxia, schizotypy and schizophrenia. He postulated that people who are born with a genetic predisposition to schizophrenia develop an integrative neural deficit which he called schizotaxia. Meehl (1962) suggested that all people with schizotaxia also develop a personality organisation that he labeled schizotypy. He proposed that as a result of the interaction between schizotypy and environmental factors, some people with schizotypy will develop schizophrenia (Meehl, 1962).

The range of schizophrenia-spectrum disorders can include schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, brief psychotic
disorder, schizotypal personality disorder, paranoid personality disorder, schizoid personality disorder, borderline personality disorder, brief reactive psychoses, avoidant personality disorder, compulsive personality disorder, obsessive compulsive disorder, Asperger's disorder, childhood-onset pervasive personality disorder, shared psychotic disorder, psychotic disorders due to a general medical condition or substance, major depressive disorder with psychotic features, and bipolar disorder with psychotic features (Adler & Strakowski, 2003; APA, 1994; Shean, 2004). Today, in the DSM-IV (APA, 1994), it is noted that some of the personality disorders may precede schizophrenia, suggesting a dimensional view of these disorders. These include the schizotypal, schizoid, and paranoid personality disorders.

Different studies use varying definitions of what the schizophrenia-spectrum encompasses and these definitions are not used consistently. How researchers define what disorders are included in the schizophrenia-spectrum influences how participants are classified and how the boundaries of schizophrenia are determined. Changing the definition of the schizophrenia-spectrum has a direct impact on the proportion of people in studies who are considered to have a schizophrenia-spectrum disorder. The way in which psychosis is assessed and measured (for example, clinical features of schizophrenia versus sub-clinical or schizotypal features) also influences how psychosis is identified in the population that is being studied (Johns & van Os, 2001). These factors can lead to spurious conclusions about the population and disorders being investigated.

There is much controversy over to what degree the disorders in the schizophrenia-spectrum overlap and where the boundaries between the disorders are (Adler & Strakowski, 2003). Researchers have proposed different approaches for attempting to address this. For example, Reich (1975) suggested that the assumption underlying the schizophrenia-spectrum disorders could be that the disorders all share some common
genetic basis. Adler and Strakowski (2003) have looked at the epidemiology, course, symptoms, neuropathology, and aetiology of schizophrenia and bipolar disorder. They surmised that there is evidence of a large degree of overlap between the two disorders but a lack of clarity about the genetic basis of the two disorders and differences in brain pathology means that this is not conclusive. Likewise, in an examination of the similarities between schizophrenia and obsessive compulsive disorder, Adler and Strakowski (2003) reported that a lack of evidence prevented them from concluding that the two disorders should be considered on the same continuum.

Other research has also attempted to investigate the boundaries between the schizophrenia-spectrum disorders. In a study of 544 patients with schizophrenia, schizoaffective disorder, or bipolar disorder, Averill et al. (2004) administered a number of clinician-rated and self-rated measures. They found that the patients with schizoaffective disorder had scores that fell between the scores of the schizophrenia and bipolar groups. In addition, the schizoaffective and schizophrenia groups had similar positive and negative symptom ratings while the schizoaffective and bipolar groups had similar distress and mood symptom ratings. This suggests that the symptoms of the three disorders overlap and exist along various continua. It is probable that most affective disorders and schizophrenia do not share the same underlying factors however; there is probably a shared continuum for schizophrenia and affective disorders with psychotic symptoms (Tsuang & Lyons, 1989). Support for a dimensional approach comes from evidence such as this that demonstrates that the symptoms of some disorders share some of the same features but to different degrees of severity.

Further support for the notion of schizophrenia-spectrum disorders comes from a study by Vallès et al. (2000). In a study of the relatives of 103 patients with bipolar disorder and the relatives of 84 controls, Vallès et al. (2000) found that the relatives of the
individuals with bipolar disorder had a significantly higher risk of developing schizophrenia or bipolar disorder than the relatives of the controls. The risk was highest for relatives of female patients who had experienced an early onset of bipolar disorder. Vallès et al. (2000) concluded that there is likely to be a degree of overlap between risk for schizophrenia and risk for bipolar disorder at the severe end of the affective continuum, suggesting the presence of a continuum of severity between the two disorders.

Dimensionality has been acknowledged within the construct of schizophrenia as well as across disorders. Bleuler (1911/1950) alluded to the dimensional nature of some of the schizophrenia symptoms where “in milder cases of schizophrenia we find a number of prominent manifestations, which strongly fluctuate within the limits of what is regarded, if not as healthy, at least as ‘not mentally ill’.” (p. 294). Research has provided support for a dimensional or continuous view of psychotic features. Stefanis et al. (2002) found evidence of a dimensional structure for features of psychosis in a general population. Participants were chosen for their age group which was thought to be representative of when males are most likely to have psychotic experiences of varying severity. They observed depressive, negative, and positive symptoms in a sample of 932 healthy men. Furthermore, the 3 dimensions were correlated with each other. This led them to conclude that the features of psychosis are in fact dimensional and are present to some degree in the general population (Stefanis et al., 2002). Similar factors to those evaluated by Adler and Strakowski (2003) have been considered by Johns and van Os (2001) but for psychosis symptoms in general as opposed to specific disorders. Johns and van Os (2001) surmised that there is strong evidence for a dimensional view of hallucinations and delusions. It is probable that psychosis is not entirely continuous but that the distribution can be described as being in between a dichotomous and continuous distribution (Johns & van Os, 2001).
Other promising research has also considered the suggestion of a dimensional approach. Bell, Dudgeon, McGorry, and Jackson (1998) investigated the schizophrenia construct using 11 different diagnostic systems with 497 participants with a schizophrenia-spectrum disorder. They found that the proportion of the sample classified as having schizophrenia varied widely across the 11 diagnostic systems. Bell et al. (1998) analysed the results of the 11 systems to determine the factor structure. They found that there were 3 underlying factors with 7 of the diagnostic systems loading on 1 of the factors, 3 loading on 2 of the factors and 1 diagnostic system loading on all 3 factors. This suggests that the symptoms included in the diagnostic systems share a degree of variance. However, none of the diagnostic systems considered neuropsychological impairment, a key symptom of schizophrenia.

Despite evidence for the conceptualisation of schizophrenia-spectrum disorders, the field would not necessarily benefit from adopting yet another diagnostic category. If the category were to be widely used then people who do not meet strict schizophrenia criteria would be included in the schizophrenia continuum and with this comes stigma, problems with labeling, public confusion, and possible legal effects (Reich, 1975). In addition, further research would be needed that investigates the reliability and consistency of the diagnosis, a task which would involve a large undertaking. Instead of adopting yet another category, it may be more practicable to consider schizophrenia-spectrum disorders from a dimensional approach (Krueger & MacDonald, 2005). Ideally, this conceptualisation would be investigated by future research (Johns & van Os, 2001). A method for evaluating dimensionality, called taxometric analysis, will be considered in Chapter 5.

Many researchers have suggested that diagnostic systems would benefit from developments in the understanding of the aetiology of schizophrenia. This is consistent
with a dimensional approach to schizophrenia. The DSM-IV does not make a provision for the aetiology of schizophrenia or for identifying people at risk. The focus instead has been on treatment rather than prevention and it is thought that this is partly due to the limited research in this area (Regier et al., 2002). It has been suggested that a dimensional view is more consistent with theories on the aetiology of schizophrenia where individuals may have varying degrees of risk for developing schizophrenia (Tsuang & Faraone, 2002). The aetiology of schizophrenia will be considered briefly next.

\textit{Aetiology of Schizophrenia}

Traditionally, theories on the aetiology of schizophrenia have focused on genetic factors. However, research has demonstrated that genetic factors alone do not account for the development of schizophrenia. More recently, theories on the aetiology of schizophrenia have also considered the role of environmental and developmental factors and how these interact with genetic factors. These theories come under the category of neurodevelopmental theories. The components of these theories will be briefly considered next.

Evidence on the causal role of genetic factors has been derived from twin, adoption, and genetic linkage studies and the rates of concordance in families (Tsuang, Stone, & Faraone, 1999a). These studies indicate that genes potentially contribute to risk for schizophrenia (see Table 2.2). If an individual has a first-degree relative with schizophrenia, then that individual’s risk of developing schizophrenia increases to 10%. Gottesman (1991) has stated that if an individual has a monozygotic twin with schizophrenia then the risk increases to 48%. Percentage of risk for other first- and second-degree relatives is shown in Table 2.2. It is thought that multiple genes are involved in the heritability of schizophrenia and the expression of these is affected by a
number of environmental and social factors. To date, up to seven separate genes have been recognised as being related to schizophrenia (Harrison & Owen, 2003; Wilcox, Faraone, Su, Van Eerdewegh, & Tsuang, 2002). However, it is important to note that none of these findings have been replicated.

Table 2.2
Morbid Risk of Schizophrenia for Relatives of Individuals with Schizophrenia (Adapted from Gottesman, 1991 and Tsuang, 2000)

<table>
<thead>
<tr>
<th>Relationship</th>
<th>% Shared genes</th>
<th>Risk (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>General population</td>
<td>N.A.</td>
<td>1</td>
</tr>
<tr>
<td>Spouses of patients</td>
<td>N.A.</td>
<td>2</td>
</tr>
<tr>
<td>Third-degree relatives</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First cousins</td>
<td>12.5</td>
<td>2</td>
</tr>
<tr>
<td>Second-degree relatives</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uncles/aunts</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Nieces/nephews</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>Grandchildren</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>Half-siblings</td>
<td></td>
<td>6</td>
</tr>
<tr>
<td>First-degree relatives</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parents</td>
<td>50.0</td>
<td>6</td>
</tr>
<tr>
<td>Siblings</td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>Children</td>
<td></td>
<td>13</td>
</tr>
<tr>
<td>Siblings with 1 schizophrenic parent</td>
<td></td>
<td>17</td>
</tr>
<tr>
<td>Dizygotic twin</td>
<td></td>
<td>17</td>
</tr>
<tr>
<td>Monozygotic twin</td>
<td>100</td>
<td>48</td>
</tr>
<tr>
<td>Children with 2 schizophrenic parents</td>
<td>100</td>
<td>46</td>
</tr>
</tbody>
</table>

Despite the evidence of the role of genetics, 63% of people with schizophrenia do not have a first-degree or second-degree relative with schizophrenia and 89% of individuals with schizophrenia do not have a parent with schizophrenia (Gottesman &
Erlenmeyer-Kimling, 2001). Therefore, it is apparent that genes and heritability by themselves are not able to account for the development of schizophrenia. Theories and models of the past few decades have considered the role of both genetic and environmental factors in the development of schizophrenia.

It is thought that environmental factors include events that occur in the prenatal and perinatal periods. Research has looked at the effects of maternal nutrition and infection, impaired sexual dimorphisms, obstetric complications, season of birth, birth order, urban birth, small head size, and physical abnormalities (e.g., Buka et al., 2001a, 2001b; Goldstein et al., 2002; Hultman, Sparén, Takei, Murray, & Cnattingius, 1999; Lewis & Levitt, 2002; Marcelis, Navarro-Mateu, Murray, Selten, & van Os, 1998; Pedersen & Mortensen, 2001; Zornberg, Buka, & Tsuang, 2000). Many studies have observed a correlation between these factors and schizophrenia. For example, Cannon (1997) reviewed studies of obstetric complications and schizophrenia and concluded that complications during birth were a greater risk factor for schizophrenia than prenatal factors such as virus exposure in individuals with a genetic predisposition. However, some studies of these factors have produced inconclusive evidence. As a result, researchers are not able to conclude that it is the factors themselves that are contributing to the development of schizophrenia. Instead it may be that exposure to some other factor that has not been accounted for in the study has a contributing role (Lewis & Levitt, 2002).

Environmental factors such as stress may also be associated with increased risk of schizophrenia. For example, research has shown that some minority groups, such as African-American people, have higher psychiatric hospital admission rates and incidence rates of schizophrenia than others (Rabkin, 1979). It is possible that this may be a reflection of the social stress of being member of a minority group (Mueser & McGurk,
2004). Some research, however, has shown that African-American people tend to be over-diagnosed with schizophrenia (Whaley, 2001).

In addition to environmental factors, it is also thought that developmental abnormalities observed during childhood and adolescence may be indicative of developing schizophrenia later in life. These abnormalities include problems with motor development, social development, and academic performance (Lewis & Levitt, 2002).

Exactly how genetic and environmental factors interact is not clear. The picture is obviously a very complex one. Ongoing research into the aetiology of schizophrenia is attempting to clarify this. Two contrasting neurodevelopmental theories of risk for schizophrenia, which are the focus of this research, will be considered in more detail in Chapters 3 and 4.

Summary

The conceptualisation of schizophrenia had changed somewhat since the time of Kraepelin and Bleuler. Many improvements have been made in how the construct is defined in an attempt to improve the diagnosis of schizophrenia. Despite this, there are still criticisms of the classification criteria in the more commonly used systems, such as the DSM-IV. It does not seem likely that the issues surrounding the diagnosis of schizophrenia will be resolved in the near future. As a result, research needs to look to other ways in which the disorder may be conceptualised. One option involves incorporating associated features of schizophrenia, such as neuropsychological impairment, into classification systems. Another option is to consider schizophrenia from a dimensional approach. This could also incorporate a focus on the aetiology of schizophrenia. Alternatively, another area that has been explored is that of risk for schizophrenia. This incorporates both the aetiology of schizophrenia and a dimensional
view of the construct. There are limited provisions in the classification systems for identifying signs and symptoms that may precede schizophrenia. This has been part of the impetus behind an increase in the research area of risk for schizophrenia over the past few decades. Chapters 3 and 4 will introduce and discuss two contrasting theories on risk for schizophrenia and the evidence for these.
CHAPTER 3

Schizophrenia, Schizotaxia, and Schizotypy: Meehl’s Perspective

It is apparent that there are many problems surrounding the classification systems that are used with schizophrenia. In addition, relatively little is known about the aetiology of the disorder. Approximately 40 years ago, a new research approach was developed in an endeavor to overcome some of these problems. This chapter outlines Meehl’s (1962, 1989, 1990b) theory about the constructs of schizotaxia and schizotypy, both of which are thought to be precursors for schizophrenia. Meehl’s theory addresses the nature of predisposition and risk for schizophrenia. A second theory of schizotaxia, proposed by Tsuang and colleagues (1999b, 2000a, 2000b) is discussed in Chapter 4. To avoid confusion, \text{schizotaxia}_{Meehl} \text{ and } \text{schizotaxia}_{Tsuang} \text{ will be used, respectively, to distinguish these constructs.}

\textit{Meehl’s Theory of Risk}

\textit{Background to Meehl’s theory.} Both Kraepelin (1919/2002) and Bleuler (1911/1950) argued that genetic factors play an important role in the aetiology of schizophrenia. Rado (1960) also emphasised the role of genetics when he considered risk for schizophrenia. He was the first to coin the term \textit{schizotype} as shorthand for the idea of a schizophrenic phenotype exhibited by the person who has a genetic risk for schizophrenia (Rado, 1960). Rado (1960) considered the expression and symptoms of schizotypy to arise from two main features: a reduced capacity for pleasure and a distorted
perception of one’s own physical body, called kinesthetic diathesis. In his conceptualisation, these features had a prevailing psychodynamic nature. Rado (1960) proposed that the term *schizotypal organisation* be used to describe the pathology of the schizotype and that the way in which the pathology is expressed be called *schizotypal behaviour*. Rado (1960) delineated four stages of schizotypal behaviour that varied in degree of severity of symptoms. These included compensated schizotypal behaviour where the individual may lead a relatively normal life; decompensated schizotypal behaviour (also called pseudoneurotic schizophrenia) where the individual displays excessive fear or rage; disintegrated schizotypal behaviour which is behaviour that betrays a fully developed psychosis; and deteriorated schizotypal behaviour which occurs when the individual does not receive appropriate care for his or her psychosis, and the individual’s functioning deteriorates to an extremely low level (Rado, 1960).

**Schizotaxia** and **hypokrisia**. Meehl (1962, 1989, 1990b) used Rado’s terms in a theory in which he attempted to explain risk for schizophrenia and the development of schizophrenia while also taking into consideration the heterogeneity of the disorder. He did this by combining both genetic and environmental factors in a multi-layered model. Meehl (1962) proposed that people who are at risk of developing schizophrenia are born with a genetic mutation, which he postulated to be in the form of a single dominant gene. He called this the *schizogene* (Golden & Meehl, 1978). Meehl (1962) conjectured that the genetic mutation gives rise to an integrative neural defect, which he labeled *schizotaxia*. This word comes from *schizo*, which means split, and *ataxia*, the Greek word for a disruption in ordering or arrangement (Meehl, 1990b). Meehl viewed schizotaxia as a brain state, an inherited neurological defect that is at the heart of the predisposition to schizophrenia. Meehl (1962, 1989) conjectured that the integrative neural defect occurs as a change in the function of cells in the central nervous system and
that every cell is affected. More specifically, he proposed that schizotaxia\textsubscript{Meehl} is manifested in the form of problems with synaptic signaling, arising from “a functional parametric aberration of the synaptic control system” (Meehl, 1990b, p. 14). Meehl (1989, 1990b) used the term hypokrisia, (which stems from Greek words that mean a paucity of separation, differentiation, or discrimination) to describe the subtle problem that characterises the schizotaxic\textsubscript{Meehl} brain.

Hypokrisia refers to “a slight quantitative aberration in the synaptic control over the spiking of a neuron” (Meehl, 1989, p. 938). Specifically, people with the schizotaxic\textsubscript{Meehl} brain do not experience the typical spike that a pattern of stimulation would produce in nonschizotaxic\textsubscript{Meehl} brains. In normal brains, a presynaptic impulse arrives at a cell and depolarises the membrane of the cell (Meehl, 1990b). If the depolarisation of the cell is of sufficient size, and a spike arrives at the same time at the axon hillock (the junction between the axon and the cell body), then the cell fires (Meehl, 1990b). The probability of the cells firing can be represented as a geometric hypersurface in hyperspace, with hills and valleys associated with the temperospatial input patterns. Meehl (1990b) conjectured that if an “aberration of the synaptic control system” (p. 14) is present, then the consequence of this involves geometric changes to the hypersurface so that the hypersurface becomes both elevated and dedifferentiated. As a result, the pattern of spikes in the schizotaxic\textsubscript{Meehl} brain is flattened compared to the pattern of spikes in the nonschizotaxic brain. Meehl (1989) viewed hypokrisia as the core defect of schizotaxia\textsubscript{Meehl}. However, he stated that it would be premature to speculate further about the impact of hypokrisia on other functions of the central nervous system before more is known about how these functions normally operate (Meehl, 1990b).

\textit{Schizotypy}. Meehl (1962) proposed that as a result of environmental effects such as social learning and reinforcement schedules, all individuals with schizotaxia\textsubscript{Meehl} develop a
Schizotypal personality organisation. The social learning and reinforcement schedules are those normally encountered by all people and are provided initially by the individual’s primary caregiver and then also by others around him or her, such as siblings, teachers, peers, and other family members (Meehl, 1989). Meehl (1989) speculated that if an appropriate intervention was to be developed and individuals could be identified as schizotypic when they are infants, then there is the potential for the schizotypal personality organisation to not develop to its full extent.

Meehl (1962) described four main symptoms that he considered indicative of schizotypy: cognitive slippage, interpersonal aversiveness, anhedonia, and ambivalence. Cognitive slippage refers to the varying forms and severity of thought disorder (Meehl, 1962). He likened this to Bleuler’s associative loosening (Meehl, 1990b). Interpersonal aversiveness or social fear involves the inability to trust others, the belief that one is unlovable, and holding the assumption that one will be rejected (Meehl, 1962). The third symptom, anhedonia, refers to Rado’s (1960) symptom of a diminished capacity for pleasure (Meehl, 1962). The fourth symptom, ambivalence, was taken from Bleuler’s (1911/1950) group of fundamental symptoms and involves the presence of contradictory emotions, will, and intellect.

*Cognitive slippage, anhedonia, and aversive drift.* Of the four symptoms of schizotypy, Meehl (1962) proposed that either cognitive slippage or anhedonia might be viewed as the key or primary symptom. In regards to cognitive slippage, he stated, “any characterisation of schizophrenic or schizotypic behaviour which purports to abstract its essence but does not include the cognitive slippage must be deemed unsatisfactory” (Meehl, 1962, p. 831). He hypothesised that cognitive slippage is due to slippage at the level of the synapse in the brain, which is caused by the genetic mutation. Meehl (1962) proposed that if synaptic slippage occurs over a long period then aversive drift develops.
This is because of an imbalance between the functioning of positive and negative feedback systems. In schizotypal people, aversive drift refers to a shift towards the negative system resulting from an increase in negative feedback (Meehl, 1962). He also generated a hypothesis involving cognitive slippage and an individual's inhibition impairment. Meehl (1962) suggested that people with schizotypy have problems controlling or inhibiting associations and this arises from cognitive slippage. Furthermore, he proposed that this also contributes to another difficulty that schizotypal people have: an inability to turn off painful thoughts about themselves and others.

In respect to the symptom of anhedonia, Meehl (1962) asserted that it "is one of the most consistent and dramatic behavioural signs of the disease" (p. 829). Meehl (1990b) observed that in schizotypal people, new activities, people, and places are initially rewarding but that with time, these factors all become somewhat negative, despite the presence of typically rewarding aspects of the factors. He hypothesised that anhedonia arises because of defective synaptic wiring which is caused by the genetic mutation. Again, Meehl (1962) used the notion of aversive drift to explain why anhedonia develops. He postulated that people have one area of their brain that controls reward systems involved in the experience of pleasure and a separate area that controls reward systems involved in the experience of aversive or negative affect. Meehl (1962, 1990b) hypothesised that in people with schizotypy, the aversive system functions normally but that the pleasure system is deficient in the amount of synaptic activity it has and the strength of connections between cells. Therefore, the pleasure system does not function normally. With time and experience, the strength of links to the aversive system become stronger and links to the pleasure system become weaker (Meehl, 1990b). As a result, aversive drift occurs where the experience of negative affect becomes stronger and the individual has a diminished capacity to experience pleasure (Meehl, 1962).
In later work, Meehl (1989) stated that he had relabeled anhedonia as hypohedonia and that he did not consider hypohedonia to have as much of an influential role as he had in 1962. He reiterated this point in a major review of his theory in 1990, drawing an additional distinction between primary and secondary hypohedonia. Meehl (1990b) described primary hypohedonia as a genetic capacity for hedonia expressed as a polygenic potentiator that can increase the probability of a person with schizotypy decompensating to schizophrenia. He described secondary hypohedonia as a clinical feature or effect of schizotypy that arises due to either aversive drift or primary hypohedonia (Meehl, 1990b, 2001b). In later work, Meehl (1989, 1990b) viewed cognitive slippage and aversive drift as the two main features of schizotypy that account for various secondary phenomena; including secondary hypohedonia, secondary cognitive slippage, ambivalence, and interpersonal aversiveness.

Development of schizophrenia. In addition to the features of schizotypy, Meehl (1989) conjectured that there are a number of other inherited polygenic factors that act as potentiators of schizophrenia. If a schizotypal individual experiences a considerable amount of these polygenic factors then the probability of them decompensating to schizophrenia is increased. These factors include social introversion, anxiety, aggression, sex drive, energy level, polymorph-perverse eroticism, mesomorphic toughness, arousal, inhibition, dominance, perception-cognition, and various abilities in addition to primary hedonia. Meehl (1990b) viewed people displaying schizotypy as schizotypes with varying degrees of compensation or decompensation. He asserted that a person could be schizotypal and have schizophrenia at the same time (Meehl, 1990b). As a result, schizophrenia is superimposed on the latent disposition (schizotypy) that does not disappear because of schizophrenia. Meehl (1990b) summarised his theory with the statement “the reason for the molar slippage in the schizophrenic is that the schizotaxia...
brain has slippage at the synapse” (p. 15). The “molar slippage in the schizophrenic” refers to the cognitive and affective symptoms seen in people with schizophrenia.

To summarise, Meehl (1962, 1989, 1990b) proposed that people who are at risk of developing schizophrenia are born with a genetic mutation which gives rise to schizotaxiaMeehl, an integrative neural defect. SchizotaxiaMeehl is characterised by a problem called hypokrisia. As a result of exposure to environmental factors, all individuals with schizotaxiaMeehl develop a schizotypal personality organisation. Schizotypy is characterised by two main features, cognitive slippage and aversive drift, that account for various secondary phenomena. The development and course of schizophrenia in the schizotypal person is determined by the interaction of the person (schizotaxiaMeehl) with the environment, with potentiators impacting on the interaction of these. Meehl’s (1989) conceptualisation of the path from the presence of the schizogene through to the development or decompensation of schizophrenia is shown in Figure 3.1. One of the advantages of Meehl’s theory is that it incorporates both genetic and environmental factors; these will be considered in more detail next.
Figure 3.1. Meehl’s (1989, 1990b) conceptualisation of the causal pathways in schizophrenia (from Meehl, 1989, p. 941).
Genetic and environmental factors. Meehl (1990b) stressed that the schizotypic brain is inherited but schizotypy is not. He maintained the view that all individuals with schizotypy inherit the predisposition for schizophrenia from their biological parents. In addition to hereditary factors, environmental factors have a significant role in Meehl’s theory of risk for schizophrenia. Schizotypy is a sine qua non for schizotypy and schizophrenia, a necessary but not sufficient requirement (Meehl, 1990b). Meehl (1990b) proposed that schizotypy develops as a result of the social learning and reinforcement schedules that a person with schizotypy is exposed to. He quoted Bleuler’s statement that “one cannot have a delusion about Jesuits if he has never learned about Jesuits” to demonstrate this and make the point that a person does not inherit schizotypy just as the content of a person’s delusion is not inherited (Meehl, 1962, 1990a, 1990b).

Meehl (1990b) considered two types of environmental factors to influence the decompensation of schizotypy to schizophrenia: exposure to certain environmental factors in early childhood and stressors in adulthood that are present around the time of decompensation. In 1962, Meehl believed that having a schizophrenogenic mother was a key factor that contributed to the development of schizophrenia in people with schizotypy. He later acknowledged that the notion of the schizophrenogenic mother had been rejected but did conjecture about the role of child-rearing practices and parental attitudes and believed these factors to have a role in the decompensation of schizotypy to schizophrenia (Meehl, 1989). Meehl (1989) also predicted that the mother would have more of an impact upon this decompensation than the father would. This was because he proposed that the schizogene came from the compensated schizotypal mother who would provide a dominant, controlling, and ambivalent environment along with aversive social learning (Meehl, 1990b). The father would provide the polygenic potentiators as well as an anxious, insecure role model (Meehl, 1990b). In addition, Meehl (1990b) suggested that
any form of trauma in childhood would increase the probability of decompensation of
schizotypy to schizophrenia. In regards to stressors in adulthood, Meehl (1990b)
described luck as a crucial factor that could contribute to decompensation.

**Base rate of schizotypy.** Meehl has estimated the incidence of schizotaxia, schizotypy, and
the percentage that decompensate to schizophrenia. He asserted that 10% of
the general population is schizotypal (Meehl, 1989). The basis of this assertion was
that every individual who develops schizophrenia must have a parent who carries a genetic
predisposition for schizophrenia (Meehl, 1990b). However, only approximately 10% of
people with schizophrenia have a parent with schizophrenia, therefore, according to
Meehl, the other 90% of people with schizophrenia have a parent who carries the genetic
predisposition but does not experience symptoms of schizophrenia. Meehl (1962) also
conjectured that a small proportion (around 10%) of schizotypal people decompensate and
develop schizophrenia. Schizophrenia occurs in approximately 1% of the general
population; therefore, schizotypy occurs in 10% of the general population and the base
rate of schizotypy and schizotaxia is .10 (Meehl, 1990b). Meehl (1990b) estimated
that approximately 35% to 40% of psychiatric patients are schizotypes. However, this
estimate depends considerably on the nature of psychiatric services provided in a
particular area, who has access to the services, and whether services are limited to
particular patient groups. In addition, those classified as having schizophrenia may
include people with genuine schizophrenia and other forms of psychosis.

**A genophenocopy of schizophrenia.** Meehl (1962) postulated that all people with
schizophrenia must have a schizotaxic brain and that a nonschizotaxic person
could not develop clinical schizophrenia but could potentially develop some other form of
psychosis. Meehl (1962) described a situation where it would be possible for a
genophenocopy of schizophrenia to occur. He labeled the syndrome SHAITU, which
stands for submissive, hypohedonic, anxious, introverted, traumatised, and unlucky. Meehl (1990b) proposed that if features of SHAJTU interact with stress, then individuals could potentially present as having a disorder similar to clinical schizophrenia. However, Meehl (1990b) speculated that the content of hallucinations and delusions in SHAJTU would be different to those seen in someone with schizophrenia decompensated from schizotypy. He also suggested that the symptoms that arise from hypokrisia in people with a schizotaxicMeehl brain would not be observed in someone with the SHAJTU syndrome. Meehl (1990b) has predicted that 85% to 90% of people currently diagnosed as having schizophrenia are schizotaxicMeehl while 10% to 15% are genophenocopies, or have the SHAJTU syndrome. It has also been suggested that people with affective disorders may present with symptoms of schizophrenia and these would need to be differentiated from schizophrenia that arises from schizotaxiaMeehl (Siever, 1990).

In order to separate genuine or schizotaxiaMeehl schizophrenia from its genophenocopy, Meehl (1990b) advocates the use of taxometric analysis procedures. Meehl (1992) defined taxometric analysis as a statistical procedure that is used to firstly help to test for evidence of the presence of a taxon, in this case of schizotypy, and secondly, classify individuals as members of the taxon or its complement. These procedures will be described in more detail in Chapter 5. Meehl (1990b) predicted that his theory of schizotaxiaMeehl will be invalidated if taxometric analyses of neurological indicators with appropriate samples do not provide evidence of a taxon.

**Strengths and criticisms of Meehl’s theory.** Meehl’s (1962, 1990b) theory has been acknowledged as being very complex but as having many strengths (e.g., Chapman, 1990; Widiger, 1990). One strength of his theory is that it highlights the problems that occur with the reliance on diagnostic systems. Currently, an individual is required to present with hallucinations or delusions in order to be first diagnosed with schizophrenia.
according to the DSM-IV (APA, 1994). However, Meehl's theory provides an opportunity for identifying people who may be at risk of developing schizophrenia and for identifying milder cases of schizophrenia. The advantage to this is that a person may be identified before they present with what are basically the end-state symptoms of the disorder. Research has shown that early identification is associated with better prognosis (e.g., Bottlender, Strauss, & Möller, 2000; Cannon et al., 2002; Johannessen et al., 2001; McGorry et al., 2002; Morrison et al., 2004; Woods et al., 2003; Wyatt, 1995).

Another advantage is that the components of Meehl's theory are conceptually independent of one another. This means that the notions of hypokrisia, a single gene, the development of schizotaxiaMeehl to schizotypy, and schizotypy as risk for schizophrenia can be subscribed to as a whole or researchers can subscribe to one or two of these concepts. As a result, if evidence suggests that one of these notions should be rejected, then this does not invalidate the other concepts.

Meehl's theory also has advantages for research in the area. When an individual is experiencing the symptoms of schizophrenia in an active episode, the symptoms interfere with their ability to communicate or take part in research (Holzman, 1990). If people at risk are able to be identified early then their contribution to research will probably be more considerable than if they were to progress to schizophrenia before being identified. The use of participants who are relatives of people with schizophrenia is advocated in an attempt to further understand the primary symptoms of schizotaxiaMeehl and schizotypy (Holzman, 1990; Meehl, 1990b). Meehl (1990a) suggests that the investigation of the psychophysiology and soft neurology of schizotaxiaMeehl may support the notion of a schizogene more than the symptoms of anhedonia, which are further along the causal pathway, would support this notion. He acknowledges, however, that this is not
necessarily practicable and that indicators of schizotypy can still be investigated if conclusions are made cautiously (Meehl, 1990a).

Meehl’s (1962) theory was somewhat controversial for the thinking at the time. Indeed, the concepts of the schizophrenogenic mother and a single schizogene are still controversial today. However, it should be acknowledged that Meehl (1989) later rejected the notion of a schizophrenogenic mother. There have also been problems identified with how to test the construct of schizotaxia_Meehl directly and whether this is achievable.

There was minimal research directly after the publication of Meehl’s 1962 article that focused on his theory and this may have been because of the controversy associated with it. Some authors (e.g., Jang, Woodward, Lang, Honer, & Livesley, 2005; Tsuang et al., 1999a) claim that Meehl’s conceptualisation of schizotypy exists in the diagnostic nomenclature of the DSM as schizotypal personality disorder. However, as will be discussed in the next section, schizotypy and schizotypal personality disorder are not the same constructs.

*Meehl’s Schizotypy and Schizotypal Personality Disorder*

Some aspects of Meehl’s (1962, 1989, 1990b) conceptualisation of schizotypy overlap with the DSM-IV’s (APA, 1994) conceptualisation of schizotypal personality disorder but there are also many differences. Meehl’s conceptualisation is somewhat broader than that included in the DSM-IV. According to the DSM-IV (APA, 1994), schizotypal personality disorder is characterised by a number of social and interpersonal deficiencies which are pervasive in nature. In particular, the disorder is typified by the individual experiencing discomfort with relationships and a reduced ability to have relationships. In addition, individuals with schizotypal personality disorder tend to exhibit
cognitive or perceptual distortions and odd behaviour. These deficits are usually manifest by early adulthood and occur in a variety of situations.

More specifically, according to the DSM-IV (APA, 1994) the deficits of schizotypal personality disorder include ideas of reference, where events are interpreted as having special meaning for the individual, but not delusions. The individual may think that they have magical control or may exhibit odd beliefs that are incongruous with their culture and have a strong influence on their behaviour, for example, repeating an action a set number of times to avoid a negative outcome. The individual may experience unusual perceptual sensations as well as unusual speech and evidence of odd thoughts. There may be a degree of suspiciousness or paranoid ideation evident in an individual with schizotypal personality disorder. In addition, their affect is limited and may be inappropriate in their interactions with others. The individual may be odd, eccentric, or peculiar in their behaviour or appearance. Interpersonal relationships are problematic and this is often seen with a lack of close friends. Lastly, excessive social anxiety is often present in people with schizotypal personality disorder and this does not decrease with familiarity.

In order to meet DSM-IV (APA, 1994) criteria for schizotypal personality disorder, an individual must display 5 or more of the 9 deficits described above. A diagnosis of schizotypal personality disorder is only given if the individual’s symptoms do not occur exclusively during the course of schizophrenia, a mood disorder with psychotic features, another psychotic disorder, or a pervasive developmental disorder (APA, 1994).

The DSM-IV (APA, 1994) reports that approximately 3% of the general population has schizotypal personality disorder and it may precede schizophrenia. This value is noticeably less than Meehl’s (1990b) conjecture that 10% of the general population has his conceptualisation of schizotypy. Research with people with schizotypal personality
disorder has estimated that their first-degree relatives have a morbid risk of 18.9% of developing schizotypal personality disorder themselves (Battaglia, Bernardeschi, Franchini, Bellodi, & Smeraldi (1995).

Research has indicated that there is some degree of overlap between schizotypy and schizotypal personality disorder. Torgersen et al. (2002) studied the schizotypal personality features in a group of 663 people from a genetic spectrum perspective of schizophrenia. They assessed schizotypal personality disorder and other psychopathology, dividing the participants into 4 groups: (a) participants with schizotypal personality disorder who were first-degree relatives (including monozygotic or dizygotic twins) of an index twin with schizophrenia; (b) participants with schizotypal personality disorder who did not have a first-degree relative with schizophrenia; (c) participants with other axis I and II disorders who did not have a first-degree relative with schizophrenia or schizotypal personality disorder; and (d) participants without psychopathology and without a first-degree relative with schizophrenia or schizotypal personality disorder. Participants were assessed for schizotypy features using Baron’s (1980, cited in Torgersen et al., 2002) Schedule for Interviewing Borderlines.

Torgersen et al. (2002) found that participants with schizotypal personality disorder and a first-degree relative with schizophrenia tended to exhibit negative features of schizotypy while people with schizotypal personality disorder and no first-degree relatives with schizophrenia tended to exhibit positive features of schizotypy such as magical thinking, depersonalisation, illusions, and social anxiety. Relative to participants with schizotypal personality disorder who did not have a first-degree relative with schizophrenia, participants with schizotypal personality disorder who were first-degree relatives of an individual with schizophrenia had higher scores for only two features of schizotypy: inadequate rapport and odd communication. Participants with schizotypal
personality disorder and a first-degree relative with schizophrenia had higher scores on all
the features of schizotypy compared to participants with other axis I and axis II disorders
and without a first-degree relative with schizophrenia and compared to participants
without psychopathology and without a first-degree relative with schizophrenia. Torgersen et al. (2002) suggested that there is a difference between schizotypal personality disorder as it is observed within the genetic spectrum of schizophrenia compared to outside of the spectrum. Indeed, Chang and Lenzenweger (2005) have highlighted that Meehl’s schizotypy “refers to an unobservable personality organisation that contains the liability for schizophrenia” (p. 85) and “is not restricted to those phenotypic features typically associated with DSM-defined schizotypal personality disorder” (p. 85).

It is apparent that Meehl’s concept of schizotypy is broader than the diagnostic construct of schizotypal personality disorder although there is some degree of overlap. As noted earlier, there was minimal research directly after the publication of Meehl’s 1962 article that focused on his theory. In later years, however, following revisions and further clarification, research based on Meehl’s theory expanded rapidly and included research such as the study described by Torgersen et al. (2002). As a result, a large field of research on risk and predisposition for schizophrenia exists today.

Risk for Schizophrenia

Risk for schizophrenia can be considered in two main forms: risk factors and risk markers (Compton, 2004). Risk factors are attributes that are considered to have a causal role in the aetiology of schizophrenia (Compton, 2004); these were discussed in Chapter 2. Risk markers are considered to be indicators of vulnerability to developing schizophrenia and can be in the form of biological, neuropsychological, or psychological variables. The risk markers assessed in studies of risk for schizophrenia include neurological soft signs,
neuropsychological impairments, behavioural factors, intellectual functioning, brain structure, and self-reported personality. Research has found that risk for schizophrenia has been associated with a large variety of phenomena, including impairments in attention (e.g., Jones, Cardno, Sanders, Owen, & Williams, 2001; Michie et al., 2000; Saoud et al., 2000), verbal memory impairments (Lyons et al., 1995), problems with executive functioning (Saoud et al., 2000), neurological soft signs such as impairments in fine motor coordination (e.g., Obiols, Serrano, Caparrós, Subirá, & Barrantes, 1999), poorer intellectual functioning (Davidson et al., 1999), deviant brain structure as measured by MRI (Faraone et al., 2003), high scores on measures of schizotypy (e.g., Lenzenweger & Loranger, 1989), and poor social adjustment (Malmberg, Lewis, David, & Allebeck, 1998).

There are two key methodological approaches to the identification of individuals at risk for schizophrenia. Studies of biological risk involve the assessment of relatives of people with schizophrenia. Adoption (e.g., Kety, 1987, 1988), twin (e.g., Kendler et al., 1991), and family studies (e.g., Saoud et al., 2000) fall within this approach as these methods are presumed to identify genetic high-risk groups (Lenzenweger & Moldin, 1990). The second approach identifies individuals with a psychometric risk for schizophrenia and involves assessing groups of participants for attributes that are thought to precede schizophrenia. Usually, these groups are identified psychometrically with measures thought to assess for predisposition for schizophrenia. The groups are then compared to control groups on any number of biological, neuropsychological, or psychometric measures to determine if there are any differences between the groups. These groups are called psychometric high-risk groups (Lenzenweger & Moldin, 1990).

Among the genetic high-risk research are several large-scale longitudinal studies. These studies have three main goals, namely, to determine in what way are those
identified as being at risk different to those not at risk, to shed light on the aetiology of schizophrenia, and to evaluate the long-term outcome of those at risk. These studies include, among others, the Edinburgh High-Risk Study (e.g., Hodges, Byrne, Grant, & Johnstone, 1999) and the New York High-Risk Project (e.g., Cornblatt, 2002; Cornblatt, Dworkin, Wolf, & Erlenmeyer-Kimling, 1996; Dworkin et al., 1990; Ott, Roberts, Rock, Allen, & Erlenmeyer-Kimling, 2002). The participants in these research projects have been selected on the basis of having a first-degree relative with schizophrenia. These studies have produced various findings. For example, researchers from the New York High-Risk Project found that the neuropsychological domain of attention was significantly impaired in the high-risk sample compared to control samples. This suggests that impairments in attention may be a risk marker for schizophrenia. The long-term outcome of these studies is yet to be determined.

Another type of genetic high-risk research involves studies where a cross-sectional approach is used. For example, the Danish Adoption Study of Schizophrenia (e.g., Kendler, Gruenberg, & Strauss, 1982; Kety, 1987, 1988) examined several variables thought to be critically involved in the aetiology of schizophrenia. Participants in the study were 34 individuals with schizophrenia who had been adopted as infants, 34 individuals without a psychiatric history who had been adopted and the biological and adoptive relatives of the two groups. The researchers of the Danish Adoption Study found that 7.3% of the biological relatives of the schizophrenic adoptees had a schizophrenia-spectrum disorder compared to 1.9% of the biological relatives of the controls (Kety, 1988). In addition, the rates of schizophrenia-spectrum disorders in the nonbiological adoptive relatives of the schizophrenic adoptees were the same as in the control samples. These results support the notion that risk for schizophrenia involves a genetic component.
The genetic high-risk studies have contributed to our understanding of risk for schizophrenia in people who have a first-degree relative with schizophrenia. The designs of these studies have many strengths, especially the longitudinal studies. However, there is one noticeable weakness: the genetic high-risk studies do not capture all those who may be at risk for schizophrenia. According to Gottesman (1991), 89% of people with schizophrenia do not have a parent affected with schizophrenia, while 81% of people with schizophrenia do not have a first-degree relative with schizophrenia. This means that over 80 percent of people who develop schizophrenia are not included in research that only uses relatives of people with schizophrenia.

Furthermore, if genetics accounted fully for the transmission of schizophrenia it would be expected that if a monozygotic twin had schizophrenia then their cotwin would also have schizophrenia. This is not the case as was shown in Table 2.2 in Chapter 2. Gottesman (1991) pooled the data on lifetime risk from approximately 40 European studies and reported that monozygotic twins have an average lifetime risk of 48% of developing schizophrenia if one twin has schizophrenia; dizygotic twins have a risk of 17%; while the children of parents, both with schizophrenia, have an average lifetime risk of 46%. This suggests that there are other factors, in addition to genetics, that play a role in risk for schizophrenia.

Consequently, the psychometric high-risk approach has several important advantages over the genetic high-risk approach. By assessing symptoms in groups that are not pre-selected based on having schizophrenia in their family, a greater variety and number of individuals who may potentially develop schizophrenia in the future are available for assessment. Because participants still choose whether or not to take part, this method is not completely random, however, selection of participants using the psychometric high-risk approach is more random than selecting on the basis of biology.
Another advantage of the psychometric high-risk approach involves the issue of consent which is more complicated with the genetic high-risk approach than the psychometric high-risk approach. With the genetic high-risk approach, if researchers do not have a participant pool already established then consent is firstly required from the management of mental health services and hospitals to approach their patients. Because of privacy and confidentiality reasons, the researchers usually cannot directly ask the patients if they would take part in the study, therefore consent and agreement is required from staff to ask their patients. Then, consent is required from individuals with schizophrenia so that researchers can contact their first-degree relatives. Lastly, consent needs to be obtained from the first-degree relatives to take part. With the psychometric high-risk approach, the general population is approached through advertisements and course requirement studies at university, and is generally more accessible.

Participants recruited through the genetic high-risk approach may be at higher risk of developing other psychiatric illnesses. However, participants recruited through the psychometric high-risk approach may be less likely to have a history of psychiatric illness or a history of substance use, and less likely to be on psychiatric medication. All of these factors may impact on the study of risk for schizophrenia; studies that employ the psychometric high-risk approach will be less likely to be affected by these potentially confounding factors.

The psychometric high-risk approach to studying risk for schizophrenia has many advantages over the genetic high-risk approach. Research that has focused on Meehl’s (1962, 1989, 1990b) theory of risk for schizophrenia has utilised the psychometric high-risk method of recruitment. A selection of this research will be considered next.

Research on Meehl’s Schizotypy
In the decade after the publication of Meehl’s (1962) original paper on his theory of schizotypia, very few published studies were carried out that focused on his theory. However, over the last few decades, research studies that have focused on Meehl’s (1962, 1989, 1990a) theory and concepts have increased dramatically in number. Some of these studies are consistent with Meehl’s conceptualisation of schizotypy, while others are not. This problem reflects a conceptual issue where researchers have perhaps misinterpreted or misrepresented components of Meehl’s (1962, 1989, 1990b) theory of risk for schizophrenia.

Initially, schizotypy was assessed using measures such as the Object Sorting Test, the Minnesota Multiphasic Personality Inventory, and the Rorschach and Thematic Apperception Tests. Grove (1982) identified a number of problems with studies that have used these tests. Some of the problems were methodological in nature, and contributed to difficulties with replicating results. Among these problems were a poor choice of indicators from the tests; and questionable psychometric properties of the tests. Grove (1982) suggested that research in this area would improve if studies employed indicators that were more representative of schizotypy symptoms with good psychometric properties.

In 1964, Meehl produced an unpublished manual containing information on 25 signs of schizotypy. These included intense ambivalence, anhedonia, body-image aberrations, chaotic sexuality, cognitive slippage, countertransference strain on the clinician, deflated self-esteem, dependency or demandingness, feeling different from others, distrust of others, failure to achieve, flat affect, hatred of mother, magical ideation, micropsychotic episodes, extreme narcissism, pan-anxiety, poor outcome, psychosomatic or neurological signs, intense rage, repetition of material, self-injury, social fear, suicidal ideation or attempt, and special signs which included 17 heterogeneous indicators of schizotypy. Meehl (1964) emphasised that the checklist was not a psychometric instrument but that he
had produced it for clinicians and researchers to use in the study of the correlates of schizotypy. Consequently, while trying to address the problems identified by Grove (1982), a number of researchers have designed psychometric assessment tools that they purport to be based on aspects of Meehl’s checklist of schizotypy. However, it is not entirely clear that all of these psychometric measures are truly representative of the attributes of Meehl’s schizotypy. This is evident where some measures are consistent with Meehl’s conceptualisation of risk for schizophrenia whereas others are more closely aligned with DSM classifications for disorders such as schizotypal personality disorder yet still claim to be representative of Meehl’s schizotypy. Measures purported to be consistent with Meehl’s schizotypy will be considered next.

The Chapman scales. Some of the more well-known and frequently used measures of schizotypy include a series of self-report scales with a true-false format developed by Chapman and Chapman and colleagues. Their measures reportedly assess various aspects of schizotypy and include the Perceptual Aberration Scale (PAS; Chapman, Chapman, & Raulin, 1978) designed to assess an individual’s experience of distortions in perceptions of their own body image as well as distortions in other objects; the Magical Ideation Scale (MIS; Eckblad & Chapman, 1983) designed to evaluate people’s beliefs about magical influences on their own experiences; the Referential Thinking Scale (REF; Lenzenweger, Bennett, & Lilienfeld, 1997) designed to assess simple and guilty ideas of reference; the Physical Anhedonia Scale (PhA; Chapman, Chapman, & Raulin, 1976) which has items that assess pleasure obtained from activities such as touching, eating, smelling, and movement; and the Social Anhedonia Scale (SAS; Chapman et al., 1976) which has items that assess pleasure obtained from things such as having a conversation, and social activities.
Research with these measures has been carried out with the first-degree relatives of people with schizophrenia; participants from clinical populations; and non-clinical participants with no genetic link to schizophrenia. Studies employing these measures have attempted to elucidate the correlates of risk for schizophrenia. Generally, participants are screened with a psychometric measure of schizotypy and divided into groups based on their scores on the measure. The participants are then administered one or a combination of assessment tools that assess psychopathology, psychophysiological or neuropsychological functioning to determine if the factors are correlates of schizotypy. The psychometrically-different groups are compared in regards to their performance on the psychopathological, neuropsychological or psychophysiological measures. In addition to the correlates of schizotypy, studies have considered the psychometric measures as predictors of psychosis. Studies employing these methods have found mixed results.

**Neuropsychological correlates of schizotypy.** A number of studies have used the psychometric approach with the psychometric measures of Chapman and Chapman and colleagues to investigate the types of neuropsychological symptoms displayed by people who obtain high scores on the measures. For example, Lenzenweger, Cornblatt, and Putnick (1991) divided a group of 726 first-year university students into 2 groups based on high and low scores on the PAS. They administered a test of sustained attention, the Continuous Performance Test, Identical Pairs version (Cornblatt, Risch, Faris, Friedman, & Erlenmeyer-Kimling, 1988) to the participants. Lenzenweger et al. (1991) found that people in the schizotypy group had significantly lower hit rate scores and poorer discriminability, relative to the nonschizotypy group. They concluded that people with a psychometrically identified attribute of schizotypy had a subtle deficit in sustained attention, which may be an indicator of schizophrenia risk or liability. In addition, studies of people identified as schizotypal on the basis of PAS scores have found that relative to
controls, schizotypal people have impaired spatial memory (Park, Holzman, & Lenzenweger, 1995), report greater cognitive slippage (Gooding, Tallent, & Hegyi, 2001), communication deficits (Miller & Chapman, 1983), longer reaction times on attention tests (Lenzenweger, 2001), higher errors of failure to maintain set on a test of executive functioning (Park et al., 1995), make more perseverative errors, and achieve fewer categories on a test of executive functioning (Gooding et al., 2001; Gooding, Kwapisil, & Tallent, 1999).

Other studies have employed different psychometric tools with the psychometric approach to determine schizotypy group membership and neuropsychological function. These studies have also found differences in neuropsychological functioning between groups with high scores and low scores on measures reported to assess for attributes of schizotypy. For high-scoring schizotypy groups impairments have been observed in the domains of verbal memory (e.g., Calev, Venables, & Monk, 1983); attention and verbal fluency (Lemos Giráldez, Inda Caro, López Rodrigo, Paino Piñeiro, & Besteiro González, 2000); and working memory (Martínez Suárez, Lemos Giráldez, Inda Caro, Paino Piñeiro, & López Rodrigo, 1999).

Yet other studies have selected participants using the genetic high-risk approach described previously and recruited individuals with schizophrenia, their first-degree relatives, and control participants. Researchers typically administer tests of neuropsychological functioning to the participants and then determine schizotypy status using psychometric measures of schizotypy (e.g., Laurent et al., 1999, 2001). The relationship between impairments in neuropsychological functioning and schizotypy status using this approach has produced mixed results. These studies have often found, as expected, that the schizophrenia group is significantly more impaired on the neuropsychological tasks (in the domains of attention and executive functioning) relative
to the first-degree relatives, who are in turn significantly more impaired on many of the tasks than the controls. However, these studies are particularly interesting in that they have found little or no significant correlations between schizotypy status as assessed by psychometric measures and neuropsychological performance in the first-degree relatives of people with schizophrenia. For example, Franke, Maier, Hardt, Hain, and Comblatt (1994) found no evidence of a correlation between attention deficits and high scores on the PhA and PAS in relatives of individuals with schizophrenia while Chen et al. (1998) found some evidence of a correlation. Laurent et al. (2000) administered a battery of neuropsychological tests and schizotypy measures to relatives of individuals with schizophrenia and found only one correlation between impaired executive functioning and high scores on the PhA. These results raise questions as to the suitability of deficits in attention and executive functioning as consistent indicators or markers of schizotypy and whether the measures reported to be representative of Meehl’s schizotypy are consistent with this notion. This also highlights the need for research in this area to assess the general population when assessing for schizotypy rather than the narrow population of first-degree relatives of individuals with schizophrenia when attempting to clarify the answers to these questions. It may be that studies using the genetic high-risk approach are not consistent with Meehl’s construct of schizotypy and are instead consistent with another construct of risk for schizophrenia which will be considered in more detail in Chapter 4.

**Psychopathological correlates of schizotypy.** In addition to the neuropsychological correlates of schizotypy, studies have investigated psychopathological correlates and the ability of the measures to predict development of psychosis in individuals and relatives. Eckblad and Chapman (1983) administered the MIS to 1,512 university students and 28 participants with high scores were invited to take part in a diagnostic interview, along with
27 control participants. Eckblad and Chapman (1983) found that compared to participants with low magical ideation scores, the participants with high magical ideation scores had significantly more psychotic-like symptoms and schizotypal symptoms.

Other research has considered the relationship between attributes of schizotypy in psychiatric patients and risk of schizophrenia-spectrum disorders in their first-degree relatives. Lenzenweger and Loranger (1989) divided a group of 101 non-psychotic psychiatric inpatients into 2 groups based on their scores on the PAS: a schizotypy group and a nonschizotypy group. They then determined the psychiatric status of the first-degree relatives of the participants and found that, compared to the relatives of the nonschizotypy group, significantly more of the first-degree relatives of people in the schizotypy group had been treated for schizophrenia. There were no significant differences between the two groups for unipolar or bipolar depression (Lenzenweger & Loranger, 1989). This suggests that the experience of perceptual distortions, thought to be consistent with an attribute of Meehl’s construct of schizotypy, may be an indicator of risk for schizophrenia.

In addition to risk of psychosis in relatives of schizotypy high-scoring individuals, the ability of the scales of Chapman, Chapman, and colleagues to predict psychosis in individuals has been investigated in a large-scale study (Chapman & Chapman, 1985, 1987; Chapman, Chapman, KwapiI, Eckblad, & Zinser, 1994). At recruitment to the study, in the late 1970’s and early 1980’s, 7,800 undergraduate university students were administered the PAS, MIS, PhA, SAS, and the Impulsive Nonconformity Scale (NonCon; Chapman et al., 1984) (Chapman & Chapman, 1985). Participants \( n = 534 \) with scores that were 1.96 \( SD \) or higher above the mean were selected to take part in the follow-up study. A total of 5 groups were formed: a PerMag group \( n = 193 \) based on PAS and MIS scores, a NonCon group \( n = 74 \), a PhA group \( n = 75 \), a combined score group \( n = 33 \)
based on having a high sum of the \( z \) scores across all 4 scales, and a control group (\( n = 159 \)) with scores lower than 0.5 \( SD \) below the mean. The participants also completed an interview based on the assessment of psychotic-like experiences. Chapman and Chapman (1987) re-assessed 439 of the participants 25 months after recruitment. They found that three participants, all of whom were in the PerMag group, had developed psychosis. One of these had schizophrenia.

Chapman et al., (1994) conducted follow-up assessments 10 to 15 years later with 503 of the original 534 participants. Participants took part in a diagnostic interview that evaluated their overall functioning and psychopathology, as well as family history of psychopathology. They found that 14 of the 503 participants met criteria for a DSM-III-R diagnosis of psychosis at follow-up, including schizophrenia, psychosis NOS, delusional disorder, psychotic bipolar, and psychotic major depression. Of these participants, 10 were in the PerMag group and only this group was significantly different from the control group. Fifteen percent of the PerMag group reported that they had a family member with psychosis. The PerMag and NonCon groups both reported significantly higher psychotic-like experiences at follow-up than the control group. Chapman et al. (1994) divided the PerMag group into 2 groups based on their reports of psychotic-like experiences at first interview. They found that, in the group who reported moderate psychotic-like experiences at first interview, there were more participants with psychosis (9 of 66) than in the group who reported low psychotic-like experiences at first interview (1 of 125). They concluded that their first interview and follow-up assessments of psychotic-like experiences were both a valid indicator and outcome measure of risk for psychosis.

Research employing another psychometric measure thought to be consistent with Meehl’s schizotypy, the Structured Interview for Schizotypy (SIS; Kendler, Lieberman, & Walsh, 1989) has found similar results concerning the development of psychosis. Miller
et al. (2002) reported results from the Edinburgh High-Risk Study on schizotypy and subsequent psychosis development. Three groups of participants were recruited from 1994 onwards: (i) 155 high-risk participants aged 16 to 25 who have at least two first-degree or second-degree relatives with schizophrenia; (ii) 36 non-affected individuals with no family history of schizophrenia to serve as controls for the high-risk group; and (iii) 37 participants aged 16 to 26 with first-episode schizophrenia but no relatives with schizophrenia. Miller et al. (2002) evaluated the clinical status of the 212 participants that had been recruited to the high-risk group by mid-1996. This identified a subgroup of 78 participants whom had high levels of social withdrawal and odd behaviour as measured by the SIS. Seven of these participants had developed schizophrenia over a 39-month period while none of the control participants had developed psychosis. These preliminary results suggest that there may be a relationship between high schizotypy scores and subsequent development of psychosis.

Miller et al. (2002) also reviewed the clinical status of all the participants in 1999. They divided the high-risk participants into 113 non-symptomatic psychosis high-risk participants and 38 symptomatic psychosis high-risk participants. Miller et al. (2002) found that the symptomatic high-risk participants scored higher on the SIS than the non-symptomatic high-risk participants. The non-symptomatic high-risk group SIS scores were similar to control group scores. These preliminary results suggest that there may be a relationship between high schizotypy scores and symptoms of psychosis. However, the long-term outcome of the Edinburgh High-Risk Study is yet to be determined.

*Psychophysiological correlates of schizotypy.* Research about schizotypy has also investigated psychophysiological correlates such as impaired visual information-processing (e.g., Nakano & Saccuzzo, 1985), dysfunctional smooth pursuit oculomotion or eye tracking (e.g., Holahan & O’Driscoll, 2005; Iacono, 1993), dysfunctions in prepulse
inhibition of the startle response (e.g., Evans, Gray, & Snowden, 2005) and dysfunctional 
regulation of responses to sensory input with the P50 wave (e.g., Croft, Dimoska, 
Gonsalvez, & Clarke, 2004; Freedman et al., 1993). Studies of these correlates have also 
employed one of two approaches where participants are identified using the psychometric 
high-risk approach or the genetic high-risk approach, with fairly consistent results. 
However, research in this area involves a conceptual problem. Some researchers report 
that they have investigated the psychophysiological correlates of schizotypy while others 
report that they have investigated the psychophysiological correlates of schizotaxiaMeehl. 
The problem with this discrepancy is that it is questionable as to whether schizotaxiaMeehl 
can be measured directly. Meehl (1962, 1989, 1990b) proposed that the schizotaxicMeehl 
brain is characterised by hypokrisia. However, he acknowledged that it is not necessarily 
practicable to measure hypokrisia (Meehl, 1990a). Furthermore, the studies claiming to 
investigate schizotaxiaMeehl have often utilised self-report measures as screening methods 
and therefore are similar in methodology to many of the studies described in the previous 
section that claim to evaluate schizotypy. As a result of this misunderstanding or 
misrepresentation of Meehl’s concepts, the studies that claim to assess schizotaxiaMeehl 
would be more appropriately classified as assessing Meehl’s schizotypy.

Many studies involving measures purported to be consistent with Meehl’s 
schizotypy have utilised the scales of Chapman, Chapman, and colleagues for assessing 
schizotypy and have found results suggesting that individuals with high-schizotypy scores 
are different to individuals with low-schizotypy scores in a number of areas of 
functioning. However, some researchers have observed inconsistent results with the 
 scales of Chapman and Chapman. For example, Clementz, Grove, Katsanis, and Iacono 
(1991) administered the PAS and the PhA to a group of 54 individuals with schizophrenia 
and 146 of their first-degree relatives, as well as 178 control participants. They found that
individuals with schizophrenia had the highest scores on the PAS, but unexpectedly, the relatives of individuals with schizophrenia had lower scores on the PAS than the controls. The pattern of results with the PhA was as expected, individuals with schizophrenia had the highest scores, followed by their first-degree relatives, and the controls had the lowest scores. Clementz et al. (1991) did not find a relationship between perceptual aberration and the presence of schizophrenia in first-degree relatives but did find a relationship between physical anhedonia and the presence of schizophrenia in first-degree relatives. Consequently, they query the usefulness of the PAS for evaluating risk for schizophrenia in first-degree relatives.

In response to this, Lenzenweger (1994) has suggested that relatives may have adopted a defensive and nondisclosing attitude when completing the study, as they may have known that they were selected based on their relative’s status. However, ambiguity surrounding results of studies that have utilized the PhA (e.g., Chapman et al., 1994) has been highlighted by Lenzenweger (1994). He has suggested that researchers consider using other psychometric measures of schizotypy that have been demonstrated to have more consistent results, and higher reliability and validity.

To summarise, research with the Chapman scales and other psychometric measures that has utilised the psychometric high-risk approach has produced a number of findings. The interpretations of these findings are that relative to individuals with low scores on the scales, individuals with high scores also experience a range of abnormalities in neuropsychological and psychophysiological functioning, and are more likely to display symptoms of psychosis and schizotypal personality disorder. In addition, individuals with high scores are more likely to have a first-degree relative with psychosis, and are more likely to develop psychosis themselves. Studies that have used the genetic high-risk approach have found conflicting results with the relationship between schizotypy status
and neuropsychological impairment, suggesting that the methods may measure different constructs. Consequently, there are many psychometric issues that need to be resolved. The conflicting results of studies that use different methods to evaluate schizotypy may be a result of the heterogeneous nature of the structure of schizotypy. The structure of schizotypy will be considered next.

The Structure of Schizotypy

Research has shown that people identified as having schizotypy (either through psychometric measures or as first-degree relatives of people with schizophrenia) differ from nonschizotypal individuals in terms of clinical, neuropsychological, and psychophysiological indicators. Another line of research in the area of schizotypy has attempted to identify whether schizotypy is multidimensional, and if so, the components of schizotypy. Many of these studies have used factor analysis to determine this and there have been some consistent results.

For example, Kendler et al. (1991) conducted comprehensive assessments of schizotypy with 29 pairs of twins from the general population; 13 twin pairs were monozygotic and 16 were dizygotic. They assessed the twins for schizotypal signs, symptoms, and traits using both an interview and a range of self-report questionnaires, as well as a number of neuropsychological tests for attention, and smooth pursuit eye movements. They found that 3 of the 58 twins met criteria for schizotypal personality disorder and 23 of the 58 twins displayed schizotypal traits. Kendler et al. (1991) conducted a factor analysis using the assessment measures. Analyses of the clinically-rated symptoms yielded evidence for two schizotypy factors: a positive factor and a negative factor. The positive factor included psychotic-like symptoms, interpersonal sensitivity, social anxiety, and speech organisation. The negative factor included social
isolation, irritability, low impulsivity, problems with rapport, and problems with affect regulation. Analyses of the self-report data also yielded evidence for two factors: a positive trait factor and trait anhedonia. A high degree of correlation was observed between the positive symptom and positive trait factors and a moderate degree of correlation between the negative symptom and trait anhedonia factors. The negative symptom factor was significantly correlated with neuropsychological dysfunction in the domain of attention and dysfunction in eye tracking. Kendler et al. (1991) concluded that their results are consistent with the notion that schizotypy is heterogeneous and involves more than one dimension. Other researchers (e.g., Linney et al., 2003) have supported this conclusion.

Other research has found evidence for more than two factors of schizotypy. In a study involving the relatives of people with schizophrenia, Nuechterlein et al. (2002) found evidence for three factors of schizotypy. These included a positive schizotypy factor, a negative schizotypy factor, and a cognitive disorganisation factor which consisted of odd or eccentric behaviour or appearance, and three neuropsychological indicators of schizotypy. Other studies have also found evidence of these three factors in both clinical and normal populations (e.g., Calkins, Curtis, Grove, & Iacono, 2004; Rossi & Daneluzzo, 2002; Suhr & Spitznagel, 2001a, 2001b; Vollema & Hoijtink, 2000) while some researchers have found evidence for a social impairment or adjustment factor as opposed to a cognitive disorganisation factor (e.g., Venables & Rector, 2000).

Vollema and van den Bosch (1995) reviewed the self-report measures of schizotypy available at the time and associated studies that had used factor analysis to determine the structure of schizotypy. They found that there was consistent evidence and good construct validity for two factors of schizotypy: positive and negative factors. They identified another two factors: nonconformity and social anxiety or cognitive disorganisation factors
but reported that these were lacking in validation studies. Vollema and van den Bosch (1995) highlighted the similarities between the factors identified in schizotypy and the multidimensional nature of schizophrenia. Fanous, Gardner, Walsh, and Kendler (2001) evaluated positive and negative symptoms in a group of individuals with psychosis (schizophrenia, simple schizophrenia, schizoaffective disorder, delusion disorder, schizophreniform disorder, brief reactive psychosis, and psychosis NOS) and schizotypy symptoms in their relatives, (total \(n = 1891\)). Fanous et al. (2001) found that positive and negative symptoms of schizophrenia significantly predicted positive and negative symptoms of schizotypy. However, there were more significant relationships between negative symptoms of schizophrenia and schizotypy than there were for positive symptoms. This ties in with Meehl’s (1990b) quasi-dimensional view of the schizophrenia-spectrum, rather than a fully dimensional view (Claridge, 1997). Meehl’s (1962, 1990b) view is that individuals either do or do not have schizotypy, the precursor for schizophrenia, and that the pathway between schizotypy and schizophrenia occurs along a dimension.

The results are somewhat mixed for the structure of schizotypy. Research has clearly identified that schizotypy is multidimensional and consists of at least two factors. However, there is variation in the studies that have produced these results in terms of the type of analysis they use (factor vs. cluster), the sample size used, and the assessment measures that are used. Another aspect of schizotypy that has recently been investigated is whether the underlying structure of the construct is dimensional or categorical. This type of research has employed taxometric analysis procedures (e.g., Golden, 1982; Golden & Meehl, 1979; Meehl 1973; Meehl & Yonce, 1994, 1996; Waller & Meehl, 1998), which are statistical procedures that can be used to assist distinguishing evidence of latent taxa from distributions of continuous data. The results of these procedures are analysed to
determine if there is evidence for the underlying structure of a construct (such as schizotypy) in terms of whether it is taxonic or dimensional. These procedures and associated research on schizotypy are described in more detail in Chapter 5.

Summary

Meehl (1962, 1989, 1990b) proposed a theory of risk for schizophrenia where he considers liability for the disorder in the form of schizotaxia and schizotypy. Meehl’s theory is quasi-dimensional in that all those with schizotaxia, an inherited integrative neural defect, develop a personality organisation called schizotypy, and some schizotypal individuals will decompensate and develop schizophrenia. In addition to genetic factors, environmental factors impact on the development of both schizotypy and schizophrenia.

Meehl’s theory highlights problems with diagnostic systems and the reliance on end-state symptoms while the components of the theory are conceptually independent of each other and have benefits for research in the area of risk for schizophrenia. Research in this area has traditionally involved two approaches: a psychometric high-risk approach and a genetic high-risk approach, with the former having many advantages over the latter. Research into Meehl’s schizotypy has resulted in the development of a number of psychometric measures. Studies that have employed these measures have attempted to elucidate the correlates of risk for schizophrenia. In addition, some studies have concluded that some of the psychometric measures can be used to predict the development of psychosis. Some research appears to be consistent with Meehl’s notion of schizotypy while other research is associated with conceptual problems relating to the misrepresentation or a misunderstanding of Meehl’s concepts. Research has also investigated the structure of schizotypy and identified that schizotypy is multidimensional in nature involving at least two factors.
Most research has focused on Meehl’s conceptualisation of schizotypy as his conceptualisation of schizotaxia. Meehl is difficult to measure directly. Recently, a group of researchers have developed a new conceptualisation of schizotaxia, based on modifications of Meehl’s theory of risk for schizophrenia. They propose that this is measurable and have established research criteria for the construct. This theory and associated research will be considered in Chapter 4.
CHAPTER 4

Schizophrenia, Schizotaxia, and Schizotypy: Tsuang et al.'s Perspective

Most past research has focused on Meehl's (1962, 1989, 1990b) conceptualisation of schizotypy. Recently, however, Tsuang and colleagues (e.g., Faraone, Green, Seidman, & Tsuang, 2001; Tsuang et al., 1999b, 2000a, 2000b; Tsuang & Faraone, 1999) have developed a theory and conducted research based on their interpretation and conceptualisation of both schizotaxia\textsubscript{Meehl} and schizotypy. Tsuang and colleagues' theory contrasts with Meehl's theory in many ways.

Tsuang and Colleagues' Theory of Risk

Background to Tsuang and colleagues' theory. As discussed in Chapter 2, Tsuang et al. (2000a) are one group of many researchers who have highlighted various difficulties and problems with the current diagnostic criteria for schizophrenia. To address some of these problems, Tsuang and colleagues have attempted to develop a conceptualisation of schizophrenia that enhances what is known about the aetiology of the disorder. They have used Meehl's (1962, 1989, 1990b) theory of risk for schizophrenia as a starting point for their reformulation of the construct of schizophrenia. Recall that Meehl (1962, 1989, 1990b) proposed that people who are at risk of developing schizophrenia are born with a genetic mutation, which he postulated to be in the form of a single dominant gene. He conjectured that the genetic mutation produces an integrative neural defect, schizotaxia\textsubscript{Meehl}, which at its essence consists of an underlying, subtle problem called
hypokrisia. Meehl (1962, 1989, 1990b) further proposed that as a result of environmental effects such as social learning and reinforcement schedules, all individuals with schizotaxia, develop a schizotypal personality organisation. He asserted that the development and course of schizophrenia in the schizotypal person is contributed to by the interaction between a number of polygenic potentiators and the social environment. Tsuang and colleagues have used some of Meehl's concepts in their theory of risk for schizophrenia but their interpretation and, thus, their definition of these concepts is somewhat different to that of Meehl's. For the remainder of this thesis, to avoid confusion, \textit{schizotaxia}_{Tsuang} will be used to denote Tsuang and colleagues' conceptualisation of schizotaxia.

\textit{Schizotaxia}_{Tsuang} Like Meehl, Tsuang et al. (Faraone et al., 2001; Tsuang, 2000; Tsuang et al., 1999b, 2000a, 2000b; Tsuang & Faraone, 1999) have used the term schizotaxia to describe a genetic predisposition to schizophrenia. They have advocated for a multifactorial version of the diathesis-stress model that also incorporates neurodevelopmental factors (Figure 4.1). Tsuang and colleagues conjecture that the pathophysiology of risk for schizophrenia is a more subtle and milder version of the pathophysiology of clinical schizophrenia. They propose that predisposition to schizophrenia arises from the interaction of a number of genetic and environmental factors. Tsuang et al. (1999b) suggest that the nature of the interaction may be additive but do not limit their model to this method.
According to Tsuang (2000), the neurodevelopment of the embryo is determined by genes but is then modified by environmental factors. As a result, they propose that early environmental factors may interact with a genetic predisposition to produce a vulnerability to developing schizophrenia, called schizotaxiaTsuang (Tsuang & Faraone, 1999).
Examples of environmental factors include viral infection during pregnancy (e.g., influenza), in particular in the second trimester, and obstetric complications (e.g., hypoxia) (Tsuang, 2000; Tsuang & Faraone, 1999). Tsuang and colleagues further propose that the vulnerability for schizophrenia is manifested in children in the form of neurodevelopmental abnormalities or target features. They define target features as "clinical or neurobiological characteristics that are expressions of the underlying predisposition to an illness" (Tsuang & Faraone, 1999, p. 2). Examples of target features include cell and structural abnormalities in the brain (Seidman, 1997; Seidman et al., 2002a) as well as cognitive impairments (Tsuang, 2000; Tsuang & Faraone, 1999).

Tsuang and Faraone (1999) assert that the presence of neurodevelopmental abnormalities and target features does not mean that an individual will definitely develop schizophrenia.

**Development of schizophrenia.** Tsuang and colleagues suggest that neurodevelopmental abnormalities and environmental factors interact to result in further brain abnormalities and the development of schizophrenia. These environmental factors may include psychosocial stressors such as exposure to a dysfunctional family environment, the early absence of a father, and living in an urban area (Tsuang, 2000). The interaction occurs in the context of the first 20 to 30 years of life during which the brain continues to change and mature (Tsuang, 2000). Tsuang and colleagues propose that the onset of schizophrenia develops into psychosis when the individual with schizophrenia is exposed to further environmental stressors such as expressed emotion, life events, and biological factors, which can all impact upon brain dysfunction (Tsuang, Stone, & Faraone, 2001). In turn, psychosis can contribute to chronic schizophrenia. Furthermore, Tsuang and colleagues predict that the brain undergoes additional, often subtle, changes after the onset of psychosis and during the development of chronic schizophrenia; they refer to this as *neurodegeneration*. 
There are a number of similarities between the theories of Meehl and Tsuang et al. Both have conceptualised risk for schizophrenia from a neurodevelopmental viewpoint and considered the impact of environmental factors in this process. In addition, like Meehl (1990b), Tsuang et al. (2001) have noted that there may be phenocopies of schizophrenia that occur as a result of factors other than genetic transmission. For example, psychosis due to drug use, psychosis due to brain trauma, and infections during pregnancy (Tsuang et al., 2001). Tsuang and colleagues consider these individuals to be sporadic or non-familial cases.

It is possible to identify six main differences between schizotaxia\textsubscript{Meehl} and schizotaxia\textsubscript{Tsuang}. These differences relate to the proposed aetiology of schizotaxia, how schizotaxia is observed, the outcome of schizotaxia, the relationship between schizotaxia and other disorders, and the use of the term schizotypy.

Firstly, there is a difference in how the genetic component of the aetiology of schizotaxia has been conceptualised. Tsuang et al. (1999b) view the genetic influence as arising from a multifactorial process involving multiple genes while Meehl (1962, 1990b) proposed that schizotaxia\textsubscript{Meehl} arises from a single dominant gene. Faraone et al. (2001) state that they agree with Meehl’s conceptualisation of schizotaxia as the “underlying defect among people genetically predisposed to schizophrenia” (p. 1). They disagree, however, with Meehl’s theory that the genetic mutation that leads to schizotaxia\textsubscript{Meehl} involves a single dominant gene. Instead, Tsuang and colleagues (Faraone et al. 2001, Stone et al., 2001, Tsuang, Stone, Tarbox, & Faraone, 2002b) state that research on genetic linkage carried out after Meehl developed his theory suggests that multiple genes are involved. However, research involving specific chromosomes has not been well replicated.
The second difference also relates to the aetiology of schizotaxia\textsubscript{Tsuang}. Schizotaxia\textsubscript{Tsuang} arises from the interaction between multiple genes and negative environmental effects such as obstetric complications. In contrast, schizotaxia\textsubscript{Meehl} has a purely genetic basis, involving a single dominant gene and environmental factors have a subsequent effect upon schizotypy, rather than a role prior to the development of schizotaxia\textsubscript{Meehl}. Tsuang (2001) states that schizotaxia\textsubscript{Tsuang} and schizotaxia\textsubscript{Meehl} are similar in that both are viewed as the neurobiological substrate for schizophrenia, however, schizotaxia\textsubscript{Tsuang} results from the combination of both genes and environmental insults prior to and during birth. According to Tsuang, Stone, and Faraone (2002a), schizotaxia\textsubscript{Tsuang} is the product of both genetic and environmental factors because it can be very difficult to isolate the neurobiological effects of genes from the neurobiological effects of some environmental events such as prenatal infection.

The third difference involves the study of schizotaxia and how it is observed. Schizotaxia\textsubscript{Tsuang} is characterised by the presence of negative symptoms and various neuropsychological impairments (Stone et al., 2001, Tsuang et al., 2002b). In addition, Tsuang and colleagues are working towards developing a diagnostic construct for schizotaxia\textsubscript{Tsuang}. This is different to Meehl's (1962, 1990b) conceptualisation because he did not view schizotaxia\textsubscript{Meehl} and hypokrisia as being able to be measured by single assessment tools such as neuropsychological tests.

The fourth difference relates to the outcome of schizotaxia. Schizotaxia\textsubscript{Tsuang} is a stable construct whose likely outcome is neither schizotypy nor schizophrenia. In contrast, people with schizotaxia\textsubscript{Meehl} develop schizotypy and a small proportion of these will develop schizophrenia. People with a schizotaxia\textsubscript{Meehl} brain develop schizotypy as a result of the interaction between schizotaxia\textsubscript{Meehl} and environmental effects. Therefore, schizotypy is the clinical manifestation or phenotype of schizotaxia\textsubscript{Meehl}. Meehl (1989)
conceded that it may be possible for an individual with a schizotaxic\textsubscript{Meehl} brain not to develop schizotypy if in the future an appropriate intervention were to be developed and individuals could be identified as schizotaxic\textsubscript{Meehl} when they are infants. In contrast, schizotaxia\textsubscript{Tsuang} does not usually lead to schizotypy; and schizotypy is one of many possible outcomes of schizotaxia\textsubscript{Tsuang}. Schizotaxia\textsubscript{Tsuang} is manifested by neuropsychological impairments and the presence of mild negative symptoms, and does not always result in the development of schizophrenia.

The fifth difference also relates to perspectives of the relationship between schizotaxia and other disorders. As described in the previous point, Tsuang et al. hold a categorical view of the relationship where schizotaxia\textsubscript{Tsuang} does not necessarily lead to either schizotypy or schizophrenia. In addition, when schizophrenia does occur, it signals the end of schizotaxia\textsubscript{Tsuang}. This contrasts with Meehl's quasi-dimensional view where schizotaxia\textsubscript{Meehl} nearly always leads to schizotypy and some schizotypal people develop schizophrenia. The presence of schizophrenia does not signal the end of schizotaxia\textsubscript{Meehl} or schizotypy. Furthermore, Tsuang and colleagues have imposed a categorical view on Meehl's theory. Stone et al. (2001) have stated that according to Meehl (1962), "schizotaxia referred to a genetically mediated, subtle neurointegrative defect that progressed usually to either schizotypy or schizophrenia, depending on environmental circumstances" (p. 435). Most aspects of this statement are consistent with what Meehl's theory; however, according to Meehl all individuals who develop schizophrenia also have both schizotaxia\textsubscript{Meehl} and schizotypy. Stone et al. (2001) have interpreted Meehl's theory as schizotaxia\textsubscript{Meehl} leading to either schizotypy or schizophrenia, not both.

The last notable difference relates to the degree of overlap between schizotypy and schizotypal personality disorder and how these terms are used, a conceptual problem that was highlighted in Chapter 3. This problem underlies most of the differences described
between the theories of Meehl and Tsuang et al. Tsuang and colleagues equate schizotypy with schizotypal personality disorder and tend to use the terms interchangeably (e.g., Stone, Faraone, Seidman, Olson, & Tsuang, 2005). Consequently, they view schizotypy as the same as schizotypal personality disorder. However, schizotypy is clearly different to schizotypal personality disorder as it appears in diagnostic systems such as the DSM-IV (APA, 1994) and the ICD-10 (WHO, 1992). The most prominent difference is that schizotypy is much broader than schizotypal personality disorder and therefore encompasses more areas of functioning. Schizotypy includes some of the symptoms that Faraone et al. (2001) consider indicative of schizotaxiaTsuang. Therefore, it appears that Tsuang and colleagues’ schizotaxiaTsuang may be at the same level of analysis as Meehl’s schizotypy. In addition, both schizotaxiaTsuang and schizotypy involve genetic and environmental factors; are measurable and observable constructs; and the outcome for both is not necessarily schizotypal personality disorder.

There are a number of conceptual similarities and differences between the theories of Meehl and Tsuang and colleagues. The last difference noted above has particular relevance, as the goal of this thesis is to determine whether schizotypy and schizotaxiaTsuang are related or independent. To do this, an exploration of the relationship between schizotypal personality disorder and schizotaxiaTsuang is warranted and this will be considered in the following section.

*SchizotaxiaTsuang and Schizotypal Personality Disorder*

Tsuang et al. have advocated for the development of a diagnostic entry for schizotaxiaTsuang but only after future research has established that it is a valid construct. As part of the validation process, they acknowledge that it needs to be determined that schizotaxiaTsuang is different from schizotypal personality disorder in order for a separate
category to be established (Faraone et al., 2001). The first step needs to consider whether schizotaxia\textsubscript{Tsuang} is related to schizotypal personality disorder (Tsuang et al., 2002b).

Indeed, Tsuang and colleagues believe that there are differences between the two constructs, especially in regards to the proportion of people who are likely to develop the constructs. Tsuang and colleagues estimate that the proportion of first-degree relatives who have schizotaxia\textsubscript{Tsuang} is higher than for schizotypal personality disorder. Faraone et al. (1995a, 1995b) reported that 20% to 50% of relatives of individuals with schizophrenia experience the negative symptoms and neuropsychological impairment features of schizotaxia\textsubscript{Tsuang}. In contrast, Faraone et al. (2001) assert that less than 10% of relatives of individuals with schizophrenia develop schizotypal personality disorder. In addition, Faraone et al. (2001) state that most people with schizotaxia\textsubscript{Tsuang} will never develop schizotypal personality disorder or schizophrenia.

These figures suggest that schizotaxia\textsubscript{Tsuang} occurs more frequently than schizotypal personality disorder in relatives of individuals with schizophrenia. However, this is in contrast to other reports that Tsuang and colleagues have made. For example, Tsuang and Faraone (1994) reported that the incidence of schizotypal personality disorder in relatives is between 4% and 15% with a further 27% of relatives expressing symptoms of schizotypal personality disorder that fall short of the minimum required for diagnosis. In addition, research by other authors has indicated that 23% of first-degree relatives may be at risk of developing a schizophrenia-spectrum disorder including schizotypal personality disorder (Tienari et al., 2003) and a morbid risk of 18.9% of developing schizotypal personality disorders for first-degree relatives of individuals with schizotypal personality disorder (Battaglia et al., 1995).

If Faraone et al. (2001) are accurate in their statement that less than 10% of relatives develop schizotypal personality disorder then this means that it occurs at around the same
rates in first-degree relatives as the more severe disorder of schizophrenia. Research has shown that approximately 10% of first-degree relatives of individuals with schizophrenia also develop schizophrenia (Gottesman, 1991). This ranges from 6% for parents of schizophrenics to 17% for a sibling of an individual with schizophrenia who also has a parent with schizophrenia (Gottesman, 1991). This raises the question as to whether schizotaxia_{Tsuang} occurs more frequently than schizotypal personality disorder in relatives of individuals with schizophrenia as conjectured by Tsuang and colleagues, or is the occurrence of the two constructs actually quite similar?

**Heterogeneity of schizotypal personality disorder.** Tsuang and colleagues have cited the heterogeneous nature of schizotypal personality disorder as further evidence of the differences between the disorder and schizotaxia_{Tsuang} (Faraone et al., 2001). They claim that the heterogeneous nature of schizotypal personality disorder has arisen from the results of the two methods in which it has historically been researched. The clinical method, involves identifying people based on the mild schizophrenia symptoms that they experience, the people are considered personality disordered patients (Tsuang et al., 2002b). In contrast, the family research method involves identifying relatives of individuals with schizophrenia who also display mild schizophrenia symptoms (Faraone et al., 2001; Kendler, 1985). Tsuang and colleagues surmise that evidence from these studies indicates that there may be two forms of schizotypal personality disorder: a clinical form and a familial form. The clinical form is dominated by positive schizotypal symptoms while the familial form is dominated by negative schizotypal symptoms (Faraone et al., 2001). Tsuang and colleagues view schizotaxia_{Tsuang} and negative schizotypal personality disorder (or familial schizotypy) as very similar (Tsuang et al., 2002b).

**Relationship between schizotaxia_{Tsuang} and schizotypal personality disorder.** The relationship between schizotaxia_{Tsuang} and positive and negative schizotypal personality
disorder as conceptualised by Tsuang and colleagues can be seen in Figure 4.2 below. The figure shows the two constructs as relatively separate entities with some overlap. In addition, the figure suggests that all those with familial schizotypal personality disorder (dominated by negative symptoms) have schizotaxia\textsubscript{Tsuang}. Tsuang and colleagues reported that they view schizotaxia\textsubscript{Tsuang} as a form of schizotypal personality disorder; namely negative schizotypal personality disorder characterised by negative schizotypal symptoms, plus neuropsychological impairments (Tsuang et al., 2002a). They believe that a "significant portion of these negative schizotypal individuals show neuropsychological deficits" but the exact percentage is unknown (M. T. Tsuang, personal communication, August 20, 2003).

\textbf{Figure 4.2.} Tsuang and colleagues' conceptualisation of the relationship between schizotaxia and schizotypy.
schizotaxia\textsubscript{Tsuang} and schizotypal personality disorder (adapted from Faraone et al., 2001, p. 6).

Tsuang and colleagues have not stated the degree of overlap or similarity between schizotaxia\textsubscript{Tsuang} and negative schizotypal personality disorder however, they acknowledge that research needs to determine this (Tsuang et al., 2002a). Faraone et al. (2001) propose that the circles do not overlap completely because the schizotaxia\textsubscript{Tsuang} construct is broader than the negative schizotypal symptom subset. Consistent with this notion, Tsuang has stated that individuals with schizotypal personality disorder who are first-degree relatives of people with schizophrenia make up a small subset of individuals with schizotaxia who are also relatives (M. T. Tsuang, personal communication, August 20, 2003). In addition, M. T. Tsuang has proposed that if an individual with negative schizotypal personality disorder also has a neuropsychological impairment and is a first-degree relative of an individual with schizophrenia then they may meet criteria for schizotaxia (personal communication, August 20, 2003).

Diagnostic implications. As a result of these notions, Tsuang and colleagues propose that familial or negative symptom schizotypal personality disorder should be joined with schizotaxia\textsubscript{Tsuang} and be considered diagnostically as one group. To support this proposal, they have cited research showing that individuals with negative schizotypy often have neuropsychological impairments that are similar to the impairments of schizotaxia\textsubscript{Tsuang} while individuals with positive schizotypy do not. Tsuang et al. (2002b) propose that research such as this suggests that negative schizotypy reflects schizotaxia\textsubscript{Tsuang} while positive schizotypy does not; and, therefore, most individuals with clinical schizotypal personality disorder do not have the genetic liability for schizophrenia (schizotaxia\textsubscript{Tsuang}). The main problem with using evidence such as this is that some
studies classify individuals as having schizotypy based on the criteria for schizotypal personality disorder while others use the much broader construct of schizotypy.

To summarise, in the development of their construct of schizotaxia\textsubscript{Tsuang}, Tsuang and colleagues have proposed that schizotaxia\textsubscript{Tsuang} is different from schizotypal personality disorder with some modifications to how schizotypal personality disorder is conceptualised. They suggest that negative schizotypal personality disorder is part of schizotaxia\textsubscript{Tsuang} and positive schizotypal personality disorder is a separate construct. The next step in developing the construct of schizotaxia\textsubscript{Tsuang} has involved considering the role of target features.

**Target Features**

Target features have an essential role in Tsuang and colleagues’ theory of risk for schizophrenia. Target features are the “clinical or neurobiological characteristics that are expressions of the underlying predisposition to an illness” (Tsuang & Faraone, 1999, p. 2). Tsuang and colleagues propose that they result from the interaction of genetic and environmental factors. However, it does not seem possible to separate out aspects of target features related to the genetic predisposition from aspects of target features related to the impact of environmental factors (Tsuang & Faraone, 1999).

**Role of target features.** Tsuang and colleagues believe that research on target features in various groups provides information and support for their theory. Studies with children of individuals with schizophrenia have provided information about the relationship between target features and the development of schizophrenia (Tsuang & Faraone, 1999). For example, research has demonstrated that children of individuals with schizophrenia have impaired attention, motor skills, and social functioning; and that impairment in these areas may be predictive of later psychosis. Tsuang and colleagues
state that research with adult first-degree relatives of individuals with schizophrenia has a different role because the adults may be partway through or no longer in the age band of risk. Instead, they view the study of target features in adult first-degree relatives as providing information about the pathophysiology of risk for schizophrenia. Schizotypal personality disorder and various neurobiological impairments are considered as target features in adult first-degree relatives (Tsuang & Faraone, 1999).

The assessment of target features is determined by their nature (Tsuang & Faraone, 1999). Tsuang and colleagues have highlighted the heterogeneity of target features considered to be present in individuals at risk of schizophrenia. They point out that it is uncertain exactly what and how many factors are required to meet a threshold level for developing a vulnerability to schizophrenia; therefore, the features are likely to be heterogeneous (Tsuang et al., 1999b). As a result, Tsuang and colleagues emphasise the use of a wide range of measures that assess a number of features.

Participants used in research of target features. Tsuang and colleagues have supported the use of non-psychotic members of genetic high-risk groups as participants in studies of target features. These groups involve children at risk of developing schizophrenia and first-degree relatives of individuals with schizophrenia. Tsuang and colleagues state, however, that target features should not be investigated in individuals with schizophrenia. They suggest that when psychosis is manifested, the target features may be confounded by neurodegeneration, the effects of pharmacotherapy, and the chronic nature of schizophrenia (Tsuang & Faraone, 1999).

Benefits of target features. There may be a number of benefits and advantages to the study of target features. Tsuang and Faraone (1999) state that target features may be advantageous in understanding schizophrenia because they avoid confounds associated with the disease itself. However, this is only the case if the research is conducted with
individuals without psychosis. Target features may also be used in the future to identify children at high risk of schizophrenia and may contribute to the development of pharmacotherapy for the prevention of schizophrenia (Tsuang & Faraone, 1999). Lastly, Tsuang and colleagues anticipate that the study of target features will contribute to what is known about the genetics of schizophrenia through the process of genetic linkage analysis (Tsuang & Faraone, 1999). Genetic linkage analysis is a process that “finds mutant DNA by analysing the coinheritance of marker DNA segments and a disorder in families” (Faraone et al., 1995b, p. 286).

Before the potential benefits and advantages of target features can be realised, the features need to be rigorously investigated to determine that they are in fact indicators of risk for schizophrenia. Tsuang and colleagues have started this. In selecting target features for the study of risk for schizophrenia, they have considered a number of factors. Most importantly, features have been selected on the basis of being consistently present and stable in individuals with schizophrenia, occurring with less frequency in other psychiatric illnesses, and observed in milder forms in individuals considered to be at risk of developing schizophrenia (Kremen et al., 1994). These factors have all been considered in selecting criteria for schizotaxia_Tsuang.

Research Criteria for Schizotaxia_Tsuang

Tsuang et al. (2000a) have stated that it is too early to develop clinical criteria for schizotaxia_Tsuang and instead Tsuang and colleagues have recently developed research criteria. Tsuang and colleagues have used these criteria in studies to validate their conceptualisation of schizotaxia_Tsuang. The first step in the development of research criteria for schizotaxia_Tsuang has involved an evaluation of which target features have the necessary reliability, sensitivity, and specificity required to be incorporated into the
syndrome to ensure that it is valid and can be used as the target for interventions in the future (Stone et al., 2001). It is predicted that the target features, which consistently meet these standards for schizotaxia_Tsuang, will be included in diagnostic systems in the future (Tsuang & Faraone, 1999).

**Target features for schizotaxia_Tsuang.** According to Tsuang and colleagues, there are a wide range of target features for schizotaxia_Tsuang that cover diverse areas of functioning. The clinical expressions of schizotaxia_Tsuang include psychiatric symptoms such as schizotypal personality disorder; psychophysiological abnormalities such as smooth-pursuit eye tracking and suppression of P50; brain abnormalities such as enlarged ventricles and reduced amygdale and hippocampal volume; neuropsychological deficits such as impaired attention, memory, and executive functioning; and psychosocial functioning deficits (Faraone et al., 2001; Tsuang et al., 2002a). Tsuang and colleagues selected neuropsychological impairments and negative symptoms as preliminary research criteria that they thought warranted further investigation.

**Neuropsychological impairments.** Research has demonstrated that individuals with schizophrenia and their relatives experience deficits of varying severity in numerous domains of neuropsychological functioning. Tsuang and colleagues have conducted a number of studies investigating the nature of these impairments. For example, Faraone et al. (1995b) conducted a study with relatives of individuals with schizophrenia and controls in an attempt to clarify inconsistent findings of previous studies. They aimed to determine which neuropsychological domains might be consistent and reliable indicators of the genetic predisposition to schizophrenia, schizotaxia_Tsuang. Faraone et al. (1995b) recruited 35 non-psychotic first-degree relatives of 25 individuals with chronic schizophrenia and 72 controls to take part in the study. Inclusion criteria consisted of age 18 to 59 years, English as a first language, and an eighth-grade education. Exclusion criteria included a
Schizotaxia and Schizotypy 92

history of psychosis, substance abuse in the past 6 months, history of head injury or neurological problems, intellectual disability, brain surgery, or a medical illness that may affect neuropsychological functioning. The relatives and controls were administered 16 neuropsychological tests which evaluated 10 neuropsychological domains including executive functioning, verbal ability, visual-spatial ability, verbal memory, visual memory, learning, perceptual-motor speed, mental control-encoding, auditory attention, and motor ability.

When the results were analysed, Faraone et al. (1995b) found that compared to controls, the relatives had poorer performance and more variability in the domains of executive functioning, verbal memory, and auditory attention. Faraone et al. (1995b) also assessed the proportion of the relatives that were considered to be impaired for each domain. They defined impairment as a score lower than the 3rd percentile of the control group. Significant differences were found for the verbal memory and auditory attention domains with a higher proportion of relatives (an average of 21.4%) impaired on these two domains. Faraone et al. (1995b) also found that the relatives had poorer performance than the controls in the domains of verbal ability and mental control-encoding but similar variability. These differences were not accounted for by psychopathology among the relatives. Faraone et al. (1995b) concluded that the domains of executive functioning, verbal memory, and auditory attention may act as risk indicators for schizotaxia-Tsuang.

Additional studies have been carried out by Tsuang and colleagues to determine the effect of other factors on the neuropsychological target features. These studies have considered sex differences, age factors, the stability of neuropsychological impairments, and the effects of genetic loading on neuropsychological functioning.

Kremen et al. (1997) evaluated sex differences in the neuropsychological functioning of relatives of individuals with schizophrenia. They hypothesised, based on
past research, that male relatives would have a greater degree of neuropsychological impairment than female relatives would. The participants included the 35 relatives and 72 controls from the study by Faraone et al. (1995b) and an additional 19 relatives recruited after the Faraone et al. (1995b) study. The same inclusion and exclusion criteria were used as in the study by Faraone et al. (1995b). Twelve domains of neuropsychological functioning were assessed.

Kremen et al. (1997) evaluated group × sex interactions. Significant results were observed for verbal memory and motor function and results approached significance for mental control, and auditory attention. Male relatives performed significantly worse on tests of motor function relative to male controls while females relatives were similar to controls (Kremen et al., 1997). Surprisingly, female relatives performed significantly worse on tests of verbal memory, auditory attention, and mental control than female controls while male relatives were similar to controls (Kremen et al., 1997). Kremen et al. (1997) found that these differences were not due to poorer performance in general by the female relatives, psychopathology among the relatives, or ethnicity. They speculated that males who are at risk of developing schizophrenia may be more vulnerable to developing a neuropsychological impairment and consequently more likely to develop psychosis. Kremen et al. (1997) suggested that the development of psychosis would exclude males such as this from their sample. Furthermore, they proposed that females may have a higher threshold than males for developing psychosis and are able to endure greater neuropsychological impairment.

The nature of neuropsychological impairments in different age groups has also been researched by Tsuang and colleagues. The participants in Faraone et al.'s (1995b) study were aged between 18 and 59 years to ensure that any impairment in neuropsychological functioning were not due to the effects of aging. Faraone et al. (1996b) conducted a study
to determine the nature of neuropsychological impairments in elderly relatives. The participants were 22 first-degree relatives aged 60 or older of individuals with schizophrenia and 14 controls also aged 60 or older. Faraone et al. (1996b) administered the 11 neuropsychological tests on which relatives under the age of 60 had performed significantly different to controls.

Faraone et al. (1996b) found that there were no significant differences between the neuropsychological performance of the relatives and the controls. They speculated that their unexpected results may have been due to the small size of the sample, and the predominance of parents in the sample versus siblings and children of individuals with schizophrenia in previous studies. Faraone et al. (1996b) suggested that parents may be less likely to manifest a neuropsychological impairment because they have needed to be competent to have a relationship and reproduce. Furthermore, they proposed that individuals who have a liability for schizophrenia may have an increased mortality therefore relatives with a neuropsychological impairment would not be included in older samples if the impairment increased risk of mortality (Faraone et al., 1996b). Consequently, they concluded that their proposal that impairments in verbal memory, executive functioning, and auditory attention may act as indicators for risk of schizophrenia may be restricted to young samples only.

The stability of neuropsychological impairments has been investigated by Faraone et al. (1999). They conducted a follow-up study of the participants of the studies by Faraone et al. (1995b) and Kremen et al. (1997) 3 to 6 years after they were initially recruited. Of the 54 relatives and 72 controls that took part in the first study, 39 relatives and 45 controls took part in the follow-up. Faraone et al. (1999) readministered those neuropsychological tests on which relatives had performed significantly different to controls as well as a further test of executive functioning. They observed significant
differences between the relatives and controls on verbal memory and attention tasks but not for the Wisconsin Card Sorting Test (WCST; Grant & Berg, 1948; Heaton, Chelune, Talley, Kay & Curtiss, 1993). This is probably because the WCST is not suitable for re-testing if a participant has determined the rule of the test. Faraone et al. (1999) found that the interaction between group and time was not significant suggesting that the differences in neuropsychological test scores between the relatives and controls had remained stable over time. They concluded that this provides further evidences as to the possibility of using these impairments as indicators of schizotypia.

Tsuang and colleagues have also investigated the effect of having a single relative with schizophrenia compared to having two relatives with schizophrenia on an individual's neuropsychological functioning (Faraone et al., 2000). They hypothesised that, based on a multifactorial model of schizophrenia where multiple genes are involved in the transmission of schizophrenia, relatives of individuals with schizophrenia should experience varying degrees of neuropsychological impairment depending on their genetic loading. Faraone et al. (2000) recruited 41 non-psychotic individuals with one relative with schizophrenia and 36 non-psychotic individuals with two relatives with schizophrenia. In addition, 100 controls were included in the study. Participants were administered a neuropsychological battery that assessed executive functioning, verbal memory, visual memory, auditory attention, intelligence and achievement. Faraone et al. (2000) found that on tests of verbal and visual memory, the relatives with two family members with schizophrenia had poorer performance than both control subjects and relatives with one family member with schizophrenia. Relatives with one family member with schizophrenia had poorer performance than control subjects on visual memory only. Faraone et al. (2000) concluded that this provides support for a multifactorial model of
schizophrenia where multiple genes are involved in the transmission of risk for schizophrenia.

Tsuang and colleagues have conducted a series of studies investigating the suitability of neuropsychological impairments as target features for schizotypia\textsubscript{Tsuang}. They have found consistent evidence for impairments in the domains of executive functioning, verbal memory, and attention. In addition, they have observed some sex differences in impairments, the impairments appear to only occur in first-degree relatives under the age of 60, the impairments are likely to be more severe if an individual has more than one relative with schizophrenia and the impairments remain relatively stable. Tsuang and colleagues have also looked at negative symptoms for research criteria for schizotypia\textsubscript{Tsuang}.

**Negative symptoms.** In addition to neuropsychological impairments, Tsuang and colleagues have considered the role of negative symptoms in relatives of individuals with schizophrenia. They have cited evidence showing that relatives of individuals with schizophrenia tend to display negative symptoms of schizotypal personality disorder such as interpersonal dysfunction, flat affect, and physical anhedonia. However, some of the evidence that Tsuang and colleagues cite is based on studies looking at schizotypal personality disorder (e.g., Battaglia & Torgersen, 1996) and other evidence comes from studies that are based on Meehl’s conceptualisation of schizotypy (e.g., Grove et al., 1991). This is a problem for Tsuang and colleagues because schizotypal personality disorder and schizotypy are not the same construct. Because Tsuang and colleagues have cited studies that look at both schizotypal personality disorder and schizotypy, the conclusion can be made that relatives of individuals with schizophrenia who are thought to have schizotypia\textsubscript{Tsuang} may also be schizotypal as based on Meehl’s (1962, 1990b) definition. However, this was probably not Tsuang and colleagues' intention.
Positive symptoms do not appear to be as evident in relatives of individuals with schizophrenia as negative symptoms and neuropsychological impairments are. However, Faraone et al. (2001) suggest that future research needs to consider whether people with schizotaxia_Tsuang demonstrate mild forms of positive symptoms as well. It would be necessary to determine this before diagnostic criteria for schizotaxia_Tsuang are established.

Research criteria for schizotaxia_Tsuang. As a result of the studies described above, Tsuang and colleagues have proposed research criteria for schizotaxia_Tsuang (Table 4.1).

<table>
<thead>
<tr>
<th>Type of Impairment/Criterion</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Neuropsychological Impairment</td>
<td>Impairment in 1 of 3 domains $\geq 2$ standard deviations below normative means.</td>
</tr>
<tr>
<td></td>
<td>Impairment in a second domain $\geq 1$ standard deviation below normative means.</td>
</tr>
<tr>
<td></td>
<td>Neuropsychological domains include executive functioning, verbal memory, and attention.</td>
</tr>
<tr>
<td>Presence of Negative Symptoms</td>
<td>Six or more items rated 3 or higher on the Scale for the Assessment of Negative Symptoms (Andreasen, 1984).</td>
</tr>
<tr>
<td>Inclusion Criteria</td>
<td>Aged 19-50 years</td>
</tr>
<tr>
<td></td>
<td>English as first language</td>
</tr>
<tr>
<td></td>
<td>Estimated IQ $\geq 70$</td>
</tr>
<tr>
<td></td>
<td>First-degree relative of individual with schizophrenia</td>
</tr>
<tr>
<td>Exclusion Criteria</td>
<td>History of psychosis</td>
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<tr>
<td></td>
<td>Substance abuse in past 6 months</td>
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<tr>
<td></td>
<td>History of head injury or neurological problems</td>
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<td></td>
<td>Medical illness with neurological effects</td>
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<td></td>
<td>History of electroconvulsive treatment</td>
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</tbody>
</table>
Tsuang and colleagues' basis for the selection of these target features for schizotaxia_{Tsuang} was the presence of consistent research evidence for these deficits in non-psychotic relatives of individuals with schizophrenia. In addition, the two types of criteria are distinct from one another (Tsuang et al., 2002a). The main criteria are negative symptoms and a neuropsychological impairment; however, variations of these criteria have been put forward by Faraone et al. (2001) who define schizotaxia_{Tsuang} as being characterised by psychiatric signs and symptoms, neuropsychological impairment, and a social dysfunction. This is because Tsuang and colleagues predict that both the neuropsychological impairments and the psychiatric symptoms such as negative symptoms have an impact on the social functioning of individuals with schizotaxia_{Tsuang}. They view the negative symptoms and neuropsychological impairment of schizotaxia_{Tsuang} as similar but less severe than the type of difficulties that individuals with schizophrenia experience. However, Tsuang et al. (2000b) have emphasised that the features of schizotaxia_{Tsuang} are separate from the prodromal symptoms of schizophrenia. In addition, they claim that the symptoms of schizotaxia_{Tsuang} appear in individuals long before prodromal symptoms.

**Inclusion and exclusion criteria.** Participants who have taken part in research by Tsuang and colleagues into the construct of schizotaxia_{Tsuang} have been required to meet a number of inclusion and exclusion criteria (see Table 4.1). The inclusion criteria of Tsuang and colleagues have raised an important question. They have stated that an individual needs to have a first-degree relative with schizophrenia in order to meet the criteria for schizotaxia_{Tsuang}. Does this mean that if an individual has a neuropsychological impairment and moderate negative symptoms but does not have a first-degree relative with schizophrenia that they do not meet criteria for schizotaxia_{Tsuang}? Faraone et al. (2001) have acknowledged that theoretically there may be individuals with
schizotaxia\textsuperscript{Tsuang} who do not have a first-degree relative. Research indicates that a large proportion of individuals at risk for schizophrenia would meet this criterion. As discussed in Chapter 3, 63% of individuals with schizophrenia do not have a first- or second-degree relative with schizophrenia (Gottesman & Erlenmeyer-Kimling, 2001). Consequently, a majority of people who are at risk of developing schizophrenia would be excluded from research into schizotaxia\textsuperscript{Tsuang} that utilises the current criteria. Despite this, Tsuang and colleagues have only included first-degree relatives of individuals with schizophrenia in their research into schizotaxia\textsuperscript{Tsuang}.

Benefits of schizotaxia\textsuperscript{Tsuang} criteria. Tsuang et al.'s (2000b) conceptualisation of schizotaxia\textsuperscript{Tsuang} has many benefits. There are benefits from utilising symptoms that are closer to the underlying aetiology than the end-state symptoms of psychosis in schizophrenia. Symptoms that are closer to the aetiology of a disorder are thought to be more specific and sensitive (Tsuang et al., 2000b). The symptoms of schizotaxia\textsuperscript{Tsuang} may also be used to identify those at risk or preschizophrenic and to develop treatment strategies. Tsuang and colleagues also believe that schizotaxia\textsuperscript{Tsuang} will contribute to genetic studies of schizophrenia. If their estimates of the frequency of schizotaxia\textsuperscript{Tsuang} are correct, 20% to 50% of relatives of individuals with schizophrenia may have schizotaxia\textsuperscript{Tsuang}. This may contribute to distinguishing individuals who have a genetic predisposition from individuals who do not (Tsuang et al., 2000b). Ongoing research into the genetic aetiology is being carried out in an attempt to elucidate the exact genes that are involved in the transmission of schizophrenia (e.g., Takahashi, Faraone, Lasky-Su, & Tsuang, 2005). It is thought that Tsuang and colleagues theory of risk for schizophrenia may complement these efforts.
Following the development of their theory of risk and research criteria for schizotaxia\textsuperscript{Tsuang}, Tsuang and colleagues have conducted a number of studies investigating the validity of the construct as well as the effectiveness of treatment for schizotaxia\textsuperscript{Tsuang} (e.g., Stone et al., 2001; Tsuang et al., 1999b).

\textit{Concurrent validity of schizotaxia\textsuperscript{Tsuang}.} The concurrent validity of schizotaxia\textsuperscript{Tsuang} has been evaluated by Stone et al. (2001). They recruited 27 first-degree relatives of individuals with schizophrenia to take part in a study where they compared participants who did and did not meet criteria for schizotaxia\textsuperscript{Tsuang} on a number of self-rated and clinician-rated measures of clinical functioning. Inclusion and exclusion criteria for the study consisted of the criteria described in Table 4.1. Negative symptoms were assessed using the Scale for the Assessment of Negative Symptoms (Andreasen, 1984) and a moderate impairment was defined as having 6 or more items with a rating of 3 or higher. Three domains of neuropsychological functioning were assessed: attention, verbal memory, and executive functioning. Attention was assessed using the Auditory Continuous Performance Test, with Interference (Seidman et al., 1998) and the Visual Continuous Performance Test, Identical Pairs version (Comblatt et al., 1988). Verbal memory was assessed with the Logical Memory subtest from the Wechsler Memory Scale-Revised (Wechsler, 1987), and the Selective Reminding Test (Buschke, 1973). Executive functioning was assessed using the Delayed Alternation Test and the Object Alternation Test (Seidman et al., 1995). To meet criteria for a neuropsychological impairment, participants were required to have a deficit in any one criterion measure of one neuropsychological domain equal to two or more standard deviations below norms and a deficit in any one criterion measure of a second neuropsychological domain equal to one or more standard deviations below norms.
In addition, Stone et al. (2001) evaluated psychopathology using components of the Diagnostic Interview for Genetic Studies (DIGS; Faraone et al., 1996a; Nurnberger et al., 1994), schizotypal personality disorder with the Structured Interview for Schizotypy (SIS; Kendler et al., 1989), family history with the Family Interview for Genetic Studies (Maxwell, 1982), global functioning with the DSM-IV Global Assessment of Functioning Scale (GAF; APA, 1994), physical anhedonia with the PhA (Chapman et al., 1976), further psychological symptoms with the Symptom Checklist-90-R (SCL-90-R; Derogatis, 1993), and social functioning with the Social Adjustment Scale Self-Report (SAS, Weissman & Bothwell, 1976).

Eight of the participants met criteria for schizotaxia_Tsuang while 19 did not (Stone et al., 2001). Of the 19 relatives who did not meet criteria for schizotaxia_Tsuang, 5 met criteria for the presence of negative symptoms and 6 met criteria for a neuropsychological impairment. Data were incomplete for 2 of the participants who did not meet criteria for schizotaxia_Tsuang and therefore some analyses were performed with only 17 or 18 participants in the nonschizotaxia_Tsuang group (Stone et al., 2001).

Participants in the schizotaxia_Tsuang group had significantly poorer functioning than the nonschizotaxia_Tsuang group as measured by one comparison from each of the GAF, PhA, SCL-90-R, and SAS measures of clinical functioning (Stone et al., 2001). In addition, many participants in both groups met criteria for a variety of past or present psychopathology; however, there were no significant differences between the two groups. None of the relatives met criteria for schizotypal personality disorder or any other schizophrenia-spectrum disorder. In the schizotaxia_Tsuang group, 4 (50%) met criteria for a past substance abuse diagnosis. However when these were compared to individuals in the schizotaxia_Tsuang group who did not have a past substance abuse diagnosis, there were no significant differences in neuropsychological functioning or clinical functioning.
on their previous findings about genetic loading, Stone et al. (2001) expected that the schizotaxia_{Tsuang} group would contain more relatives with more than one relative with schizophrenia than the nonschizotaxia_{Tsuang} group; however, both groups had approximately the same proportion of individuals with one relative with schizophrenia and individuals with more than one relative with schizophrenia.

Stone et al. (2001) surmised that the schizotaxia_{Tsuang} relatives were different from the nonschizotaxia_{Tsuang} relatives in a number of areas of clinical functioning and this was independent of age, education, paternal education, IQ, gender, genetic loading or other psychopathology. They concluded that this overall finding offers evidence for the construct validity of their conceptualisation of schizotaxia_{Tsuang} and helps to provide criteria for distinguishing between relatives who do and do not have schizotaxia_{Tsuang}.

There are a number of limitations of this study. Stone et al. (2001) have emphasised the preliminary nature of their findings. They have also recognised that the small sample size was a limitation and that the results would have been strengthened with a control group.

**Predictive validity of schizotaxia_{Tsuang}**. In addition to investigating the concurrent validity of schizotaxia_{Tsuang}, Tsuang and colleagues have considered the predictive validity of the construct. They have done this by investigating treatment options for schizotaxia_{Tsuang} using the antipsychotic medication risperidone. Tsuang et al. (1999b) have based their use of risperidone with non-psychotic individuals on evidence that the features of schizotaxia_{Tsuang} are milder forms of the symptoms of schizophrenia. Furthermore, Tsuang et al. (1999b) state that risperidone has been shown to reduce positive symptoms and some negative symptoms of schizophrenia, has fewer side effects than other antipsychotics, and may improve some aspects of neuropsychological functioning. This has provided them with the impetus to conduct a pilot study that has assessed the ability of risperidone to treat and reduce the features of schizotaxia_{Tsuang}. 
Tsuang and colleagues consider this the first trial in an evaluation of the predictive validity of schizotypy.

In their pilot study, Tsuang et al. (1999b) recruited participants from an ongoing family study of schizophrenia. They approached 36 people and 12 of these agreed to be evaluated for eligibility for the study. This also included participants from the study by Stone et al. (2001). The measures, inclusion and exclusion criteria were the same as that used by Stone et al. (2001) except Tsuang et al. (1999b) did not administer the additional clinical measures. Participants were considered eligible for the study if they experienced negative symptoms to a moderate level and moderate neuropsychological impairments (Tsuang et al., 1999b). Seven participants met criteria for a negative symptom impairment while 4 of the participants met criteria for both negative symptoms and a neuropsychological impairment.

Following recruitment, Tsuang et al. (1999b) began a 6 week trial of risperidone with the 4 participants. The initial dose was .25 mg per day, which was increased to a maximum of 2.0 mg per day over the first two weeks. Participants’ negative symptoms were assessed at baseline, and at weeks 2, 4, and 6. Neuropsychological functioning was assessed at baseline, and after week 6. Physical health and side effects of the medication were also monitored during this time (Tsuang et al., 1999b).

Tsuang et al. (1999b) reported individual results for each of the 4 participants. They found that 3 of the 4 participants made subjective reports of experiencing improvement in their functioning. Tsuang et al. (1999b) reported that 3 of the 4 participants demonstrated a reduction in their negative symptoms with a decrease in the number of items rated as moderate or higher. Furthermore, Tsuang et al. (1999b) stated that all 4 participants experienced an improvement in their attentional skills, namely, an improvement in hit rate.
of between 20% and 40% on the auditory task. Improvements in memory and executive functioning were either very mild or temporary (Tsuang et al., 1999b).

Subsequent articles have surmised the results of the group of 4 and an additional 2 participants as a whole group (e.g., Tsuang, Stone, Tarbox, & Faraone, 2002c). One of the 6 participants did not experience any improvements. Five of the 6 participants experienced a reduction in negative symptoms ranging from 25% to 50% and an improvement in hit rate on the auditory attention task. There were selective improvements in the selective reminding test total recall score and total errors for the objective alternation task (Tsuang et al., 2002c). Tsuang et al. (1999b) concluded that their study demonstrated the positive effects of risperidone over a 6 week trial for relatives of individuals with schizophrenia who may have a liability for developing schizophrenia in the form of schizotypy.

There are a number of limitations associated with this study involving practice effects, the results, the age of the participants, inconsistencies in the reports of side-effects, and whether the people conducting the assessments were blind to the purpose of the study. The main improvement for neuropsychological functioning in the participants related to attention. This may not be due solely to the effects of the medication as Tsuang et al. (1999b) acknowledged that they had to consider the possibility of practice effects on participants' performance on this task. In addition, the age of 4 of the participants in the study ranged from 33 to 43 years (the ages of the additional 2 participants were not reported) which has an impact on the ability to generalise the results to prevention of schizophrenia. It has been suggested that the 4 participants may have been beyond the at-risk age-band of developing schizophrenia and therefore not representative of relatives at risk of developing schizophrenia (Remington & Shammi, 2004). This means that Tsuang and colleagues can only make inferences about the ability of risperidone to alleviate some
symptoms in individuals who may be at risk of developing schizophrenia; they cannot claim that risperidone may prevent schizophrenia.

Furthermore, it is not clear whether the researchers completing the negative symptom ratings were blind to the purpose of the study or whether they knew that the participants were being assessed to evaluate the effects of risperidone. Rigorous testing that involves more participants who have been randomly selected is required before Tsuang et al. (1999b) can proceed with their proposal that risperidone may reverse, to some degree, the neuropsychological impairments and negative symptoms that relatives of individuals with schizophrenia may experience.

Research by other groups of authors has occurred following Tsuang and colleagues' treatment trials. Rybakowski, Dróżdż, and Borkowska (2003) have described research they conducted using risperidone. They recruited 8 participants who had impairments in their smooth pursuit eye-tracking, neuropsychological performance, and subjective reports of social functioning; and a first- or second-degree relative with schizophrenia. Rybakowski et al. (2003) reported that following 12 months of treatment with 1 to 2 mg per day of risperidone, 7 of the 8 participants demonstrated significant improvements in neuropsychological functioning and improvements in occupational and social functioning. Three of the participants reportedly met criteria for schizotypal personality disorder. One participant had ceased to take the medication after 8 months and 2 months later had a first psychotic episode (Rybakowski et al., 2003). The results of this study need to be interpreted with caution as they are presented in the form of a letter to the editor of a journal and all of the participants were relatively high functioning, and included a pharmacist, a farmer, an economist, a priest, and 4 students.

Based on the preliminary findings of investigations into the construct of schizotaxia, Tsuang and colleagues have concluded that there is evidence for the
validation of schizotaxia\textsubscript{Tsuang}. Tsuang et al. (2002a) state that there are two lines of supporting evidence. The first of these is from the study by Stone et al. (2001) which evaluated the concurrent validity of schizotaxia\textsubscript{Tsuang}, and the second concerns the predictive validity examined in the studies by Tsuang et al. (1999b, 2002c). Tsuang et al. (2002a) state there is a third line of evidence from unpublished data from a large multisite study of the genetics of schizophrenia. In the unpublished study, all participants with schizophrenia, schizophrenia-related disorders, and other psychoses were excluded and one subgroup remained. The subgroup was characterised by negative symptoms, part of the criteria for schizotaxia\textsubscript{Tsuang} (Tsuang et al., 2002a).

It is important to emphasise the preliminary nature of these findings and many limitations of the studies. Much more research is needed before schizotaxia\textsubscript{Tsuang} can be considered as a valid diagnostic entry in classification systems and before further treatment studies take place.

\textit{Future Directions for Schizotaxia\textsubscript{Tsuang}}

As research continues into the construct of schizotaxia\textsubscript{Tsuang}, there are a number of potential implications for the diagnosis of schizotaxia\textsubscript{Tsuang} and other disorders; and issues to consider for the treatment of alleviating the symptoms of schizotaxia\textsubscript{Tsuang}, preventing the development of schizophrenia, and future treatment protocols. In addition, research into this construct raises a number of questions that need to be resolved.

\textit{Diagnostic implications}. Tsuang and colleagues have proposed that their reformulation of schizophrenia has two major implications for current diagnostic systems. Firstly, the traditional emphasis on psychosis would have to change; and secondly, the tendency to use signs and symptoms that are considered representative of end-state problems rather than signs and symptoms that are closer to the aetiology of schizophrenia
would need to change (Tsuang et al., 2000a). There would also be implications for how schizotaxiaTsuang and schizotypal personality disorder are conceptualised in diagnostic systems.

If schizotaxiaTsuang were to be included in diagnostic systems, then the dimensional or categorical nature of the construct would need to be determined. Tsuang and colleagues appear to have a categorical perspective of the relationship between schizotaxiaTsuang and other schizophrenia-spectrum disorders. However, Faraone et al. (2001) have acknowledged that there is uncertainty as to whether schizotaxiaTsuang is a discrete entity or a quantitative trait with variations in degree of severity. Tsuang and colleagues predict that if future research evaluates the nature of the criteria for schizotaxiaTsuang, then schizophrenia may be reconceptualised as two categories: schizotaxiaTsuang and schizotaxiaTsuang with psychosis (Tsuang & Faraone, 2002). SchizotaxiaTsuang with psychosis would be equivalent to schizophrenia (Tsuang et al., 2000a). The establishment of another diagnostic category, however, would increase the number of individuals who are labeled as having a psychiatric disorder. This has both positive and negative effects. Implications include how others will react to an individual, how the individual feels about themselves, accessibility to health insurance, and the need for counseling to cope with the information (Tsuang et al., 2000a).

Research into schizotaxiaTsuang may also affect how schizotypal personality disorder is conceptualised in diagnosis systems. If research were successful in establishing diagnostic criteria for schizotaxiaTsuang as well as determining the degree of comorbidity between schizotaxiaTsuang and schizotypal personality disorder, then changes would be necessary for the formulation of schizotypal personality disorder in the DSM (Faraone et al., 2001; Tsuang et al., 2002b). Tsuang and colleagues propose that schizotaxiaTsuang would be defined as “the syndrome of negative symptoms and neuropsychological
dysfunction observed among relatives of schizophrenia patients” (Faraone et al., 2001, p. 7) and schizotypal personality disorder would be defined by the presence of mild positive schizophrenia symptoms only. This would have a potentially large impact on diagnostic systems as well as theories as to how these constructs relate to schizophrenia. Further research is needed before these changes should be achieved.

*Treatment for schizophrenia* Tsuang and colleagues have not been hesitant in their endeavours to develop a treatment protocol for schizophrenia. They have speculated about treatment for schizophrenia in the form of both pharmacotherapy and psychological treatments. They suggest that pharmacotherapy for adults who are older than the at-risk age-band should focus on alleviating the symptoms of schizophrenia, namely neuropsychological impairment, negative symptoms, and problems with social functioning (Faraone et al., 2001). In regards to psychological intervention, Faraone et al. (2001) propose that treatment would involve firstly, clinicians carrying out an assessment to have a thorough understanding of the individual’s neuropsychological strengths and weaknesses; and secondly, clinicians helping individuals with schizophrenia to learn cognitive behavioural techniques to manage their difficulties.

In addition to alleviating the symptoms of schizophrenia, Tsuang and colleagues have speculated about the use of treatment to prevent the development of schizophrenia. They suggest that if an antipsychotic has a positive effect on the symptoms of schizophrenia, then it can probably be used in the future for the prevention of schizophrenia. They have based this proposal on the assumption that the features of schizophrenia share the same aetiology as preschizophrenic individuals (Tsuang et al., 2000b). Tsuang and colleagues predict that in the future, when technology has advanced further, ethical and effective intervention for schizophrenia will be achieved. The current treatment research focus with schizophrenia has been on adults. Tsuang and
colleagues expect that with time and after the validity of the construct and benefits of treatment have been clearly established then research with adolescents may be able to take place, followed by research with children thought to be at risk of developing schizophrenia.

Treatment for schizotaxia Tswana raises a number of ethical issues. Any treatment would need to consider whether the person’s symptoms were of a severity such that they warranted medication, as well as the potential side-effects of any medication versus the benefits (if any) in reducing symptoms of schizotaxia Tswana. In most cases, schizotaxia Tswana will not develop into schizophrenia; therefore treatment is not warranted (Tsuang et al., 2000a). This is especially pertinent when the impairments of schizotaxia Tswana are not clinically significant or causing an individual any distress. In addition, the risks of carrying out drug trials with children and the implications of labeling children with a psychiatric disorder may have more detrimental effects than research carried out with adults (Tsuang et al., 2000b). The advantages and disadvantages of this would need to be carefully considered.

Further research and questions. Before treatment strategies with adolescents and children can be developed, more research with adults is required, particularly studies with large samples that involve double-blind methodologies and investigate other types of treatments. Furthermore, it would need to be established that schizotaxia Tswana can be used to identify children at high risk of developing schizophrenia. It may be that other impairments and abnormalities in domains of functioning need to be considered for the conceptualisation of the syndrome of schizotaxia Tswana. Stone et al. (2001) draw on evidence that has shown relatives to have structural and functional differences in their brain compared to controls as well as other features such as smooth-pursuit eye tracking and biochemical abnormalities. They propose that these factors may need to be
incorporated into the category of schizotaxia\textsubscript{Tsuang} if it is shown that there are differences in these factors between relatives who do and do not meet criteria for schizotaxia\textsubscript{Tsuang} as it stands now. In addition, treatment programs need to ensure that the factors that are used to identify people at risk are in fact useful and effective in identifying those at risk (Faraone, Brown, Glatt, & Tsuang, 2002).

Research is also needed that establishes whether the construct of schizotaxia\textsubscript{Tsuang} can be used to predict schizophrenia and other schizophrenia-spectrum disorders as well as the effect of the schizotaxia\textsubscript{Tsuang} treatment protocol on schizophrenia-spectrum disorders (Faraone et al., 2001). There have been very few treatment studies for schizotypal personality disorder that involve the atypical antipsychotic drugs. Recent research has suggested that these medications may be effective in alleviating some of the symptoms of schizotypal personality disorder. Keshavan, Shad, Soloff, and Schooler (2004) administered a 26 week course of olanzapine to a group of 11 participants with schizotypal personality disorder. They found that there were significant reductions in the participants’ positive and negative symptoms and improvements in overall functioning. The most common side effect was weight gain. If schizotypal personality disorder is part of the schizophrenia-spectrum, then this raises the question as to whether treatment of schizotypal personality disorder reduces the risk of subsequently developing schizophrenia.

It is apparent that the early stages of research into the construct of schizotaxia\textsubscript{Tsuang} have created numerous other research questions. The construct of schizotaxia\textsubscript{Tsuang} needs to be clearly established, defined, and validated before further treatment research can take place. One of the long-term goals of Tsuang and colleagues is to eventually develop a treatment protocol that can be used with children at risk of developing schizophrenia. Before this can happen it needs to be determined if schizotaxia\textsubscript{Tsuang} criteria can be applied
to children. This would need to consider if the same measures that are used with adults would be used with children. In addition, it would need to be established that research can accurately determine which children do and which do not develop schizophrenia.

Other questions surrounding the nature of people who meet criteria for schizotaxia\textsubscript{Tsuang} would also need to be resolved. For example, what types of psychopathology do people with schizotaxia\textsubscript{Tsuang} develop? Are there people with schizotaxia\textsubscript{Tsuang} in the psychiatric outpatient and inpatient population? Is there a higher proportion than normal of people with schizotaxia\textsubscript{Tsuang} also diagnosed with particular psychiatric disorders? What proportion is diagnosed with schizotypal personality disorder? Stone et al. (2001) suggested that future research needs to include larger samples that can consider the impact of any potential confounds such as substance abuse more effectively or how individuals who do and do not meet criteria for schizotaxia\textsubscript{Tsuang} differ in regards to psychiatric disorders. Most importantly, all of the above past research and future research directions have stemmed from Tsuang et al.'s theory of risk for schizophrenia which was developed on the basis of Meehl’s (1962, 1989, 1990b) theory. There are many similarities between schizotaxia\textsubscript{Tsuang} and schizotypy and it is possible that the two constructs are at the same level of analysis. Before further research into the construct of schizotaxia\textsubscript{Tsuang} can take place, it needs to be established whether schizotaxia\textsubscript{Tsuang} and schizotypy are sufficiently different from each other. Consequently, the relationship between the constructs as conceptualised by Meehl and Tsuang et al. needs to be determined, namely the degree of similarity or overlap between schizotaxia\textsubscript{Tsuang} and Meehl’s schizotypy.

Summary
Tsuang et al. have used Meehl’s (1962, 1989, 1990b) theory of risk for schizophrenia as the starting point for their own theory of risk for schizophrenia. There are some similarities between the theories but also many differences. One of the key distinctions concerns differences between schizotaxia_{Meehl} and schizotaxia_{Tsuang}. These differences relate to the aetiology of schizotaxia, how the construct is observed, the outcome of schizotaxia, and the relationship between schizotaxia and other schizophrenia-spectrum disorders. In addition, Tsuang and colleagues use the terms schizotypy and schizotypal personality disorder interchangeably in their theory and research yet there are conceptual differences between the two constructs. Schizotaxia_{Tsuang} has many similarities to Meehl’s schizotypy and therefore may be related yet Tsuang et al. maintain that they are conceptually different. Tsuang and colleagues have conducted a limited number of studies that have started to investigate the validity of the construct of schizotaxia_{Tsuang}, as well as treatment protocols to alleviate the symptoms of schizotaxia_{Tsuang} in relatives of individuals with schizophrenia. Their goals are for schizotaxia_{Tsuang} to enter the diagnostic nomenclature and for treatments to be available to high-risk groups including first-degree relatives and children thought to be at risk of developing schizophrenia with the aim of preventing schizophrenia. Before these goals can be achieved, more research into the construct of schizotaxia_{Tsuang} is required, especially further research that investigates the relationship between schizotaxia_{Tsuang} and schizotypy to determine whether they are related. The present study has attempted to do this and will be described in Chapters 7 and 8. Before that, Chapter 5 will introduce and describe a statistical approach, taxometric analysis, which was employed in the current study.
CHAPTER 5

Taxometric Analysis and the Structure of Schizotypy

A taxon is a type, class, or nonarbitrary category (Meehl, 1992). Meehl and his colleagues (e.g., Golden, 1982; Golden & Meehl, 1979; Meehl 1973; Meehl & Yonce, 1994, 1996; Waller & Meehl, 1998) have developed a range of statistical procedures that can be used to assist distinguishing evidence of latent taxa from distributions of continuous data. These statistical methods are known collectively as taxometric or coherent cut kinetic procedures. The results of these procedures are analysed to determine if there is evidence for the underlying structure of a construct (such as schizotypy, psychopathy, or depression) in terms of whether it is taxonic or dimensional. If a taxometric procedure suggests a qualitative boundary within a sample, then the construct under investigation is said to be taxonic. Otherwise the construct under investigation is presumed to be dimensional. Meehl (1992) defines taxometric analysis as a statistical procedure that is used firstly to help provide evidence of the presence of a taxon, and secondly, to classify individuals as members of the taxon or its complement.

Specific taxometric procedures include MAXCOV-HITMAX (Maximum Covariance, Making Hits Maximum; Meehl, 1973), MAMBAC (Mean Above Minus Below A Cut; Meehl & Yonce, 1994), MAXEIG-HITMAX (Maximum Eigenvalue, Making Hits Maximum; Waller & Meehl, 1998), and L-Mode (Latent Mode; Waller & Meehl, 1998). These procedures were initially developed by Meehl to try to further understand genetic risk and predisposition for schizophrenia (1973, 1979, 1995a). Unlike many organic diseases in medicine, such as Huntington’s disease or cancer, many mental illnesses or constructs do not have a specific pathology or pathognomonic signs that can
be used as the gold standard for comparing suspected cases with for the purpose of illness identification (Meehl, 2001a). As a potential solution to this problem, Meehl developed a set of procedures that use imperfect indicators or variables to identify hypothesised taxa. All involve visual analysis of graphical output from the procedures. If the graphical outputs possess characteristics suggesting latent taxonicity, parameter estimates and classifications are determined.

The General Covariance Mixture Theorem

The General Covariance Mixture Theorem is the basis of Meehl’s taxometric procedures. The General Covariance Mixture Theorem was developed by Meehl (1965, 1968, cited in Waller & Meehl, 1998; 1973; Meehl & Golden, 1982) to describe the covariance between two indicators of a latent taxon in data obtained from a sample comprising taxon and nontaxon (or complement) members. The General Covariance Mixture Theorem describes the variance in a total sample. It does this through the use of the taxon and complement variances, the taxon base rate, and the differences between the means of the latent class indicators (Waller & Meehl, 1998). The covariance of two indicators, $x$ and $y$, in a sample, is equal to the product of the covariance of the two indicators within the taxon multiplied by the taxon’s prevalence or base rate, the covariance of the two indicators in the complement group multiplied by the complement base rate, and the differences between the means of the latent class indicators. The theorem in equation form is:

$$cov(xy) = Pcov_t(xy) + Qcov_c(xy) + PQ\left(\bar{x}_t - \bar{x}_c\right)\left(\bar{y}_t - \bar{y}_c\right)$$ (Waller & Meehl, 1998, p.12),

where

$$cov(xy) = \text{the covariance of } x \text{ and } y \text{ in the total sample}$$
$P = \text{the base rate of taxon members in the total sample}$

$Q = 1 - P$ is the base rate of nontaxon members in the total sample

$P_{cov}(xy) = \text{the weighted indicator covariance in the taxon class}$

$Q_{cov}(xy) = \text{the weighted indicator covariance in the complement class}$

$PQ = \text{the weighted cross product of the latent class mean differences (Waller & Meehl, 1998, p.12).}$

**MAXCOV-HITMAX**

Meehl and others have demonstrated that the covariance of two indicators that are sensitive to some construct (e.g., risk for schizophrenia) but uncorrelated within a homogeneous group (e.g., those at risk; those not at risk) is maximised in a mixed group sample when the ratio of the groups approximates 1:1 (or 50%). A maximisation of covariance is detected from a plot of covariances from intervals with different mixture ratios. The point of maximum covariance is influenced by the proportion of taxon members in the overall sample. A plot of all output covariances is used to detect whether the latent structure is taxonic or dimensional. Then, base rate estimates can be determined and individuals in the sample can be classified as either taxon or complement members. This entire process involves the framework of the General Covariance Mixture Theorem.

Usually, the parameters required in the General Covariance Mixture Theorem are not known (Meehl, 1973). In these cases, the MAXCOV-HITMAX (MAXCOV; Meehl, 1973) procedure can be applied to the framework of the theorem and used to determine the values of the parameters. The MAXCOV procedure is applied when the assumption is held that there is no nuisance covariance, or within-group correlation, present. If the nuisance covariance for both the taxon and complement groups is equal to zero, then the
covariance of the total sample is described by the taxon base rate and the means of the indicators (Waller & Meehl, 1998). That is, the equation becomes:

\[ \text{cov}(xy) = P \left( \bar{x} - \bar{x}_c \right) \left( \bar{y} - \bar{y}_c \right). \]

Due to the imperfect nature of indicators, it is practically impossible to remove all nuisance covariance; however, the MAXCOV procedure can still be used in these situations. Studies where the nuisance correlation has been less than .50 have shown that MAXCOV still yields accurate estimates of the point of maximum covariance (Meehl & Golden, 1982).

The point of maximum covariance is called the Hitmax. This is the interval at which the functions of the complement and taxon intersect (Meehl, 1973; Waller & Meehl, 1998). This is also the point at which the maximum number of accurate taxon classifications occurs (Golden & Meehl, 1979). Within this slab or interval, half of the individuals belong to the taxon and half belong to the complement (Meehl & Yonce, 1996).

MAXCOV is an example of bootstrap taxometrics which means that the parameters of the indicators are not known and the indicators used are often fallible. Despite this, the nature of the statistical interactions between the indicators can be evaluated to determine the parameters (Cronbach & Meehl, 1955; Meehl, 1973). Bootstrap taxometrics are appropriate to use when investigating psychopathology constructs because there is no gold standard as such to compare data to (Meehl, 1995a).

Waller and Meehl (1998) have put forward a number of requirements necessary for a MAXCOV analysis. Specifically, there must be at least 3 indicator variables available for analysis, and one of these indicators must be quantified on a continuous scale; there
needs to be minimal correlation between pairs of variables within groups (minimum
nuisance covariance); and the indicators need to have means with a large degree of
separation (indicative of high validity; Waller & Meehl, 1998). Indicators that are shown
to have a high level of nuisance covariance should be removed and not included in the
MAXCOV analysis (Golden & Meehl, 1979). In line with this, Meehl and Yonce (1996)
have suggested that a nuisance correlation of up .30 is acceptable. Meehl (1995b) has
developed an extension of the MAXCOV-HITMAX procedure, called generalized
MAXCOV, for use with samples that have a large amount of nuisance covariance. This
procedure warrants further testing and will not be a focus of the current discussion. A
large degree of indicator separation has been defined by Meehl (1995a) as indicators that
have a mean separation of greater than 1.25 standard deviations.

Meehl (1992, 1995a) also recommended that taxometric analyses not be conducted
with samples smaller than 300. Meehl has stated that he does not approve of taxometric
research employing samples smaller than this. Nonetheless, research has shown that
MAXCOV analyses can be conducted with samples of 200 individuals and still provide
accurate results (Golden & Meehl, 1979). Furthermore, in later research, Meehl and
Yonce (1996) conceded that MAXCOV could be used with samples of 100 individuals if
other conditions were met, including a base rate of approximately .50, a separation of 2
standard deviations on the indicators, and minimal nuisance covariance.

If the above criteria are met, the first step in the MAXCOV procedure involves
designating three of the available variables as input and output indicators, one input and
two outputs (Meehl & Yonce, 1996). Participants are ranked or ordered on the basis of
the input indicator scores (Meehl, 1973; Waller & Meehl, 1998). The input scores are
then separated into sub-samples, called slabs, which do not overlap with each other. The
slabs have equal widths, ranging from \( \frac{1}{4} \) to \( \frac{1}{2} \) of a standard deviation, depending on the
size of the sample (a larger slab width is used for a smaller sample). Alternatively, the slabs could be defined using a fixed number of cases or individuals (Meehl & Yonce, 1996). This is not recommended if the base rate is smaller than .50 (Meehl & Yonce, 1996). The covariance between the output variables is then calculated across individuals in each slab. If the input indicator is sensitive to a latent group structure, then the output covariances will vary according to slab population and the proportion of taxon members in the slab (Waller & Meehl, 1998). This means that if the structure is taxonic, then the output covariances will increase from zero to a maximum value and then decrease to zero. If the indicators measure a dimension, then the output covariances will be similar across the slabs (Waller & Meehl, 1998).

If the output covariances are plotted in terms of sequential slabs, then the pattern for a latent taxon will be distinct from a pattern for a latent dimension (Waller & Meehl, 1998). Specifically, if the data are taxonic, when the output covariances are plotted a peak will be observed among the output covariance. If the data are dimensional, then a relatively flat line should be produced when covariances are plotted.

The taxonic plot can also be used to estimate the Hitmax (Waller & Meehl, 1998). The maximum point of a MAXCOV plot is used for this estimate and is an approximation of the cut-off score used to distinguish between taxon and complement members. The structure of a latent taxon will differ depending on the proportion of taxon members in the overall sample (Meehl & Yonce, 1996). If the proportion, or base rate, is approximately .50, then the highest point of covariance will occur in the middle of the distribution as the slab will contain an even mixture of taxon and complement members (Waller & Meehl, 1998). The slab at which the highest point of covariance occurs will change accordingly if the proportion of taxon members, or base rate, is greater or less than .50. Examples of taxonic and nontaxonic covariance plots can be seen in Figure 5.1.
After the structure of a sample has been determined, the reduced General Covariance Mixture Theorem can be used to estimate the overall base rate of the group (Golden & Meehl, 1979; Waller & Meehl, 1998). Recall, the covariance between the output indicators was described by

$$\text{cov}(xy) = PQ \left( \bar{x}_r - \bar{x}_c \right) \left( \bar{y}_r - \bar{y}_c \right).$$

This can be further reduced to

$$\text{cov}_r(xy) = pqK.$$
where

\( i \) represents a particular interval or slab

\[ p_i = \text{the conditional taxon rate measured as proportion of taxon members in a slab} \]

\[ q_i = \text{the conditional complement rate measured as 1 - } p_i \]

\[ K = \text{the cross product of the latent validities of } x \text{ and } y. \]

The slab that contains the hitmax score occurs when \( p_i \approx .50 \) and therefore, \( p_i q_i \approx .25 \). As a result, the covariance at the hitmax slab can be represented by

\[
\text{cov}_{h}(xy) = \frac{1}{4} K,
\]

and subsequently rearranged to

\[
K = 4\text{cov}_{h}(xy).
\]

As \( K \) is a constant, and the base rate of one slab is known, then the overall base rate can be calculated. To do this, the equation is further reduced to

\[
Kp_i^2 - Kp_i + \text{cov}_i(xy) = 0.
\]

For each sub sample or slab the conditional taxon rate, \( p_i \), is multiplied by the sample size of the slab to estimate the number of taxon members within each slab, \( n_i \). These values are added together to provide an estimate of the number of taxon members in the whole sample. This estimate is then divided by the total sample size, \( N \), to calculate the grand taxon base rate, \( P \)

\[
P = \left( \sum n_i \right) / N.
\]
Repeated analyses can be conducted where each separate indicator is designated as the input indicator with two output indicators (Meehl & Yonce, 1996). The order of the output indicators is not important. For example, if 4 indicators are available, 12 separate MAXCOV analyses can be conducted, providing 12 estimates of the taxon base rate. These analyses provide estimates of the hitmax for the other indicators and the base rates can be averaged to determine a mean base rate (Meehl, 1973).

Identification of Taxon and Complement Members

Bayes’s Theorem is used in the MAXCOV procedure to identify individual taxon members (Meehl, 1973). An estimate of the taxon base rate is required, as well as estimates of the valid positive rates and false positive rates for each taxon indicator. A valid positive rate is defined as the probability that a taxon member scores higher than the hitmax point for a particular indicator (Waller & Meehl, 1998). A false positive rate is defined as the probability that a nontaxon member scores higher than the hitmax point for a particular indicator (Waller & Meehl, 1998). These rates are then applied using Bayes’s Theorem and a probability value of taxon membership is produced for each individual. As with the covariance values, the distribution of probabilities can be plotted and observed to see whether there is further support for a taxon. If the structure is taxonic then the distribution of taxon membership probabilities will resemble a U shape, as seen in Figure 5.2. If the structure is dimensional, then the distribution of taxon membership probabilities is more evenly distributed and the plot will look more consistent, as seen in Figure 5.3.
Figure 5.2. Example of taxon membership distribution for taxonic data (adapted from Waller & Meehl, 1998).

Figure 5.3. Example of taxon membership distribution for nontaxonic data (adapted from Waller & Meehl, 1998).
Consistency Tests

Consistency tests are viewed as a necessary component of taxometric analysis (Meehl, 1992, 1995a, 2004). Consistency tests can be used to evaluate the level of agreement between the indicators in regards to the taxon profile that they each produce (Waller & Meehl, 1998). This acts as an estimate of the reliability and validity of the latent structure. There are a number of different consistency tests that can be carried out with taxometric procedures. One of these involves examining the variance of the estimates of the base rate across the multiple MAXCOV analyses (Meehl, 1973). The taxonic structure is supported if the variance is small whereas large variance is inconsistent with a latent taxonic structure.

Another consistency test involves calculating the observed and predicted covariance of each indicator combination (Meehl, 1973). Ideally the difference between the observed and predicted matrices would produce a null matrix, however, the indicators are not perfect, and, therefore, it is sufficient for the observed and predicted covariance matrices to be similar (Waller & Meehl, 1998). The similarity between the observed and predicted covariances can be determined using a goodness-of-fit index (GFI). The GFI can be interpreted as a multivariate $R^2$, and produces a value that ranges from 0.00 to 1.00. A higher value is indicative of a better taxonic fit.

Further consistency tests can be carried out by applying other taxometric procedures to the data (Meehl & Yonce, 1994, 1996). These include MAMBAC, MAXEIG-HITMAX, and L-Mode. All of the procedures produce at least one estimate of the underlying base rate and these can be compared to determine the degree of convergence across the procedures. In addition, the plots produced by the procedures can also be compared. Meehl and colleagues have reportedly developed a total of 13 taxometric analysis procedures (Meehl, 1999). The more well-known ones are MAXCOV,
MAMBAC, MAXEIG, and L-Mode. Of these, MAXEIG and L-Mode have only been developed recently and have appeared in fewer studies than MAMBAC and MAXCOV. Grove and Meehl (1993) have developed another taxometric analysis procedure called MAXSLOPE (Maximum Slope) which is similar to MAXCOV but requires only 2 indicators and produces a maximum regression slope. In addition, Golden (1991) developed an extension of the MAXCOV procedure called the taxonomic regression method.

**MAMBAC**

The MAMBAC (mean above minus below a cut; Meehl & Yonce, 1994) procedure can be used as a consistency test to evaluate the results of a MAXCOV analysis. The MAMBAC procedure also involves the taxometric analysis of a number of indicator variables. The difference in mean scores between group members who fall above and below a series of cuts made along the indicator variables is calculated. Plots of the difference scores are used to infer whether the latent structure is taxonic or dimensional.

The indicator requirements for a MAMBAC analysis are the same as for a MAXCOV analysis, however, a minimum of 2 indicators, not 3, are required (Meehl & Yonce, 1994). The MAMBAC procedure involves designating one variable as the input indicator and another variable as the output indicator. A series of cuts are made along the input indicator. Meehl and Yonce (1994) have recommended, based on Monte Carlo runs, that a minimum of 15 individuals should fall above or below the cut at each end of the distribution and this value be used to set the interval (measured in standard deviations) at which cuts should be made. A sufficient number of cuts have to be made to ensure that the shape of the subsequent plot produced by this procedure is clear. A mean output indicator score is calculated for individuals who fall above the cut and a mean output
indicator score is calculated for individuals who fall below the cut (Meehl & Yonce, 1994). Following this, the mean of the group below is subtracted from the mean of the group above the cut. The difference score that is produced constitutes one data point on a MAMBAC plot (Meehl & Yonce, 1994).

The difference scores are plotted with the number of cuts on the input indicator the same as the number of data points on the MAMBAC plot. If the plot forms a peak, then this is indicative of a taxonic structure (Meehl, 1995a). If the plot resembles a dish-shaped curve, then a dimensional structure may be inferred. This can be repeated with each possible input-output combination, depending on the number of variables available (Meehl & Yonce, 1994). For example, 4 indicators results in 12 plots. Each input-output combination produces an estimate of the base rate.

Occasionally, a taxonic sample will produce a MAMBAC plot that is not clearly taxonic, i.e., does not form a clear peak, yet all other plots are clearly taxonic (Meehl & Yonce, 1994). In this situation, the opposite indicator combination will often produce a taxonic plot. That is, if the combination of input = x and output = y did not produce a clear peak, then the combination of input = y and output = x may produce a taxonic plot (Meehl & Yonce, 1994).

Unlike Meehl's recommendations of the size of samples to be used with MAXCOV, MAMBAC can be used with small samples (e.g., 100). It is recommended, however, that larger samples be used or, if this is not possible, that more than 2 indicators be used (Meehl & Yonce, 1994).

MAXEIG-HITMAX

The MAXEIG-HITMAX (MAXEIG) procedure is very similar to MAXCOV; however, instead of using the degree of covariance between indicators, eigenvalues are
calculated (Waller & Meehl, 1998). The multivariate equivalent of covariance is the eigenvalue (Ruscio, Ruscio, & Keane, 2002). The MAXEIG procedure is able to be applied to relatively small samples. In order to do this, overlapping windows are used in the analysis for the input variable whereas with MAXCOV, the slabs for the input variable do not overlap with each other. The number of individuals in each window or interval is determined by the degree of overlap between each window (Waller & Meehl, 1998). One variable is designated as the input indicator and the other variables are the output. The eigenvalues for the output indicators are then calculated for individuals in each window. These can be plotted as with the MAXCOV procedure. This process can be repeated in turn with each variable as the input indicator.

**L-Mode**

The L-Mode procedure begins with a factor analysis of all the indicators (Waller & Meehl, 1998). The distribution of the scores is then reviewed on the basis of the first principal factor that the analysis produces. If the plot of the factors scores resembles a bimodal distribution, then the structure is said to be taxonic (Waller & Meehl, 1998). If the plotted factor scores have a unimodal distribution, then the structure is said to be dimensional.

An estimated base rate is also produced by the L-Mode procedure (Waller & Meehl, 1998). This can be done in two ways. Firstly, an estimate can be obtained from the location of each mode and then averaged. Secondly, the L-Mode procedure categorises a proportion of the sample as taxon members and this can be used as an estimate of the base rate. These base rates can in turn be compared to the base rates generated by the other taxometric procedures. The proportion of the sample categorised as taxon members by the
L-Mode procedure can be compared to the proportion categorised as taxon members by the MAXCOV procedure.

Ideally, a researcher would use at least two of the taxometric procedures described above when examining the latent structure of a construct. The conclusions he or she makes about the nature of a construct under investigation must be backed up by multiple consistency tests in order for the conclusions to be well founded (Meehl, 1995a). After a taxometric analysis has been carried out, inferences on the latent structure depend on both the nature of the indicators and the sampling population (Meehl, 2004). It is important to know additional information about the research sample as a whole as well as differences between the taxon and complement groups (Lenzenweger, 2004). For example, this may include information on diagnostic criteria that individuals meet or other features that have been screened for during the assessment. Preferably, these other measures have not been part of the taxometric analysis (Lenzenweger, 2004).

**Taxometric Analysis in Research**

Taxometric analysis has been used in research to examine a number of different constructs, including personality traits and mental illnesses, for the presence of manifest taxa consistent with latent taxa or manifest dimensions consistent with latent dimensions. Before describing some of these studies, it is important to make a distinction between manifest and latent structure. Manifest structure involves the identifiable characteristics of a construct that are measured or assessed (Ruscio & Ruscio, 2004b). Latent structure involves the underlying structure of the construct that is present, irrespective of how it is assessed or measured (Ruscio & Ruscio, 2004b). Studies that have used a variety of taxometric procedures to consider the manifest and latent taxon or dimension of a number of constructs include research on posttraumatic stress disorder (Ruscio et al., 2002), eating
disorders (Williamson et al., 2002; for a review of taxometric analysis of eating disorders see Williamson, Gleaves, & Stewart, 2005), dementia (Golden, 1982), Jungian preferences or attitudes (Arnau, Green, Rosen, Gleaves, & Melancon, 2003), sexual orientation (Gangestad, Bailey, & Martin, 2000), children at risk for schizophrenia (Erlenmeyer-Kimling, Golden, & Comblatt, 1989), personality factors (Gangestad & Snyder, 1985), depression (Haslam & Beck, 1994; Ruscio & Ruscio, 2000), psychopathy (Harris, Rice, & Quinsey, 1994; Marcus, John, & Edens, 2004), dissociation (Waller, Putnam, & Carlson, 1996), and schizotypy (e.g., Lenzenweger & Korfine, 1992; Tyrka et al., 1995a). Some of these studies will be described next.

The MAMBAC, MAXEIG, and L-Mode procedures were used by Ruscio et al. (2002) to examine the underlying structure of posttraumatic stress disorder (PTSD). Their sample comprised 1,230 male participants who were all war veterans and data came from two common measures of PTSD. The scores on the measures were used to create three types of indicator sets. Each of the three taxometric procedures produced results that were consistent with PTSD being conceptualised as a latent dimension as opposed to a latent taxon. Ruscio et al. (2002) were able to use a number of consistency tests to further confirm their findings of a manifest dimensional structure as measured by the PTSD assessment tools that they used.

The constructs of sexual orientation and gender identity were investigated by Gangestad et al. (2000) using MAXCOV and MAMBAC. In a study of 4,901 male and female twins, they found that there was evidence of an underlying taxon for both sexual orientation and gender identity with 12% to 15% of men and 5% to 10% of women having taxa membership related to homosexual preference. These findings contradicted past research that suggested that sexual orientation is a dimensional construct and provided support for a categorical conceptualisation of the manifest structure.
Depression and its subtypes have been evaluated by various researchers using taxometric analysis procedures (e.g., Haslam & Beck, 1994; Ruscio & Ruscio, 2000). Haslam and Beck (1994) used the MAXCOV procedure to look at the nature of 5 subtypes of depression. They found that only one of the subtypes was distinctly categorical whereas the results for the other subtypes were consistent with a latent dimensional structure. However, their interpretation of the plot upon which they based their conclusion about the taxonic subtype has been questioned. Haslam and Beck’s (1994) dimensional findings were supported in a study by Ruscio and Ruscio (2000) who used both MAXCOV and MAMBAC procedures with three well-known measures of depression administered to a clinical sample. They created indicator variables using three distinct methods and found there was no evidence for the taxonicity of depression. Ruscio and Ruscio (2000) concluded that the manifest structure of depression is dimensional.

The manifest structure of cognitive vulnerability to depression has also been found to be dimensional (Gibb, Alloy, Abramson, Beevers, & Miller, 2004), as has the manifest structure of depression in a community sample with depressive symptomatology (Slade & Andrews, 2004).

Various dimensional models of eating disorders have been proposed as a result of the controversy surrounding the DSM-IV classification of eating disorders into categories (Williamson et al., 2002). MAMBAC and MAXCOV procedures were used in a study by Williamson et al. (2002) to investigate this controversy further. They found in their sample of 341 clinical and non-clinical female participants that eating disorders (anorexia nervosa, bulimia nervosa, binge eating disorder, and eating disorder not otherwise specified) formed a category that was distinct from people without eating disorders. In addition, when each disorder was evaluated independently, all of the disorders (apart from anorexia nervosa) produced evidence of a manifest taxonic structure. The findings for
anorexia nervosa were mixed. Williamson et al. (2002) also investigated the eating disorders group as a whole by excluding non-clinical participants and found that some of the eating disorder features were suggestive of the presence of a manifest taxon and others were suggestive of a manifest dimension. Their findings were inconsistent with an overall latent dimensional model of eating disorders.

Waller et al. (1996) examined the structure of dissociation using various taxometric analysis procedures. Historically, dissociation has been viewed as a dimensional construct; however, Waller et al. (1996) showed that this may not be the case. In their study, 456 individuals (228 normal controls and 228 people diagnosed with multiple personality disorder) completed the Dissociative Experiences Scale (DES; Bernstein-Carlson & Putnam, 1986) and their responses were evaluated using the MAMBAC, MAXSLOPE, and MAXCOV procedures. Analyses were conducted across the whole group rather than within groups. This produced an 8-item version of the DES, called the DES-T that contained items that were all thought to be taxonic indicators. Waller et al. (1996) found evidence for a manifest dissociative taxon with a distinct difference between pathological dissociation (e.g., amnesia, depersonalization) and nonpathological dissociation (e.g., daydreaming). Furthermore, they found that the indicators for nonpathological dissociation were suggestive of a manifest dimensional structure.

Haslam (2003b) conducted a review of 21 studies that had used taxometric analysis to examine the structure of personality disorders including schizotypal, antisocial, and borderline personality disorders. He concluded that 80% of the studies produced evidence of a manifest taxon for personality disorders. This was inconsistent with a prevailing view that personality disorders are dimensional constructs. However, he also argued that those studies that produced mixed or nontaxonic results tended to be methodologically flawed or weak and had various limitations (Haslam, 2003b).
Taxometric analysis procedures have also been used to determine whether constructs are assessed appropriately in research. Ruscio and Ruscio (2002) used three taxometric analysis procedures to evaluate the manifest structure of depression as measured by the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979). The basis of their study came from the trend for research to use university student (or analogue) samples to investigate depression and classify the participants on the basis of cut-off scores on the BDI. Ruscio and Ruscio (2002) found evidence for the manifest dimensional structure of depression as measured by the BDI. They concluded that the use of cut-off scores in research with analogue samples may produce inaccurate results that misrepresent the true nature of depression. Instead, they suggest that the measurement method used should match the structure of depression, that is, a continuous description would be more appropriate than a discrete description.

In addition, Meehl’s taxometric analysis procedures have been subjected to, and validated by, numerous Monte Carlo studies with artificial data samples. These have included studies of MAXCOV (e.g., Haslam & Cleland, 1996; Meehl, 1995a; Meehl & Yonce, 1996), and MAMBAC (e.g., Cleland & Haslam, 1996; Meehl, 1995a; Meehl & Yonce, 1994). Furthermore, Cleland and Haslam (1996; Haslam and Cleland, 1996) have investigated the ability of MAXCOV and MAMBAC to provide true results when the data are skewed. They found that both procedures are not affected by the presence of skewed data and produce reliable results under these conditions. It is advised, however, that caution needs to be exerted when interpreting MAXCOV results of small base rates produced by skewed indicators. A Monte Carlo study has also shown that MAXCOV is superior to cluster analysis for certain conditions (when there are reductions in effect size, indicator and base rate; and increases in nuisance covariance; Beauchaine & Beauchaine,
investigate whether the taxonic structure they had observed was due to the dichotomous item format. He administered a number of schizotypy measures, including the PAS (Chapman et al., 1978), the Magical Ideation Scale (MIS, Eckblad & Chapman, 1983), and the Referential Thinking Scale (REF; Lenzenweger et al., 1997) to a sample of 429 undergraduate university students. The total scores of the scales were used as indicators for a MAXCOV analysis. Lenzenweger (1999) observed a pattern consistent with a latent taxon when the data was plotted. In addition, the median base rate was .13. The consistency between the findings of these three studies provides support for the use of MAXCOV in identifying a true taxon for schizotypy.

Meyer and Keller (2001) also administered the PAS and the MIS to a sample of 809 tertiary education students from Germany taking courses in various vocational and industrial skills such as travel agents and beauticians. In addition, the Physical Anhedonia Scale (PhA; Chapman et al., 1976) was administered and a MAXCOV analysis was used. Meyer and Keller (2001) observed a taxonic structure for the PAS and PhA with estimated base rates of .12 and .15. This provides evidence of manifest taxa for perceptual distortions and physical anhedonia. The structure for the MIS, however, was suggestive of a latent dimension, indicating that the schizotypal features of magical ideation and odd beliefs may not be a manifest taxon in this sample.

In addition to body image, perceptual distortions, and odd beliefs, another trait thought to be a core feature of schizotypy, anhedonia, has been examined with taxometric procedures. Blanchard, Gangestad, Brown, and Horan (2000) investigated the construct of social anhedonia and whether it is a taxonic indicator of schizotypy. Recall, that Meehl (1962) initially proposed that social anhedonia is an essential feature observed in people with schizotypy. He later amended this to propose that social anhedonia is not a core feature of schizotypy and is observed in nonschizotypal individuals as well (Meehl, 1989,
Blanchard et al. (2000) wanted to determine which of these views was most accurate. They administered the Revised Social Anhedonia Scale (RSAS; Eckblad, Chapman, Chapman, & Mishlove, 1982, cited in Blanchard et al., 2000) to 1,526 undergraduate university students. MAXCOV and MAXEIG procedures were applied to the data and a manifest taxonic structure with a mean base rate of .083 was observed. Blanchard et al. (2000) concluded that their results conflict with Meehl’s (1989, 1990b) more recent view that social anhedonia is a dimensional construct. However, Meehl never stated whether social anhedonia is taxonic or dimensional, but instead his later theoretical revision involved the notion that social anhedonia is not a key feature of schizotypy and is also observed in nonschizotypal people. It is not clear in Blanchard et al.’s (2000) study that they assessed Meehl’s notion, however, their conclusion that social anhedonia is consistent with a latent taxon appears to be accurate.

In another study, Horan, Blanchard, Gangestad, and Kwapil (2004) administered the RSAS, PAS, and MIS to a large group of undergraduate university students to determine if the three measures of schizotypy shared a latent taxonic structure. Both MAXCOV and MAMBAC procedures were used in the study. Horan et al. (2004) observed a taxonic structure for RSAS which replicated Blanchard et al.’s (2000) results. There was also evidence for a manifest taxon for the PAS. Base rates approximating Meehl’s (1990b) proposed .10 were observed for the taxonic results. They did not observe support for a latent structure for the MIS which is consistent with Meyer and Keller’s (2001) findings. In addition, the results did not support the hypothesis that the three measures shared a common manifest taxon. Linscott (2005) found evidence of a manifest taxon for schizotypy (including magical ideation and perceptual aberration) and a manifest dimension for hypohedonia (physical and social anhedonia), which is inconsistent with the findings of Horan et al. (2004). In addition, the two constructs were shown to be
independent of one another. Furthermore, Linscott (2005) found that there was an association between schizotypy and clinical distress and diminished attention.

Taxometric analysis has also been used to investigate schizotypy in an at-risk population. Tyrka et al. (1995a) used the MAXCOV procedure to evaluate schizotypy in a sample of 311 individuals; 207 who had mothers with a schizophrenia-spectrum disorder, and 104 normal controls. The children had initially been assessed at the age of 15 then followed up at 25 and 39 years. Tyrka et al. (1995a) used premorbid behavioural indicators and observed evidence of a taxon in the sample of 15 year olds with an estimated base rate of .48. In the taxon group, 81% were from the sample that had mothers with a schizophrenia-spectrum disorder and 19% were normal controls. In addition, 67% of the sample who later developed schizophrenia were taxon members and 73% of the sample who later developed schizotypal, paranoid, or schizoid personality disorder were taxon members. These results suggest that being a taxon member at age 15 was predictive of developing a schizophrenia-spectrum disorder. In all, 40% of the taxon developed a schizophrenia-spectrum disorder while 16% of the complement developed a schizophrenia-spectrum disorder. Tyrka, Haslam, and Cannon (1995b) also used taxometric analysis procedures to evaluate the group at age 25 and 39 years. Again, they observed evidence of a latent taxon at both ages. Furthermore, being a taxon member was predictive of developing a schizophrenia-spectrum disorder, where 60% of the taxon at age 25 and 56% of the taxon at age 39 later met criteria for a schizophrenia-spectrum disorder (Tyrka et al., 1995b).

Research with clinical populations that employs taxometric procedures to evaluate schizotypy has been very limited. In a comprehensive literature search, only one published article was identified that applied taxometric procedures to schizotypy in a clinical population. Golden and Meehl (1979) used responses on the Minnesota
Multiphasic Personality Inventory (MMPI) as indicators for the schizoid taxon, or schizotaxiaMeehl. Recall, that Meehl’s view is that all who have schizotaxia will develop schizotypy, therefore, this study was also looking at indicators of schizotypy. Golden and Meehl (1979) applied taxometric procedures to the MMPI responses of 211 male psychiatric inpatients. They observed a base rate for schizotaxiaMeehl/schizotypy approximating .40 in their clinical sample. This high base rate is due to the nature of the population sampled by Golden and Meehl (1979). It is likely that the inpatient population is characterised by individuals with chronic and severe mental illnesses such as schizophrenia and in addition, schizotypy is considered to be part of the schizophrenia-spectrum disorders and a precursor for schizophrenia. Furthermore, Meehl (1962, 1990b) outlined in his theory of risk for schizophrenia that all individuals with schizotaxia develop schizotypy and some of these individuals later develop schizophrenia. Assuming that Meehl’s theory is correct, it is logical that individuals with schizophrenia and other related disorders will also display schizotypal features and that the proportion of individuals in the clinical population who display these features would be higher than in the general population. Much more research is required that examines the structure of schizotypy using taxometric analyses with clinical populations.

Criticisms of Taxometric Analysis Procedures

A number of researchers have pointed out limitations and criticisms of the taxometric analysis procedures. Garb (1996) has criticised Meehl’s (1995a) statement that in order for a taxometric analysis procedure to be carried out a researcher needs to be fairly accurate when hypothesising that a taxon actually exists. Garb (1996) has said that it is not always clear if a single taxon actually exists for a construct under investigation and questions the rigidity of using a guideline such as Meehl’s. In later research, Meehl
(2004) has conceded that taxometric analysis can be used even when a researcher does not hold a strong view that a taxon is present. In line with this, the importance of adhering to guidelines that have been provided when using taxometric analysis has been emphasised in research (Cole, 2004). The purpose of this is to avoid oversimplifying the procedures of taxometric analysis in order to prevent the production of misleading results about the latent structure of a construct.

A number of limitations of the MAXCOV procedure have been highlighted. Miller (1996) has shown that the covariance curve for the MAXCOV approach is not always flat for a nontaxonic set of data. In addition, it has been shown that consistency tests do not always correctly identify inconsistency for nontaxonic data. If a MAXCOV analysis is carried out correctly, however, then it should involve at least three indicators, and, therefore, a researcher would be presented with at least three plots to observe to examine for consistency or inconsistency (Meehl, 1996). Miller (1996) has also criticised a reliance on visual observations of MAXCOV graphs to conclude whether a taxon is present or not. He states that this is because the appearance of a curve can be affected by the y-axis scale and lead people to make inaccurate conclusions. Various studies by Meehl and Yonce (1994, 1996; Meehl, 1996) have shown that taxonic and nontaxonic plots of taxometric procedures can be accurately sorted by both psychologists and nonpsychologists.

A large amount of the research on schizotypy using taxometric analysis has involved rating scales such as the PAS and MIS. Beauchaine and Waters (2003) have cautioned against researchers only using rating scales as indicators when carrying out taxometric procedures. They demonstrated pseudotaxonicity in a study where they manipulated the information that raters had about the characteristics of a sample they were rating. When the data were evaluated using taxometric analysis (MAXCOV and MAMBAC), they
found that one group of raters provided results indicative of a taxon and another group of raters provided results indicative of a dimension. Beauchaine and Waters (2003) concluded that human bias can result in the creation of artefactual categories when people are completing ratings of other individuals. Other researchers (e.g., Haslam, 2003a) have suggested that artefactual categories may also occur when individuals complete rating scales about themselves.

There are relatively few studies using taxometric research methods, however this number is increasing. An increase in the quantity of taxometric findings currently available is required in order to support and increase confidence in previous findings (Haslam, 2003a). This includes more research using the MAXEIG and L-Mode procedures as well as research in other areas of psychopathology such as psychosis.

*Implications of Taxometric Analysis Research*

The findings of research using taxometric analysis have implications for a number of aspects of psychopathology, including aetiology, theory, assessment, classification and diagnosis. Recall that Meehl initially developed the taxometric analysis procedures while trying to further understand predisposition for schizophrenia in terms of schizotaxia. There is still a lot of research that needs to be carried out but research into schizotypy will hopefully robustly clarify in the future the transmission mode of risk for schizophrenia. Taxometric analysis may help to discern the causal factors for the construct of schizotypy. This depends on whether research can consistently show that schizotypy is a taxonic construct or a dimensional construct. For a dimensional construct it is likely that a number of minor factors are contributing to the structure, however, for a taxonic construct, it is likely that one dichotomous factor is involved (Haslam, 2003a).
In terms of the theory behind conceptualisations of constructs, taxometric analysis findings may also be beneficial. It is important to remember that if a taxon is identified, this does not automatically provide support for a biological model (Haslam, 2003a). The conceptualisation of a construct influences how that construct is assessed. For example, if taxometric analysis identifies a dimensional construct then an assessment tool that employs a particular cut point for viewing a factor as absent or present is misleading (Haslam, 2003a). Taxometric analysis may also impact on assessment by highlighting the problems associated with measurement tools (Lenzenweger, 2004).

The conceptualisation of a construct also has an impact on classification and diagnosis. Many of the problems inherent within the DSM-IV were discussed in Chapter 2. Schmidt, Kotov, and Joiner (2004) have proposed that the DSM-IV would be improved if taxometric analysis procedures were applied to each diagnostic construct contained within the classification system. They suggest that this will address problems with the reliability and validity of DSM-IV diagnoses, criticisms of a failure to consider the aetiology of disorders, and the categorical approach used in the DSM-IV. Classification systems such as the DSM-IV have the potential to be affected by findings of taxometric analyses, whether support is provided for either a categorical or dimensional structure. When a categorical construct is identified, current classification systems can be further defined and this may increase accuracy of diagnosis. When a dimensional construct is identified, the classification system can be revised and consequently, dimensional conceptualisations can be introduced (Haslam, 2003a).

Summary

Research using taxometric analysis procedures to identify latent taxa or dimensions has revealed that it is not appropriate to apply a generic dimensional structure. To date,
empirical studies have covered a number of areas of psychopathology, including schizotypy. However, more research is needed using the various procedures that Meehl and his colleagues (e.g., Golden, 1982; Golden & Meehl, 1979; Meehl, 1973; Meehl & Yonce, 1994, 1996; Waller & Meehl, 1998) have developed to increase confidence in the robustness of the methods.

The research involving schizotypy has provided support for conceptualising schizotypy as taxonic in the general population (Lenzenweger & Korfine, 1992, 1995; Tyrka et al., 1995a, 1995b). There is, however, a dearth of research that has utilised taxometric analysis procedures for investigating schizotypy in a psychiatric population. This is in contrast to the increasing quantity of research that has examined depression, eating disorders, and other personality traits in clinical populations. In addition to increasing confidence in taxometric procedures in general, future research needs to be conducted to evaluate the application of taxometric procedures when investigating the structure of schizotypy in clinical populations. It needs to be determined whether the structure of schizotypy and related features in clinical populations mirrors that seen in general populations. This research would need to proceed with caution to ensure that the appropriate guidelines for taxometric analysis are observed and that the construct being evaluated is in fact schizotypy and not features of other psychiatric disorders. Research that looks at schizotypy and risk for schizophrenia in general populations is valuable and meaningful, however, research on schizotypy with clinical populations also has the potential to make a notable contribution to knowledge about the pathways involved in the aetiology of schizophrenia as well as current theories related to this.
CHAPTER 6

The Overlap of Schizotypy and Schizotaxia

The Present Study

Over the past century there have been many problems associated with the diagnosis of schizophrenia and how the construct is defined. Many improvements have been made since schizophrenia was first introduced into the diagnostic nomenclature. However, there are still criticisms of the classification criteria that are currently used for diagnosing schizophrenia. Some of these criticisms relate to the categorical nature of diagnostic systems, the type of criteria used to define schizophrenia, the focus on psychosis in diagnosis, and the questionable reliability and validity of the construct of schizophrenia as it is used in diagnostic systems. As a result of these problems, research has considered other ways in which schizophrenia can be conceptualised. It has been suggested that diagnostic systems would be improved if the underlying aetiology of schizophrenia was incorporated into the construct. Research in this area has investigated incorporating a dimensional approach into the definition of schizophrenia by considering risk for schizophrenia.

Two contrasting theories of risk for schizophrenia have been considered in the context of this thesis. One of these theories was developed by Meehl. He used the term \textit{schizotaxia}_{Meehl} to describe a genetic liability for schizophrenia (Meehl, 1962, 1989, 1990b). Meehl hypothesised that as the result of the interaction between \textit{schizotaxia}_{Meehl} and environmental factors, most people who have \textit{schizotaxia}_{Meehl} develop a personality
organisation that he labelled schizotypy. He proposed that approximately 10% of schizotypal people decompensate after exposure to further environmental factors and develop schizophrenia. Meehl (1990b) conjectured that 10% of the general population has schizotypy and that schizotypy is observed in 35% to 40% of the psychiatric population.

A second theory of risk for schizophrenia was developed by Tsuang and colleagues. Tsuang et al. (1999b) proposed that early environmental insults interact with a genetic predisposition to produce a vulnerability to developing schizophrenia, called schizotaxia\textsubscript{Tsuang}. Tsuang and colleagues have conjectured that schizotaxia\textsubscript{Tsuang} is manifested in the form of neurodevelopmental brain abnormalities known as target features. They have proposed that schizophrenia develops as a result of the interaction between the neurodevelopmental abnormalities and environmental factors. Tsuang and colleagues have reported that 20% to 50% of relatives of individuals with schizophrenia experience the symptoms of schizotaxia\textsubscript{Tsuang} (Faraone et al., 1995a, 199b). They have not incorporated Meehl’s construct of schizotypy into their theory but predict that there is some degree of overlap between schizotaxia\textsubscript{Tsuang} and negative schizotypal personality disorder.

There are some similarities between the theories of Meehl and Tsuang et al. but also many differences. One of the key distinctions concerns differences between schizotaxia\textsubscript{Meehl} and schizotaxia\textsubscript{Tsuang}. These differences relate to the aetiology of schizotaxia (genetic versus genetic and environmental), how the construct is observed (nonmeasurable versus measurable), the outcome of schizotaxia (schizotypy and maybe schizophrenia versus maybe schizophrenia), and the relationship between schizotaxia and other schizophrenia-spectrum disorders (quasidimensional versus categorical). However, many of the differences may not actually be differences and may have resulted from difference conceptualisations of the same construct. This is because schizotaxia\textsubscript{Tsuang} has
many similarities to Meehl's schizotypy and therefore may be at the same level conceptually. Tsuang and colleagues maintain that schizotypy is not the likely outcome of schizotypia_Tsang_. This may be because they appear to have equated Meehl's schizotypy with schizotypal personality disorder. They use the terms interchangeably in their theory and research yet there are significant differences between the two constructs because Meehl's schizotypy is much broader than schizotypal personality disorder.

Two theories of risk for schizophrenia have been reviewed in this thesis. One group of researchers maintains that their theory is very different to the other theory. However, different conceptualisations of the same construct may have contributed to this conclusion. It may be more appropriate to view some of the components of the two theories as similar. Research into these theories has not previously considered this.

**Phase 1.** Research into Meehl's theory has traditionally focused on his conceptualisation of schizotypy and has resulted in the development of a number of psychometric measures. Studies employing these measures have attempted to elucidate the correlates of risk for schizophrenia. Some research appears to be consistent with Meehl's notion of schizotypy while other research involves conceptual problems relating to the misrepresentation or a misunderstanding of Meehl's concepts. Research has also investigated the structure of schizotypy and identified that schizotypy is multidimensional in nature involving at least two factors. Another aspect of schizotypy that has recently been investigated is whether the underlying structure of the construct is dimensional or categorical. This type of research has utilised taxometric analysis procedures (e.g., Golden, 1982; Golden & Meehl, 1979; Meehl 1973; Meehl & Yonce, 1994, 1996; Waller & Meehl, 1998), which are statistical procedures that can be used to assist distinguishing evidence of latent taxa from distributions of continuous data.
Research using taxometric analysis procedures to investigate the construct of schizotypy has provided support for conceptualising schizotypy as taxonic in the general population (Lenzenweger & Korfine, 1992, 1995; Tyrka et al., 1995a, 1995b). There is, however, a dearth of research that has utilised taxometric analysis procedures for investigating schizotypy in a psychiatric population. It needs to be determined whether the structure of schizotypy and related features in clinical populations mirrors that seen in general populations. This has the potential to make a significant contribution to what is known about the aetiology of risk for schizophrenia. Consequently, the first aim of the present study is to investigate the manifest structure of Meehl’s schizotypy in a sample of psychiatric patients. This will be achieved in Phase 1 (Chapter 7), by using a self-report measure of schizotypy, the Thinking and Perceptual Style Questionnaire (TPSQ; Linscott & Knight, 2004) and taxometric analysis procedures, namely MAXCOV and MAMBAC. It is hypothesised that, consistent with the trend observed in the general population, there will be evidence of a manifest taxonic structure for schizotypy in the psychiatric participants.

Phase 2. Research into Meehl’s schizotypy using the psychometric approach has found that, relative to nonschizotypy groups, schizotypy groups tend to display impairments in a range of neuropsychological functions (e.g., Gooding et al., 2001; Lenzenweger et al., 1991; Park et al., 1995). Impairments have been observed in the domains of attention, verbal memory, working memory, and executive functioning. There is an increasing amount of research involving taxometric analysis and schizotypy but few studies have examined the neuropsychological and psychopathological functioning of schizotypy and nonschizotypy groups identified through taxometric analysis, and no study has considered this in a psychiatric population. The second aim of the current study is to determine if schizotypy group membership is associated with poorer functioning.
To date, Tsuang and colleagues have conducted a limited number of studies with a small number of participants who have relatives with schizophrenia to investigate the criteria for schizotaxia\textsubscript{Tsuang} and the effectiveness of medications at alleviating the symptoms of schizotaxia\textsubscript{Tsuang}. Their goals are for schizotaxia\textsubscript{Tsuang} to enter the diagnostic nomenclature and for treatments to be available to high-risk groups including first-degree relatives and children thought to be at risk of developing schizophrenia with the aim of preventing schizophrenia. However, before further research is conducted that creates further classification categories and looks at psychopharmacological treatments for Tsuang and colleagues' schizotaxia\textsubscript{Tsuang}, it is essential that research examines the robustness of the schizotaxia\textsubscript{Tsuang} criteria. In addition, it has not been established how Tsuang and colleagues' categorical conceptualisation of schizotaxia\textsubscript{Tsuang} is related to Meehl's quasidimensional view of schizotaxia\textsubscript{Meehl} and schizotypy. Meehl's schizotypy is broader than the construct of schizotypal personality disorder yet many researchers, including Tsuang and colleagues, use the terms interchangeably. Meehl views the schizophrenia spectrum disorders as quantitatively different while Tsuang and colleagues view the schizophrenia spectrum disorders as qualitatively different.

Despite these differences, it appears that Meehl's schizotypy and schizotaxia\textsubscript{Tsuang} may actually be very similar conceptually; however this has not been evaluated to date. Furthermore, the construct of schizotaxia\textsubscript{Tsuang}, as conceptualised by Tsuang et al. (1999b), has not been assessed in a psychiatric population, let alone in conjunction with Meehl's schizotypy. Consequently, the third aim of the present study is to investigate the nature of the relationship between Meehl's schizotypy and Tsuang et al.'s schizotaxia\textsubscript{Tsuang} in a psychiatric population.

Aims two and three will be achieved in Phase 2 (Chapter 8), by administering a number of neuropsychological tests and a measure of negative symptoms to sub-samples
of taxon and complement members from the Phase 1 participants. A diagnostic interview will also be conducted at the time. The taxon and complement groups will be compared in terms of their scores on the neuropsychological and negative symptoms measures. It is hypothesised that, compared to nonschizotypy group members, schizotypy group members will have poorer functioning on a range of measures. In addition, the participants in the schizotypy (taxon) and nonschizotypy (complement) groups will be grouped according to whether they meet criteria for Tsuang and colleagues’ schizotaxia\textsubscript{Tsuang}, to create four groups. Statistical analyses will be used to examine the relationship between the four groups to determine whether there is dependence or independence present. It is hypothesised that, as schizotypy and schizotaxia\textsubscript{Tsuang} are conceptually very similar, there will be evidence of dependence between schizotypy and schizotaxia\textsubscript{Tsuang}. 
CHAPTER 7

The Overlap of Schizotypy and Schizotaxia:

Phase 1

The Identification of Schizotypy in a Mixed Psychiatric Sample

The aim of Phase 1 was to investigate the latent structure of Meehl's (1962, 1990b) schizotypy in a mixed sample of psychiatric patients. This was undertaken by administering a self-report measure of schizotypy, the Thinking and Perceptual Style Questionnaire (TPSQ; Linscott & Knight, 2004) to a group of psychiatric inpatients and outpatients. Then, taxometric analysis procedures, MAXCOV and MAMBAC, were applied to the responses to determine if there was evidence of two distinct groups, a schizotypy group and a nonschizotypy group. It was hypothesised that there would be evidence of a manifest taxonic structure for schizotypy in the psychiatric participants.

Method

Participants

Phase 1 participants were inpatients and outpatients from various mental health services located in Dunedin, New Zealand. The mental health services were all operated by the Otago District Health Board. The participants were recruited to the study by their primary healthcare professional. These included psychologists, psychiatrists, psychiatric...
district nurses, primary nurses, occupational therapists, social workers, and case managers. A total of 109 patients were recruited, including 70 females and 39 males. Their ages ranged from 19 to 64 years with a mean age of 39.5 years ($SD = 10.4$). Participants were required to have English as a first language, be aged 18 or older, and be capable of providing informed consent (as judged by their healthcare professional). In addition, the presence of a psychiatric illness was required. Exclusion criteria included the presence or history of a head injury, and/or neurological problems; a substance abuse diagnosis in the past 6 months; and intellectual disability. Ethical approval for this study was obtained from the Otago Ethics Committee.

**Schizotypy Measure**

The Thinking and Perceptual Style Questionnaire (TPSQ; Linscott & Knight, 2004) was used to assess for the presence of schizotypy. The TPSQ was developed from a number of existing measures of schizotypy but is based predominantly on Meehl's (1990b) conceptualisation of schizotypy. The TPSQ is a 99-item self-report measure and consists of 9 subscales. These include physical anhedonia, social anhedonia, hallucinatory tendency, social paranoia, fear of negative social evaluation, thought disorganisation, magical ideation, self-reference ideation, and perceptual illusion. Participants are required to rate their response to each item on a 5-point Likert scale (ranging from 0 to 4). The TPSQ takes approximately 20 to 30 minutes for participants to complete. The full measure is given in the Appendix. The TPSQ has been shown to have construct and concurrent validity and good internal consistency (Linscott, 2005; Linscott & Knight, 2004).

The developers of the TPSQ administered the measure to a sample of 997 undergraduate university students (Linscott, unpublished data). The TPSQ item ratings
from the undergraduate sample were factor analysed and a number of factors were identified. An item was allocated to a subscale on the basis of empirical evidence that the item loaded more heavily onto a particular factor than onto other factors. Items were discarded from the TPSQ if they produced a loading value of less than 0.4 (Linscott, unpublished data). For practical reasons, the first 10 factors were chosen to create 10 alternative subscale scores from the TPSQ (TPSQ-A). The subscales include general thought disorder, social anhedonia, social fear, non-spiritual magical ideation, hallucinations, self-reference ideation, perceptual illusion about changing appearance, solitary pursuits, perceptual illusion about dyscontrol, and thought disorder related to concentration.

Procedure

The patients were asked during their usual appointment with their primary healthcare professionals if they would consider participating in the study. If they agreed, the staff member then gave them a questionnaire pack to take away with them and complete in their own time. The packs contained instructions for completing the questionnaire pack, an information sheet about the study, a consent form to participate, the TPSQ, a consent form to view the participant’s psychiatric files at HealthCare Otago, and the researcher’s contact details. In addition, a voucher form was included for participants to indicate whether they would prefer a $10 petrol voucher or a movie voucher as a token of appreciation for participating in the study. A postage-paid envelope was included with each questionnaire pack for returning responses in.

When a participant’s response was received by the researcher, the information was checked to ensure that it was complete. The participant was sent their voucher of choice
and their medical files were reviewed to ensure that they met inclusion criteria and did not need to be excluded from the study if selected for Phase 2.

Data Analysis

The TPSQ-A produced a total of 10 subscale scores that could have potentially been used as indicators. It was not practical or appropriate to conduct analyses using all 10 indicators. Suitable indicators were selected on the basis of criteria put forward by Waller and Meehl (1998) and Meehl (1999). They suggest that, firstly, any significant nuisance covariance should be identified and removed. This was done by calculating correlation matrices to determine the degree of correlation between pairs of subscales. If subscales were highly correlated with each other then this was interpreted as potentially high nuisance covariance and so only one of the highly correlated scales was included in the analyses. Subscales that were negatively correlated with many other subscales were also removed from the analyses. The remaining indicators were then analysed using the MAXCOV-HITMAX analysis R modules obtained from the taxometric analysis website (Meehl, Waller, & Yonce, 2001; R Development Core Team, 2003).

The MAXCOV-HITMAX procedure produced a covariance plot for each indicator combination and a plot of the distribution of the Bayesian probabilities of taxon membership. The Bayesian probabilities of taxon membership were used to identify individual taxon and complement members.

Several consistency tests were used to determine if there was evidence of results that corroborated with the MAXCOV graph findings. These included estimates of the base rate, which is an approximation of the proportion of people in the sample who are taxon members; the variance of the base rate estimates produced by each indicator combination;
the mean indicator validity, which is the separation between indicators; and a goodness of fit index (GFI).

Lastly, further consistency tests were carried out with the MAMBAC procedure, another taxometric analysis method (Meehl & Yonce, 1994). The MAMBAC procedure produced a series of graphs and the observed shapes of the plotted graphs for each indicator combination and the conclusions formed from these were compared to the plot of the MAXCOV procedure. In addition, the MAMBAC procedure produced base rate estimates and these were compared to the estimate produced by the MAXCOV procedure to examine the similarity of the estimates across analyses.

Results

Approximately 1% of responses for the TPSQ were missing. The missing values were calculated by pro-rating from the mean value of the completed items on the subscale. The mean and standard deviation of scores of the subscales of the TPSQ-A are shown in Table 7.1.
### Table 7.1

<table>
<thead>
<tr>
<th>TPSQ-A Subscale</th>
<th>M</th>
<th>SD</th>
<th>Number of Items</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Thought Disorder</td>
<td>18.74</td>
<td>8.99</td>
<td>11</td>
</tr>
<tr>
<td>Social Anhedonia</td>
<td>19.39</td>
<td>9.31</td>
<td>11</td>
</tr>
<tr>
<td>Social Fear</td>
<td>16.18</td>
<td>7.04</td>
<td>7</td>
</tr>
<tr>
<td>Nonspiritual Magical Ideation</td>
<td>9.20</td>
<td>7.12</td>
<td>9</td>
</tr>
<tr>
<td>Hallucinations</td>
<td>8.52</td>
<td>6.92</td>
<td>7</td>
</tr>
<tr>
<td>Self-reference Ideation</td>
<td>6.53</td>
<td>5.63</td>
<td>6</td>
</tr>
<tr>
<td>Perceptual Illusion (changing appearance)</td>
<td>1.94</td>
<td>2.83</td>
<td>3</td>
</tr>
<tr>
<td>Solitary Pursuits</td>
<td>5.64</td>
<td>3.63</td>
<td>4</td>
</tr>
<tr>
<td>Perceptual Illusion (dyscontrol)</td>
<td>2.61</td>
<td>3.01</td>
<td>4</td>
</tr>
<tr>
<td>Thought Disorder (concentration)</td>
<td>8.26</td>
<td>3.20</td>
<td>4</td>
</tr>
</tbody>
</table>

### Selection of Indicator Variables

The 10 subscales of the TPSQ-A were analysed to examine the degree of correlation among the subscales. It is apparent from the General Covariance Mixture Theorem that in two-group structures, the covariance is maximised where each group is equally represented. The salience of this maximum is reduced where the separation of groups on the indicators is small or where the covariance of the indicators within the groups is large.

Indeed, the General Covariance Mixture Theorem allows one to predict the degree of correlation that should be observed in a two group mixture. For example, if the latent group comprises 50% of the population sample (i.e., \( p = 0.5 \), \( q = 0.5 \)) and mean separations on the indicators are 1.2 standard deviations on standardised indicators, then the correlations among indicators that are not prone to nuisance covariance should be in the vicinity of

\[
pq\left(\bar{x} - \bar{\epsilon}\right)\left(\bar{y} - \bar{\epsilon}\right) = 0.50 \times 0.50 \times 1.2 \times 1.2 = 0.36\ (\text{Waller & Meehl, 1998}).
\]
Correlations substantially greater than this arise where nuisance covariance is problematically high. Therefore, correlations among the TPSQ-A indicators were examined to identify those subscales that had a high level of nuisance covariance (Meehl, 1999). The purpose of this was to reduce the likelihood of including redundant subscales; thereby reducing the risk that nuisance covariance obscures covariance peaks in MAXCOV plots. Ideally, nuisance covariance should be reduced as much as possible.

The matrix of correlations among the 10 subscales is shown in Table 7.2. The data from 4 participants who were identified as outliers were removed prior to creating the correlation matrix and applying the taxometric analysis procedures.

<table>
<thead>
<tr>
<th>TPSQ-A Subscale</th>
<th>SA</th>
<th>SF</th>
<th>Mins</th>
<th>HS</th>
<th>SI</th>
<th>Plc</th>
<th>SolP</th>
<th>PlD</th>
<th>TDc</th>
</tr>
</thead>
<tbody>
<tr>
<td>TDg</td>
<td>.31</td>
<td>.53</td>
<td>.29</td>
<td>.61</td>
<td>.38</td>
<td>.29</td>
<td>.09</td>
<td>.49</td>
<td>.71</td>
</tr>
<tr>
<td>SAg</td>
<td>.44</td>
<td>-.06</td>
<td>.10</td>
<td>-.05</td>
<td>.15</td>
<td>.28</td>
<td>.40</td>
<td>.32</td>
<td>.59</td>
</tr>
<tr>
<td>SF</td>
<td>.13</td>
<td>.30</td>
<td>.10</td>
<td>.22</td>
<td>.10</td>
<td>.40</td>
<td>.59</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mlns</td>
<td>.45</td>
<td>.68</td>
<td>.39</td>
<td>-.09</td>
<td>.35</td>
<td>.25</td>
<td>.59</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HS</td>
<td>.51</td>
<td>.44</td>
<td>-.04</td>
<td>.58</td>
<td>.48</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SI</td>
<td>.34</td>
<td>-.03</td>
<td>.32</td>
<td>.27</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plc</td>
<td>.13</td>
<td>.42</td>
<td>.19</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SolP</td>
<td>.13</td>
<td>.07</td>
<td>.40</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PlD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. TDg = General Thought Disorder, SAg = Social Anhedonia, SF = Social Fear, Mlns = Nonspiritual Magical Ideation, HS = Hallucinations, SI = Self-reference Ideation, Plc = Perceptual Illusion (changing appearance), SolP = Solitary Pursuits, PlD = Perceptual Illusion (dyscontrol), TDc = Thought Disorder (Concentrate).

Subscales were chosen as indicators firstly, by excluding all subscales that had negative correlations, and secondly, by excluding all high correlations. A total of 5 of the subscales of the TPSQ-A were selected for the taxometric analysis procedures, the
correlation among these 5 subscales is shown in Table 7.3. As can be seen in Table 7.3, a high degree of correlation was observed between the Nonspiritual Magical Ideation and Self-reference Ideation subscales. It was thought that these subscales were not independent and measured the same construct. They were, therefore, combined to form one indicator. When the Nonspiritual Magical Ideation and Self-reference Ideation subscales were combined, the 5 subscales formed 4 indicators, as shown in Table 7.4.

<table>
<thead>
<tr>
<th>Table 7.3</th>
<th>Correlation Among 5 TPSQ-A Subscales Selected for Taxometric Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>TPSQ-A Subscale</td>
<td>MIns</td>
</tr>
<tr>
<td>SF</td>
<td>.13</td>
</tr>
<tr>
<td>MIns</td>
<td></td>
</tr>
<tr>
<td>HS</td>
<td></td>
</tr>
<tr>
<td>SI</td>
<td></td>
</tr>
</tbody>
</table>

Note. SF = Social Fear, MIns = Nonspiritual Magical Ideation, HS = Hallucinations, SI = Self-reference Ideation, Plc = Perceptual Illusion (changing appearance).

<table>
<thead>
<tr>
<th>Table 7.4</th>
<th>Indicator Composition for Taxometric Analysis Procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indicator</td>
<td>TPSQ-A Subscale/s</td>
</tr>
<tr>
<td>1</td>
<td>3 (SF)</td>
</tr>
<tr>
<td>2</td>
<td>4 + 6 (MIns + SI)</td>
</tr>
<tr>
<td>3</td>
<td>5 (HS)</td>
</tr>
<tr>
<td>4</td>
<td>7 (Plc)</td>
</tr>
</tbody>
</table>

Note. SF = Social Fear, MIns = Non-spiritual Magical Ideation, SI = Self-reference Ideation, HS = Hallucinations, Plc = Perceptual Illusion (changing appearance).
MAXCOV-HITMAX analyses were then conducted on the four subscales. Analyses were conducted on all possible triplets of the indicators and used slab widths of 0.33 standard deviations. The resulting covariance plot is shown in Figure 7.1.

![Figure 7.1. Median covariance for each slab or z-value (dots) and smoothed covariance curve (solid line) for taxonic indicators.](image)

Smoothed data are presented in Figure 7.1. The curve was smoothed using Tukey's method. As can be seen in Figure 7.1, the smoothed data shows a peaked distribution with the peak to the right. This is indicative of a taxonic distribution with a base rate that is less than half.
The MAXCOV-HITMAX procedure produced a median base rate of .32 and a mean base rate of .38 ($SD = .17$). The mean indicator validity or separation between indicators was 1.02 ($SD = .42$). The MAXCOV procedure also produced an estimate of the base rate from the number of people who fell above the hitmax point and this was .43. In addition, the analysis calculated the probability of each participant belonging to the taxon group. This was achieved through the application of Bayes theorem to the true and false positive rates for each of the four taxon indicators. The distribution of the probability of Bayesian taxon membership is shown in Figure 7.2. The figure forms a shape that resembles a ‘u’, which is consistent with the presence of a latent taxon. As can be seen in Figure 7.2, none of the participants were identified as having a probability between the values of .21 and .50 of being taxon members.

![Probability of Taxon Membership](image-url)

Figure 7.2. Distribution of probabilities of taxon membership.
Consistency Tests

A number of consistency tests were carried out to determine the reliability and validity of the taxonic structure suggested by the MAXCOV results. First, the variance of the estimates of the base rate produced by the MAXCOV analysis was calculated. Each indicator produced up to 3 base rate estimates. The base rate could not be calculated for 4 of the 12 combinations. The 8 base rate estimates that were produced can be seen in Table 7.5.

Table 7.5
Base Rate Estimates Produced by MAXCOV Analysis for Each Indicator Combination

<table>
<thead>
<tr>
<th>Input Indicator</th>
<th>Output Indicators</th>
<th>Base Rate Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2, 3</td>
<td>.47</td>
</tr>
<tr>
<td>1</td>
<td>3, 4</td>
<td>.30</td>
</tr>
<tr>
<td>2</td>
<td>1, 3</td>
<td>.37</td>
</tr>
<tr>
<td>2</td>
<td>3, 4</td>
<td>.34</td>
</tr>
<tr>
<td>3</td>
<td>1, 2</td>
<td>.75</td>
</tr>
<tr>
<td>3</td>
<td>2, 4</td>
<td>.21</td>
</tr>
<tr>
<td>4</td>
<td>1, 2</td>
<td>.28</td>
</tr>
<tr>
<td>4</td>
<td>1, 3</td>
<td>.28</td>
</tr>
</tbody>
</table>

Note. MAXCOV-HITMAX = Maximum Covariance Hitmax.

The variance of the 8 base rate estimates produced by MAXCOV was .17. This value is somewhat inconsistent with a taxonic structure. It is influenced by the presence of an outlier base rate estimate produced by the combination of input indicator 3 and output indicators 1 and 2. After removing the outlying base rate estimate of .75, the variance of the 7 remaining base rate estimates produced by MAXCOV was .08. The median base rate became .29 and the mean base rate became .32.
For the second consistency test, the similarity between the observed and predicted covariance matrices produced by the MAXCOV analysis was used to determine the goodness of fit of the taxonic model to the data. The GFI was .98 and this value is indicative of a high degree of similarity between the observed and predicted covariance matrices.

Finally, another taxometric procedure, MAMBAC, was used as a consistency test to evaluate the degree of agreement and reliability of the base rate estimate produced by the MAXCOV analysis. Both the base rate estimates and plots produced by this procedure were used as further tests of consistency.

MAMBAC

The MAMBAC procedure was conducted using the same 4 indicators as used in the MAXCOV analysis. The MAMBAC procedure produced a plot for each possible input-output combination of indicators with one serving as the input indicator and another as the output indicator. With four indicators, this resulted in a total of 12 plots. Each plot was obtained by graphing the mean of the output indicator score differences between individuals who fell above and below a cut made along the input indicator. These plots can be seen in Figure 7.3. The first plot for indicator 1 resembles a straight line while the second and third plots for indicator 1 resemble a peak, indicating a taxonic distribution. The plots for indicators 2, 3 and 4 all resemble a peak which is indicative of a taxonic distribution. In summary, 11 of the 12 plots are suggestive of a latent taxon.
Figure 7.3. Plots of MAMBAC output
As with MAXCOV, the MAMBAC procedure produced estimates of the base rate. A mean base rate estimate (and standard deviation) was calculated from the base rate estimates for each indicator. The mean base rate estimates for both taxometric procedures can be seen in Table 7.6 below.

Table 7.6
Base Rate Estimates for Each Taxometric Analysis Procedure

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Median Base Rate Estimate</th>
<th>Mean Base Rate Estimate</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAXCOV-HITMAX</td>
<td>.29</td>
<td>.32</td>
<td>.08</td>
</tr>
<tr>
<td>MAMBAC</td>
<td>.51</td>
<td>.56</td>
<td>.18</td>
</tr>
</tbody>
</table>

Note. MAXCOV-HITMAX = Maximum Covariance Hitmax; MAMBAC = Mean Above Minus Below A Cut; SD = Standard Deviation.

In summary, the distribution of taxon membership probabilities and the smoothed covariance curve for the MAXCOV procedure are indicative of the presence of a latent taxon. The peaked line seen in 11 of the 12 MAMBAC plots is consistent with the MAXCOV observation and also points to the presence of a taxonic distribution. The median base rates produced by the MAXCOV and MAMBAC procedures were .29 and .51 respectively. The two estimates are not consistent with each other. Possible reasons for this will be outlined in the discussion.

Discussion

The aim of Phase 1 was to investigate the latent structure of Meehl’s (1962, 1990b) schizotypy in a mixed sample of psychiatric patients. It was hypothesised that, consistent
with the trend observed in the general population, there would be evidence of a manifest taxonic structure for schizotypy in the psychiatric participants. This was determined by a taxometric analysis of 4 indicators formed from 5 subscales of the TPSQ-A. The MAXCOV-HITMAX procedure yielded a covariance curve with a peak to the right. This is consistent with a manifest taxonic structure with a low base rate. Furthermore, the figure of the distribution of the probability of Bayesian taxon membership formed a ‘u’ shape, which also suggests the presence of a manifest taxon.

A number of consistency tests were carried out to determine if there was additional support for the MAXCOV findings. These included an examination of the variance of the base rate estimates produced by each indicator combination, the GFI, and the results yielded by the MAMBAC procedure. The variance of the base rate estimates was relatively small (.08) which is consistent with a taxonic structure. In addition, the GFI was very high (.98) which is indicative of a good taxonic fit. Lastly, the MAMBAC analyses yielded 12 plots, although only 11 of these plots formed a clear peak. According to Meehl and Yonce (1994), if one plot is not clearly taxonic yet all other plots are definitely taxonic in a MAMBAC analysis, then the opposite indicator combination will often produce a taxonic plot. This was the case with the current study. The combination of input indicator = 1 and output indicator = 2 produced an ambiguous plot which was not clearly taxonic yet the combination of input indicator = 2 and output indicator = 1 produced a plot which was taxonic. Consequently, the MAMBAC plots also corroborate the finding of a manifest taxon. These consistency tests all support the taxonic structure identified by the MAXCOV analysis.

An additional consistency test produced conflicting results. The median and mean base rates produced by the MAXCOV and MAMBAC procedures are not consistent with each other. This could be due to a number of reasons. Firstly, the inconsistency could
have occurred as a result of the small sample size. This may have resulted in an unstable estimation of the base rate. Meehl (1992, 1995a) and Lenzenweger (2004) recommended that taxometric analyses not be carried out with samples smaller than 300 and the current study had a sample of 105 participants. However, research has shown that MAXCOV analyses can be conducted with samples of 200 participants and still provide accurate results. In addition, Meehl and Yonce (1996) stated that MAXCOV analyses can be conducted with samples of 100 participants if the base rate approximates .50, the indicator separation is 2 standard deviations, and there is minimal nuisance covariance. The current study had a sample size of 105 participants, and there was minimal nuisance covariance. However, the base rate and the mean indicator validity were lower than that recommended by Meehl and Yonce (1996) for a small sample size. This may have resulted in the inconsistency in the base rate estimates produced by the MAXCOV and MAMBAC procedures.

Secondly, the MAMBAC procedure has some problems associated with base rate estimates. The MAMBAC procedure has been shown to overestimate small base rates in some circumstances and this could be the case with the current study (Ruscio & Ruscio, 2004a). As a result, it is probable that the estimates yielded by the MAXCOV analysis may be most representative of the actual base rate for this sample. In addition, Ruscio and Ruscio (2004a) have found that MAMBAC analyses frequently yield consistent base rate estimates between research, simulated taxonic and simulated dimensional data. Consequently, they suggest that finding evidence of consistency between base rate estimates with a MAMBAC analysis offers weak evidence of a taxonic structure. This means that the lack of consistency between the base rate estimates yielded by the MAXCOV and MAMBAC analyses for the current study is not necessarily a problem.
Although the median base rate estimates yielded by the MAXCOV and MAMBAC procedures are not as narrow as would be preferred, these values are not unreasonable considering the sample size and problems that can occur with MAMBAC analyses. Ruscio and Ruscio (2004a) caution against relying solely on coherence between base rate estimates to draw a taxonic conclusion as sometimes nontaxonic data can produce consistent base rate estimates. This emphasises the importance of using multiple consistency tests and this has been adhered to in the current study. Furthermore, all of the other consistency tests conducted in this study provide corroborating evidence for a taxonic structure. In light of this, the evidence of the current taxometric analyses suggests a manifest taxonic structure.

The results of the current study suggest evidence for a taxonic model of schizotypy using indicators from a self-report questionnaire, the TPSQ-A, with a sample of psychiatric patients. These results are consistent with previous research that has identified the manifest structure of schizotypy as taxonic in the general population (e.g., Blanchard et al., 2000; Korfine & Lenzenweger, 1995; Lenzenweger & Korfine, 1992, 1995; Linscott, 2005). The estimated median base rate of .29 is likely to be the most representative estimate for this psychiatric patient taxon. As expected, this is higher than the base rate observed in the general population. Meehl (1989, 1990b) proposed that schizotypy has a base rate approximating .10 in the general population and this has been supported by research with non-clinical populations, mainly university students. Meehl (1990b) estimated that the base rate of schizotypy in psychiatric patients is 35% to 40%; however, this estimate is influenced by the nature of the psychiatric services that are provided and which people are considered to be patients. The base rate estimate observed in the current study with psychiatric patients is lower than that estimated by Meehl (1990b) but the value appears to be reasonable.
Meehl (1964). However, to date, there have been very few studies of schizotypy with a psychiatric population. Lenzenweger and Loranger (1989) administered the PAS to a group of nonpsychotic psychiatric patients to determine schizotypy status and then evaluated the mental health of the first-degree relatives of the patients to investigate risk for schizophrenia. They found that, compared to the relatives of the nonschizotypy group, significantly more of the first-degree relatives of people in the schizotypy group had been treated for schizophrenia. Lenzenweger and Loranger (1989) used mean scores on the PAS for determining schizotypy status and consequently, they only assessed for one of Meehl’s (1964) schizotypy characteristics. The current study employed the TPSQ-A which considers a range of Meehl’s (1964) schizotypy characteristics.

The current study has an additional strength in that schizotypy has only been previously evaluated once in a psychiatric population with taxometric analysis. Golden and Meehl (1979) applied taxometric procedures to the MMPI responses of 211 male psychiatric inpatients. They observed a base rate for schizotypy approximating .40 in their clinical sample. Many studies have used taxometric analysis to evaluate schizotypy in the general population but Golden and Meehl’s (1979) study is the only published study that has used taxometric analysis to evaluate schizotypy with a psychiatric population. Ruscio and Ruscio (2004a) suggest that with some constructs it may be more appropriate to carry out a taxometric analysis that has higher power with a clinical population than to conduct the analysis with a community sample where the size of the putative taxon is likely to be much smaller. According to Ruscio and Ruscio (2000), taxometric analyses involving moderate sized base rates that are near .50 are more powerful than taxometric analyses involving more extreme base rates that are near 0 or 1. More studies such as this with psychiatric populations are required.
Limitations

There are a number of limitations associated with the current study. One of these involves the small sample size and the potential impact this may have had on the results yielded by the analyses, as discussed above. The sample size was 105 after 4 outliers were removed. When recruitment began, a target sample size of 150 participants was set. However, there were many difficulties associated with achieving this number and these became apparent as recruitment proceeded. The major reason for this is the nature of the population used in the current study. Studies conducted with psychiatric patients typically have a low recruitment rate and this can be due to a number of reasons. The current study was reliant on health care staff at mental health services to recruit participants at their appointments. Staff members at such services are typically busy and remembering to ask their patients if they would like to take part in a study may not be a high priority. Attempts were made to overcome this problem with frequent reminders about the study at staff meetings, advertisements in waiting rooms, and movie vouchers given to staff when they recruited a participant. Despite these attempts, the number of participants recruited to the study was lower than expected. Because of the objectives of the study and the pressure of time, recruitment for Phase 1 was ceased after 11 months and a total of 109 participants were recruited.

A second limitation concerns the use of only one psychometric self-report measure to evaluate schizotypy in the current study. Some taxometric analysis research has involved multiple self-report measures that purport to assess various characteristics of schizotypy (e.g., Horan et al., 2004; Lenzenweger, 1999; Meyer & Keller, 2001). Although the TPSQ-A assesses a wider range of schizotypy characteristics than the traditional measures of schizotypy such as the PAS and MIS, the current study may have been limited by the use of only one measure. In addition, Lenzenweger (2004) advocates
for applying taxometric analysis to other types of measures, for example, biobehavioural indicators such as deficits in attention and memory, and not just self-report measures. He suggests that if researchers are limited to the use of self-report measures, then they should consider using multiple measures of different aspects of schizotypy. Future research could employ multiple measures to evaluate schizotypy in a psychiatric population.

A third potential limitation concerns the number of taxometric procedures that were used in the current study. Many studies that have investigated schizotypy in the general population have used one taxometric analysis procedure (e.g., Lenzenweger & Korfine, 1992, 1995; Meyer & Keller, 2001) while others have utilised multiple taxometric analysis procedures (e.g., Blanchard et al., 2000; Horan et al., 2004). It is recommended that at least two procedures be employed when conducting taxometric analysis research (Ruscio & Ruscio, 2004a). The present study met this requirement by using both MAXCOV and MAMBAC procedures.

During and directly after the time in which the current study and analyses were completed, the amount of published research utilising taxometric analysis increased and more detailed recommendations and guidelines were developed (e.g., Lenzenweger, 2004; Ruscio & Ruscio, 2004a). Ruscio and Ruscio (2004a, 2004b) recommend that researchers create simulated comparison data to test whether research data are suitable for taxometric analysis. In addition, the comparison data can be used for interpreting results. Furthermore, Lenzenweger (2004) has recommended that it is important for researchers to determine how taxon members are different to complement members other than belonging to two different groups. This step was not carried out in Phase 1 of the current study but in Phase 2 the neuropsychological functioning, negative symptoms, and diagnostic status of a subgroup of taxon and complement members were compared.
Summary

The results of the current study suggest the presence of a manifest taxonic structure of schizotypy in the psychiatric participant sample. In Chapter 9, the implications of these findings will be considered more thoroughly. Specifically, I will consider the degree to which the evidence, although apparently consistent with a discontinuous model of schizotypy, can be viewed as consistent with Meehl’s quasi-dimensional theoretical model. Before then, Phase 2 of the present study will investigate two questions in Chapter 8. Firstly, is schizotypy group membership associated with poorer functioning; and secondly, what is the nature of the relationship between schizotypy and schizotaxiaTsuang? This was undertaken by firstly comparing the functioning of the schizotypy and nonschizotypy groups on a range of measures. Secondly, it was determined who in the Phase 2 sample met criteria for schizotaxiaTsuang. Then, the degree of overlap between schizotypy and schizotaxiaTsuang was evaluated and the observed dependence or independence of the two constructs was established.
CHAPTER 8

The Overlap of Schizotypy and Schizotaxia:

Phase 2

The Identification of Schizotaxia in Schizotypal and Nonschizotypal Groups

Phase 1 of this study involved the application of an empirical-statistical process to the responses of a mixed sample of psychiatric patients to a self-report measure of schizotypy. The taxometric analysis procedures identified two groups of people: one group whose members most probably are schizotypal in Meehl’s (1962, 1990b) sense, and one group whose members most probably are not schizotypal. The first aim of Phase 2 was to determine if schizotypy group membership is associated with poorer functioning. This was undertaken by comparing the schizotypy and nonschizotypy groups on a range of neuropsychological and psychopathological measures. It was hypothesised that, compared to nonschizotypy group members, schizotypy group members would have poorer functioning on a range of measures. The second aim of Phase 2 was to determine the nature of the relationship between Meehl’s (1962, 1990b) schizotypy and Tsuang et al.’s (1999b, 2000a, 2000b) schizotaxia_{Tsuang}. This was undertaken by, firstly, determining who in the Phase 2 sample met Tsuang et al.’s (1999b) criteria for schizotaxia_{Tsuang}. Then, the degree of overlap between Meehl’s (1962, 1990b) schizotypy and Tsuang et al.’s (1999b) schizotaxia_{Tsuang} was evaluated and the observed dependence or independence of the two constructs was established. It was hypothesised that, as schizotypy and schizotaxia_{Tsuang}
neurological or substance abuse problems; 1 was excluded on the basis of an estimated IQ below 70; 1 was deceased; and 1 had moved out of the area.

The demographic details of the two groups can be seen in Table 8.1. The two groups were similar in regards to proportion of males and females and mean age was not significantly different between the groups. The nonschizotypy group had a significantly greater mean number of years of education compared to the schizotypy group, t(27) = 2.76, *p* = .005. The Otago Ethics Committee reviewed and granted ethical approval for this study.

<table>
<thead>
<tr>
<th>Table 8.1</th>
<th>Demographic Characteristics of the Schizotypy and Nonschizotypy Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>Schizotypy M (SD)</td>
</tr>
<tr>
<td>N</td>
<td>14</td>
</tr>
<tr>
<td>Females</td>
<td>6</td>
</tr>
<tr>
<td>Males</td>
<td>8</td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>42.5 (8.6)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>12.9 (1.9)</td>
</tr>
</tbody>
</table>

**Measures and Materials**

**Estimate of IQ**

The Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) was used as a measure of IQ. The Vocabulary and Matrix Reasoning subtests from this measure were used for a 2-subtest estimate. For two of the participants, the Vocabulary and Block Design subtests from the Wechsler Adult Intelligence Scale, Third edition (WAIS-III; Wechsler, 1997a) were used to estimate IQ. Axelrod (2002) compared performance on the WASI to performance on the WAIS-III in a mixed sample of neuropsychological and
psychiatric participants. He found that the WASI IQ scores were not consistently accurate in predicting WAIS-III scores as the WASI scores tended to both over and underestimate WAIS-III scores. Axelrod (2002) cautioned against using the WASI when accurate evaluations of an individual's abilities are required. Indeed, the developers of the WASI emphasise that the WASI should only be used as a screening tool and that other measures should be used if a more accurate evaluation is required (Wechsler, 1999). The WASI was used in the current study to obtain an estimate of IQ to ensure that all of the participants had a level of intellectual functioning above the cut-off for mental retardation.

Attention

Two measures were used to assess attention: a simplified version of the Visual Continuous Performance Test, Identical Pairs version (VCPT-IP; Cornblatt et al., 1988) and the Paced Auditory Serial Addition Task (PASAT; Gronwall, 1977).  

The VCPT-IP was a simplified form of that described by Cornblatt et al. (1988). The test was administered using an iMac computer with stimuli presented on a screen measuring 22cm x 29cm. Stimuli consisted of 4-digit numbers (e.g., 6 4 3 2). Each stimulus was 7.5cm wide and 2cm high and when viewed at a distance of 60cm, subtended a visual angle of 2.2° x 1.4°. Each stimulus had an onset asynchrony of 1000msec and was presented on the screen for 50msec. There were three types of stimuli: (a) target stimuli that were identical to their immediately preceding stimuli; (b) catch stimuli that shared three digits in common with their immediately preceding stimuli; and (c) filler stimuli that did not have any digits in common with their immediately preceding stimuli. There were a total of 160 trials made up of 32 target trials, 32 catch trials, and 96 filler trials.

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1 It had been intended to use the Auditory Continuous Performance Test with Interference (ACPT-INT; Seidman et al., 1998) as Tsuang et al. (1999) had used in their study, however, undertakings to provide the stimuli and details of this test were not fulfilled by the authors of the test.
trials. Participants were instructed to press the space bar of the computer keyboard as quickly as they could when 2 identical stimuli were presented in a row on the computer screen. These responses were recorded by the computer. Comblatt et al. (1988) found that their version of the VCPT-IP had good test-retest reliability when they established normative data with a group of 120 adults and adolescents. In addition, on this version of the task, people with schizophrenia had impaired performance relative to depressed people and normal controls (Comblatt et al., 1989). A meta-analysis of studies using the VCPT with schizophrenia patients found an association between negative symptoms of schizophrenia and poor performance on the VCPT (Nieuwenstein, Aleman, & de Haan, 2001). Furthermore, Linscott (2005) has found that the VCPT is sensitive to the schizotypy versus nonschizotypy classification in undergraduate students.

The PASAT (Gronwall, 1977) was administered using an audiotaped presentation of 61 digits (numbers 1 to 9). The digits were presented at four different speeds, beginning with 2.4 sec between each digit, 2.0 sec, 1.6 sec, and lastly 1.2 sec between each digit. The same sequence of 61 digits was used for each of the four trials. The four trials were preceded by extensive instructions, a non-paced practice, and a paced practice of the task. Participants were instructed to add the first digit that they heard to the second digit that they heard and report the sum out loud. The participant was then required to remember the second digit and add it to the third digit that they heard and report the sum out loud, and so on. A scoring sheet was used to record verbal responses to each digit presentation. The PASAT has been shown to have good construct validity as a measure of attention and speed of information processing (Larrabee & Curtiss, 1995). Scores on the PASAT are affected by age and IQ and these need to be taken into account when evaluating PASAT performance (Brittain et al., 1991; Wiens, Fuller, & Crossen, 1997).
**Verbal Memory**

Verbal memory was assessed using the Logical Memory subtest from the Wechsler Memory Scale, Third Edition (WMS-III; Wechsler, 1997b), and Buschke’s Selective Reminding Test (SRT; Buschke, 1973). A digital audio recorder was used to record participants’ immediate and delayed recall responses to the Logical Memory subtest. Participants’ responses were also manually recorded on a scoring booklet. Toulopoulou et al. (2003) administered 8 memory measures, including the Logical Memory subtest from the first edition of the WMS (Wechsler, 1945) to a group of 62 schizophrenia patients, 98 of their relatives, and 66 controls. Participants with schizophrenia and their relatives performed significantly worse than controls on the Logical Memory subtest. Their impairment on the Logical Memory subtest was larger than for any of the other memory measures (Toulopoulou et al., 2003).

The SRT consists of a 12-word list of unrelated words that was read out loud to participants. After each trial, participants were only told the words that they had been unable to recall and then asked to recall the total list again, up to a maximum of 12 trials. A cued-recall task, a multiple-choice recognition task, and a delayed recall task were also administered as part of the SRT. A scoring sheet was used to record participants’ verbal immediate and delayed recall responses. The SRT has good construct validity with a group of mixed outpatients (Larrabee & Curtiss, 1995). People with schizophrenia have been shown to have impaired performance on the SRT compared to normal controls (Goldberg, Weinberger, Pliskin, Berman, & Podd, 1989).

**Executive Functions**

Tsuang et al. (1999b) used the Delayed Alternation Test (DAT; Seidman et al., 1995) and the Object Alternation Test (OAT; Seidman et al., 1995) to assess executive
functions. Seidman et al. (1995) administered the Wisconsin Card Sorting Test (WCST; Grant & Berg, 1948; Heaton et al., 1993), the DAT, and the OAT to a group of 18 schizophrenic patients and 14 controls. The patients demonstrated a significant impairment on all three measures when compared to controls. Seidman et al. (1995) also observed a significant correlation of .69 between the DAT error scores and WCST perseveration scores and a significant correlation of .59 between the DAT and WCST perseveration scores for the schizophrenia patients. Faraone et al. (2000) have suggested that the WCST should not be used when assessing subtle deficits in neuropsychological functioning. They found that the scores on the WCST were not stable over time (four years) in a sample of relatives of people who had schizophrenia. Faraone et al. (2000) concluded that the WCST is too easy. It is possible that Faraone et al. (2000) observed instability in their results because the WCST is recommended for use as a one-off test. Once a person has learnt the rule, re-testing does not usually serve a purpose unless the person has experienced neurological impairment (e.g., a head injury).

The evidence for and against using the WCST to assess neuropsychological impairment in relatives of individuals with schizophrenia is mixed. Faraone et al. (2000) observed an impairment in WCST scores for relatives of people who had schizophrenia; however, Laurent et al. (2001) found no difference between relatives and normal controls. With further analysis, Laurent et al. (2001) observed a significant difference when a subgroup of relatives with high scores for negative features of schizotypy was compared to relatives with low scores. The high scoring group had impaired performance relative to the low scoring group.

For practical reasons, such as test accessibility, and based on Seidman et al.'s (1995) evidence, the WCST (Grant & Berg, 1948; Heaton et al., 1993) was substituted for the DAT and OAT and used to assess executive functioning in the current study. Responses
were recorded using a modified version of the scoring sheet, as suggested by Lezak (1995).

Clinical Measures

Several modules of the Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2001b) were used to assess for psychopathology. The modules included those for mood episodes, psychotic and associated symptoms, psychotic disorders, mood disorders, substance use disorders, anxiety disorders, and eating disorders as well as the overview. There are very few studies that have examined the reliability and validity of the SCID-I for DSM-IV-TR axis I disorders. Zanarini et al. (2000) observed kappa values ranging from .57 to 1 for pairs of SCID-I raters and kappa values ranging from .35 to .78 for a test-retest interval of 7-10 days. Despite the wide range of kappa values, Zanarini et al. (2000) advocated the use of the SCID-I in research to obtain reliable diagnoses.

The Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1984) was used to assess for the presence of negative symptoms. This is a 25-item experimenter-rated scale and each item is rated on a 0-5 scale (not at all, questionable, mild, moderate, marked, severe). The SANS has 5 subscales: affective flattening or blunting; alogia; avolition-apathy; anhedonia-asociality; and attention. Schuldberg, Quinlan, Morgenstern, and Glazer (1990) evaluated the SANS with a mixed sample of 339 psychiatric outpatients. They found that the SANS had good interrater reliability and internal consistency, and moderate test-retest reliability over a period of 2 years. Andreasen (1989) has reported good reliability of the 5 subscale scores and high intercorrelations between the individual items.
Rater Training

The SCID-I and the SANS were administered by a postgraduate student of clinical psychology who had received instruction and training in psychometric test administration, scoring, and interpretation. In addition, specific training in the administration of the SCID-I and the SANS was undertaken. Training for the SCID-I involved completing the SCID-I video training course (First, Gibbon, Spitzer, & Williams, 2001a). This involved viewing 11 hours of detailed training on the SCID-I modules and as well as practice with videotaped interviews with patients. Nine written cases and six role plays contained in the SCID-I user's guide (First et al., 2001a) were also completed. The level of agreement between the experimenter's ratings and the ratings provided in the training package was evaluated by comparing the final diagnoses that were produced. The percentage of occurrence agreement was 94.1%.

Training for the SANS involved watching a videotape of three case vignettes. Ratings of these vignettes were completed and compared to those provided with the training package (Andreasen, 1984). The level of agreement between the experimenter's ratings and the ratings provided with the videotapes was evaluated by comparing the rating for each item for the 25 items. The percentage of occurrence agreement was 80%.

Procedure

Phase 2 was typically carried out over two to three sessions with each participant. Additional appointments were made as appropriate in order to minimise fatigue in participants. There was a maximum of 3 weeks between each session. Sessions ranged from 1½ hours to 2½ hours and included breaks as necessary. Participants were reimbursed $25 per session. The researcher was blind to group membership until all participants had completed participation and all ratings had been made.
Once the nature and purpose of the study had been described to participants, informed consent was obtained. Participants completed the WMS-III and WASI (or WAIS-III) subtests, the VCPT-IP, the PASAT, mental status tests of the SANS, the SRT, and the WCST. Tests were administered in this order for all participants. All tests were administered using procedures specified in their respective administration manuals.

The second session typically began by reviewing what would be involved in the session and ensuring that the participant was happy for the session to be audiotaped. The SCID-I (First et al., 2001b) interview was then initiated. Administration of the SCID-I took between 1½ and 2½ hours. Participants were offered breaks during the session.

The Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1984) was completed after the participant had completed all assessments involving their participation. Ratings for this were based on the information obtained and observations made during the sessions.

**Data Analysis**

The dependent measures of Phase 2 are shown in Table 8.2. The kurtosis and skewness of the two groups on each of the dependent measures were calculated to examine the distribution of the scores, except for the SCID-I diagnoses. Parametric and non-parametric tests were used to investigate differences between the schizotypy and nonschizotypy groups on scores of the dependent measures, except for the SCID-I diagnoses. Independent t-tests were also used to investigate differences between the groups and the normative samples on scores of the neuropsychological tests and of the SANS. Cohen's measure of effect size, $d$, was calculated for each parametric and non-parametric test. The sequentially rejective Bonferroni test (Holm, 1979) was used to correct for type I error that may have arisen due to the use of multiple tests.
Table 8.2
*Dependent Measures used in Phase 2*

<table>
<thead>
<tr>
<th>Domain</th>
<th>Test</th>
<th>Test Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>IQ</td>
<td>Wechsler Abbreviated Scale of Intelligence</td>
<td>IQ estimate</td>
</tr>
<tr>
<td>Global Functioning</td>
<td>Structured Clinical Interview for DSM-IV-TR Axis I Disorders</td>
<td>Global Assessment of Functioning</td>
</tr>
<tr>
<td>Psychopathology</td>
<td>Structured Clinical Interview for DSM-IV-TR Axis I Disorders</td>
<td>Diagnosis code</td>
</tr>
<tr>
<td>Attention</td>
<td>Visual Continuous Performance Test, Identical Pairs</td>
<td>Catches/false alarms</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Random errors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Commissions/total errors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Discriminability (d')</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time interval score</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total score</td>
</tr>
<tr>
<td></td>
<td>Paced Auditory Serial Addition Task</td>
<td></td>
</tr>
<tr>
<td>Verbal Memory</td>
<td>Logical Memory Subtest</td>
<td>Logical Memory I total score</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Logical Memory II total score</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Logical Memory II percent retention</td>
</tr>
<tr>
<td></td>
<td>Selective Reminding Test</td>
<td>Total recall</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Long term storage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Short term retrieval</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Continuous long term retrieval</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Random long term retrieval</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reminders</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cued recall</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Multi-choice</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30 minute delayed recall</td>
</tr>
<tr>
<td>Executive Functions</td>
<td>Wisconsin Card Sorting Test</td>
<td>Percent total errors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Percent perseverative errors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Percent nonperseverative errors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Number of categories completed</td>
</tr>
<tr>
<td>Negative Symptoms</td>
<td>Scale for the Assessment of Negative Symptoms</td>
<td>Total sum</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Number of items scored as 3 or more</td>
</tr>
</tbody>
</table>
Individual scores on the dependent measures were compared to normative samples for selected tests. This was only carried out for the test score criteria for schizotaxia\textsubscript{Tsuang} proposed by Tsuang et al. (1999b). These comparisons were conducted to determine whether individuals met Tsuang et al.'s (1999b) criteria for an impairment. An impairment was defined as a score equal to or greater than either 1 or 2 standard deviations below the mean of the normative samples. This replicated Tsuang and colleagues' definitions.

A 2 x 2 chi-square analysis was conducted to investigate whether the relationship between classification of schizotypy and schizotaxia\textsubscript{Tsuang} was dependent or independent. This was done by evaluating whether the observed counts for each schizotaxia\textsubscript{Tsuang} and schizotypy combination were significantly different from the expected counts. Logistic regression analyses were carried out to determine the ability of Tsuang et al.'s (1999b) criteria for schizotaxia\textsubscript{Tsuang} to predict who would have Meehl's (1962, 1990b) schizotypy. The aim of this was to ascertain if the raw scores or the impairment classifications on the neuropsychological tests and the negative symptom scores could identify people with schizotypy or characteristics of schizotypy.

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2 The test scores used to assess Tsuang et al.'s (1999b) criteria for schizotaxia form a subset of the dependent measures shown in Table 8.2. These tests scores included the hit rate and $d'$ of the Visual Continuous Performance Test, Identical Pairs; time interval score of the Paced Auditory Serial Addition Task; Logical Memory II total score and percent retention of the Logical Memory subtest; total recall and random long term retrieval scores of the Selective Reminding Test; percent total errors and percent perseverative errors scores of the Wisconsin Card Sorting Test; and the number of items scored as 3 or more on the Scale for the Assessment of Negative Symptoms.
Results

Comparisons between Schizotypy and Nonschizotypy Groups

Responses and data were complete for all 29 participants, apart from scores on the PASAT. During the administration of Phase 2, it became apparent that many of the participants were experiencing difficulty completing the PASAT. Nearly half of the participants were unable to master the practice tasks and consequently did not meet practice criteria for the task to be administered. As PASAT performance data were not available for approximately half of the participants, the PASAT scores were excluded from data analysis. One participant from the schizotypy group did not complete the VCPT-IP and their data were excluded from analyses involving the VCPT.

The distributions of the dependent measures were examined by calculating the kurtosis and skewness. Normality was rejected if the kurtosis or skewness values were significant for a particular dependent measure. The kurtosis, skewness and associated $p$-values for the schizotypy and nonschizotypy groups can be seen in Tables 7.3 and 7.4. The sequentially rejective Bonferroni test (Holm, 1979) was applied to correct for type I error generated through the use of multiple tests. As can be seen in Table 8.3, normality was rejected for the schizotypy group for the VCPT random errors score, VCPT total errors score, SRT multi-choice score, and the WCST percent of nonperseverative errors score after applying the sequentially rejective Bonferroni test. As can be seen in Table 8.4, normality was rejected for the nonschizotypy group for IQ, VCPT random errors score, VCPT total errors score, LMII percent retention score, SRT short term storage score, SRT multi-choice score, and the WCST percent of nonperseverative errors score after applying the sequentially rejective Bonferroni test.
Table 8.3
Skewness and Kurtosis of Distribution of Schizotypy Group for Each Dependent Measure

<table>
<thead>
<tr>
<th>Domain</th>
<th>Test Score</th>
<th>Kurtosis</th>
<th>p-value</th>
<th>Skewness</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IQ</td>
<td>IQ</td>
<td>-.04</td>
<td>.975</td>
<td>.49</td>
<td>.411</td>
</tr>
<tr>
<td>Global Functioning</td>
<td>GAF</td>
<td>-.71</td>
<td>.550</td>
<td>.23</td>
<td>.698</td>
</tr>
<tr>
<td>Attention</td>
<td>VCPT targets/hit rate</td>
<td>-1.61</td>
<td>.201</td>
<td>-.24</td>
<td>.698</td>
</tr>
<tr>
<td></td>
<td>VCPT mean reaction time</td>
<td>1.62</td>
<td>.198</td>
<td>-1.23</td>
<td>.046</td>
</tr>
<tr>
<td></td>
<td>VCPT catches/false alarms</td>
<td>-.84</td>
<td>.496</td>
<td>.93</td>
<td>.133</td>
</tr>
<tr>
<td></td>
<td>VCPT random errors</td>
<td>10.45</td>
<td>.000</td>
<td>3.14</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>VCPT commissions/total errors</td>
<td>5.21</td>
<td>.001</td>
<td>2.17</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>VCPT d'</td>
<td>.32</td>
<td>.790</td>
<td>.26</td>
<td>.676</td>
</tr>
<tr>
<td>Verbal Memory</td>
<td>LMI total score</td>
<td>.36</td>
<td>.759</td>
<td>.09</td>
<td>.878</td>
</tr>
<tr>
<td></td>
<td>LMII total score</td>
<td>.49</td>
<td>.678</td>
<td>.37</td>
<td>.532</td>
</tr>
<tr>
<td></td>
<td>LMII percent retention</td>
<td>-.89</td>
<td>.452</td>
<td>-.43</td>
<td>.477</td>
</tr>
<tr>
<td></td>
<td>SRT total recall</td>
<td>-.31</td>
<td>.796</td>
<td>-.09</td>
<td>.886</td>
</tr>
<tr>
<td></td>
<td>SRT long term storage</td>
<td>.47</td>
<td>.694</td>
<td>-.45</td>
<td>.456</td>
</tr>
<tr>
<td></td>
<td>SRT short term retrieval</td>
<td>-.31</td>
<td>.792</td>
<td>.32</td>
<td>.598</td>
</tr>
<tr>
<td></td>
<td>SRT CLTR</td>
<td>-1.15</td>
<td>.338</td>
<td>.53</td>
<td>.377</td>
</tr>
<tr>
<td></td>
<td>SRT RLTR</td>
<td>-1.15</td>
<td>.336</td>
<td>.27</td>
<td>.657</td>
</tr>
<tr>
<td></td>
<td>SRT reminders</td>
<td>-.36</td>
<td>.762</td>
<td>.03</td>
<td>.960</td>
</tr>
<tr>
<td></td>
<td>SRT cued recall</td>
<td>-.68</td>
<td>.568</td>
<td>-.42</td>
<td>.483</td>
</tr>
<tr>
<td></td>
<td>SRT multi-choice</td>
<td>4.12</td>
<td>.003</td>
<td>-2.20</td>
<td>.000</td>
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<tr>
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<td>SRT 30 minute delayed recall</td>
<td>.01</td>
<td>.995</td>
<td>-.42</td>
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<tr>
<td>Executive Functions</td>
<td>WCST % total errors</td>
<td>-1.02</td>
<td>.392</td>
<td>-.17</td>
<td>.780</td>
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<td>WCST % perseverative errors</td>
<td>-1.24</td>
<td>.304</td>
<td>.05</td>
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<td>WCST % nonperseverative errors</td>
<td>2.39</td>
<td>.059</td>
<td>1.52</td>
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<td>WCST categories completed</td>
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<td>Negative Symptoms</td>
<td>SANS total sum</td>
<td>.89</td>
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<td>.291</td>
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<td>SANS n of items scored as ≥ 3</td>
<td>-1.00</td>
<td>.107</td>
<td>-.30</td>
<td>.620</td>
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</tbody>
</table>

Note. GAF = Global Assessment of Functioning; VCPT = Visual Continuous Performance Test, Identical Pairs; LMI/II = Logical Memory I and II; SRT = Selective Reminding Test; CLTR = continuous long term retrieval; RLTR = random long term retrieval; WCST = Wisconsin Card Sorting Test; SANS = Scale for the Assessment of Negative Symptoms.
<table>
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<th>p-value</th>
<th>Skewness</th>
<th>p-value</th>
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<tr>
<td>Attention</td>
<td>VCPT targets/hit rate</td>
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<td>.681</td>
<td>-.80</td>
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<td>VCPT mean reaction time</td>
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<td>.253</td>
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<td>.462</td>
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<td>VCPT catches/false alarms</td>
<td>-.18</td>
<td>.877</td>
<td>.99</td>
<td>.088</td>
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<td>VCPT random errors</td>
<td>12.54</td>
<td>.000</td>
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<td>VCPT commissions/total errors</td>
<td>10.16</td>
<td>.000</td>
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<td>.000</td>
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<tr>
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<td>VCPT d'</td>
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<td>.763</td>
<td>-.14</td>
<td>.807</td>
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<td>Verbal Memory</td>
<td>LMI total score</td>
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<td>.01</td>
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<td>LMII total score</td>
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<td>-.11</td>
<td>.850</td>
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<td>LMII percent retention</td>
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<td>.022</td>
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<td>SRT long term storage</td>
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<td>SRT short term retrieval</td>
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<td>SRT CLTR</td>
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<td>.763</td>
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<td>SRT RLTR</td>
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<td>.780</td>
<td>.97</td>
<td>.096</td>
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<td>SRT reminders</td>
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<td>.229</td>
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<td>.037</td>
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<td>SRT multi-choice</td>
<td>4.79</td>
<td>.000</td>
<td>-2.27</td>
<td>.000</td>
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<td>SRT 30 minute delayed recall</td>
<td>-1.12</td>
<td>.334</td>
<td>-.46</td>
<td>.427</td>
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<td>Executive Functions</td>
<td>WCST % total errors</td>
<td>-.94</td>
<td>.416</td>
<td>.75</td>
<td>.196</td>
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<td>WCST % perseverative errors</td>
<td>-.72</td>
<td>.530</td>
<td>.75</td>
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<td>WCST % nonperseverative errors</td>
<td>2.43</td>
<td>.048</td>
<td>1.53</td>
<td>.009</td>
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<td>WCST categories completed</td>
<td>-.47</td>
<td>.683</td>
<td>-1.03</td>
<td>.075</td>
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<td>Negative Symptoms</td>
<td>SANS total sum</td>
<td>-1.77</td>
<td>.138</td>
<td>.28</td>
<td>.628</td>
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<td>SANS n of items scored as ≥ 3</td>
<td>-1.07</td>
<td>.356</td>
<td>.73</td>
<td>.210</td>
</tr>
</tbody>
</table>

Note. GAF = Global Assessment of Functioning; VCPT = Visual Continuous Performance Test, Identical Pairs; LMI/II = Logical Memory I and II; SRT = Selective Reminding Test; CLTR = continuous long term retrieval; RLTR = random long term retrieval; WCST = Wisconsin Card Sorting Test; SANS = Scale for the Assessment of Negative Symptoms.
Independent *t*-test analyses were conducted to determine if there were significant differences between the schizotypy and nonschizotypy groups. A parametric independent samples one-sided *t*-test was used for dependent measures that were normally distributed. A nonparametric independent samples equivalent, the Mann-Whitney test, was used for dependent measures that were not normally distributed. The sequentially rejective Bonferroni test (Holm, 1979) was applied to correct for type I error generated through the use of multiple tests. This correction was made for tests, both parametric and nonparametric, within the 6 domains. Cohen’s measure of effect size, $d$, was also calculated by subtracting the mean of the nonschizotypy group from the mean of the schizotypy group, and then dividing by the standard deviation of the nonschizotypy group.

Table 8.5 shows the means, standard deviations, observed $p$-values, and $d$, for the schizotypy and nonschizotypy groups for each dependent measure. Compared to the nonschizotypy group, the schizotypy group had significantly lower mean scores for the global functioning score ($t(27) = 2.36$, $p = .01$), LMI ($t(27) = 2.94$, $p = .00$) and LMII total scores ($t(27) = 3.96$, $p = .00$), and SRT long term storage score ($t(27) = 2.13$, $p = .02$). Compared to the nonschizotypy group, the schizotypy group had a significantly lower median score for estimate of IQ ($U(15, 14) = 54.00$, $p = .01$) and LMII percent retention score ($U(15,14) = 58.50$, $p = .02$). The schizotypy group had a significantly higher median score than the nonschizotypy group for the VCPT random errors score ($U(15,13) = 55.00$, $p = .03$) and WCST percent of nonperseverative errors score ($U(15,14) = 49.50$, $p = .01$). Levene’s test for equality of variances showed that equal variances were not observed on *t*-tests for the VCPT targets score ($p = .02$) and negative symptoms total score ($p = .00$).
### Table 8.5

*Parametric and Nonparametric T-tests of Differences on Test Scores between the Schizotypy and Nonschizotypy Groups*

<table>
<thead>
<tr>
<th>Domain</th>
<th>Test Score</th>
<th>Schizotypy</th>
<th>Nonschizotypy</th>
<th>p-value</th>
<th>d</th>
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<td></td>
<td>M/Mdn SD/R</td>
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<td></td>
<td></td>
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<tr>
<td></td>
<td><strong>IQ</strong></td>
<td>104/55</td>
<td>115/36</td>
<td>.013</td>
<td>-.95</td>
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<tr>
<td>Global Functioning</td>
<td>GAF</td>
<td>57.9/9.21</td>
<td>67.1/11.54</td>
<td>.013</td>
<td>-.80</td>
</tr>
<tr>
<td>Attention</td>
<td>VCPT targets/hit rate</td>
<td>18.5/9.30</td>
<td>23.7/6.29</td>
<td>.054</td>
<td>-.82</td>
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<tr>
<td></td>
<td>VCPT mean reaction time (ms)</td>
<td>530/88.86</td>
<td>562/88.31</td>
<td>.170</td>
<td>-.37</td>
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<tr>
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<td>VCPT catches/false alarms</td>
<td>5.2/5.60</td>
<td>5.0/4.52</td>
<td>.468</td>
<td>.03</td>
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<td></td>
<td>VCPT random errors</td>
<td>2.0/3.40</td>
<td>0.0/3.90</td>
<td>.026</td>
<td>.14</td>
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<td>VCPT commissions/total errors</td>
<td>5.0/47.0</td>
<td>4.0/53.0</td>
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<td>.12</td>
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<td>VCPT d'</td>
<td>1.62/1.13</td>
<td>2.13/1.19</td>
<td>.132</td>
<td>-.43</td>
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<td><strong>Verbal Memory</strong></td>
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<td>LMI total score</td>
<td>26.1/9.44</td>
<td>37.3/10.93</td>
<td>.004</td>
<td>-1.02</td>
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<td>LMII total score</td>
<td>13.8/6.66</td>
<td>24.5/7.77</td>
<td>.000</td>
<td>-1.37</td>
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<td>LMII % retention</td>
<td>77.5/69.0</td>
<td>89.0/61.0</td>
<td>.021</td>
<td>-1.06</td>
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<td>SRT total recall</td>
<td>100.4/23.38</td>
<td>114.7/16.66</td>
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<td>.86</td>
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<td>SRT long term storage</td>
<td>88.4/31.80</td>
<td>110.1/22.38</td>
<td>.021</td>
<td>-.97</td>
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<tr>
<td></td>
<td>SRT short term retrieval</td>
<td>20.5/45.0</td>
<td>10.0/38.0</td>
<td>.026</td>
<td>.83</td>
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<td>SRT CLTR</td>
<td>60.3/45.33</td>
<td>86.0/37.98</td>
<td>.054</td>
<td>-.68</td>
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<td>SRT RLTR</td>
<td>20.1/15.66</td>
<td>17.1/17.12</td>
<td>.313</td>
<td>.18</td>
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<td>SRT reminders</td>
<td>53.2/21.50</td>
<td>40.1/15.57</td>
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<td>.84</td>
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<td>SRT cued recall</td>
<td>8.5/2.03</td>
<td>9.1/2.03</td>
<td>.204</td>
<td>-.31</td>
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<td>SRT multi-choice</td>
<td>12.0/3.0</td>
<td>12.0/2.0</td>
<td>.440</td>
<td>-.27</td>
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<tr>
<td></td>
<td>SRT 30 minute delayed recall</td>
<td>7.7/3.58</td>
<td>9.2/2.37</td>
<td>.098</td>
<td>-.63</td>
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<tr>
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<td><strong>Executive Functions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WCST % total errors</td>
<td>39.9/17.45</td>
<td>28.7/17.67</td>
<td>.050</td>
<td>.63</td>
</tr>
<tr>
<td></td>
<td>WCST % perseverative errors</td>
<td>19.7/11.68</td>
<td>19.3/12.74</td>
<td>.467</td>
<td>.03</td>
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<td>WCST % nonperseverative errors</td>
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<td>7.0/24.0</td>
<td>.007</td>
<td>1.70</td>
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<td>WCST categories completed</td>
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<td>4.67/1.80</td>
<td>.093</td>
<td>-.57</td>
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<td><strong>Negative Symptoms</strong></td>
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<td></td>
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<tr>
<td></td>
<td>SANS total sum</td>
<td>24.6/10.09</td>
<td>19.9/15.98</td>
<td>.175</td>
<td>.29</td>
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<td>SANS n of items scored as ≥ 3</td>
<td>3.93/3.32</td>
<td>2.87/3.52</td>
<td>.206</td>
<td>.30</td>
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</table>

*Note.* *a* = Nonparametric t-test used; GAF = Global Assessment of Functioning; VCPT = Visual Continuous Performance Test, Identical Pairs; LMI/II = Logical Memory I and II; SRT = Selective Reminding Test; CLTR = continuous long term retrieval; RLTR = random long term retrieval; WCST = Wisconsin Card Sorting Test; SANS = Scale for the Assessment of Negative Symptoms; *M* = Mean; *Mdn* = Median; *SD* = Standard Deviation; *R* = Range; *d* = Cohen's measure of effect size.
Comparisons were made between the means of both groups and the means of the normative samples for each neuropsychological test score and total negative symptom score. For the VCPT-IP, the schizotypy and nonschizotypy groups' scores were compared to a normative sample of 283 undergraduate university students. For the Logical Memory scores, raw scores were transformed into scaled scores. The schizotypy and nonschizotypy groups were then contrasted with reference group norms provided in the WMS-III manual. The reference group (n = 200) was aged 20 to 34 years, and had a mean scaled score of 10 (SD = 3). The SRT raw scores required a numerical adjustment based on gender. This was based on normative data produced in a study by Larrabee, Trahan, Curtiss, and Levin (1988). Adjustments were only made to the scores of male participants. The WCST scores were compared to the scores of a normative group (n = 384) who were census-matched to represent the population of the United States. These norms were taken from the WCST manual.

For the total score on the SANS, the schizotypy and nonschizotypy groups were compared to mental health centre outpatient norms (n = 393) provided by Schuldberg et al. (1990). The ratings for each individual for 3 of the SANS items were omitted so that comparisons could be made to normative data. These included ratings for the physical anergia, sexual interest/activity, and work inattentiveness items. Normative data were not available for the number of items rated as 3 or higher on the SANS. In addition, normative data from a non-psychiatric population was not available for the SANS. Of the normative groups mentioned above, only the SANS normative data were from a clinical population. The parametric independent samples one-sided t-test and nonparametric one-sample sign test were used, as appropriate, to determine if differences between the schizotypy and normative groups and between the nonschizotypy and normative groups
were significant. The sequentially rejective Bonferroni test (Holm, 1979) was applied to correct for type I error generated through the use of multiple tests. Cohen’s measure of effect size, \( d \), was calculated by subtracting the mean of the normative group from the mean of the experimental group and then dividing by the standard deviation of the normative group.

Table 8.6 shows the means, standard deviations, \( p \)-value, and \( d \) for the schizotypy and normative groups for each dependent measure. In comparison to the normative group, the schizotypy group had a significantly lower mean score for the VCPT hit rate score \((t(12) = -3.61, p = .00)\), VCPT d’ score \((t(12) = -3.45, p = .00)\), LMI total scaled score \((t(13) = -5.39, p = .00)\), LMII total scaled score \((t(13) = -5.05, p = .00)\), SRT total recall score \((t(13) = -2.28, p = .02)\), SRT long term storage score \((t(13) = -2.27, p = .02)\), and WCST number of categories completed \((t(13) = -2.39, p = .02)\). The schizotypy group had a significantly higher mean score than the normative group for the VCPT mean reaction time score \((t(12) = 1.91, p = .04)\), SRT reminders score \((t(13) = 4.32, p = .00)\), WCST percent total errors score \((t(13) = 3.33, p = .00)\), WCST percent perseverative errors score \((t(13) = 2.09, p = .03)\), and SANS total sum \((t(13) = -1.84, p = .04)\).

Table 8.7 shows the means, standard deviations, \( p \)-value, and \( d \) for the nonschizotypy and normative groups for each dependent measure. The nonschizotypy group had a significantly lower mean score than the normative group for the VCPT hit rate \((t(14) = -2.58, p = .01)\), LMII percent retention score \((S(13) = 11, p = .01)\), SRT multiple choice score \((S(15) = 12, p = .01)\), and SANS total sum \((t(14) = -2.20, p = .02)\). The nonschizotypy group had a significantly higher mean score than the normative group for the VCPT mean reaction time score \((t(14) = 3.48, p = .00)\), SRT reminders score \((t(14) = 2.91, p = .01)\), and the WCST percent perseverative errors score \((t(14) = 1.87, p = .04)\).
<table>
<thead>
<tr>
<th>Domain</th>
<th>Test Score</th>
<th>Schizotypy</th>
<th>Normative</th>
<th>p-value</th>
<th>d</th>
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<td></td>
<td>M</td>
<td>SD</td>
<td></td>
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<td>VCPT targets/hit rate</td>
<td>18.5</td>
<td>9.30</td>
<td>27.9</td>
<td>4.28</td>
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<td>VCPT mean reaction time (ms)</td>
<td>530</td>
<td>88.86</td>
<td>483</td>
<td>61.19</td>
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<tr>
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<td>VCPT catches/false alarms</td>
<td>5.2</td>
<td>5.60</td>
<td>4.6</td>
<td>4.02</td>
</tr>
<tr>
<td></td>
<td>VCPT random errors</td>
<td>4.8</td>
<td>8.79</td>
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<tr>
<td></td>
<td>VCPT commissions/total errors *</td>
<td>9.6</td>
<td>12.73</td>
<td>6.5</td>
<td>9.43</td>
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<tr>
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<td>VCPT d</td>
<td>1.62</td>
<td>1.13</td>
<td>2.70</td>
<td>1.19</td>
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<td>Verbal Memory</td>
<td>LMII % retention scaled score</td>
<td>8.4</td>
<td>3.96</td>
<td>10.0</td>
<td>3.00</td>
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<tr>
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<td>LMI total scaled score</td>
<td>5.9</td>
<td>2.88</td>
<td>10.0</td>
<td>3.00</td>
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<td>LMII total scaled score</td>
<td>6.5</td>
<td>2.59</td>
<td>10.0</td>
<td>3.00</td>
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<tr>
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<td>SRT total recall</td>
<td>102.6</td>
<td>23.28</td>
<td>116.8</td>
<td>13.56</td>
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<tr>
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<td>SRT long term storage</td>
<td>91.4</td>
<td>31.74</td>
<td>110.7</td>
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<tr>
<td></td>
<td>SRT short term retrieval</td>
<td>18.3</td>
<td>12.77</td>
<td>11.4</td>
<td>7.87</td>
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<tr>
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<td>SRT CLTR</td>
<td>65.9</td>
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<td>SRT RLTR</td>
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<td>14.89</td>
<td>14.0</td>
<td>10.95</td>
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<td>21.41</td>
<td>26.3</td>
<td>12.71</td>
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<tr>
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<td>SRT cued recall</td>
<td>8.5</td>
<td>2.03</td>
<td>8.9</td>
<td>2.09</td>
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<tr>
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<td>SRT multi-choice *</td>
<td>11.6</td>
<td>0.94</td>
<td>12.0</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>SRT 30 minute delayed recall</td>
<td>8.1</td>
<td>3.63</td>
<td>10.2</td>
<td>1.88</td>
</tr>
<tr>
<td>Executive Functions</td>
<td>WCST % total errors</td>
<td>39.9</td>
<td>17.45</td>
<td>24.3</td>
<td>15.11</td>
</tr>
<tr>
<td></td>
<td>WCST % perseverative errors</td>
<td>19.7</td>
<td>11.68</td>
<td>13.2</td>
<td>9.90</td>
</tr>
<tr>
<td></td>
<td>WCST % nonperseverative errors *</td>
<td>20.1</td>
<td>15.39</td>
<td>11.1</td>
<td>7.74</td>
</tr>
<tr>
<td></td>
<td>WCST categories completed</td>
<td>3.64</td>
<td>2.24</td>
<td>5.07</td>
<td>1.63</td>
</tr>
<tr>
<td>Negative Symptoms</td>
<td>SANS total sum</td>
<td>21.1</td>
<td>8.87</td>
<td>25.5</td>
<td>16.1</td>
</tr>
</tbody>
</table>

* = Nonparametric t-test used; VCPT = Visual Continuous Performance Test, Identical Pairs; LMI/II = Logical Memory I and II; SRT = Selective Reminding Test; CLTR = continuous long term retrieval; RLTR = random long term retrieval; WCST = Wisconsin Card Sorting Test; SANS = Scale for the Assessment of Negative Symptoms; M = Mean; SD = Standard Deviation; d = Cohen’s measure of effect size.
Table 8.7
Parametric and Nonparametric T-tests of Differences on Test Scores between the Nonschizotypy and Normative Groups

<table>
<thead>
<tr>
<th>Domain</th>
<th>Test Score</th>
<th>Nonschizotypy</th>
<th>Normative</th>
<th>p-value</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td></td>
<td>VCPT targets/hit rate</td>
<td>23.7</td>
<td>6.29</td>
<td>27.9</td>
<td>4.28</td>
</tr>
<tr>
<td></td>
<td>VCPT mean reaction time (ms)</td>
<td>562</td>
<td>88.31</td>
<td>483</td>
<td>61.19</td>
</tr>
<tr>
<td></td>
<td>VCPT catches/false alarms</td>
<td>5.0</td>
<td>4.52</td>
<td>4.6</td>
<td>4.02</td>
</tr>
<tr>
<td></td>
<td>VCPT random errors a</td>
<td>3.7</td>
<td>10.11</td>
<td>1.9</td>
<td>6.62</td>
</tr>
<tr>
<td></td>
<td>VCPT commissions/total errors a</td>
<td>8.7</td>
<td>13.22</td>
<td>6.5</td>
<td>9.43</td>
</tr>
<tr>
<td></td>
<td>VCPT d'</td>
<td>2.13</td>
<td>1.19</td>
<td>2.70</td>
<td>1.19</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Verbal Memory</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>LMI total scaled score</td>
<td>8.9</td>
<td>2.99</td>
<td>10.0</td>
<td>3.00</td>
</tr>
<tr>
<td></td>
<td>LMII total scaled score</td>
<td>10.2</td>
<td>2.78</td>
<td>10.0</td>
<td>3.00</td>
</tr>
<tr>
<td></td>
<td>LMII % retention scaled score a</td>
<td>12.2</td>
<td>2.88</td>
<td>10.0</td>
<td>3.00</td>
</tr>
<tr>
<td></td>
<td>SRT total recall</td>
<td>116.7</td>
<td>16.70</td>
<td>116.8</td>
<td>13.56</td>
</tr>
<tr>
<td></td>
<td>SRT long term storage</td>
<td>113.0</td>
<td>22.49</td>
<td>110.7</td>
<td>18.70</td>
</tr>
<tr>
<td></td>
<td>SRT short term retrieval a</td>
<td>10.0</td>
<td>10.43</td>
<td>11.4</td>
<td>7.87</td>
</tr>
<tr>
<td></td>
<td>SRT CLTR</td>
<td>91.2</td>
<td>39.14</td>
<td>92.2</td>
<td>28.16</td>
</tr>
<tr>
<td></td>
<td>SRT RLTR</td>
<td>15.1</td>
<td>17.87</td>
<td>14.0</td>
<td>10.95</td>
</tr>
<tr>
<td></td>
<td>SRT reminders</td>
<td>38.1</td>
<td>15.68</td>
<td>26.3</td>
<td>12.71</td>
</tr>
<tr>
<td></td>
<td>SRT cued recall</td>
<td>9.1</td>
<td>2.03</td>
<td>8.9</td>
<td>2.09</td>
</tr>
<tr>
<td></td>
<td>SRT multi-choice a</td>
<td>11.7</td>
<td>0.59</td>
<td>12.0</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>SRT 30 minute delayed recall</td>
<td>9.6</td>
<td>2.32</td>
<td>10.2</td>
<td>1.88</td>
</tr>
<tr>
<td></td>
<td>Executive Functions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WCST % total errors</td>
<td>28.7</td>
<td>17.67</td>
<td>24.3</td>
<td>15.11</td>
</tr>
<tr>
<td></td>
<td>WCST % perseverative errors</td>
<td>19.3</td>
<td>12.74</td>
<td>13.2</td>
<td>9.90</td>
</tr>
<tr>
<td></td>
<td>WCST % nonperseverative errors a</td>
<td>9.5</td>
<td>6.19</td>
<td>11.1</td>
<td>7.74</td>
</tr>
<tr>
<td></td>
<td>WCST categories completed</td>
<td>4.67</td>
<td>1.80</td>
<td>5.07</td>
<td>1.63</td>
</tr>
<tr>
<td></td>
<td>Negative Symptoms</td>
<td>17.4</td>
<td>14.27</td>
<td>25.5</td>
<td>16.1</td>
</tr>
</tbody>
</table>

Note. * = Nonparametric t-test used; VCPT = Visual Continuous Performance Test, Identical Pairs; LMI/II = Logical Memory I and II; SRT = Selective Reminding Test; CLTR = continuous long term retrieval; RLTR = random long term retrieval; WCST = Wisconsin Card Sorting Test; SANS = Scale for the Assessment of Negative Symptoms; M = Mean; SD = Standard Deviation; d = Cohen's measure of effect size.
Diagnoses from the SCID-I and Psychiatric Records

Table 8.8 shows the frequency of each DSM-IV-TR research diagnosis as produced by the SCID-I interview for both the schizotypy and nonschizotypy groups. Research diagnoses were divided into past and current diagnoses. If participants currently met criteria for a particular research diagnosis, then this was not included as a past diagnosis. Comorbidity was present and 17% of the whole group received more than one current SCID-I diagnosis. As can be seen in Table 8.8, the schizotypy group had a total of 20 current and 20 past diagnoses, whereas the nonschizotypy group had a total of 8 current and 19 past diagnoses. The 20 current diagnoses held by the schizotypy group were spread across 10 people, and the 8 current diagnoses held by the nonschizotypy group were spread across 7 people. Four people (29%) of the schizotypy group and 8 people (53%) of the nonschizotypy group did not meet criteria for a current diagnosis. The most common current SCID-I diagnoses across the whole sample of 29 participants were panic disorder with and without agoraphobia (17% of the sample received this diagnosis), major depressive disorder (14%), social phobia (14%), and posttraumatic stress disorder (14%). Across the whole sample, a total of 12 people met criteria for a current or past schizophrenia-spectrum disorder (including major depressive disorder with psychotic features, bipolar I disorder, schizophrenia, and schizoaffective disorder). Of these people, 5 (17%) met criteria for a current schizophrenia-spectrum disorder, 3 from the schizotypy group and 2 from the nonschizotypy group. In the schizotypy group, 6 people met criteria for a past substance abuse or dependence diagnosis (a total of 8 past substance abuse or dependence diagnoses were given), whereas in the nonschizotypy group 1 person met criteria for a past substance abuse or dependence diagnosis (1 diagnosis was given). None of the participants met criteria or exhibited any symptoms of current substance abuse or dependence.
Table 8.8
Frequency of Current and Past DSM-IV-TR Research Diagnoses from SCID-I Interviews for Schizotypy and Nonschizotypy Groups

<table>
<thead>
<tr>
<th>Domain</th>
<th>Diagnosis</th>
<th>Schizotypy C</th>
<th>Schizotypy P</th>
<th>Nonschizotypy C</th>
<th>Nonschizotypy P</th>
<th>Whole Group Total C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mood</td>
<td>Major Depressive Disorder (MDD)</td>
<td>3</td>
<td>5</td>
<td>1</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>MDD with Psychotic Features</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Dysthymic Disorder</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Bipolar I Disorder</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Depressive Disorder NOS</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Psychosis</td>
<td>Schizophrenia</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Schizoaffective Disorder</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Substance</td>
<td>Alcohol Abuse/Dependence</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Cannabis Abuse/Dependence</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Opioid Abuse/Dependence</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Panic Disorder with/without Agoraphobia</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Agoraphobia without Panic Disorder</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Social Phobia</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Specific Phobia</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Obsessive Compulsive Disorder</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Posttraumatic Stress Disorder</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Generalised Anxiety Disorder</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Eating</td>
<td>Anorexia Nervosa</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Binge Eating Disorder</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>No Current Diagnosis</td>
<td></td>
<td>4</td>
<td>-</td>
<td>8</td>
<td>-</td>
<td>12</td>
</tr>
<tr>
<td>Total Number of Diagnoses</td>
<td></td>
<td>20</td>
<td>20</td>
<td>8</td>
<td>19</td>
<td>28</td>
</tr>
</tbody>
</table>

Note. C = Current; P = Past.

Further analyses were conducted to determine if significant differences between the schizotypy and nonschizotypy groups on 8 of the test scores were due to the 6 people in the schizotypy group who met criteria for a past substance abuse or dependence diagnosis. Comparisons were made between the schizotypy subgroup with a past substance abuse or dependence diagnosis \((n = 6)\) and the schizotypy subgroup without a past substance abuse or dependence diagnosis \((n = 8)\). Kurtosis and skewness were examined and multiple
testing was corrected for. With a parametric independent samples one-sided \( t \)-test, the subgroup with a past substance diagnosis had a significantly lower mean GAF of 52.83 \( (SD = 7.76) \) compared to the subgroup without a past substance diagnosis \( (M = 61.75, SD = 8.70) \), \( t(12) = -1.99, p = .04, d = -1.03 \). With the Mann-Whitney test, a nonparametric independent samples test, the subgroup with a past substance abuse diagnosis had a significantly lower median LMII percent retention score of 56 \( (R = 44) \) than the subgroup without a past substance abuse diagnosis \( (Md = 85.5, R = 69) \), \( U(6, 8) = 8.00, p = .02, d = -1.06 \). There were no significant differences between the two subgroups for the other 6 test scores.

Participants' current clinical diagnoses were also obtained from their psychiatric records at their relevant mental health service. In addition to current clinical diagnoses, past substance abuse or dependence diagnoses were obtained. It is important to note that these are working clinical diagnoses that had been recorded in the participant's file by their psychiatrist or psychologist and were obtained at the time that participants took part in Phase 1. The time between Phase 1 and Phase 2 participation ranged between 9 and 18 months. As a result, the clinical diagnoses in participants' psychiatric files obtained at the time of Phase 1 may no longer have been applicable when participants took part in Phase 2. The clinical diagnoses from Phase 1 are shown in Table 8.9. It is unknown which diagnostic system, if any, diagnoses were based on. Where possible, diagnoses have been grouped according to DSM-IV-TR classifications. In brackets next to each diagnostic label are the terms that are included within that category, as they appeared in participants' psychiatric records. As can be seen in Table 8.9, the schizotypy group \( (n = 14) \) had a total of 23 current clinical diagnoses and the nonschizotypy group \( (n = 15) \) had a total of 24 current clinical diagnoses. Across the whole sample, the most common clinical diagnoses from participants' psychiatric records were variations of major depressive disorder (41%).
Table 8.10
Frequency of Current and Past DSM-IV-TR Diagnoses from SCID-I Interviews for Participants Who Did and Did Not Meet Criteria for Schizotaxia_Tsuang

<table>
<thead>
<tr>
<th>Domain</th>
<th>Diagnosis</th>
<th>Schizotaxia_Tsuang</th>
<th>Nonschizotaxia_Tsuang</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Current</td>
<td>Past</td>
<td>Current</td>
</tr>
<tr>
<td>Mood</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mood</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mood Major Depressive Disorder</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Mood Major Depressive Disorder with Psychotic Features</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Mood Dysthymic Disorder</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Mood Bipolar I Disorder</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Mood Depressive Disorder NOS</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Psychosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Schizoaffective Disorder</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Substance</td>
<td>Alcohol Abuse/Dependence</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Substance</td>
<td>Cannabis Abuse/Dependence</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Substance</td>
<td>Opioid Abuse/Dependence</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Panic Disorder with and without Agoraphobia</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Agoraphobia without history of Panic Disorder</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Social Phobia</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Specific Phobia</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Obsessive Compulsive Disorder</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Posttraumatic Stress Disorder</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Generalised Anxiety Disorder</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Eating</td>
<td>Anorexia Nervosa</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Eating</td>
<td>Binge Eating Disorder</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>No Current Diagnosis</td>
<td>1</td>
<td>-</td>
<td>11</td>
</tr>
<tr>
<td>Total Number of Diagnoses</td>
<td>10</td>
<td>12</td>
<td>18</td>
</tr>
</tbody>
</table>

\(^{a}n = 7\)

\(^{b}n = 22\)
of the participants who did not meet criteria for schizotaxia\textsubscript{Tsuang}, 1 (5\%) met diagnostic criteria for a current schizophrenia-spectrum disorder and 1 participant met criteria for a past schizophrenia-spectrum disorder. The schizophrenia-spectrum disorders included major depressive disorder with psychotic features, schizophrenia, and schizoaffective disorder.

A chi-square analysis was conducted to determine whether the observed counts for each schizotaxia\textsubscript{Tsuang} and schizophrenia-spectrum disorder combination were significantly different from the expected counts. Fisher’s Exact test was used for this analysis as some of the expected counts had values of less than 5. A significant result was observed indicating that there was a relationship between classification of Tsuang et al.’s (1999b) schizotaxia\textsubscript{Tsuang} and classification of a current or past schizophrenia-spectrum disorder, with $\chi^2 (1) = 7.47$, Fisher’s Exact $p = .02$. In addition, 4 (57\%) of the participants who met criteria for schizotaxia\textsubscript{Tsuang} and 3 (14\%) of the participants who did not meet criteria for schizotaxia\textsubscript{Tsuang} met criteria for a past substance abuse or dependence diagnosis.

**The Relationship between Schizotaxia\textsubscript{Tsuang} and Schizotypy**

After an individual’s schizotaxia\textsubscript{Tsuang} classification status had been determined, their schizotypy classification from Phase 1 was revealed. Prior to this the experimenter had been blind to individuals’ schizotypy or nonschizotypy group membership. This process produced four groups of participants, based on their schizotaxia\textsubscript{Tsuang} and schizotypy group status. Table 8.11 shows the observed counts for each schizotaxia\textsubscript{Tsuang} and schizotypy combination. As can be seen in Table 8.11, 8 participants who were classified as schizotypy members did not meet criteria for schizotaxia\textsubscript{Tsuang}. Only 1 participant who was not classified as a schizotypy member met criteria for schizotaxia\textsubscript{Tsuang}. The largest group
(N = 14) was made up of those who did not meet criteria for schizotaxia\textsubscript{Tsuang} and were also classified as nonschizotypy group members.

Table 8.11

<table>
<thead>
<tr>
<th>Schizotaxia\textsubscript{Tsuang}</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>6</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>No</td>
<td>8</td>
<td>14</td>
<td>22</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>15</td>
<td>29</td>
</tr>
</tbody>
</table>

A chi-square analysis was conducted to determine whether the observed counts for each schizotaxia\textsubscript{Tsuang} and schizotypy combination were significantly different from the expected counts. Fisher's Exact test was used for this analysis as some of the expected counts had values of less than 5. A significant result was observed indicating that the classifications of Tsuang et al.'s (1999b) schizotaxia\textsubscript{Tsuang} and Meehl's (1962, 1990b) schizotypy were not independent, with $\chi^2 (1) = 5.18, p = .04$, Fisher’s Exact $p = .03$.

Logistic Regression Analysis

As a statistically significant relationship was observed between Tsuang et al.'s schizotaxia\textsubscript{Tsuang} and Meehl's schizotypy, a standard logistic regression analysis was conducted to determine the ability of Tsuang et al.'s schizotaxia\textsubscript{Tsuang} criteria to predict who would have Meehl's schizotypy.

Each participant was categorised as having an impairment or no impairment for each of the 9 test scores that were used to determine the schizotaxia\textsubscript{Tsuang} classification. The
logistic regression analysis showed that the combination of 9 test scores was a significantly reliable model of schizotypy outcome, $\chi^2(9, N = 29) = 31.09, p = .00$. The Hosmer and Lemeshow goodness of fit statistic was $\chi^2(6, N = 29) = 0.0, p = 1.00$, indicating the model was a good fit of the data. The model accounted for 65.8% to 87.7% of the variance in classification of Meehl’s schizotypy. As shown in Table 8.12, Tsuang et al.’s schizotaxia$^\text{Tsuang}$ criteria were able to correctly identify 85.7% of people with Meehl’s schizotypy. Tsuang et al.’s schizotaxia$^\text{Tsuang}$ criteria correctly identified 93.3% of people without Meehl’s schizotypy. Overall, 89.7% of classifications were accurate.

<table>
<thead>
<tr>
<th>Actual Classification</th>
<th>Predicted Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Schizotypy</td>
</tr>
<tr>
<td>Schizotaxia$^\text{Tsuang}$</td>
<td>85.7%</td>
</tr>
<tr>
<td>No Schizotaxia$^\text{Tsuang}$</td>
<td>6.7%</td>
</tr>
</tbody>
</table>

As part of the logistic regression analysis, the ability of each test score (in terms of presence of absence of impairment) to predict Meehl’s schizotypy was determined. This involved calculating the Wald statistic to determine if each test score was a significant predictor. As can be seen in Table 8.13 the results from the analysis were all nonsignificant, meaning that none of the 9 impairment test scores were individually predictive of Meehl’s schizotypy. The odds ratio and regression coefficient (natural log of the odds ratio) are used to determine the weight of a predictor if it is observed to be significant.
<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>B</th>
<th>SE</th>
<th>Odds Ratio</th>
<th>Wald</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Attention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VCPT targets/hit rate</td>
<td>-49.1</td>
<td>5036.8</td>
<td>0.0</td>
<td>0.0</td>
<td>NS</td>
</tr>
<tr>
<td>VCPT d'</td>
<td>-82.4</td>
<td>6874.7</td>
<td>0.0</td>
<td>0.0</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Verbal Memory</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMII % retention</td>
<td>33.0</td>
<td>21242.7</td>
<td>0.0</td>
<td>0.0</td>
<td>NS</td>
</tr>
<tr>
<td>LMII total score</td>
<td>-249.2</td>
<td>24647.6</td>
<td>0.0</td>
<td>0.0</td>
<td>NS</td>
</tr>
<tr>
<td>SRT total recall</td>
<td>150.2</td>
<td>18038.2</td>
<td>--</td>
<td>0.0</td>
<td>NS</td>
</tr>
<tr>
<td>SRT RLTR</td>
<td>-248.2</td>
<td>24049.2</td>
<td>0.0</td>
<td>0.0</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Executive Functions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCST % total errors</td>
<td>-65.6</td>
<td>5900.6</td>
<td>0.0</td>
<td>0.0</td>
<td>NS</td>
</tr>
<tr>
<td>WCST % perseverative errors</td>
<td>-33.2</td>
<td>4591.5</td>
<td>0.0</td>
<td>0.0</td>
<td>NS</td>
</tr>
<tr>
<td><strong>SANS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of items rated as ≥ 3</td>
<td>-130.7</td>
<td>11107.2</td>
<td>0.0</td>
<td>0.0</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Note.* VCPT = Visual Continuous Performance Test, Identical Pairs; LMII = Logical Memory II; SRT = Selective Reminding Test; WCST = Wisconsin Card Sorting Test; SANS = Scale for the Assessment of Negative Symptoms; B = Regression Coefficient or Natural Log of Odds Ratio; SE = Standard Error; NS = nonsignificant.

Another logistic regression analysis was conducted using the raw scores of the 9 test scores rather than converting them into the presence or absence of an impairment. The logistic regression analysis showed that the combination of the 9 types of raw test scores was a significantly reliable model of schizotypy outcome, \( \chi^2 (9, N = 29) = 21.066, p = .01 \). However, the goodness of fit statistic for this model was \( \chi^2 (8, N = 29) = 9.1, p = .33 \). The model accounted for 51.6% to 68.9% of the variance in classification of Meehl's schizotypy. This analysis showed that Tsuang et al.'s schizotaxia–Tsuang criteria in the form of raw scores were able to correctly identify the same percentage of people with and without Meehl's schizotypy as when the scores were categorised in terms of presence or absence of impairment (Table 8.12). Again, the ability of each raw test score to predict Meehl's schizotypy was determined. As can be seen in Table 8.14 the results of this part
of the analysis were also all nonsignificant, suggesting that none of the raw scores were individually predictive of Meehl’s schizotypy.

Table 8.14  
Summary of Standard Logistic Regression Analysis of Tsuang et al.’s (1999b) Schizotypia (Tsuang) Criteria Using Raw Scores

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>B</th>
<th>SE</th>
<th>Odds Ratio</th>
<th>Wald</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Attention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VCPT targets/hit rate</td>
<td>-0.11</td>
<td>0.13</td>
<td>0.89</td>
<td>0.79</td>
<td>NS</td>
</tr>
<tr>
<td>VCPT d’</td>
<td>-0.36</td>
<td>1.13</td>
<td>0.70</td>
<td>0.10</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Verbal Memory</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMII % retention</td>
<td>0.02</td>
<td>0.22</td>
<td>1.20</td>
<td>0.30</td>
<td>NS</td>
</tr>
<tr>
<td>LMII total score</td>
<td>-0.21</td>
<td>0.05</td>
<td>1.02</td>
<td>2.92</td>
<td>NS</td>
</tr>
<tr>
<td>SRT total recall</td>
<td>-0.05</td>
<td>0.05</td>
<td>0.95</td>
<td>0.91</td>
<td>NS</td>
</tr>
<tr>
<td>SRT RLTR</td>
<td>-0.10</td>
<td>0.07</td>
<td>0.91</td>
<td>2.08</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Executive Functions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCST % total errors</td>
<td>0.37</td>
<td>0.24</td>
<td>1.45</td>
<td>2.48</td>
<td>NS</td>
</tr>
<tr>
<td>WCST % perseverative errors</td>
<td>-0.57</td>
<td>0.34</td>
<td>0.57</td>
<td>2.75</td>
<td>NS</td>
</tr>
<tr>
<td><strong>SANS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of items rated as ≥ 3</td>
<td>0.18</td>
<td>0.22</td>
<td>1.20</td>
<td>0.70</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Note.* VCPT = Visual Continuous Performance Test, Identical Pairs; LMII = Logical Memory II; SRT = Selective Reminding Test; WCST = Wisconsin Card Sorting Test; SANS = Scale for the Assessment of Negative Symptoms; B = Regression Coefficient or Natural Log of Odds Ratio; SE = Standard Error; NS = nonsignificant.

**Results Summary**

In summary, in comparison to the nonschizotypy group, the schizotypy group was significantly impaired on a number of measures of neuropsychological functioning. In addition, both the schizotypy group and the nonschizotypy group were significantly impaired on a number of measures of neuropsychological functioning when compared to normative groups; however, the schizotypy group had a greater number of impairments than the nonschizotypy group. A greater proportion of people in the schizotypy group
received a current *DSM-IV-TR* diagnosis than those in the nonschizotypy group. Likewise, a greater proportion of people who met criteria for Tsuang et al.'s (1999b) schizotaxia,Tsuang received a current *DSM-IV-TR* diagnosis than those who did not meet criteria for schizotaxia,Tsuang. Results of a contingency analysis indicated that Tsuang et al.'s (1999b) schizotaxia,Tsuang and Meehl's (1962, 1990b) schizotypy are not independent in this mixed psychiatric sample. Furthermore, Tsuang et al.'s (1999b) set of criteria for schizotaxia,Tsuang are able to reliably distinguish between schizotypal and nonschizotypal cases. However, none of the individual criteria are able to reliably predict schizotypy group membership. The implications of these results will be considered in the discussion.

**Discussion**

**First Aim**

The first aim of Phase 2 was to determine if schizotypy group membership is associated with poorer functioning. It was hypothesised that, compared to the nonschizotypy group, the schizotypy group would have poorer functioning on a range of measures. This was carried out by investigating the differences between the schizotypy and nonschizotypy groups on a number of tests that assessed neuropsychological functioning, negative symptoms, and global functioning. The groups were also compared to normative groups.

The results of the current study partially supported the hypothesis that the schizotypy group would have poorer functioning on a range of measures. It was found that, relative to the nonschizotypy group, the schizotypy group was impaired on a total of 8 test scores involving the neuropsychological domains of attention, verbal memory, and
executive functioning. In addition, the schizotypy group had lower IQ and global functioning scores than the nonschizotypy group. However, there were no significant differences between the schizotypy and nonschizotypy group in regards to negative symptoms. Compared to normative samples, both the schizotypy and nonschizotypy groups were impaired in the domains of attention, verbal memory, and executive functioning. The schizotypy group had more negative symptoms than a normative group and the nonschizotypy group had less negative symptoms than a normative group. The schizotypy group had more significant impairments when compared to normative samples than the nonschizotypy group did. Overall, these results indicate that a group of individuals identified as schizotypal by taxometric analysis procedures, had poorer functioning in a range of domains than a group of individuals identified as nonschizotypal.

These results are consistent with other studies that have found differences in functioning between schizotypy and nonschizotypy groups. Other studies have found that schizotypy groups have impaired attention, verbal memory, and executive functioning relative to nonschizotypy groups (e.g., Gooding et al., 2001; Lenzenweger et al., 1991; Park et al., 1995). However, these studies have identified schizotypal and nonschizotypal individuals on the basis of psychometric scores. Only one other study has investigated functioning in schizotypy and nonschizotypy groups identified through taxometric analysis procedures. Linscott (2005) used MAXCOV and MAXEIG procedures which yielded results consistent with a manifest taxonic structure for schizotypy. He found that individuals in the schizotypy group had significantly impaired attention and more psychological distress than individuals in the nonschizotypy group. The results of the current study are consistent with this finding as schizotypy group membership was associated with impairments in the domains of attention, executive functioning, verbal memory, and global functioning, and lower IQ.
The psychopathology of the two groups was evaluated in the current study with a diagnostic interview. The schizotypy group met criteria for a greater number of current diagnoses than the nonschizotypy group and both groups had a similar number of past diagnoses. A similar number of people from both groups met criteria for a current schizophrenia-spectrum disorder. More people in the nonschizotypy group did not meet criteria for a current diagnosis than the schizotypy group. These results indicate that the schizotypy group had more current difficulties with psychopathological functioning than the nonschizotypy group did. This is consistent with the findings of previous studies that have found that schizotypal individuals display more psychopathological symptoms than nonschizotypal individuals (e.g., Chapman et al., 1994; Eckblad & Chapman, 1983; Miller et al., 2002).

The difference in psychopathological functioning between the two groups raises the question as to whether individuals were members of the schizotypy group as a result of their mental health difficulties rather than having schizotypal characteristics. A diagnostic interview was used at Phase 2 to determine past diagnoses. In addition, diagnoses from psychiatric records were obtained at the time participants took part in Phase 1, which was between 9 and 18 months before participants took part in Phase 2. Participants in the schizotypy and nonschizotypy groups had a similar number of past diagnoses based on the diagnostic interview. Furthermore, the two groups had a similar number of diagnoses obtained from their psychiatric records. The two groups had a similar number of past diagnoses from the depression-spectrum, while the nonschizotypy group had more past substance abuse or dependence diagnoses and the schizotypy group had more past diagnoses from the anxiety-spectrum. This means that the two groups may have been similar in psychopathological functioning at the time that schizotypy group status was ascertained. This conclusion is made on the basis of the frequency of disorders rather than
the severity of disorders. Future research in this area could compare the severity of the psychopathological functioning of the schizotypy and nonschizotypy groups. It would not be surprising if it was found that the schizotypy group had more severe psychopathology as schizotypy is considered by many researchers to be a predisposition to schizophrenia and consequently more individuals with schizotypy may have a schizophrenia-spectrum disorder.

More people in the schizotypy group met criteria for a past substance abuse or dependence diagnosis than the nonschizotypy group. This may have had an impact on differences in functioning between the two groups. The results of analyses to determine the impact of past substance diagnoses found that individuals in the schizotypy group with a past substance abuse or dependence diagnosis were significantly more impaired than individuals in the schizotypy group without a past substance abuse or dependence diagnosis in regards to global functioning and retention of verbal memory. There were no other significant differences. This suggests that overall, having a past substance abuse or dependence diagnosis did not fully account for the obtained differences between the schizotypy and nonschizotypy group.

Second Aim

The second aim of Phase 2 was to determine the nature of the relationship between Meehl's (1962, 1990b) schizotypy and Tsuang et al.'s (1999b) schizotaxia_{Tsuang}. It was hypothesised that, as schizotypy and schizotaxia_{Tsuang} are conceptually very similar, there would be evidence of dependence between the two constructs. This was carried out by firstly determining which participants of a sub-sample of schizotypy and nonschizotypy group members from Phase 1 met Tsuang et al.'s (1999b) criteria for schizotaxia_{Tsuang}. Secondly, the degree of overlap between schizotypy and schizotaxia_{Tsuang} was established.
Recall, an individual was classified as having met criteria for schizotaxiaTsuang if they had: (a) an impairment in one neuropsychological domain equal to 1 standard deviation or greater below norms; (b) an impairment in another neuropsychological domain equal to 2 standard deviations or greater below norms; and (c) a negative symptom impairment. Individuals met criteria for the presence of negative symptoms if they had 6 or more items rated as at least moderately impaired (a rating of 3 or higher). A total of 7 people out of 29 met these criteria for schizotaxiaTsuang, 6 were from the schizotypy group and 1 from the nonschizotypy group. A greater proportion of individuals who met criteria for schizotaxiaTsuang also met criteria for a current psychiatric diagnosis relative to the individuals who did not meet criteria for schizotaxiaTsuang. Relative to the nonschizotaxiaTsuang group, a greater proportion of individuals in the schizotaxiaTsuang group met criteria for a current or past schizophrenia-spectrum disorder. In addition, a statistically significant result was observed when this relationship was evaluated. This indicates that there is a relationship between schizotaxiaTsuang and meeting criteria for a present or past schizophrenia-spectrum disorder. These results suggest that individuals who met criteria for schizotaxiaTsuang had poorer psychological functioning than individuals who did not meet criteria for schizotaxiaTsuang.

The second step in determining the nature of the relationship between schizotypy and schizotaxiaTsuang involved establishing the degree of overlap between the two constructs. A statistically significant result was observed indicating that the classifications of Meehl’s (1962, 1990b) schizotypy and Tsuang et al.’s (1999b) schizotaxiaTsuang are not independent. This supports the hypothesis that there would be evidence of dependence between Meehl’s (1962, 1990b) schizotypy and Tsuang et al.’s (1999b) schizotaxiaTsuang. Exactly to what degree schizotaxiaTsuang and schizotypy are related cannot be stated, however, as 6 of the 7 individuals who met criteria for schizotaxiaTsuang were also
probably did not affect the outcome as, although 3 of the 29 participants reported that their medication had changed in the month prior to testing, all 3 of these participants were in the nonschizotypal group. In fact, some research has shown that medication does not necessarily have an impact on neuropsychological functioning. For example, Epstein, Keefe, Roitman, Harvey and Mohs (1996) found that the performance of a group of individuals with schizophrenia on a continuous performance task of attention was not affected by the presence or absence of typical neuroleptics. In addition, Squire, Judd, Janoswky, and Huey (1980) found that lithium carbonate affected the performance of a group of psychiatric patients on a perceptual motor test but did not affect memory and learning skills. Browne et al. (2000) found that there were no significant differences in neurological functioning between a group of medicated individuals with first-episode psychosis and a group of non-medicated individuals with first-episode psychosis. These findings suggest that the presence or a change of medication does not necessarily contribute to an impairment in neuropsychological functioning. The differences in functioning that were observed in the current study are likely not fully accounted for by medication, past substance use, and general psychopathology.

**Limitations**

There are a number of limitations of the current study. Firstly, as explained in the Method there were difficulties associated with obtaining a sufficient number of participants for each group in Phase 2. The size of the groups was sufficient for the analyses that were conducted to investigate both of the aims of Phase 2. In addition, the size of the groups is comparable to the groups investigated by Stone et al. (2001). However, it would be interesting to compare the neuropsychological functioning of the four groups formed at the end of Phase 2 and this would require larger samples.
A second limitation involves the nature of the normative groups that were used for the comparison of the schizotypy and nonschizotypy groups to normative samples. There were many differences between the normative groups and the schizotypy and nonschizotypy groups. These differences could not be controlled for and this may have an impact on the strength of the results. In addition, it is possible that the normative samples that were used were different to those used by Tsuang and colleagues. It is difficult to know for certain as Tsuang and colleagues have not stated in the literature which normative samples they have used in their studies for determining the presence or absence of an impairment. A potential solution to this would be to create new normative samples that were drawn from a New Zealand population more relevant to the sample used in this study. The use of different normative samples would probably have an impact on the proportion of individuals who meet criteria for schizotypy.

The current study did not look at the impact of psychopathology in general on neuropsychological functioning. The schizotypy group met criteria for more current diagnoses than the nonschizotypy group. This raises the question as to how this difference contributed to the variations between the two groups in terms of their neuropsychological and global functioning. This was not examined directly in the current study however, as discussed above, past research has suggested that psychopathology does not necessarily have an impact on neuropsychological functioning.

An additional limitation also concerns the assessment of psychopathology in this study. The focus was on Axis I disorders in this study. Axis II disorders were not assessed because of practical reasons such as the extra time this would take (up to an additional 3 hours), and only one researcher was involved in data collection. A few of the participants had a personality disorder diagnosis in their psychiatric records; however, an independent assessment of personality disorders would have been interesting considering
Tsuang and colleagues view of the relationship between schizotaxia\textsuperscript{Tsuang} and schizotypal personality disorder. None of the participants had a diagnosis of schizotypal personality disorder in their psychiatric records, nevertheless, as found in Phase 2, individuals’ psychiatric record diagnoses did not always match the diagnoses obtained from the diagnostic interview.

**Summary**

The results of the current study indicate that schizotypy group membership is associated with poorer functioning in the psychiatric participant sample and that schizotypy and schizotaxia\textsuperscript{Tsuang} are indeed related. In Chapter 9, the implications of these findings will be considered more thoroughly. Specifically, I will consider the degree to which the evidence can be viewed as inconsistent with Tsuang and colleagues’ assertion that schizotypy is not the likely outcome of schizotaxia\textsuperscript{Tsuang} and the implications this has for both Meehl’s and Tsuang et al.’s theories.
CHAPTER 9

General Discussion

Review of Main Results

As reviewed in Chapter 7, the results of Phase 1 of the current study suggest the presence of a manifest taxonic structure of schizotypy in the psychiatric participant sample with a base rate of approximately 29%. This is consistent with Meehl's (1962, 1989, 1990b) theory of schizotypy which holds the view that schizotypy is discontinuous. As discussed in Chapter 7, there are a number of limitations associated with Phase 1 including the small sample size, and the use of only one psychometric self-report measure.

As reviewed in Chapter 8, the results of Phase 2 of the current study indicate that schizotypy group membership is associated with poorer functioning in the psychiatric participant sample. This is consistent with past research but is relatively unique in that schizotypy was investigated using taxometric analysis with the psychiatric participants. The second finding of Phase 2 was that Meehl's (1962, 1989, 1990b) schizotypy and Tsuang et al.'s (1999b, 2000a, 2000b) schizotaxia\textsubscript{Tsuang} are related, specifically, that schizotaxia\textsubscript{Tsuang} may be a subset of schizotypy. This has not previously been investigated.

As discussed in Chapter 8, there are a number of limitations associated with Phase 2 including the small sample size, the nature of the normative groups that were used, and the way in which psychopathology was assessed.

The degree to which the evidence from Phases 1 and 2 can be viewed as, firstly, consistent with Meehl's quasi-dimensional theoretical model and, secondly, inconsistent
with Tsuang et al.'s categorical model will be considered next. This will include an examination of the implications these results have for both theories.

**Review of Two Theories**

To begin with, a brief summary of the two theories under consideration is required. As reviewed in Chapter 2, there have been a number of problems identified with diagnostic systems and how schizophrenia is conceptualised. These problems have occurred both historically and currently. In an effort to better understand the construct, researchers began to look at risk for schizophrenia and states that may precede schizophrenia. One researcher, Meehl (1962, 1989, 1990b), proposed that people who are at risk of developing schizophrenia are born with a genetic mutation, which he postulated to be in the form of a single dominant gene. He conjectured that the genetic mutation produces an integrative neural defect, schizotaxiaMeehl, which has an underlying, subtle problem called hypokrisia. Meehl (1962, 1989, 1990b) further proposed that as a result of environmental effects, such as social learning and reinforcement schedules, all individuals with schizotaxia develop a schizotypal personality organisation. He conjectured that 10% of the general population has schizotypy and that schizotypy is observed in 35% to 40% of the psychiatric population (Meehl, 1990b). Meehl (1962) proposed that approximately 10% of schizotypal people decompensate and develop schizophrenia. He further asserted that the development and course of schizophrenia in the schizotypal person is contributed to by the interaction between a number of polygenic potentiators and the social environment. According to Meehl (1990b), schizotypal individuals continue to be schizotypal when they decompensate to schizophrenia.

Another group of researchers, Tsuang and colleagues, proposed a contrasting theory of risk for schizophrenia. Tsuang et al. (1999b, 2000a, 2000b) based their definition of
schizotaxia\textsubscript{Tsuang} on their interpretation of Meehl’s original theory. They proposed that early environmental insults interact with a genetic predisposition to produce a vulnerability to developing schizophrenia, called schizotaxia\textsubscript{Tsuang}. Tsuang and colleagues conjectured that schizotaxia\textsubscript{Tsuang} is manifest in the form of neurodevelopmental brain abnormalities known as target features (Tsuang & Faraone, 1999). They proposed that schizophrenia develops as a result of the interaction between neurodevelopmental abnormalities (schizotaxia\textsubscript{Tsuang}) and environmental factors. Tsuang and colleagues have reported that 20% to 50% of relatives of individuals with schizophrenia experience the symptoms of schizotaxia\textsubscript{Tsuang} (Faraone et al., 1995a, 199b). They predicted that there is some degree of overlap between schizotaxia\textsubscript{Tsuang} and negative schizotypal personality disorder and that schizotaxia\textsubscript{Tsuang} is broader than negative schizotypal personality disorder (Faraone et al., 2001). They maintain that neither schizotypy nor schizophrenia is the likely outcome of schizotaxia\textsubscript{Tsuang}. It appears that Tsuang et al. equate Meehl’s construct of schizotypy with schizotypal personality disorder, as they tend to use the terms interchangeably.

Interpretation of Results and Implications for Theories

The results of the current study have implications for aspects of both Meehl’s and Tsuang et al.’s theories. The current study provides support for Meehl’s theory. As discussed in Chapter 7, one of the aims of the current study was to investigate the manifest structure of schizotypy in the psychiatric population. There was evidence that schizotypy is discontinuous, that is, individuals either are or are not schizotypal. This is consistent with Meehl’s (1962, 1989, 1990b) theory of schizotypy. Consequently, the current study supports one component of Meehl’s theory. The other components of Meehl’s theory including schizotaxia\textsubscript{Meehl}, hypokrisia, and the relationship between schizotaxia\textsubscript{Meehl} and
schizotypy were not investigated. Therefore, conclusions cannot be made about these aspects.

The findings from the second phase that schizotaxia_{Tsuang} and schizotypy are related does not provide direct support for Meehl's theory. This is because schizotaxia_{Meehl} is very different to schizotaxia_{Tsuang} and this is one of the key distinctions of the two theories. The differences between schizotaxia_{Meehl} and schizotaxia_{Tsuang} relate to the aetiology of schizotaxia (genetic versus genetic and environmental), how the construct is observed (nonmeasurable versus measurable), the outcome of schizotaxia (schizotypy and maybe schizophrenia versus maybe schizophrenia), and the relationship between schizotaxia and other schizophrenia-spectrum disorders (quasidimensional versus categorical). Consequently, a direct comparison of schizotaxia_{Meehl} and schizotaxia_{Tsuang} is not possible in this study.

The finding from Phase 2 that there is evidence of dependence between schizotaxia_{Tsuang} and Meehl's schizotypy is an outcome that is not predicted by Tsuang and colleagues' theory (Faraone et al., 2001; Tsuang et al., 1999b). Most individuals in the schizotaxia_{Tsuang} group were schizotypal. Furthermore, of those in the schizotypy group, 43% met criteria for schizotaxia_{Tsuang}. This indicates that schizotaxia_{Tsuang} may be a subset of schizotypy and suggests that schizotaxia_{Tsuang} is unlikely to be diagnosed among nonschizotypal individuals. However, this conclusion needs to be made with caution due to the small sample size used in this study.

The finding that schizotaxia_{Tsuang} may be a subset of Meehl’s schizotypy is contrary to Tsuang and colleagues’ prediction that schizotaxia_{Tsuang} is broader than schizotypy (Faraone et al., 2001). It is also contrasts with their view that schizotypy is not the likely outcome for schizotaxia_{Tsuang} (Tsuang et al., 2002b). This could mean that the criteria for schizotaxia_{Tsuang} are features of a construct related to risk for schizophrenia, which is in
Schizotypy is also related to schizotypy. Schizotypy has many similarities to Meehl’s schizotypy. Schizotypy includes some of the symptoms that Faraone et al. (2001) consider indicative of schizotypy. In addition, both schizotypy and schizotypy involve genetic and environmental factors; are measurable and observable constructs; and the outcome for both is not necessarily schizotypal personality disorder. Therefore, it appears that Tsuang and colleagues’ schizotypy may be at the same level of analysis as Meehl’s schizotypy.

Alternatively, the finding that schizotypy may be a subset of Meehl’s schizotypy could mean that the criteria for schizotypy are features that are part of the heterogeneous nature of schizotypy. This explanation is consistent with past research that demonstrates that neuropsychological impairments and psychopathological difficulties are correlates of schizotypy (e.g., Gooding et al., 2001; Laurent et al., 1999, 2001; Lenzenweger et al., 1991; Park et al., 1995). This suggests that perhaps schizotypy should not be considered as a separate construct. One of Tsuang and colleagues’ goals is for schizotypy to enter the diagnostic nomenclature. The findings of the current study indicate that the relationship between schizotypy and schizotypy needs to be carefully considered and investigated further before this could happen.

The results of the logistic regression analysis demonstrated that the criteria for schizotypy can be used to distinguish between schizotypal and nonschizotypal individuals. One possible explanation for this is that individuals with schizotypy have more features of schizotypy than nonschizotypal individuals. This explanation is supported by the first part of Phase 2, in which a separate set of analyses showed that schizotypy group membership is associated with poorer functioning. This interpretation
contrasts with Tsuang et al.’s (2002b) prediction that schizotypy is not the likely outcome of schizotaxia_{Tsuang}.

In their theory, Tsuang and colleagues view schizotaxia_{Tsuang} as very similar or the same as negative schizotypal personality disorder (Faraone et al., 2001). They propose that schizotaxia_{Tsuang} does not overlap with positive schizotypal personality disorder (Faraone et al., 2001). Tsuang et al. (2002b) have described an unpublished study where Stone et al. compared the ratings on the Structured Interview for Schizotypy (SIS) of individuals with schizotaxia_{Tsuang} and without schizotaxia_{Tsuang}. Global negative ratings were compared with global positive ratings and they found that participants with schizotaxia_{Tsuang} had significantly higher ratings on 2 of the 3 global negative scores and on a mean of the 3 scores than participants without schizotaxia_{Tsuang}. There were no differences between the two groups on ratings of positive symptoms (cited in Tsuang et al., 2002b). They conclude that this demonstrates that schizotaxia_{Tsuang} and negative schizotypal personality disorder are similar and that schizotaxia_{Tsuang} and positive schizotypal personality disorder are distinct.

A potential problem with this is that the SIS (Kendler et al., 1989) is more representative of Meehl’s (1962, 1990b) schizotypy than schizotypal personality disorder. Consequently, Stone et al. (cited in Tsuang et al., 2002b) have found a relationship between schizotaxia_{Tsuang} and negative aspects of Meehl’s schizotypy as measured by the SIS. They have made the error of equating Meehl’s schizotypy with schizotypal personality disorder. Tsuang and colleagues use the terms interchangeably in their theory and research yet there are significant differences between the two constructs because Meehl’s schizotypy is much broader than schizotypal personality disorder. Consequently, when Tsuang et al. refer to schizotypy it is difficult to know if they are referring to
Meehl's (1962, 1990b) schizotypy or schizotypal personality disorder. Irrespective of this, Tsuang and colleagues (Faraone et al., 2001; Stone et al., cited in Tsuang et al., 2002b) state that schizotaxia_{Tsuang} is related to negative schizotypal personality disorder and distinct from positive schizotypal personality disorder. The results of the current study do not support or disprove these predictions by Tsuang and colleagues. Although a relationship was observed between schizotaxia_{Tsuang} and schizotypy in the current study, the measure that was used to evaluate schizotypy, the TPSQ-A, incorporates both positive and negative features of schizotypy and consequently negative schizotypy and positive schizotypy were not assessed separately. Future research in this area could consider evaluating the relationship between schizotaxia_{Tsuang}, negative schizotypy and positive schizotypy.

In addition to the relationship between schizotaxia_{Tsuang} and schizotypy, the results of the current study need to be considered in relation to other aspects of the theories of Meehl and Tsuang et al. According to Tsuang and colleagues, individuals with schizophrenia do not have schizotaxia_{Tsuang} because the relationship between the disorders is categorical, or transitional (Faraone et al., 2001; Tsuang et al., 1999b). They view the presence of schizophrenia as signaling the end of schizotaxia_{Tsuang}. Because of this reason and the potential impact of psychosis, Tsuang and colleagues have excluded individuals with a history of psychosis from their research (Tsuang & Faraone, 1999). However, the current study has included people with a diagnosis of schizophrenia. This is because according to Meehl (1990b), individuals with schizophrenia are also schizotypal and schizotaxic_{Meehl}. In addition, as previously discussed, schizotaxia_{Tsuang} may be at the same level of analysis as schizotypy. One of the aims of the current study was to investigate the relationship between the theories of Meehl and Tsuang et al. and in order to do this, individuals with a diagnosis of schizophrenia were included. Because of the differences
between Meehl and Tsuang et al. in regards to the relationships between schizotaxia_Tsuang and schizophrenia, and schizotypy and schizophrenia, an examination of individuals who did and did not meet criteria for a schizophrenia-spectrum disorder may have implications for both Meehl’s and Tsuang et al.’s theories.

Three participants (43%) in the schizotaxia_Tsuang group met criteria for a current research diagnosis of schizophrenia-spectrum disorder and one met criteria for a past research diagnosis of schizophrenia-spectrum disorder. Two (29%) of the participants with a schizophrenia-spectrum disorder met criteria for a current research diagnosis of schizophrenia, 1 of these participants had nonschizotypy group membership. A total of 4 (57%) participants in the schizotaxia_Tsuang group and 2 (10%) participants in the nonschizotaxia_Tsuang group had a current or past research diagnosis of a schizophrenia-spectrum disorder. Although this may appear to be inconsistent with Tsuang et al.’s (1999b, 2000a, 2000b) theory, this is not necessarily the case because schizophrenia acts as an exclusion criterion in their theory and research. It is not surprising that an individual may meet criteria for both schizotaxia_Tsuang and schizophrenia as Tsuang and colleagues view the criteria for schizotaxia_Tsuang as milder forms of the features observed in individuals with schizophrenia (Kremen et al., 1994). The important factor relates to classification, as based on Tsuang and et al.’s theory, participants in the schizotaxia_Tsuang group who meet criteria for schizophrenia are no longer considered to be schizotypic_Tsuang.

An examination of schizotypal and nonschizotypal individuals shows that 6 (43%) participants from the schizotypy group and 6 (40%) participants from the nonschizotypy group met criteria for a current or past research schizophrenia-spectrum disorder. One participant from each group had schizophrenia. This is inconsistent with Meehl’s theory because an individual with schizophrenia was not schizotypal. Meehl’s (1990b) view is that schizotypal individuals continue to be schizotypal when they decompensate to
schizophrenia. However, it is possible that the individual who met criteria for schizophrenia and was not in the schizotypy group actually had a genophenocopy of schizophrenia. Meehl (1990b) has predicted that 85% to 90% of people diagnosed as having schizophrenia are also schizotaxic Meehl while 10% to 15% have genophenocopies of schizophrenia, a syndrome he calls the SHAITU syndrome.

Both theories involve genetic factors and consider these to have a significant role in risk for schizophrenia. Participants in Phase 2 were asked whether there was a history of mental illness in their family, including relatives with schizophrenia and other forms of psychosis. In the study, 3 of 29 participants were adopted, 3 of 29 reported no mental illness in their family, and the rest reported a history of mental illness in their family but approximately half of these were unsure as to the nature of the mental illness. Consequently, conclusions about the genetic contribution to schizotypy, schizotaxia Tsuang, and other psychiatric illness cannot be made from the results of the current study. In addition, the extent of any possible genetic contribution cannot be inferred from the current study as participants may have had relatives with psychiatric illness but not have known this or preferred not to report this. Furthermore, the accuracy of the verbal reports was not known and other information sources were not sought to confirm this. Future research could consider a more comprehensive investigation of family psychiatric illness using measures such as the Family Interview for Genetic Studies (FIGS; Maxwell, 1982). Based on the theories of both Meehl and Tsuang et al., it would be expected that individuals with schizotypy and/or schizotaxia Tsuang would be more likely to have relatives with psychiatric illness than individuals without schizotypy and schizotaxia Tsuang.

Future Research
test scores that make up Tsuang and colleagues’ research criteria for schizotaxia. This aspect of the study was unique in that the schizotypy and nonschizotypy groups were identified with taxometric analysis and involved psychiatric participants. More research involving psychiatric participants and taxometric analysis to investigate the manifest structure of schizotypy and neuropsychological functioning is required to confirm the results of the current study. It would also be interesting to determine if similar results for schizotypy and nonschizotypy groups of psychiatric participants are observed for other neuropsychological domains such as spatial memory. If there are differences in other domains then maybe these could also be considered risk indicators. In addition, future research could compare performance in these domains between those who meet criteria for both schizotaxia and are schizotypal to those who are just schizotypal. The current study was unable to do this due to the small size of the four groups created at the end of Phase 2.

Tsuang and colleagues chose two thresholds for a neuropsychological impairment, one impairment equal to 1 standard deviation or greater below norms and a second impairment equal to 2 standard deviations below norms (Stone et al., 2001; Tsuang et al., 1999b). In addition, a negative symptom impairment was defined as 6 or more items with a rating of 3 or higher (Stone et al., 2001; Tsuang et al., 1999b). Faraone et al. (1995b) claim that two standard deviations below norms is a “commonly used threshold of impairment in psychopathology and neuropsychological research” (p. 293). The thresholds appear to be rather arbitrary which raises the question as to what effect it would have if the criteria were modified. A total of 7 people met criteria for Tsuang et al.’s (1999b) schizotaxia in the current study. It is likely that modifying the thresholds, even slightly, for example, increasing or decreasing by 1 the required number of negative symptom items, would have an impact on the proportion of individuals who met criteria
for schizotaxia\textsubscript{Tsuang} in the current study. As part of future research that investigates the construct of schizotaxia\textsubscript{Tsuang}, it needs to be established whether these thresholds are the most suitable and informative.

The nature of the criteria and thresholds established by Tsuang and colleagues for schizotaxia\textsubscript{Tsuang} needs further investigation. According to Faraone et al., (2001), the criteria for schizotaxia\textsubscript{Tsuang} were chosen because they are more pertinent to relatives of individuals with schizophrenia than other impairments. Research by Tsuang and colleagues has shown that individuals who are relatives of people with schizophrenia experience impairments in a range of areas relative to individuals without a family history of schizophrenia. Indeed, Tsuang and colleagues have acknowledged that there may be other criteria that need to be included with the current schizotaxia\textsubscript{Tsuang} criteria (Stone et al., 2001). Future research could be conducted to determine the most appropriate criteria for the schizotaxia\textsubscript{Tsuang} construct.

The relationship between schizotaxia\textsubscript{Tsuang} and schizotypal personality disorder also needs to be investigated. Tsuang and colleagues have acknowledged that they do not know to what extent schizotaxia\textsubscript{Tsuang} and schizotypal personality disorder overlap (M. T. Tsuang, personal communication, August 20, 2003). In addition, they have proposed a reformulation of the diagnostic criteria for schizotypal personality disorder (Faraone et al., 2001). The current study has indicated that there is a relationship between Meehl’s schizotypy and Tsuang et al.’s schizotaxia\textsubscript{Tsuang}. Consequently, research needs to look at the level of comorbidity between schizotypal personality disorder and schizotaxia\textsubscript{Tsuang}.

The current study created 4 groups with the 4 possible combinations of schizotypy and schizotaxia\textsubscript{Tsuang} classifications. As part of this research, individuals current and past functioning was determined; however, it would be interesting to know what the future course and outcome of these classifications will be and how these relate to the predictions
made by Meehl and Tsuang et al. The mean age of the schizotypy group was 42.5 years and the mean age of the nonschizotypy group was 44.9 years. Consequently, they are outside of the at-risk age group for developing schizophrenia and a follow-up of these individuals would not necessarily provide useful information about the development of schizophrenia. However, information about future psychiatric difficulties would be interesting to investigate to determine if there are differences in long-term functioning between the 4 groups. Furthermore, future research could investigate schizotaxia-Tsuang and schizotypy in a younger sample of psychiatric patients that is more representative of the at-risk age group. These participants could be followed in a longitudinal study to evaluate the outcome of the constructs in regards to the development of schizophrenia and other psychiatric illnesses. Based on the theories of both Meehl and Tsuang et al., it would be expected that a small proportion of individuals with schizotaxia-Tsuang and/or schizotypy would develop schizophrenia.

Schizophrenia is characterised by positive symptoms, negative symptoms, and neuropsychological impairment. Positive and negative symptoms are included as criteria in diagnostic systems but neuropsychological impairment is not. This is despite a vast array of evidence showing that neuropsychological impairment is a characteristic of schizophrenia (e.g., Aylward et al., 1984; Cornblatt & Malhotra, 2001; Gooding & Tallent, 2002; Heinrichs & Zakzanis, 1998; Lysaker et al., 2000; Manschreck et al., 2000). Neuropsychological impairments cover a wide range of domains and, as with the other symptoms of schizophrenia, can vary across individuals. However, it has been suggested that one way to improve diagnostic systems would be to include neuropsychological impairment as part of the criteria (Lewis, 2004; Tsuang et al., 2002a). The current study has demonstrated that a group of individuals identified through taxometric analysis as schizotypal have significantly poorer functioning on a range of neuropsychological
measures compared to a group of individuals identified as nonschizotypal. Many researchers view schizotypy as a risk indicator for schizophrenia. Meehl (1962) proposed that 10% of schizotypal individuals develop schizophrenia. Considering the evidence of past research that has found that neuropsychological impairment is a characteristic of schizophrenia and evidence of the current study that neuropsychological impairment is associated with a risk indicator for schizophrenia, it is possible that diagnostic systems will benefit from the inclusion of a neuropsychological impairment criterion. Future research needs to establish the validity, reliability, specificity and treatment utility of a criterion such as this before it is included in diagnostic systems, either as a criterion for schizophrenia or as a criterion for a precursor of schizophrenia.

Future research also needs to consider the issues surrounding psychopharmacological treatment of individuals considered to be at risk of schizophrenia. As discussed in Chapter 4, Tsuang and colleagues have carried out research to investigate the impact of risperidone on symptoms of schizotaxia\textsubscript{Tsuang} (Tsuang et al., 1999b; Tsuang et al., 2002c). There are a number of limitations associated with these studies. Furthermore, the results of the current study indicate that there is a relationship between schizotaxia\textsubscript{Tsuang} and schizotypy, which may have an impact on future research in this area. Tsuang and colleagues' goals are for schizotaxia\textsubscript{Tsuang} to enter the diagnostic nomenclature and for treatments to be available to high-risk groups thought to be at risk of developing schizophrenia. Substantially more research into the construct of schizotaxia\textsubscript{Tsuang} is needed before this can be considered, which Tsuang and colleagues have acknowledged. In addition, the treatment of a risk indicator raises a number of ethical and practical issues that mirror those associated with research that has investigated the prodrome of schizophrenia and early interventions for this. Some of these issues will be discussed next.
The area of early intervention for psychosis has focused on offering individuals treatment when the first signs and symptoms of psychosis have been identified. This has considered a variety of risk indicators as well as symptoms considered to be indicative of the early or late prodromal phase (Ruhrmann, Schultzze-Lutter, & Klosterkötter, 2003; Yung et al., 2003). Research in this area has found that individuals who experience psychotic symptoms for longer prior to their first treatment have poorer treatment outcome than individuals who have a shorter duration of symptoms (e.g., Bottlender, Strauss, & Möller, 2000; Cannon et al., 2002; Johannessen et al., 2001; McGorry et al., 2002; Morrison et al., 2004; Woods et al., 2003; Wyatt, 1995). In addition, individuals with a longer duration of symptoms take longer to respond to treatment. These studies have involved psychopharmacological treatments such as risperidone and olanzapine as well as cognitive-behavioural treatments. However, the link between a positive outcome and early treatment may actually be due to a relationship between an acute course and good prognosis (Woods et al., 2003). In addition, there have also been inconsistent findings (Clarke & O'Callaghan, 2003).

The aim of early intervention is to reduce the likelihood of further deterioration. Indeed, it is desirable to avoid the end-state of terminal dementia as conceptualised by Kraepelin (1919/2002). People at risk need to be offered the best chance of recovery or a reasonable level of functioning in the event that they do decompensate to schizophrenia. However, the benefits need to outweigh the costs. It has been suggested that before prevention or treatment is implemented for those considered to be at risk, research needs to clearly establish the validity and reliability of the indicators used to predict or determine those who are at risk (Cornblatt, 2002). In addition, research needs to establish which factors are best at predicting subsequent development of schizophrenia (Maier, Cornblatt, & Merikangas, 2003). It needs to be determined how sensitive any screening tools are at
detecting people at risk who later develop schizophrenia and also whether the screening tools incorrectly classify people as being at-risk (Bentall & Morrison, 2002; Jablensky, 2000). The effects of false-positive rates of identification, the issue of stigma, and the potential side effects of being identified incorrectly need to be considered (Clarke & O'Callaghan, 2003; Corcoran, Malaspina, & Hercher, 2005; Cornblatt, Lencz, & Kane, 2001; Heinimaa & Larsen, 2002). This is especially important as current early-identification programs often involve adolescents or teenagers. Consequently, if they go through a process where they are incorrectly identified as being at-risk of developing schizophrenia then this can have a significant impact on their lives. Lastly, it needs to be ensured that the benefits of early identification and treatment programs have been maximised and risk minimised before the programs are introduced into general clinical settings (Bentall & Morrison, 2002; Maier et al., 2003). These issues have created a number of ethical debates (for a more comprehensive summary see McGlashan, 2001; Schaffner & McGorry, 2001).

All of these issues, although related to early intervention programs, have a direct relevance to the research of Tsuang and colleagues. Cornblatt et al. (2001) have cautioned that if conclusions are made hastily about research on treatment for those at risk of schizophrenia, then this will have a direct impact on how psychopharmacological treatments are used in a wide range of clinical settings involved in the treatment of schizophrenia. To overcome this, more sound and ethical research is needed in a number of areas related to risk for schizophrenia. As can be seen, there is controversy over the use of early intervention programs for individuals displaying early signs of psychosis. Tsuang and colleagues believe that the symptoms of schizotypy (Tsuang) occur long before prodromal symptoms of schizophrenia (Tsuang et al., 2000b). This controversy of early intervention is likely to be amplified with the implementation of pharmacological treatments for
individuals thought to be at risk but not showing any signs of psychosis, i.e., those classified as having schizotaxia\textsubscript{Tsuang}. Consequently, it needs to be determined that some individuals who meet criteria for schizotaxia\textsubscript{Tsuang} do actually go on to develop schizophrenia.

This thesis has involved the investigation of the manifest structure of schizotypy in a psychiatric sample and the relationship between two theories of risk for schizophrenia. The findings of this research may have little immediate impact on the area of risk for schizophrenia but has highlighted a number of issues with the way in which risk for schizophrenia is currently conceptualised. Research in this area contributes further to knowledge and understanding of the aetiology of schizophrenia. In the long-term, the goal of research in this area is to eventually locate and identify which specific genetic aspects of schizotypy or schizotaxia\textsubscript{Tsuang} are inherited. The purpose of this is to identify individuals who are at risk with the aim of reducing the likelihood that they may develop schizophrenia or another schizophrenia-spectrum disorder. Individuals have an increased prospect of a positive outcome if their difficulties are identified early.

The advantage of both of the theories that have been considered in this thesis is that they avoid focusing on psychosis as an end-state. This problem is inherent in many of the diagnostic systems that are used to diagnose schizophrenia. The development and research of these theories means that there is pressure for diagnostic systems to consider the aetiology of schizophrenia. Neurodevelopmental theories such as those considered in this thesis are currently the most comprehensive models of the aetiology of schizophrenia. These have the potential to impact upon the diagnosis of those individuals both who have schizophrenia and are at risk.

\textit{Summary}
The current study has demonstrated that, using a self-report measure of schizotypy, there is evidence of a manifest taxonic structure of schizotypy in a psychiatric sample. This has supported Meehl’s (1962, 1989, 1990b) theory that schizotypy is discontinuous. The current study also provided evidence that compared to nonschizotypy group membership, schizotypy group membership is associated with poorer functioning in a range of areas. This is consistent with past findings that have investigated schizotypy group membership in general populations (e.g., Gooding et al., 2001; Lenzenweger et al., 1991; Park et al., 1995). These findings are particularly informative because schizotypy has only previously been investigated once using taxometric analysis in a psychiatric population. In addition, the current study provided evidence that Meehl’s (1962, 1989, 1990b) schizotypy and Tsuang et al.’s (1999b, 2000a, 2000b) schizotaxia_{Tsuang} are related. This finding is unique in that the construct of schizotaxia_{Tsuang} has not previously been assessed in a psychiatric population. Furthermore, the relationship between schizotaxia_{Tsuang} and Meehl’s schizotypy has not been previously evaluated.

The results of this study supported all three hypotheses that were outlined in Chapter 6. However, there are a number of limitations and it is important to consider how these may have impacted upon the results. The current study has addressed many questions but has also drawn attention to a number of areas related to the theories under investigation that require further research and consideration. These include further research with psychiatric participants using taxometric analysis to investigate the manifest structure of schizotypy and further research into the construct of schizotaxia_{Tsuang} to clearly establish its relationships with schizotypy and schizotypal personality disorder, validity and ability to predict which individuals are at risk of developing schizophrenia.
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APPENDIX

Thinking and Perceptual Style Questionnaire

INSTRUCTIONS

This questionnaire is about personality—about how you think about things, how you view and experience life, what you value, and about unusual experiences. Some people have found that the types of things included in this questionnaire are actually quite common; they suggest that most people have had such experiences, or held such beliefs at some time in their lives. These are important because they seem to be related to characteristics such as creativity and imagination, and people's ability to perform various mental tasks.

The questionnaire contains 99 questions divided among 6 different sections. Each section begins with a brief description of the questions in that section. For each of the questions you will be asked to provide a rating on a scale like one of these shown at the right:

To do this, simply put a cross (X) in the circle which corresponds to the answer you want to give. For some of the questions you may think that there is no simple answer. In those cases just pick the answer that is best, even if it is not entirely correct. If you want to change your answer, please make it clear which one your new answer is.

Please answer honestly. Some of the questions and statement may not apply to you whereas other may apply. Still other questions may seem strange or trivial. Nevertheless, it is important that you read all the questions and answer honestly. You need not feel embarrassed or ashamed by answering one way or another. There are no right or wrong answers and we expect there to be a great variation in the way different people respond to different questions.

Occasionally, a question may mention something that has happened to you only after taking drugs (e.g., marijuana) or drinking alcohol. If this is the only time(s) you had that experience, answer as if you had not had the experience.

Please answer honestly. Some of the questions and statement may not apply to you whereas other may apply. Still other questions may seem strange or trivial. Nevertheless, it is important that you read all the questions and answer honestly. You need not feel embarrassed or ashamed by answering one way or another. There are no right or wrong answers and we expect there to be a great variation in the way different people respond to different questions.

SECTION 1

The questions that follow ask about things that some people find pleasurable or enjoyable. Please answer by thinking about how much pleasure, delight, or enjoyment you would normally get from these things. There are no right or wrong answers. Answer by placing a cross in the circle.

How much pleasure, delight, or enjoyment do you normally get from:

1. looking at art, sculpture, or architecture .................................................................
2. observing nature or wildlife .................................................................
3. public entertainment such as parades, sports games, or concerts .................................................................
4. listening to music of any kind .................................................................
5. reading or listening to poetry or stories, whether fiction or non-fiction .................................................................
6. listening to the sounds of nature or wildlife, such as the rain or birds singing .................................................................
7. hearing other people talking .................................................................
8. loud noises .................................................................
9. the taste of foods .................................................................
10. the taste of drinks .................................................................
11. the smell of food being cooked .................................................................
12. sweet fragrances such as from perfume or flowers .................................................................
13. the smell or sounds of machinery or vehicles .................................................................
14. very intense physical exercise .................................................................
15. feeling physically exhausted or worn out .................................................................
16. mild physical exercise, such as walking .................................................................
17. singing or dancing .................................................................
18. the feel of the heat from the sun or the warmth of a fire .................................................................
19. the feel of water on your skin .................................................................
20. extremes in the weather .................................................................
21. having very close friendships with others .................................................................

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Section 1 continued:

How much pleasure, delight, or enjoyment do you normally get from:

<table>
<thead>
<tr>
<th>Activity</th>
<th>None</th>
<th>A Little</th>
<th>Some</th>
<th>Quite a Bit</th>
<th>A Great Deal</th>
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</thead>
<tbody>
<tr>
<td>Making new friends</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Talking with others about your problems</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Playing with children</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Caring for friends or feeling affection or love for others</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Going out socialising in large or small groups</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Hobbies or sports that involve being around other people</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Having people visit you at home</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Meeting new people</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Physical touch with others, whether affectionate or not</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>5</td>
</tr>
<tr>
<td>Helping others with their problems</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Receiving compliments from other people</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

Section 2

Sometimes thoughts and mental images seem particularly life-like, as though they were real and not in our mind, and as if they were actually happening. The following questions ask about your experience of these types of thoughts and mental images. Please answer by placing a cross in one circle for each question. There are no right or wrong answers.

How often have you had the following experiences:

<table>
<thead>
<tr>
<th>Experience</th>
<th>Never</th>
<th>Occasionally</th>
<th>Sometimes</th>
<th>Often</th>
<th>All the Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoughts being so loud that they are distracting or disturbing</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Mental images of things or people seeming real, as if they were before your eyes</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Thoughts sounding like your own voice talking to you</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Irrelevant thoughts interrupting your work or conversations</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Thoughts seeming or sounding like a conversation between two real people</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Day-dreams being so clear that you believed it was really happening to you</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Imagined smells seeming real and actually there</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Thoughts sounding like other people talking to you</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Imagined music or noises seeming real and actually there</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

Section 3

The following questions contain reasons for feeling uncomfortable or anxious around other people, or for not wanting to be around others. How often have these things made you feel uncomfortable around others. Please answer by placing a cross in one circle for each question. There are no right or wrong answers.

How often are you made uncomfortable by

<table>
<thead>
<tr>
<th>Reason</th>
<th>Never</th>
<th>Occasionally</th>
<th>Sometimes</th>
<th>Often</th>
<th>All the Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>The concern that others may reject you or hold a low opinion of you</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>The thought that others really can't be trusted</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Worry you might look foolish or out of place</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Thinking that other people are looking for an opportunity to criticise you or put you down</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Concern that you might do or say something stupid</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Worry that others might try to humiliate you</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>The thought that others do not like you</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Concern that other people may want to harm you</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
### SECTION 4

The questions that follow are about concentration, thinking, and speaking. Please answer by indicating how often these things generally happen to you. Place a cross in the circle that gives the best answer. There are no right or wrong answers.

How often do you

<table>
<thead>
<tr>
<th>Question</th>
<th>Occasionally</th>
<th>Sometimes</th>
<th>Often</th>
<th>All the Time</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>feel like you can't concentrate on anything</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>feel like you can't direct where your thoughts are going</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>find yourself becoming easily distracted by little things</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lose track of what you were meaning to say or meaning to do</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>have difficulty making decisions</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>find yourself saying things that sound a little odd</td>
<td></td>
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<tr>
<td>see relationships between ideas or words which others do not see</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>use terms or phrases in ways that other people do not understand</td>
<td></td>
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<tr>
<td>find it difficult to get a particular phrase or word out of your mind</td>
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<tr>
<td>find that your mind seems to run too easily from one idea to the next</td>
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<td></td>
</tr>
<tr>
<td>notice that your thinking is cloudy or vague</td>
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<td></td>
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<tr>
<td>have difficulty finding the right word, having to use another word instead</td>
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<td></td>
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<tr>
<td>find yourself getting confused while you are talking</td>
<td></td>
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<td></td>
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<tr>
<td>find that your mind goes completely blank</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>notice that your thoughts are all muddled up in your mind</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>use terms or phrases that you made up (which others do not use)</td>
<td></td>
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<tr>
<td>use terms or phrases that sound a little awkward</td>
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<tr>
<td>have difficulty describing relationships between things that are clearly related</td>
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<tr>
<td>make up a new word to describe something</td>
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</tbody>
</table>

### SECTION 5

The questions below contain reasons that people use to explain things that happen to themselves or to others. You may think that some of these reasons are better than others. For each reason listed below, indicate how good a reason it is for things that happen to you or to others. Place a cross in the circle that gives the best answer. There are no right or wrong answers.

How good a reason is

<table>
<thead>
<tr>
<th>Reason</th>
<th>No Good</th>
<th>A Little Good</th>
<th>Somewhat Good</th>
<th>Quite Good</th>
<th>Good</th>
</tr>
</thead>
<tbody>
<tr>
<td>telepathy, mind-reading, or thought transference</td>
<td></td>
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<tr>
<td>telekinetic powers (ability to shift or change things by the power of thought)</td>
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<tr>
<td>a sixth sense, extrasensory perception (ESP), or special dreams</td>
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<tr>
<td>aliens and things that aliens do</td>
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<tr>
<td>rituals (such as special prayers) or good luck charms</td>
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<tr>
<td>astrology (past horoscopes), or fate, or clairvoyance</td>
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<tr>
<td>things like numbers such as 13 or cracks in the footpath</td>
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<tr>
<td>curses, or spells, or magic</td>
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<tr>
<td>interference by spirits</td>
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<tr>
<td>forgetting to do things like pray, or think happy thoughts</td>
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<tr>
<td>supernatural events or supernatural beings</td>
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<tr>
<td>déjà vu (the feeling that you are experiencing something for a second time)</td>
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</tr>
<tr>
<td>the statement, 'it was arranged for that to happen to me, or for me to see that.'</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>the statement, 'it was the answer I was waiting on.'</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Section 5 continued:

How good a reason is

15. the statement, 'Because I just needed to know or see those things.' .......................................................... 1 2 3 4 5
16. the statement, 'I was the only one allowed to hear that.' .............................................................................. 1 2 3 4 5
17. the statement, 'No-one else would know what to do with that information.' ........................................... 1 2 3 4 5

SECTION 6

The questions that follow are about changes in how some things usually seem to you.
Sometimes people experience unusual and brief changes in how their bodies look or seem, or in how parts of their bodies feel, or in how objects around them appear. For each of the questions, please answer by indicating how often you have had the experience(s) listed. Place a cross in the circle that gives the best answer. There are no right or wrong answers.

How often have you felt as though

1. part of your body (e.g., an arm, or leg, or your head) was disconnected from you ................................... 1 2 3 4 5
2. a part or all of your body was connected with some object or some other person .................................... 1 2 3 4 5
3. you no longer owned or had control over part or all of your body ............................................................. 1 2 3 4 5
4. part or all of your body no longer existed ................................................................................................... 1 2 3 4 5
5. how you looked or appeared had changed for a moment ........................................................................... 1 2 3 4 5
6. the appearance of part of your body (e.g., your face, or a limb) changed briefly ...................................... 1 2 3 4 5
7. the appearance of others who you know had changed for a brief moment ............................................... 1 2 3 4 5
8. part of you (e.g., a leg, or your hands or arms) had grown in size or length ............................................. 1 2 3 4 5
9. part of your body (e.g., an arm or leg) had become smaller or shorter ..................................................... 1 2 3 4 5
10. part of you (e.g., perhaps your brain or stomach) was rotting or had died ............................................ 1 2 3 4 5
11. part of you felt quite strange, abnormal, or unreal .................................................................................... 1 2 3 4 5
12. objects about you were changing shape or appearance, or were moving around .............................. 1 2 3 4 5
13. you had become more sensitive to colours, shapes, sounds, or smells ............................................. 1 2 3 4 5

ID:
Date: