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Schizotypy: Consideration of neurological soft signs, language and affective factors

Saskia de Leede-Smith
University of Wollongong

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Schizotypy: Consideration of neurological soft signs, language and affective factors

Saskia de Leede-Smith

Bachelor of Science (Psychology) (Hons)

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CERTIFICATION

I, Saskia de Leede-Smith, declare that this thesis, submitted in partial fulfilment of the requirements for the award of Doctor of Philosophy (Clinical Psychology), in the School of Psychology, University of Wollongong, is wholly my own work unless otherwise referenced or acknowledged. The document has not been submitted for qualifications at any other academic institution.

Saskia de Leede-Smith
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ABSTRACT

Schizotypy is regarded as a trait vulnerability marker for psychosis. The study of this personality trait in otherwise healthy samples affords opportunities to research potential co-occurring risk factors for psychosis, without the confounds inherent to psychiatric diagnosis. The current thesis is focused on the expression of schizotypy alongside neurodevelopmental, language and affective risk factors for psychosis. The specific risk factors investigated were neurological soft signs (NSS), semantic processing, affective temperament, and psychological distress. Additionally, this thesis investigated whether propensity to hallucinate had an effect on these factors when combined with psychometric schizotypy. Language abnormalities have also been extensively researched in schizotypy and along the psychosis continuum. Given the overlap between the psychosis continuum and language abnormalities such as those seen in dyslexia, this thesis also sought to determine whether risk factors for psychosis are also present in a dyslexia sample.

This thesis is made up of a combination of five studies, some of which are published and submitted manuscripts, with the remainder being manuscripts in preparation for publication. **Study One** investigated the relationship between schizotypy and distress. Affective temperament was found to mediate this relationship. Contrary to predictions hallucination predisposition was not found to exert significant effects on either the direct or indirect relationship between schizotypy and distress. **Study Two** explored whether NSS are expressed differently in those with high and low levels of schizotypy and additionally, whether hallucination predisposition interacts with this effect. Results indicated that those with high overall schizotypy express significantly more NSS, and that hallucination predisposition has additive effects on this association. **Study Three** looked at the expression of NSS in a dyslexia sample. It was found that individuals with dyslexia expressed a significantly greater amount of NSS compared to controls. Individuals with dyslexia also had significantly higher rates of schizotypy, which was found to contribute to the higher level of distress found in those with dyslexia compared to controls. **Study Four** investigated semantic processing capabilities of those with high and low positive schizotypy, as well as high and low hallucination predisposition. High levels of positive schizotypy resulted in slower reaction times compared to low positive schizotypy, whilst high hallucination predisposition

resulted in faster reaction times compared to low hallucination predisposition. **Study Five** investigated semantic processing in a dyslexia sample. There was some evidence that the dyslexia group responded slower than controls. The dyslexia group also had difficulty discriminating degree of relatedness between semantic pairs, and schizotypy was found to contribute to this effect.

This thesis concludes with a synthesis of the findings across the five studies. Theoretical and clinical implications are also considered, alongside limitations of the research and possible avenues for further enquiry. Overall, the results of this thesis indicate that individuals with high levels of schizotypy have associations with distress through affective temperament, as well as an increased expression of NSS, and abnormal semantic processing. Hallucination predisposition is not synonymous with schizotypy in its effects on these risk factors, suggesting schizotypy and propensity to hallucinate may have different mechanisms of effect. Dyslexia was associated with an increased expression of NSS, as well as semantic processing abnormalities that were contributed to by schizotypy. These findings are indicative of schizotypy and dyslexia having overlapping features, which may be suggestive of possible shared aetiologies.

KEY ABBREVIATIONS

At Risk Mental State	ARMS
Auditory Verbal Hallucinations	AVH
Clinical High Risk	CHR
Cognitive-Perceptual (schizotypy)	CP
Neurological Soft Signs	NSS
Schizotypal Personality Disorder	SPD
Psychotic-Like Experiences	PLEs
Ultra-High Risk	UHR

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de Leede-Smith, S., Roodenrys, S., Horsley, L., Matrini, S., Mison, & E. Barkus E., (2017). Neurological soft signs: Effects of trait schizotypy, psychological distress and state auditory verbal hallucination predisposition. *Schizophrenia Research: Cognition.* (Appendix J)

STATEMENT OF VERIFICATION

This statement verifies that the greater part of the work in the previously stated publications/manuscripts is attributed to the candidate. Saskia de Leede-Smith, under the guidance and supervision of her supervisors, took primary responsibility for the design of each study, all data collection and analysis, prepared the first draft of each manuscript, and prepared the papers for submission to relevant journals. Co-authors EB and SR, whom were supervisors to the candidate, contributed to this thesis by providing guidance on the design and structure of each study, and editorial suggestions for every paper. Co-authors LH, EM and SM assisted with data collection for the previously stated publications/manuscripts.

Saskia de Leede-Smith (PhD candidate)

Dr Emma Barkus (Primary supervisor)

Associate Professor Steven Roodenrys (Co-supervisor)

1 GENERAL INTRODUCTION

1.1 Overview

Schizotypy has similar clinical characteristics to schizophrenia (e.g. Kwapil et al., 2014). Due to these similarities, Fanous et al. (2007) has proposed common factors may underlie both phenomena. The clinical relevance of schizotypy is dependent on an individual's exposure to additional risk factors (van Os et al., 2009). This thesis will examine a range of psychological and neurodevelopmental risk factors that may interact with trait schizotypy to increase psychosis risk, to develop our understanding of the impact of trait risk on functioning within the general population.

Recent research has been concerned with the association between schizotypy and neurodevelopmental risk factors for psychosis (e.g. Hans et al., 2009; Soler et al., 2017). This thesis will focus on: neurological soft signs (NSS) as biological indicators of aberrant neurodevelopment, and; language processing abnormalities, represented through the use of a dyslexia sample, as well as semantic processing as a specific language domain of interest. Affective factors have also been identified as relevant in risk for psychosis (e.g. Barkus et al., 2010; Cella et al., 2013; Cohen et al., 2016), with this thesis specifically interested in affective temperament, as well as state psychological distress.

Schizotypal personality trait is one of the most reliable indicators of risk of transition to psychosis (Mason et al., 2004). The combination of schizotypal trait with changeable psychotic-like symptoms (such as hallucinations) is understudied, with many psychosis risk studies instead focused on state indicators of ultra and clinical high risk (e.g. Yung et al., 2006). Psychotic experiences such as auditory-verbal hallucinations (AVH) have an annual incidence of 2.5% in the general population, with 7.4% of these individuals transitioning to psychotic disorder (Linscott and van Os, 2013). Understanding the interaction between trait and state risk will develop our understanding of when these experiences are benign, versus when they are associated with distress. As such, the primary goal of this thesis is to determine whether affective (psychological distress and affective temperament) and neurodevelopmental (NSS and semantic processing) factors are expressed abnormally in psychometrically identified schizotypy, and additionally, whether hallucination predisposition is a contributor to this. Finally, given that dyslexia and the psychosis continuum have features in common (e.g. Richardson, 1994; Shapleske

et al., 1999), this thesis also aims to determine whether NSS and semantic processing neurodevelopmental factors are expressed abnormally in individuals with dyslexia compared to controls.

2 THE PSYCHOSIS CONTINUUM

2.1 Schizophrenia

Schizophrenia affects roughly 0.4% of the world's population (lifetime prevalence rate; McGrath et al., 2008) and is characterised by positive (auditory verbal hallucinations (AVH), delusions), negative (psychomotor poverty, anhedonia), and disorganised (thought disordered speech, disorganised or catatonic behaviour) symptoms (American Psychiatric Association (APA), 2014). Two of these symptoms must be present for a significant portion of time during a one-month period, with continuous signs of disturbance persisting for at least six months (in the form of prodromal, residual, or attenuated disturbances) (APA, 2014). Symptoms need to significantly impair functioning in at least one area of the person's life. Although these psychotic symptoms are most characteristic of schizophrenia, they are also exhibited in other disorders, including neurological disorders such as dementia and temporal lobe epilepsy. These need to be excluded prior to a diagnosis of schizophrenia. Symptom presentation in schizophrenia is extremely variable between individuals (Tsuang, Lyons, & Faraone, 1990), with it being possible for two patients to exhibit no overlapping symptoms (Wing & Agrawal, 2003; Beck et al., 2009).

Categorical distinctions are used to classify psychotic disorders including schizophrenia in the Diagnostic System of Mental Disorders, fifth edition (DSM-5; APA, 2014). These classification systems have been regarded as useful insofar as they aid communication between clinicians, ensure disorders are easily identifiable, and inform decisions related to treatment (Kraemer et al., 2004; Livesley & Jackson, 1992). Yet debate exists as to whether psychotic disorders are categorical. Commonality exists between diagnostic categories, along with heterogeneity within a category. Even when a diagnosis is made often it does not become stable until enough time has elapsed for symptoms to consolidate (McGorry et al., 2009). Furthermore, despite plenty of research into biological markers of schizophrenia, no diagnostic test has been developed that is able to categorically determine whether someone has schizophrenia or not (Wong & van Tol, 2003). This lack of diagnostic specificity for psychotic symptoms is further highlighted by the presence of these symptoms in healthy individuals (Heinrichs, 2005). Symptoms such as AVH and delusions have been consistently reported at attenuated levels in healthy population

samples (for review see Verdoux and van Os, 2002). The continuity in these experiences across non-clinical and clinical boundaries suggests the categorical nature of schizophrenia as depicted in the DSM-5 has pragmatic value from a diagnostic perspective, however does not truly represent the phenomenology of psychotic experiences.

Therefore, taking a lead from other researchers (e.g. van Os et al., 2009; Nuevo et al., 2012), in the context of this thesis, schizophrenia is conceptualised as a disorder existing along a continuum of psychotic experiences, rather than a binary phenotype (present/absent) with sudden onset. The continuum view of psychosis suggests that features of psychosis, and schizophrenia more specifically, should be evident to some extent in the general population. To this end, 28% of the general population have reported psychotic symptoms in their lifetime, with these symptoms able to be accounted for by the same aetiological factors associated with schizophrenia (van Os et al., 2009). Furthermore, prior to the onset of psychosis, symptoms have been found to become significantly more frequent (Hafner, 2000), suggesting phenomenological continuity between subclinical and clinical manifestations of psychosis.

Although the aetiology of schizophrenia has been well researched, the causes still remain unclear. Heritability estimates indicate approximately 70% of schizophrenia risk is attributable to genetic factors, believed to result from a combination of multiple genes each contributing minor effects (Sullivan, Daly and O'Donovan, 2012). Early prenatal and perinatal processes have also been implicated, including low birth weight, caesarean section, hypoxia, and being born in winter months (e.g. Cannon, Jones, & Murray, 2002; Khandaker et al., 2013). Longitudinal studies of children at familial risk who go on to develop schizophrenia have evidenced neuromotor, cognitive, social, as well as functional and structural brain changes from early childhood (e.g. Seidman et al., 2006; Lawrie et al., 2008; Arango et al., 2008; Reichenberg et al., 2010). Throughout the early prodromal phase cognitive deficits (Fusar-Poli et al., 2012) and reductions in grey matter volume are also found (Jung et al., 2012; Rapoport et al., 1999).

2.2 Schizotypal personality disorder

Although thought of as less severe on the psychosis continuum compared to schizophrenia, schizotypal personality disorder (SPD) is also defined by impairments

in psychosocial functioning, and is found in the personality disorders chapter of the DSM-5 (APA, 2014). Along the psychosis continuum, SPD is believed to sit in between schizophrenia and subclinical psychotic experiences (Esterberg and Compton, 2009). Whilst SPD is characterised by many of the same pervasive and disturbing perceptual aberrations, interpersonal dysfunctions, and disorganised speech and behaviour as that found in schizophrenia, the symptoms do not always lead to medicalization or hospitalisation (Siever & Davis, 2004). The cognitive deficits associated with SPD are also attenuated compared to that found in schizophrenia, with less severe cognitive deficits believed to be due to greater frontal lobe reserves, and the protective capacity to recruit from wider brain regions to compensate for dysfunctional areas (Buchsbaum et al., 2002). The prevalence rates of SPD range from 0.6% (Torgersen, Kringlen, & Cramer, 2001) to 4.6% (Johnson et al., 2000), with higher incidence rates in relatives of schizophrenia patients (Siever, Bernstein, & Silverman, 1996), highlighting the strong genetic liability for links to schizophrenia (Cadenhead & Braff, 2002; Kendler et al., 1993). Studies have found that the genetic relationship between schizophrenia and SPD is more pronounced in the negative symptoms (cognitive deficits, interpersonal abnormalities) than the positive symptoms (perceptual aberrations) (Ingraham & Kety, 2000; Torgersen et al., 1993). Likewise, results from a twin study found that positive and negative symptom clusters may be the result of two separate heritable dimensions, rather than a product of one underlying disorder (Kendler et al., 1991). These findings suggest that whilst the negative deficit-like symptoms may be heritable as a "spectrum phenotype", the positive symptoms may exist as an independent genetic factor related to psychosis ("psychotic phenotype"), and not acting as a distinct product of schizophrenia (Siever & Davis, 2004).

Owing to the genetic overlap between schizophrenia and SPD, many phenomenological studies have found that the presentation of SPD is markedly similar to the prodromal characteristics of schizophrenia pathology (Bedwell & Donnelly, 2005). As a consequence, a significant number of individuals diagnosed with SPD later go on to develop schizophrenia (Walker et al., 2004; Parnas et al., 2011).

2.3 Schizotypy

The existence of schizotypal personality traits (without the pervasive functional impairment of the personality disorder) has been associated with subsequent transition to schizophrenia illness, with retrospective studies demonstrating the presence of these traits before illness onset (Woods et al., 2009; Salokangas et al., 2013). Prospective studies have also shown an increased risk for psychotic disorders for those who have elevated schizotypal traits (Gooding et al., 2005; Kwapil et al., 2013). Together, the occurrence of psychotic symptoms, SPD and schizotypal traits make up the psychosis continuum and highlight the overlap between schizophrenia illness and subclinical schizotypal personality.

The term schizotypy was first introduced by Rado (1953) to describe a broad range of schizophrenia-like traits and impairment. Due to its position on the psychosis continuum, the psychometric assessment of schizotypy is able to provide valuable information for psychosis risk in the general population. Schizotypy has been linked to an increased frequency of anomalous experiences (Barkus and Lewis, 2008; Barrantes-Vidal et al., 2013a), as well as schizophrenia-spectrum symptoms (Kwapil et al., 2014).

Schizotypy can be assessed via structured clinical interview (e.g. Structured Interview for Schizotypy; Kendler, Lieberman, & Walsh, 1989), or through self-report psychometric questionnaires. The scores obtained from self-report measures have been found to correlate highly with structured interview assessments (Raine, 1991; Konings et al., 2006). For the purpose of this thesis the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) will be used. This scale was chosen because it has demonstrated validity across a range of research projects, including non-clinical college samples (e.g. Fonseca-Pedrero et al., 2014), genetically-at risk samples (e.g. Vollema et al., 2002), and; cross-sectional interview designs (e.g. Raine, 1991). Due to the SPQ's broad measurement of schizotypal phenomena it taps into the personality features of schizotypy, as well as subclinical psychotic symptoms. The SPQ is also the most conservative measure of schizotypy, and is sensitive enough at the upper limit to identify those with levels of schizotypy seen clinically in SPD (Raine, 1991). Kwapil and Chun (2015, pp. 21.) noted that the SPQ's advantage lies in its ability to provide a continuous and multidimensional measure of schizotypy in general population samples. This thesis is interested in

identifying those in the general student population who are at psychometric risk for psychosis. Accordingly, the SPQ is believed to be an appropriate measure.

The psychometric structure of schizotypy has been the focus of much research, with the majority of findings in support of a 3-factor structure underlying the personality trait (Vollema & van den Bosch, 1995; Cicero & Kerns, 2010), consisting of positive, negative, and disorganised dimensions (Raine et al., 1994; Raine, 2006). The positive dimension is also often referred to as Cognitive-Perceptual schizotypy, and is made up of unusual perceptual experiences and delusions. Negative schizotypy (Interpersonal schizotypy) is characterised by the absence of emotional, social and physical functions, such as an inability to experience pleasure (anhedonia), loss of motivation or drive (avolition), and less interest in socialising. Disorganised schizotypy consists of eccentric/odd behaviour and disordered speech and thoughts. The three dimensions of schizotypy are substantially similar to the factor structure of schizophrenia (Rossi & Daneluzzo, 2002; Wuthrich & Bates, 2006), and are also invariant across culture, gender and religious affiliation (Reynolds et al., 2000).

More recent investigations based on item-level factor analysis have revealed 4 and 5 factor structures to schizotypy (e.g. Stefanis et al., 2004; Wuthrich and Bates, 2006). For example, Bove and Epifani (2012), utilising item-level confirmatory factor analysis of the SPQ, reported 4 factors which consisted of; unusual beliefs and experiences, mistrust, social anhedonia, and eccentric/odd behaviour. However Chmielewski and Watson (2008) reported 5 SPQ dimensions, corresponding to unusual beliefs and experiences, social anxiety, social anhedonia, mistrust, and eccentricity/oddity. The psychometric structure is reported to differ significantly based on the type of analysis that occurs (subscale level or item level; Chmielewski and Watson, 2008), as well as the type of sample used to investigate factor structure (community or undergraduate; Zhang and Brenner, 2017). Given that the factor structure of schizotypy as measured by the SPQ is based on a prevailing theoretical model of three-factors (Raine et al., 1994), this will be the factor structure adopted in this thesis. Investigations into the three schizotypal personality traits come from two main approaches: the clinical (or quasi-dimensional), and the individual differences (or fully-dimensional) approaches.

The clinical approach is based on the work of Meehl (1962; 1989; 1990), who states that schizotypy is a psychological and personality organisation found in

people who possess a single gene called the schizogene. This gene is said to cause schizotaxia: a genetic vulnerability to the development of psychosis. Schizotaxia is not sufficient itself to cause psychosis, rather, it is believed to interact with environmental risk factors throughout a person's life. The gene-environment interaction determines whether an individual will develop a psychotic disorder or not (Lenzenweger, 2006). From the point of genetic vulnerability this approach is considered categorical; either an individual possesses the genetic vulnerability or they do not. The population base rate of schizotaxia is suggested to be 10%, with evidence supporting this approach obtained through taxometric studies (e.g. Haslam, Holland, & Kuppens, 2012; Waller & Meehl, 1998), although these studies are not fully conclusive (e.g. Rawlings et al., 2008). Under this model, the focus is on transitions from subclinical stages to psychosis, with 10% of schizotypes thought to decompensate into schizophrenia, corresponding with the 1% prevalence rate of schizophrenia (Meehl, 1990).

Contrastingly, the individual differences approach sees schizotypy as a normally distributed personality trait, which is present to some extent across the entire population, and at its extreme high, results in risk for schizophrenia (Claridge, 1972; 1987). Thus it sees schizotypy as both normal variations in personality, and possible, but not inevitable, predisposition to psychosis. The high prevalence of unusual perceptual aberrations and other psychotic symptoms in the general population are taken as evidence for this approach (Lincoln, 2007; Scott et al., 2008; Hanssen et al., 2005; Johns & van Os, 2001). The individual differences approach is also consistent with major theories depicting continuity between clinical and non-clinical psychotic populations (Linscott & van Os, 2010; Allardyce et al., 2007). Under these theories multiple genes and environmental factors are thought to interact, to determine an individual's expression of risk (Figure 2.1). Additionally, the model proposed by Claridge and colleagues offers explanation for the adaptive advantage often reported in association with schizotypy, such as enhanced creativity (e.g. Claridge & Blakey, 2009).

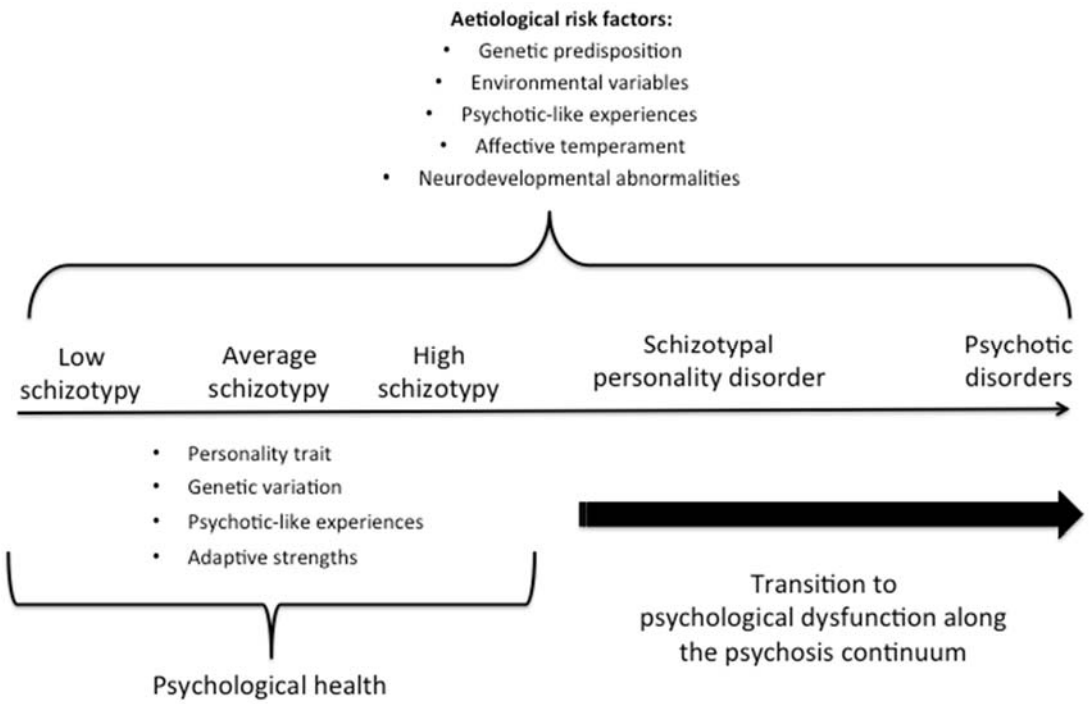

Figure 2.1. Continuum model of psychosis, with schizotypy conceptualised from an individual differences approach.

Both the clinical and individual differences approaches are alike in that they acknowledge variation in schizotypy throughout the population, however the approaches differ in their conceptualisation of the nature of the distribution (e.g. Rawlings et al., 2008; Haslam, Holland, Kuppens, 2012; Johns & van Os, 2001). The high prevalence of aberrant perceptions in the general population suggests there is not a clear distinction between what is considered a normal versus abnormal psychotic experience. As a result, for the purpose of this thesis the fully dimensional approach will be the theoretical standpoint adopted. By assuming the individual differences approach, schizotypy research is highly relevant to understanding the aetiology of psychosis. It also allows research to take place without the additional confounds of medicalization, hospitalisation and other factors intrinsic with psychotic illness. For this reason in particular, over the past several years there has been a dramatic increase in the number of studies examining the etiological similarities between schizotypy and schizophrenia (Nelson et al., 2013).

Methodologies employed by studies utilising the SPQ are numerous. Given that schizotypy is often considered a dimensional construct, many studies have used statistical methods that correspond with a continuous variable, such as regression

analyses (e.g. Kline et al., 2012; Barrantes-Vidal et al., 2013b). However other studies have chosen to adopt a grouping statistical approach, which also does not preclude the dimensional nature of schizotypy (e.g. Cameron, Kaplan and Rossell, 2014; Oestreich et al., 2015). Group approaches may be considered suitable especially in exploratory research designs, where the relationship between the variables is not well known. In these cases group approaches are appropriate, given that schizotypy has been shown to act differently at the top and bottom end of the continuum (Nettle, 2006). By using a grouping approach in these instances the initial exploration of schizotypy at different levels can be explored without assuming that the relationship remains consistent across the spectrum of schizotypy.

3 THE PSYCHOSIS CONTINUUM AND CO-OCCURRING RISK FACTORS

3.1 Auditory-verbal hallucinations (AVH)

The continuum view of psychosis suggests that signs and symptoms of the clinically diagnosed disorder are also present, often in attenuated forms, in the non-clinical population. One focus of this thesis is the experience of auditory-verbal hallucinations (AVH), as well as other hallucinatory experiences. AVH are reliably shown to be present in the general population, and therefore lie on a continuum of normal and psychotic experiences (Nuevo et al., 2012). The co-occurrence of symptoms, such as hallucinations, with trait markers like schizotypy, is under investigated in comparison to clinical and ultra-high risk markers. The study of hallucinations with schizotypy also affords opportunities to understand how these factors interact, to impact on the expression of other neurodevelopmental and affective risk factors for psychosis.

The following section is largely based on parts of a review published by the author of this thesis. See Appendix J for published version. Citation:

de Leede-Smith S, Barkus E. (2013). A comprehensive review of auditory verbal hallucinations: lifetime prevalence, correlates and mechanisms in healthy and clinical individuals. *Frontiers in Human Neuroscience, 7*, 1–25.

Auditory verbal hallucinations (AVHs) are a sensory experience that takes place in the absence of any external stimulation whilst in a fully conscious state (Beck and Rector, 2003). AVH occur with a sufficient similarity to the real percept that the individual attributes the event to be out of his/her own control (David, 2004). To date, the mechanism and pathophysiology of AVH, although widely speculated upon, are still largely unknown.

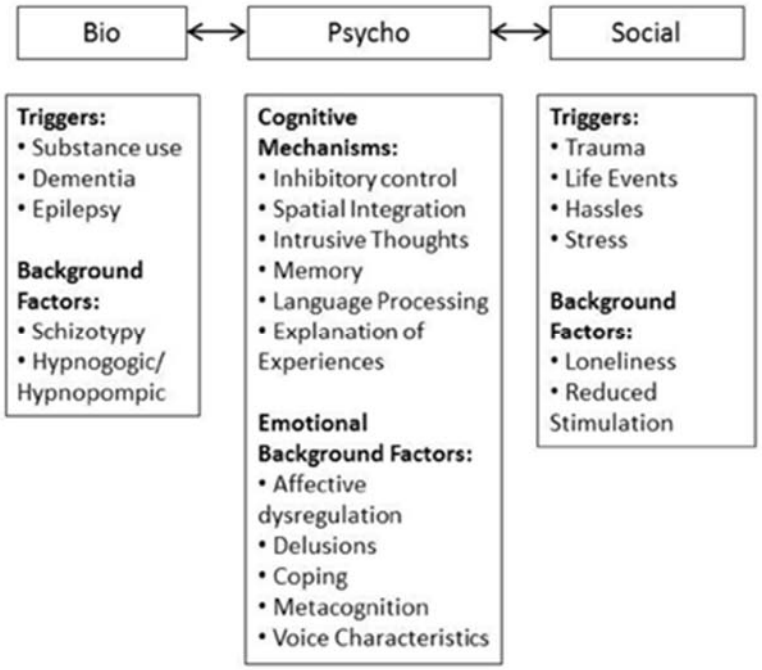

Figure 3.1. Biopsychosocial framework used in the summary of the AVH literature.

The current review aims to examine the phenomenology of AVH. We will consider the literature and data available in clinical and non-clinical groups. Extrapolating differences between clinical and non-clinical hallucinatory experiences provides an understanding of different developmental trajectories, characteristics of the experience and modes of interpretation for the voice hearer. As such, a review is timely which investigates the similarities and differences between the pathological voice hearing experience and AVH which are considered otherwise healthy modes of functioning. By integrating research in this very much evolving field, we can move forward toward a conceptualization of the intricate mechanism(s) responsible for the voice hearing experience.

The framework used in the current review is summarized in Figure 3.1. The biopsychosocial model provides a system where triggers, maintaining and moderating factors can be incorporated informatively. The domains interact with one another on a causal and mechanistic level, demonstrating the etiological complexity of AVH at any point along the lifespan and in both clinical and non-clinical groups. Domains can be conceptualized as background factors that are stable, may be biologically underpinned, and provide a backdrop against which other factors interact. These interacting factors can be mechanisms or triggers, the former contributing to maintenance and the latter initiating onset. However, the relationships

between these variables are not discrete, the content of AVH can be informed by social and personal experiences. For example, the triggering environmental stressor can provide information for AVH content. This creates an intricate picture. However, given the complexity of the AVH experience it is not surprising that the factors, which both initiate and maintain AVH are multifaceted and not mutually exclusive.

3.1.1 Prevalence of AVH and related phenomena

AVH are at their most prevalent in diagnosed psychotic disorders such as schizophrenia and schizoaffective disorder (Sartorius et al., 1986) but also occur in other disorders including bipolar disorder, substance intoxication and organic dementias. Recent research has focused on the existence of AVH in general population samples (Moritz and Larøi, 2008; Sommer et al., 2010; Daalman et al., 2011a,b; Temmingh et al., 2011; Larøi et al., 2012; Stanghellini et al., 2012). Epidemiological studies have estimated the prevalence of AVH to be between 5 and 28% in the general population (Tien, 1991; van Os et al., 2000; Johns et al., 2004; Scott et al., 2006). Johns et al. (2002) found 25% of individuals reporting hallucinatory experiences met the diagnostic criteria for a psychotic disorder; however that leaves 75% of people experiencing AVH who are considered otherwise healthy. Possible implications (which are by no means mutually exclusive) for the existence of non-clinical AVH are:

1. Healthy AVH may present as an isolated symptom and may not be related to any sort of predisposition for a psychotic disorder (Daalman et al., 2011a,b).
2. AVH may form part of a genetic predisposition toward psychotic illness. They can co-occur alongside other attenuated psychotic symptoms including paranoid ideation, odd/unusual behaviour, delusions and inefficient cognitive processing (Krabbendam et al., 2005).
3. AVH may lie on a continuum of risk ranging from normal experiences to pathological psychotic (Johns and van Os, 2001) suggesting that clinically relevant AVH could be an extension of the processes occurring in otherwise healthy hallucinators.

The prevalence of voice hearing in adult non-clinical populations is roughly the same as that in children, ranging from 10 to 15% (Tien, 1991; Sommer et al., 2010). The most common experiences reported by non-clinical adults take place on average every 3 days, for 2–3 min, are controllable for around 60% of the time and cause little to no distress or disruption to daily life (e.g., Daalman et al., 2011a). However, there do seem to be some healthy individuals who experience hearing voices to the same frequency and qualities as clinical patients with schizophrenia (Honig et al., 1998; Faccio et al., 2012). Given that the majority of childhood AVH resolve prior to adolescence (Bartels-Velthuis et al., 2011b), the rates in adulthood suggest that there are a significant group of individuals who develop hallucinations during adolescence and early adulthood, which persist onward.

3.1.2 Comparison of clinical and non-clinical hallucinations in adult populations

In a comparison of the phenomenological features of adult voice hearers (Table 3.1), it is evident that components such as the localization, number of voices, and loudness of the voice hearing experience are largely consistent between clinical and non-clinical groups. Therefore, examining which features distinguish voice hearing in clinical groups from healthy voice hearers can derive meaningful information. Compared to AVH in schizophrenia (referred to as a "clinical" population in this section and including those with psychosis), non-clinical AVH have been found to occur much less frequently, and usually occur after specific conditions such as high stress or sleep deprivation (Larøi et al., 2012). The most commonly reported difference between healthy and clinical voice hearers is the emotional valence of the voice (Honig et al., 1998; Choong et al., 2007; Sommer et al., 2010), with a negative emotional appraisal of the voice having a predictive value of 88% for the presence of a psychotic disorder (Daalman et al., 2011a). Other phenomenological differences between the groups include a reduction in perceived control for psychotic AVH, as well as a higher frequency of AVH, and later age of onset (average of 21 years) when compared to healthy voice hearers (average of 12 years) (Daalman et al., 2011a). On the other hand, factors such as the loudness of the voice, attribution of source and perceived location all remain largely consistent between the groups, which is suggestive of AVH differing primarily in terms of severity, rather than them

being separate phenomena. Some authors have gone as far as to say that voice hearing may be adaptive for some healthy individuals (Faccio et al., 2012).

Apart from differences in those factors that may predispose individuals to experience AVH, there are a number of cognitive capacities that also distinguish clinical and non-clinical voice hearers, both of whom are distinguishable from healthy volunteers. These differences in cognitive capacities lend weight toward the view that there may only be a partial overlap in the healthy and clinical AVH experiences. A cognitive factor that has been found to distinguish clinical from non-clinical AVH is inhibitory control. Inhibitory control and intentional cognitive inhibition specifically, is the ability to inhibit intrusive memories and thoughts. Poor intentional cognitive inhibition has been specifically related to AVH above and beyond any other negative or positive psychotic symptoms (Waters et al., 2003). This poor inhibitory control has been replicated and extended in subsequent studies concerned with the prevalence and frequency of AVH in schizophrenia (Badcock et al., 2005; Soriano et al., 2009) and healthy individuals with high hallucinatory predisposition (Paulik et al., 2007). The relationship between AVH and intentional cognitive inhibition may be associated with executive resources in the prefrontal cortex (Badcock and Hugdahl, 2012). Whilst it seems that both clinical and healthy AVH groups have problems in inhibitory control along a gradient of severity (Waters et al., 2003; Paulik et al., 2007), Paulik et al. (2008) suggests the source of intrusions may be related to emotional dysregulation in non-clinical groups, whereas for clinical populations the source may relate more to impaired memory processes. This would account for the greater frequency of intrusions in clinical compared to non-clinical groups (Badcock et al., 2008; Daalman et al., 2011b).

Table 3.1. *Phenomenological characteristics of AVH in clinical and non-clinical groups.*

	Clinical (confirmed psychotic disorder) AVH	**Non-clinical AVH**	**Can distinguish between clinical and non-clinical groups?**
Localisation	- Inside head (near ears) (Daalman et al., 2011) - Heard via the ears (78%) (Romme & Secher, 2000)	- Inside head (further from body) (Daalman et al., 2011) - Heard via ears (57%) (Romme & Escher, 2000)	No
Explanation of origin	- 50% External (Daalman et al., 2011; Nayani & David, 1996) - Either inside or outside the head (hard to distinguish) (Stephane et al., 2003; Copolov, Trauer & Mackinnon, 2004; Nayani & David, 1996)	- 60% external, 40% internal (Daalman et al., 2011) - External source-mostly benevolent spirits (Sommer et al., 2010)	No
Loudness	- Little softer than own voice (Daalman et al., 2011)	- Little softer than own voice (Daalman et al., 2011) - 36% rated their voices as 'normal' in loudness (Lawrence, Jones & Cooper, 2010)	No
Voices speaking in third person	- 50% (Daalman et al., 2011) - 39% (Romme & Escher, 2000)	- 25% (Daalman et al., 2011) -27% (Romme & Escher, 2000)	Yes
Controllability	- 20% of the time (Daalman et al., 2011) - 17% of the time (Romme & Escher, 2000)	- 60% of the time (Daalman et al., 2011) - 87% of the time (Romme & Escher, 2000)	Yes
Number of different voices	- 11.44 (Daalman et al., 2011)	- 7.62 (Daalman et al., 2011) - 51% heard only one voice (Lawrence, Jones & Cooper, 2010)	Yes
Frequency	- One every hour (Daalman et al., 2011; Honig et al., 1999)	- One every 3 days (Daalman et al., 2011; Honig et al., 1999)	Yes

		- 25% heard voices several times a day, 37% had not heard lately (Lawrence, Jones & Cooper, 2010)	
Duration	- 40 minutes (Daalman et al., 2011) - Continuous (Honig et al., 1999)	- 2-3 minutes (Daalman et al., 2011)	Yes
Types of voices experienced	- Commenting voices (72%) (Romme & Escher, 2000)	- Commenting voices (18%), voices speaking with each other (11%) (Sommer et al., 2010) - Commenting voices (47%) (Romme & Escher, 2000)	Yes
Mean age first experiencing voices	- 21 years (Daalman et al., 2011) - 11% onset before 12 years (Honig et al., 1999)	- 14 years (Sommer et al., 2010) -12 years (Daalman et al., 2011) - 40% onset before 12 years (Honig et al., 1999)	Yes
Disturbance to daily functioning	- Moderate to severe distress, disruption (Daalman et al., 2011) - Significant disturbances to daily functioning (Honig et al., 1999) - Disrupting daily life in 100% of voice hearers (Romme & Escher, 2000) - Significant distress and disruption to the person (Evensen et al., 2011)	- Disrupting daily life in 9% of voice hearers (Sommer et al., 2010) - Almost no discomfort, disruption to daily life (Daalman et al., 2011) - Disrupting daily life in 20% of voice hearers (Romme & Escher, 2000)	Yes
Emotional valence of voice	- Majority of voices are unpleasant/annoying (Daalman et al., 2011) - 100% of voice hearers experience negative voices (Honig et al., 1999; Romme & Escher, 2000)	- 4% of voice hearers experience negative content only (Sommer et al., 2010) - Seldom unpleasant voices/content (Daalman et al., 2011) - 53% of voice hearers experience negative voices (Honig et al., 1999; Romme & Escher, 2000) - Are evaluative of others but have mundane content (Leudar et al., 1997)	Yes

Effect on individual	- Frightening effect (78%); upsetting effect (89%) (Romme & Escher, 2000) - Feelings of anxiety or depression (Hoffman et al., 2008; Freeman & Garety, 2003) - 75% moderate-severe anxiety ratings, 81% moderate-severe depression ratings (Chadwick et al., 2000)	- Frightening effect (none); upsetting effect (27%) (Romme & Escher, 2000) - Over 50% fell within the normal range for anxiety and depression measures (Lawrence, Jones & Cooper, 2010)	Yes
Childhood trauma	- 33% Childhood sexual abuse (Honig et al., 1999) - 53% childhood sexual abuse (Read & Argyle, 1999) - 38% childhood sexual abuse (Offen et al., 2003) - Experience of early trauma (Fowler et al., 2006) - 75% experienced some sort of traumatic event (Escher et al., 2004)	- Significantly more prevalent than healthy controls (Sommer et al., 2010)	No
Family history axis I disorders	- Increased risk of AVH in those who have biological relatives with the disorder (Aukes et al., 2008; Goldman et al., 2009; Erlenmeyer-Kimling et al., 1997)	- Sig more prevalent than healthy controls (Sommer et al., 2010)	No

Compared to healthy non-voice hearers, higher levels of negative affect are common to AVH in schizophrenia (Delespaul et al., 2002) and otherwise healthy voice hearers (van't Wout et al., 2004; Allen et al., 2005) both during hallucinations and also when hallucinations are not present (for review see Freeman and Garety (2003)). This is suggestive of emotional arousal possibly premeditating hallucination onset, or being a factor involved in the occurrence of these perceptual experiences (Slade and Bentall, 1988). Anxiety has the most predictive power for the predisposition to hallucinate in non-clinical groups (Paulik et al., 2006), over and above depression and stress ratings. Anxious non-clinical individuals have been shown to have a greater number of hallucinatory experiences (Allen et al., 2005), whilst in clinical voice hearers, there is a significant relationship between positive symptoms (hallucinations) and anxiety, rather than depression (Norman et al., 1998). Depression in clinical groups however, has been specifically associated with AVH of greater severity compared to their non-depressed counter parts (Smith et al., 2006). This points to a dynamic whereby higher depression ratings may be indicative of greater severity of the AVH to the individual, whilst higher anxiety is more strongly related to the level of distress those AVH illicit (Hartley et al., 2012).

Another area of dissimilarity between clinical and non-clinical AVH groups concerns lateralization of language functions during verbal fluency tasks (Diederen et al., 2010). Decreased lateralization of language function has been well documented in the schizophrenia literature (for review see Li et al. (2009)). In healthy participants, verbal fluency tasks typically activate the prefrontal cortex in the left hemisphere, which has also been reported in healthy voice hearers (Diederen et al., 2010). This implies that the failure to establish left hemisphere dominance for language is not a specific mechanism that underlies AVH. However, it does not rule out the possibility that decreased language lateralization may be related to the pathological nature of AVH specifically, such as the frequency of negative emotional content which differentiates them from healthy hallucinatory experiences.

A comparison of the previously discussed phenomenological characteristics of AVH in adults across clinical and non-clinical groups has been provided in Table 3.1. When comparing information regarding the perceptual quality of the voice hearing experience in adult populations, it can be seen that features such as the localization, number of voices, and loudness of the voice hearing experience are largely consistent between clinical and non-clinical voice hearers. Antecedent

features that may be associated with the onset of the voice hearing experience also seem similar between clinical and non-clinical groups. This could point to common developmental trajectories for AVH in both groups, with similar environmental and biological factors associated with the onset of AVH. As a result it can be asserted that it is not the experience of voice hearing per se, or features predisposing AVH onset that are associated with psychological dysfunction.

The most notable differences between healthy and clinical voice hearers seem to be the emotional valence of the voice and the distress voice hearing elicits. This seems to be particularly in regard to the controllability and the increased frequency of the experience for clinical voice hearers. These differences may stem from an interaction between:

1. Cognitive mechanisms: appraisal of the content; coping; thoughts/delusions related to the experience; and, inhibitory control;
2. Emotional regulation: appraisal of the emotional tone of the experience; metacognitive processes underpinning emotions and general metacognitive capacity. These dictate the emotional tone and loading of thoughts, specifically through experiential avoidance (Goldstone et al., 2012) or metacognitive beliefs in general (e.g., Varese et al., 2011).

One of the major cognitive mechanisms suggested as a component cause in the generation of AVH experiences is a lack of inhibitory control. Instinctively appealing, such a conceptualization satisfies the notion reported in many phenomenological studies of a lack of personal control over the generation and subsequent experience of voice hearing in both clinical and non-clinical groups. Impairments in intentional cognitive inhibition (the conscious active suppression of mental processes/thoughts) specifically have been put forward as factors linked to AVH experiences. This relationship is independent of any association to other positive, negative and disorganized symptoms of schizophrenia (Waters et al., 2003), demonstrating its specific association to AVH as a symptom unto itself. Intentional cognitive inhibition deficits follow a gradient of severity whereby non-clinical hallucinators demonstrate an impairment intermediate to clinical hallucinators (at the extreme) and healthy members of the general population (where little/no deficit exists) (Waters et al., 2003; Paulik et al., 2007). This relationship mirrors our

observations of the phenomenology of clinical and non-clinical AVH experiences, lending to its significance in the generation of hallucinatory phenomena.

If deficits in intentional cognitive inhibition are implicated in the experience of AVH for all individuals, what component must interact with this dysfunction to create clinically significant AVH experiences in some people, but not in others? This difference is believed to lie in the way in which emotions are regulated, appraised and controlled for clinical vs. non-clinical groups. High levels of negative affect, primarily anxiety, depression and stress, have been documented both prior to and at AVH onset for clinical voice hearers (for review see Freeman and Garety (2003)). Such emotional states are suggested to be involved in the development of the AVH rather than a consequence of it, as levels of negative affect have been found to fall (rather than rise) at the end of a hallucinatory episode, and increase immediately prior to an episode (Delespaul et al., 2002). So how is it that this dysregulation of emotion acts to create differences in the appraisal of AVH for clinical and non-clinical voice hearers? It has been put forward that high states of anxiety act to exacerbate deficits in intentional cognitive inhibition by increasing intensity above a critical threshold (Slade and Bentall, 1988) which act to create distressing intrusive thoughts (Paulik et al., 2006). Under this hypothesis, the individuals control over intrusive cognitive events is compromised even further by a heightened state of arousal, which impairs that person's ability to function rationally and with clarity. It is also hypothesized that under this increased state of arousal, the individual's control regarding the feasibility of their metacognitive beliefs is compromised. Patients with AVH score higher on metacognitive beliefs in relation to uncontrollability and worry (Baker and Morrison, 1998). When these metacognitive beliefs occur in the context of AVH, they may act to exacerbate the negative emotional states, which are already present as a result of AVH onset. The interplay between these beliefs and an already heightened mood state may dictate the appraisal of a negative emotional tone for the individual, and place emphasis on ways of thinking associated with paranoia, anxiety and distress. Although feasible, this line of reasoning requires further research before claims to its plausibility can be made.

What seems to be pertinent to present research is the identification of features that allow these experiences to be dealt with in a beneficial manner. What strategies do non-clinical voice hearers adopt which allow them to regulate their experiences in an emotionally beneficial manner? It seems that they may possess coping strategies

that allow them to deal with their experiences in the face of highly stressful or traumatic events. Research concerning the adaptive strategies of non-clinical voice hearers has suggested that an increased use of adaptive emotional regulation strategies (such as reappraisal) may allow the individual to adequately cope with the distressing nature of their experiences (Larøi, 2012). In contrast, clinical voice hearers have been found to use a greater number of maladaptive emotional regulation strategies (such as suppression) (van der Meer et al., 2009; Badcock et al., 2011). As a result, this leaves them in a position where they are unable to appropriately cope with their experiences, resulting in higher levels of distress and a negative emotional appraisal of the voice hearing experience. However, the precise mechanisms and processes which are involved in regulating the emotional appraisal associated with hallucinatory experiences has not yet been disseminated. As such, an understanding of these mechanisms is pertinent to the conceptualization of the differing developmental pathways leading to either: (a) clinically relevant AVH which cause distress and impairment, or; (b) healthy AVH experiences which allow the individual to function adaptively in society.

3.1.3 Significance of the schizotypal personality trait

Under a continuum model of psychosis, schizotypy is believed to represent a trait-like marker of schizophrenia personality which is evident in the general population (Johns et al., 2004). Schizotypy is readily regarded as a biological precursor for hallucinatory experiences, with a common etiologic component being identified between hallucinatory symptoms and schizotypy in non-clinical (Mata et al., 2000, 2003) and clinical (Grove et al., 1991; Kwapil, 1998; Gooding et al., 2005) groups. Accordingly, an increase in this personality trait has been conceptualized as part of the at-risk mental health criteria (ARMS; e.g., Wood et al., 2011). Individuals who score highly on schizotypy are more likely to display a propensity for anomalous experiences including AVH (e.g., Barkus et al., 2007). It involves qualities such as odd behaviour, unusual perceptual experiences, aloofness, introversion, and cognitive disorganization (Raine, 2006). The personality trait is reported to decrease with age (Rössler et al., 2007), being at its peak in adolescence (Fossati et al., 2007), although there are limited investigations of its base rate in children. The most robust difference of healthy voice hearers compared to the general population is a significantly greater level of overall schizotypy (Sommer et al., 2010). Since AVH

are a positive symptom of psychotic illness, voice hearers would be expected to display a significant increase in positive schizotypy only, as it is a trait vulnerability for the experience of hallucinatory phenomena (Tsakanikos and Reed, 2005). However, the difference between healthy voice hearers and controls reflects a general increase in all schizotypal dimensions. This could be indicative of the presence of AVH being associated with subclinical levels of all schizotypal phenomena. In combination with an increased family loading for psychosis (Sommer et al., 2010), these findings may be suggestive of a genetic predisposition for psychosis for those experiencing AVH who have increased schizotypal levels and a genetic liability. Evidence for an etiologic component linking hallucinatory predisposition and schizotypy has also been illustrated by Mata et al. (2003) through the identification of relatives of psychotic patients who display significantly elevated schizotypy levels compared to controls.

It seems clear that an understanding of the phenomenology of clinical voice hearing as a symptomatic component of psychosis has reached a stage of competent understanding. Perhaps the time has come for psychosis research to begin focusing on stable risk components such as schizotypy, rather than symptoms like AVH. It has become clear that AVH are a transdiagnostic symptom which cannot give us an indication of outcome, especially one specific to psychosis. In clinical staging models (Wood et al., 2011) early phases must focus on stable rather than transitory features of pathology which are able to separate high-risk individuals from their counterparts. Clinical features such as AVH seem no longer able to provide us with such a distinction. As a result, a move toward early indicators of risk, such as neurological soft signs and schizotypy appear to be a much more feasible line of enquiry.

In regards to the measurement of schizotypy and AVH, it is acknowledged that the Cognitive-perceptual factor of the SPQ and the LSHS tap into similar experiences, however the nature of the constructs are conceptually different, with Cognitive-perceptual schizotypy recognised as a trait component of schizotypal personality, and hallucination predisposition a state and more dynamic factor which is more fluid and can change in response to situational variables. There are 9 questions under Cognitive Perceptual schizotypy which relate specifically to unusual perceptual experiences, with hallucinations being a component of these. The other 24 questions capture ideas of reference, odd beliefs or magical thinking, and paranoid

ideation/suspiciousness. It also needs noting that many of the items on the LSHS relate to vivid imagery with only the few later items capturing what would be considered clinically relevant hallucinations. Conceptually there is a proposed difference in the nature of the constructs, with the questions on the Cognitive-perceptual schizotypy subscale worded to capture experiences and behaviours in general, and therefore are more stable and trait like, while hallucination predisposition as represented by specific measures such as the LSHS could be viewed as a state and more dynamic factor. Therefore Cognitive Perceptual schizotypy and LSHS do not overlap sufficiently for them to be considered the same construct. Indeed there are people in the general population who experience florid auditory hallucinations but do not experience mental health difficulties (de Leede-Smith & Barkus, 2013; Johns et al., 2014). Additionally, correlations between LSHS and the SPQ subscales have been calculated, with the relationship between LSHS and Cognitive-Perceptual schizotypy found to be moderate and significant (r =.613, n = 746, p =-.000). Whilst this provides evidence for a statistical relationship between the two variables the correlation is only moderate, pointing to the fact there is not a complete overlap. Therefore although there is some degree of statistical overlap, there is the potential for a distinction in their impact on psychosis risk.

To this end, the current thesis is interested in understanding hallucinations from a neurodevelopmental perspective. Hallucination proneness is a subclinical state indicator believed to represent an increased propensity to hear voices/other noises, and see/feel things which are not actually there. People who are predisposed to hallucinations have unusual perceptual experiences, however they are not usually frequent enough to place them in the category of auditory/visual/tactile hallucinations per se. Hallucination proneness is understood to be *state* in nature, given that an individual's propensity to experience AVH shifts depending on their current environment. Lack of social support, increased stress, and discrimination has been associated with the experience of hallucinations (e.g. Wickham et al., 2014).The interaction between state hallucinations with trait factors along the psychosis continuum, such as schizotypal personality, and affective temperament has seen little research attention. These studies are warranted, given that the specificity of the hallucinatory experience as an indicator of risk on its own is limited. Accordingly, these non-clinical but phenotypically similar trait schizotypy and state hallucination factors will be investigated in the current research thesis. Affective

temperament is another trait factor which carries significance along the psychosis continuum, and will be reviewed next.

3.2 Affective temperament

A central aim of this thesis is to investigate possible mechanisms which may contribute to increased trait risk for a psychotic disorder. Affective temperament is one major psychological construct believed to be relevant to risk for psychosis given its role in shaping the way individuals respond to and interpret stressful situations. Temperament is broadly defined as the innate functions that make up an individual's personality (Clark, 2005). These functions consist of affective traits as well as character dimensions. This thesis is concerned only with the psychobiological affective dimensions of temperament: negative temperament and positive temperament. Negative temperament results in a tendency to experience the world as problematic, threatening and frightening, with a heightened experience of aversive mood states and increased reactivity to stress. Positive temperament is associated with an enthusiastic approach disposition and a tendency to experience rewarding and pleasant emotional states (Watson et al., 1988). Maladaptive temperament can increase the likelihood of transition to psychopathology (Widiger, Varheul, & van den Brink, 1999), and therefore may be one of the mechanistic factors in the decline from healthy schizotypal personality to frank psychosis. Compared to controls, schizophrenia patients report increased negative temperament and decreased positive temperament (e.g. Horan & Blanchard, 2003a; Camisa et al., 2005; Horan, Blanchard, Clark, & Green, 2008; Barch et al., 2008). These findings are consistent at different stages of illness (recent onset versus chronic) and different patient status (inpatient versus outpatient) (Horan et al., 2008). Temporal stability has also been established, with affective temperament remaining stable over time and despite changes in symptom status (e.g. Blanchard, Horan & Brown, 2001; Kentros et al., 1997).

Increased negative and decreased positive temperament is also found in psychometrically identified schizotypy (e.g. Ross et al., 2002; Phillips & Seidman, 2008). Negative temperament has been associated with both positive and negative schizotypal traits (e.g. Gooding et al., 2002). However symptomatic states associated with increased negative temperament; such as anxiety and depression, have previously only been associated with the positive schizotypy dimension

(Lewandowski et al., 2006). This may have implications for hallucination predisposition in the at-risk state. For example: current hypotheses have emphasised that it is the interpretation of positive symptoms in a negative way (i.e. attributing the voice to a malevolent source) that determines distress, rather than the experience of hallucinations/delusions per se (Morrison & Baker, 2000). The role of affective temperament in influencing the interpretation and attribution of positive symptoms may therefore be central in increasing the risk of need for care.

Temperament abnormalities have also been preliminarily associated with functional implications across the psychosis continuum. In schizophrenia, increased negative and decreased positive temperament has been associated with heightened reactivity to stress and avoidant coping (Horan & Blanchard, 2003b). Given that heightened stress reactivity is now regarded as central in the pathogenesis of psychotic disorders (Holtzman et al., 2013), the role of affective temperament in this cascade is noteworthy. Schizotypy studies have also recorded an association between increased negative and decreased positive temperament, stress and greater use of avoidant strategies in response to aversive stimuli (Horan, Brown and Blanchard, 2007; MacAulay & Cohen, 2013). Schizotypy also independently results in psychological distress (Lewandowski et al., 2006; Preti et al., 2007; Barkus et al., 2010), which suggests it is not affective traits alone that are driving reduced functioning for those at psychometric risk.

3.3 Psychological distress

Psychological distress is defined as an affective response characterised by unpleasant and/or upsetting emotions, such as depression, anxiety, anger, and irritability, alongside cognitive problems and somatic symptoms (Préville, Potvin and Boyer, 1995). It is understood as a state construct, and therefore is changeable over time and in response to environmental factors. Psychological distress that occurs alongside psychotic-like experiences (PLE's) has been associated with increased risk of transition to psychotic disorder in UHR (Rapado-Castro et al., 2015) and clinical samples (Miller et al., 2003). Distress is a core feature of the high-risk state, with models that identify and classify individuals as UHR requiring that PLE's cause those individuals distress or impairment (Yung et al., 2005).

In trait schizotypy, the literature regarding the relevance of psychological distress is a little more complex. Kline et al (2012) found schizotypy to moderate the

relationship between PLEs and distress, such that higher levels of schizotypy were associated with more PLEs and less distress. Contrastingly, people with fewer schizotypal traits found higher levels of PLEs more distressing. The authors suggested this finding might be due to the increased prevalence of PLEs in schizotypy and overlap in the constructs (Kline et al., 2012). Kline et al. (2012) hypothesised that over time and with repeated exposure, those with high schizotypy may become used to PLEs, and thus respond in an affectively neutral way compared to others for whom this experience is less common and therefore more distressing. This is not to suggest that distress does not have clinical utility in understanding how schizotypy can contribute to risk for psychosis. To this end, Cella et al (2013), using Latent Class Analysis, identified an extreme schizotypy class believed to represent those at increased risk of psychosis. Adolescents from the general population formed three schizotypy classes: minimal schizotypy endorsement, increased positive and disorganised schizotypy endorsement, and increased overall schizotypy endorsement, which was also associated with psychological distress and a family history of psychosis. It seems that when all dimensions of schizotypy are heightened and occur in the context of familial liability, psychological distress may be indicative of an increased risk of psychopathology. What is not yet known however is what mechanisms are contributing to the association between schizotypy and distress, and whether affective temperament has a mediating role in this cascade.

4 NEURODEVELOPMENTAL FACTORS ALONG THE PSYCHOSIS CONTINUUM

An understanding of the aetiological factors that contribute to the psychosis continuum necessitates the investigation of neurodevelopmental risk factors. Accordingly, trait schizotypy, representing an important but not sufficient vulnerability to psychotic disorder, will be focused on, alongside the neurodevelopmental factors of neurological soft signs (NSS), and language processing abnormalities.

4.1 Neurological Soft Signs (NSS)

Neurological abnormalities can be divided into 2 main categories: hard signs and soft signs. Hard signs are those which are localizable to a specific region of the brain, and are usually the result of illness, injury or toxins (Woods et al., 1991). Neurological soft signs (NSS) contrastingly reflect impairments in the connections between different cortical and subcortical brain regions (Bombin, Arango, & Buchanan, 2005). NSS were originally regarded as non-localizable, however recent evidence from brain imaging studies suggests these abnormalities can be partly localizable to specific regions of the brain involved in the cerebello-thalamo-prefrontal brain network (see Zhao et al., 2014 for meta-analysis). Types of NSS vary between scales but are usually grouped into 3 main categories: sensory functioning, motor co-ordination and complex motor sequencing, with these abnormalities also correlating with a wide range of neurocognitive and neuroanatomical abnormalities (Chan et al., 2009). Although not exclusive to psychosis continuum pathology, NSS occur at a significantly higher rate in psychosis compared to healthy controls (Bombin, Arango, & Buchanan, 2005) and patients with other psychological disorders (e.g. Rigucci, 2014). As a result, it has been suggested that NSS represent "target features" of psychotic illness (Tsuang & Faraone, 1999), and are an important focus of research examining the correlates of psychosis risk.

4.1.1 NSS in patients with schizophrenia and their relatives

In the previous three decades research has consistently reported NSS to be present at significantly higher rates in patients with schizophrenia compared to healthy controls (for meta-analyses see Chan et al., 2010a; research subsequent to 2009 in Table 4.1).

Evidence indicates NSS are present to a greater extent in first episode (Dazzzan & Murray, 2002) as well as medication and treatment naïve patients (Venkatasubramanian, et al., 2003) when compared to controls. NSS in first-degree biological relatives are also reported to be intermediate between schizophrenia patients and controls (for meta-analysis see Neelam et al., 2011; research subsequent to 2009 in Table 4.1). These findings suggest that genetic processes, at least in part, underlie NSS. As a result, the utility of NSS as an endophenotype for psychotic disorders has been considered by recent research (e.g. Chan & Gottesman, 2008). An endophenotype is a trait marker which is present independent of the manifestation of the disease/illness (i.e. they are present below the level of overt psychopathological symptoms) (Gottesman & Shields, 1973). Three of the requisite criteria for an endophenotype have been established with reference to the occurrence of NSS in schizophrenia:

1. Association with illness (as NSS occur significantly more frequently in patients compared to controls; Table 4.1);
2. State-independence (NSS are present regardless of whether illness is in the active phase) (Chan et al., 2010a);
3. Familial association (NSS occur in relatives at intermediate rates between patients and healthy controls) (Chan et al., 2010b; Neelam, Garg & Marshall, 2011).

Meeting criteria for classification as an endophenotype is not essential for research investigating NSS along the psychosis continuum, however its status as a potential endophenotype highlights their importance and justifiable consideration here.

Research indicates NSS closely align with negative symptoms of schizophrenia illness (e.g. Prikryl et al., 2006; Jahn et al., 2006; Whitty et al., 2006; Compton et al., 2007; Cveti et al., 2009), with inconsistent findings regarding associations between NSS and positive symptoms (Cuesta et al., 1996; Malla et al., 1997).

Table 4.1. *Summary of studies investigating neurological soft signs in patients with schizophrenia, their relatives, and healthy controls.*

Study	**Participants**	**NSS scale**	**Findings**			
			Sensory functioning	**Motor coordination**	**Complex motor sequencing**	**Total Soft Signs**
Galindo et al. (2016)	Sch: 29 R: 24 C: 37	NSS Scale (Krebs et al., 2000)	Sch>C***	Sch, R>C***; Sch>R***	Sch, R>C***	Sch, R>C*; Sch>R*
Chan et al. (2015)	FEP: 145 (29 with PNS) C: 62	CNI	FEP>C***	FEP>C***; FEP with PNS> FEP without PNS**	FEP>C***	FEP>C***; FEP with PNS> FEP without PNS*
Arabzadeh et al. (2014)	Sch: 30 C: 30	NES	Sch>C**	Sch>C**	Sch>C**	Sch>C**
Hembram et al. (2014)	Sch(FRS): 30 R(FRS): 30 Sch(WFRS): 30 R(WFRS): 30 C: 30	Extended Standard Neurological Assessment	Sch(FRS)>R(FRS), C**; Sch(WFRS)>R(WFRS), C**	n.s.	-	Sch(FRS)>R(FRS), R(WFRS), C***; Sch(WFRS)> R(FRS), R(WFRS), C***; R(WFRS)>C***
Mayoral et al. (2012)	FEP: 110 (Sch: 53; BP: 22; OP: 35) C: 98	NES	FEP>C*** n.s. between diagnostic subgroups.	FEP>C*** n.s. between diagnostic subgroups.	FEP>C*** n.s. between diagnostic subgroups.	FEP>C*** n.s. between diagnostic subgroups.
Prikryal et al. (2012)	Sch: 68 (Remitters: 39 Non-remitters: 29)	NES	Baseline(B)-4 year check-up (C) difference: Remitters: B>C*** Non-remitters: B>C*	B-C difference: Remitters: n.s. Non-remitters: n.s.	B-C difference: Remitters: B>C*** Non-remitters: n.s.	B-C difference: Remitters: B>C* Non-remitters: C>B*
Aksoy-Poyraz et al. (2011)	Sch: 96 R(Si): 66 C: 51	NES	Sch>R(Si), C*	Sch>R(Si), C*	Sch>R(Si), C*	Sch>R(Si), C*

Mechri et al. (2010)	31 siblings of patients with Schizophrenia 60 C	NSS Scale (Krebs et al., 2000)	Siblings>C***	Siblings>C***	Siblings>C***	Siblings>C***
Cveti et al. (2009)	Sch: 66 Positive subtype (P): 36 Negative subtype (N): 30	NES	N>P***	N>P**	N>P**	N>P***

* Significant < 0.05; ** Significant < 0.01; *** Significant < 0.001; n.s.= Not significant at 0.05 level. Sch=Schizophrenia patients; PNS = Prominent negative symptoms; Sch(FRS)= Schizophrenia patients with first-rank symptoms; Sch(WFRS)= Schizophrenia patients without first-rank symptoms; R= Relatives of Schizophrenia patients; R(FRS)= Relatives of Schizophrenia patients with first-rank symptoms; R(WFRS)= Relatives of Schizophrenia patients without first-rank symptoms; R(Si)= Healthy siblings of Schizophrenia patients; FEP= First-Episode patients; BP= Patients with Bipolar disorder; OP= Patients with other psychoses; C= Healthy controls; CNI=Cambridge Neurological Inventory (Chen et al., 1995); NES=Neurological Evaluation Scale. N.B. See Chan et al (2010a) for a systematic review and meta-analysis of studies prior to March 2009.

4.1.2 NSS in schizotypy

The neurodevelopmental view of the psychosis continuum posits that individuals who are at risk of a psychotic disorder should also display attenuated markers of the illness. In the case of NSS, this should result in rates of NSS in schizotypy that are intermediate between patients with schizophrenia and healthy controls. The most robust finding is the positive correlation between schizotypy and total NSS (see Table 4.2). Significant differences between schizotypy and control groups have also been found in many studies, with schizotypy groups recording significantly greater total NSS. Total NSS has also been correlated with negative schizotypy to a stronger degree than positive schizotypy (e.g. Chan et al., 2010c; Kaczorowski et al., 2009; Barrantes-Vidal et al., 2003), which is congruous with findings in schizophrenia studies (e.g. Compton et al., 2007; Cveti et al., 2009). Given the variability in how NSS and schizotypy are associated, it is unclear which aspects of schizotypy are most closely related to NSS.

Interestingly, NSS are found to be significantly higher for those with high schizotypy and Axis 1 psychopathology (i.e. anxiety, depression), compared to both high schizotypes without psychopathology, and healthy controls (Keshavan et al., 2008; Prasad et al., 2009). These findings are in support of an individual differences approach for schizotypy; where high levels of schizotypy alone are not sufficient to increase the presence of NSS. Rather, these studies suggest that it is the combination of schizotypy along with a reduction in psychological functioning which significantly impacts on neurodevelopmental processes, to potentially increase risk for psychotic illness.

Table 4.2. *Summary of studies investigating neurological soft signs in individuals with psychometrically identified schizotypy.*

Study	Participants	Schizotypy scale	NSS scale	Statistical method	Findings			
					Sensory functioning	**Motor coordination**	**Complex motor sequencing**	**Total Soft Signs**
Theleritis et al. (2012) (Data from first assessment)	169 male conscripts: 73 High SPQ 96 C	SPQ	NES	1. Correlations: SPQ w/ NES 2. Mann Whitney test: High, Middle SPQ 3. Regression predicting NES	n.s.	n.s.	1. SPQT: .2* NegS: .27*** 2. High SPQ>C* 3. NegS *b*: .67***	1. SPQT: .19* NegS: .24** 2. High SPQ>C** 3. NegS *b*: .28**
Chan et al. (2010c)	64 High SPQ 51 C	SPQ (Chinese version)	CNI	1. Correlations: SPQ w/ NES 2. One way ANOVA: High SPQT, C	1.NegS: .273** SPQT: .24* 2. High SPQT>C*	1. NegS: .374*** SPQT: .27** 2. High SPQT>C*	-	1. PosS: .253** NegS: .422*** SPQT: .364*** 2. High SPQT>C***
Mechri et al. (2010)	31 siblings of patients with Schizophrenia 60 C	SPQ (French version)	NSS Scale	1. Correlations: SPQ w/ NSS 2. *t*-tests and Chi-squared tests	1. n.s. 2. Rel>C***	1. SPQT(rel): .46** PosS(rel): .37* 2. Rel>C***	1.SPQT(rel):.48** PosS(rel): 41* DisS(rel): .48** 2. Rel>C***	1.SPQT(rel):.46** (C): .28* DisS(rel): .4* 2. Rel>C***
Prasad et al. (2009)	74 offspring of patients with Schizophrenia spectrum disorder (Divided into: +P, -P)	PAS MIS	NES (Factors via PCA)	1. MANCOVAS: (age and sex as covariates), schizotypy, NES 2. Correlations: NES w/ schizotypy 3. MANCOVA:	[Factor: Cognitive-perceptual abnormalities] 1.Offspring>C** 2. n.s	[Factor: Repetitive Motor] 1. n.s. 2. n.s. 3. n.s.	-	1. n.s.

	86 C			Offspring +P, Offspring –P, C	3. Offspring +P> Offspring-P* Offspring +P> C**			
Kaczorowski et al. (2009)	177 healthy students	PAS PhAS SAS MIS	NES	1. Correlations: SPQ w/ NES 2. Regression predicting NES	1. PosS: .07* 2. PosS *b*: .07*	1. NegS: .11* 2. NegS *b*: .11* PosS*NegS *b:* -.07* (high NegS, low PosS performed worse)	1. NegS: .16* 2. NegS *b*: .16*	1. NegS: .11** 2. NegS *b*: .11*** PosS*NegS *b:* -.05** (high NegS, low PosS performed worse)
Keshavan et al. (2008)	75 offspring of patients with Schizophrenia spectrum disorder (Divided into: EP, NEP, WP) 82 C	PAS MI	NES (13 most reliable items)	1. ANOCOVA: (Age as covariate), composite schizotypy score, NES, PAS	1. [Factor: Cognitive-perceptual abnormalities] EP>NEP*** EP>WP*** EP>C***	1. [Factor: Repetitive Motor] n.s.	-	-
Bollini et al. (2007)	26 relatives of patients with Schizophrenia 38 C	SPQ	NES	1. Correlations: SPQ w/ NES	1. SPQT(C): .54* PosS(C): .57* NegS(C): .43* DisS(C): .43*	1. n.s.	1.NegS(C): .47* DisS(C): .54*	1. SPQT(C): .58* NegS(C): .72** DisS(C): .62*

Barkus et al. (2006)	28 Psychosis-prone (PP) 33 C	O-LIFE LSHS	NES	1. *t*-tests 2. Regression predicting PP group	1. n.s 2. n.s.	1. n.s 2. n.s.	1. n.s 2. n.s.	1. PP>C** 2. *b* = -.12*
Barrantes-Vidal et al. (2003)	270 healthy adolescents split into 4 groups: 1. High total sch 2. NegS 3. PosS 4. Normal scorers	PAS SAS PhAS	Battery of 9 signs^ (Obiols et al., 1999)	1. Cluster analysis, MANOVA w/ schizotypy scores 2. One-way ANOVA w/ clusters, NSS	-	-	-	2. PosS scored better than High sch and NegS (trend level significance *p*=.07)

* Significant < 0.05; ** Significant < 0.01; *** Significant < 0.001; n.s.= Not significant at 0.05 level. ^ Higher scores reflect better performance. w/=with; PAS=Perceptual Aberration Scale (Chapman et al., 1978); SAS=Social Anhedonia Scale (Eckblad et al., 1982); PhAS= Physical Anhedonia Scale (Chapman et al., 1976); MIS=Magical Ideation Scale (Eckblad & Chapman, 1983); O-LIFE=Oxford Liverpool Inventory of Feelings and Experiences (Mason, Claridge and Jackson, 1995); LSHS= Launay-Slade Hallucination Scale (Launay & Slade, 1981); SPQ=Schizotypal Personality Questionnaire (Raine, 1991); SPQT= Schizotypal Personality Questionnaire total score; NegS= Negative schizotypy; PosS= Positive schizotypy; DisS= Disorganised schizotypy; rel=relatives; C= Healthy controls; NES=Neurological Evaluation Scale (Buchanan & Heinrichs, 1989); NSS Scale= Neurological Soft Signs Scale (Krebs et al., 2000); CNI= Cambridge Neurological Inventory (Chen et al., 1995); PCA= Principal Component Analysis; EP = Externalising Psychopathology; NEP = Non-Externalising Psychopathology, WP = Without Psychopathology; +P = with Axis I psychopathology; -P = without Axis I psychopathology.

4.1.3 NSS, schizotypy and dyslexia: the role of the Neurodevelopmental Hypothesis

Proposed by Weinberger (1987) and Murray and Lewis (1987), the neurodevelopmental hypothesis views schizophrenia as arising from early pre- and perinatal insults, resulting in structural brain changes which confer a predisposition to the development of schizophrenia in early adult life. Findings of increased perinatal and intrauterine complications for individuals who later go on to develop psychosis support this theory (Zornberg, Buka and Tsuang, 2000; Cannon et al., 2002). The onset of psychotic symptoms and functional decline often commence in late adolescence/early adulthood. This delay in psychopathology between infancy and adulthood has been explained by an excess of functional demand in the context of maturing brain circuitry (Weinberger, 1987). Some researchers have referred to this delay as a 'second hit', which is neurodevelopmentally characterised by aberrant synaptic pruning in the adolescent/young adult brain (McGlashan and Hoffman, 2000). This second hit is believed to open up a biological window, whereby biological and environmental insults are then able to confer this neurodevelopmental vulnerability. Feinberg was the first to suggest that exuberant synaptic pruning may be implicated in the aetiology of schizophrenia (Feinberg, 1982). Recent evidence has suggested that brain dysconnectivity in schizophrenia is not purely due to excessive synaptic pruning, but also by way of disrupted myelination (Karlsgodt et al., 2010), deficits in dendritic spines during development (Glausier and Lewis, 2013), and dendritic atrophy occurring as a result of elevated cortisol (Walker et al., 2008). These contributions to brain disconnectivity are thought to occur at both early (pre and perinatal) and later (adolescent/young adulthood) stages of development (Cannon et al., 2003).

The neurodevelopmental hypothesis holds that aberrations occurring in psychosis should be present to some degree prior to full-threshold symptom onset, and it is this premise in particular which this thesis is based on. One of the first studies investigating this assertion was conducted retrospectively by Walker, Savoie and Davis (1994). Through viewing home movies, they were able to differentiate those children who went on to develop psychosis from those who did not, on the basis of neurodevelopmental anomalies in areas of motor function. Longitudinally,

language, motor, and social abnormalities have been noticed in children who later go on to develop schizophrenia (Cannon et al., 2000; Clegg et al., 2005).

Dyslexia, a language disorder believed to have neurodevelopmental origins, has also been linked to schizophrenia and the psychosis continuum in general (i.e. Bersani et al., 2006; Becker et al., 2012). The psychosis continuum refers to the spectrum of psychotic experiences which ranges from schizotypal personality at the non-clinical end, through to clinically diagnosed schizotypal personality disorder and first episode psychosis, and ending with schizophrenia as the most extreme manifestation of psychotic illness. The language deficits which occur in psychosis have been suggested as phenomenologically similar to those occurring in dyslexia (Condray, 2005). Both dyslexia and the psychosis continuum have also shown abnormalities in cortical functioning, including an absence of the typical cerebral asymmetry of the N400 in response to auditory tones (Heim et al., 2004). Specific neurological indictors of risk for future pathology such as neurological soft signs (NSS) have also been documented in dyslexia (Roongpraiwan et al., 2013; Sadhu, 2008), as well as along the psychosis continuum, in both schizophrenia (Dazzan and Murray, 2002; Bombin et al., 2005) and schizotypy (Barkus et al., 2006; Barrantes-Vidal et al., 2003). These findings are in support of a neurodevelopmental model of psychosis, which implies that neurodevelopmental deviances should be evident in some level at all stages of the psychosis continuum. They also suggest that dyslexia, as a neurodevelopmental disorder, may have similarities with the psychosis continuum, specifically in relation to language processing and NSS.

4.2 Language processing abnormalities

Language processing abnormalities have a fundamental role in psychosis pathology, both as a key diagnostic indicator (e.g. Caplan et al., 2000) and also as a risk marker (e.g. Miklowitz et al., 1991). Language related brain regions have been implicated in the pathophysiology of schizophrenia (e.g. Li, Branch and DeLisi, 2009). This has resulted in language abnormalities being regarded as neurodevelopmental factors associated with the psychosis continuum (e.g. Bearden et al., 2000; Arango, Fraguas, and Parellada, 2014). It has been proposed that the language abnormalities observed in schizophrenia mirror those exhibited in learning disorders such as dyslexia and thus could be indicative of a shared neurodevelopmental pathway (Condray, 2005;

Bersani et al., 2006). The overlap between research findings on language dysfunction in psychosis and dyslexia will be examined below.

4.2.1 Reduced language lateralisation

In schizophrenia robust findings exist for an increased prevalence of mixed and left-handedness, alongside reduced language lateralisation (e.g. Sommer et al., 2001; Collinson et al., 2009), with researchers going so far as to claim that atypical language lateralization is a biological risk marker for schizophrenia illness (e.g. Crow, 2000; Oertel et al., 2010). These findings have been supported by both behavioural (e.g. Hugdahl et al., 2007) and neuroimaging studies (e.g. van Veelen et al., 2011; Bleich-Cohen et al., 2012), however the literature does contain inconsistencies (e.g. Løberg et al., 2002; Razafimandimby et al., 2011). Mixed findings were initially proposed to be the result of methodological limitations (Sommer et al., 2001), however evidence now suggests that these findings may be the result of the absence of positive symptoms in some patients, specifically auditory verbal hallucinations (AVH; Hugdahl et al., 2007; 2008). Research indicates that for those patients who do not experience AVH, their lateralization for language function does not appear to be compromised, reflecting that of healthy controls (Løberg et al., 2002). Yet for schizophrenia patients with ongoing AVH, language lateralization is reduced (Løberg, Jørgensen, & Hugdahl, 2004), such that a greater frequency of AVH is associated with reduced left hemispheric language dominance (Plaze et al., 2006; Hugdahl et al., 2008). In line with these findings, a recent meta-analysis has revealed that schizophrenia patients who experience AVH show a significantly larger reduction of left hemisphere language lateralisation compared to non-hallucinating controls (Ocklenburg et al., 2013). This result led the authors to conclude that reduced language lateralisation represents a strong trait marker for schizophrenia patients who experience AVH.

In individuals at genetic risk for psychosis a loss of asymmetry to left hemisphere language regions has also been reported (e.g. Yücel et al., 2003; Li et al., 2012), with decreased cerebral dominance correlated with psychosis for individuals at high genetic risk (Li et al., 2007). Interestingly, Yücel et al. (2003) found no difference between those who went on to develop psychosis compared to those who did not. This could be suggestive of cerebral asymmetry reflecting language related dysfunction specifically. Longitudinal studies of pre-psychotic children are similar,

with a meta-analysis by Sommer et al (2001) concluding strong evidence exists for decreased cerebral lateralisation in schizophrenia. Pre-psychotic children demonstrated deficits in verbal ability which was associated with a reduction in left hemisphere language lateralisation (Leask & Crow, 2005). These findings indicate first, that dimension of laterality is relevant to the aetiology of psychosis. Secondly, and perhaps more importantly, these anomalies are present long before the onset of psychosis and may therefore have neuodevelopmental origins. Similar findings exist where functional and structural abnormalities are already present in brain regions associated with language processing before progression to psychosis (Callicott et al., 2003; Whalley et al., 2005). The pattern of language dysfunction and associated lateralization for individuals with high schizotypal traits is also similar to the rest of the psychosis continuum. A meta-analysis found high schizotypy to be significantly associated with non-right-handedness (Somers et al., 2009), which parallels observations found in schizophrenia (Dragovic & Hammond, 2005). Mixed handedness has also been associated with disorganized schizotypy specifically (Stefanis et al., 2006), which strengthens the notion of lateralization as a determinant of verbal ability.

4.2.2 Reading dysfunctions

Investigations into reading difficulties in patients with schizophrenia have found difficulties in reading ability and comprehension compared to the general population (e.g. Revheim et al., 2006; 2014; Roberts et al., 2013). Although reading difficulty is not necessarily a fundamental aspect of schizophrenia, it is relevant from an aetiological perspective given the focus on possible overlapping neurodevelopmental origins with dyslexia. Reading deficits are also a central aspect of dyslexia diagnostic criteria.

Relative to controls and population norms, significant deficits in reading comprehension (Hayes & O'Grady, 2003), reading rate (Revheim et al., 2006), and phonological processing (Arnott, Sali & Copland, 2011) have been reported for patients with schizophrenia. Phonological awareness and rapid naming skills particularly have been associated with schizophrenia symptomatology (Arnott, Sali and Copland, 2011). However there is no relation between comprehension and schizophrenia symptomatology (e.g. Bagner et al., 2003). Rather, poorer reading comprehension has been associated with risk (Weiser et al., 2004), and subsequent

hospitalization (Weiser et al., 2007) for psychosis. This suggests phonological processing is related to transient psychotic illness states, whilst comprehension deficits appear to be a more ingrained core dysfunction occurring irrespective of symptom fluctuation.

Abnormalities in visual processing via the magnocellular pathway have also been reported during reading tasks for patients with schizophrenia (Revheim et al., 2006). The magnocellular pathway is located in the upper dorsal section of the brain, and is responsible for signalling where objects are in space, as well as the detection of distance, movement and speed of an object as it moves through space (Wright, Bowen and Zecker, 2000). The combination of magnocellular dysfunction and phonological processing deficits found in schizophrenia overlap with findings in dyslexia samples (e.g. Revheim et al., 2014). Associated working memory impairments have been mechanistically suggested to contribute towards reading difficulties in patients with schizophrenia since increasing sentence length (rather than complexity) exacerbated problems in comprehension (Bagner et al., 2003) Within working memory systems the phonological loop is an important feature in language processing (Baddeley, 2003), thus deficient working memory may be a significant restriction on reading comprehension particularly. Given that many of the cognitive deficits associated with schizophrenia present prior to the onset of symptoms (see Fusar-Poli et al., 2012 for meta-analysis), it is possible that reading deficits are equally associated with underlying risk for the disorder rather than a consequence of subsequent symptoms. Along this line of thought poor reading accuracy and reading rate prior to diagnosis have been retrospectively reported in those who go on to develop schizophrenia (Fuller et al., 2002; Reichenberg et al., 2002), with these deficits possibly reflecting risk of future psychopathology.

4.2.3 Semantic processing and other language-relevant cognitive deficits

Behavioural research into schizophrenia language function has demonstrated an overall typical profile of relatively preserved syntactic processing, with most of the marked deviations found in semantic processing (Covington et al., 2005). Adult patients diagnosed with schizophrenia demonstrate poor semantic categorization of recalled words, suggesting dysfunctions in semantic encoding (e.g. Kareken, Moberg, & Gur, 1996; Nestor et al., 2001). Irregularities in semantic processing were also demonstrated in patients with a high IQ (Rodriguez-Ferrera, McCarthy, &

McKenna, 2001) and children (under 13) diagnosed with schizophrenia (Phillips et al., 2004).

The semantic system is one of the most frequently studied aspects of language processing in schizophrenia. The replication of semantic abnormalities in patients has led to the hypothesis of hyperactivity within semantic memory networks (Kwapil et al., 1990; Spitzer et al., 1993; 1994; Moritz et al., 2001; 2003). Most of the studies investigating semantic system dysfunctions have employed priming tasks. Priming is a faster reaction time in response to a target when it is preceded by a related prime stimulus compared to an unrelated prime stimulus. For example: healthy participants would be expected to respond to "dog" more quickly when it is preceded by "cat", rather than "lemon" (Kuperberg, 2010). Behavioural studies have indicated that under automatic conditions where the time between the stimulus and prime is less than 250ms (SOA; stimulus onset asynchrony), schizophrenia patients demonstrate increased direct (Spitzer et al., 1994; Moritz et al., 2001) and indirect priming (Weisbrod et al., 1998; Moritz et al., 2001; 2002) (where the prime target relation is only evident through some unmentioned mediating word (Neely, 1991; Kreher et al., 2006)). This results in faster reaction times to target stimuli, reflective of less conflict in neural processing. These findings have been confirmed in a meta-analysis of 36 studies by Pomarol-Clotet and colleagues (2008).

When the semantic system is studied under controlled conditions (i.e. SOA longer than 750ms) a reduction in semantic priming is usually observed in schizophrenia patients, for both behavioural (e.g. Minzenberg et al., 2002) and ERP studies (e.g. Condray et al., 1999; Hokama et al., 2003). The reduced priming observed varies significantly from healthy participants who are able to employ strategies that facilitate the processing of the related target, whilst slowing down (inhibiting) the processing of the unrelated target (Neely, 1991). For schizophrenia patients without thought disorder these control strategies do not appear to be used, suggesting dysfunctions in the semantic regulatory system (Kuperberg, 2010). This results in reaction times which are significantly longer in duration, which is believed to be due to a reduced ability to inhibit contextually inappropriate responses, therefore creating neural conflict when deciding on the most correct response.

Homographs (words that have multiple unrelated meanings) have also been used in schizophrenia research to understand how excessive activity in semantic networks can disrupt sentence processing. Patients with schizophrenia are found to

have specific dysfunctions inhibiting the context inappropriate meaning of a homograph in specific situations (e.g. Titone et al., 2000; Salisbury et al., 2002). Utilizing ERP measures, the N400 was abnormally attenuated to words incongruent to the sentence presented immediately prior, even though the words were semantically related to the principal meaning of a homograph (Sitnikova et al., 2002). Therefore it seems that the hyper-activation of the semantic network in schizophrenia leads to the spontaneous activation of dominant word meanings which can be difficult to inhibit, even when they are irrelevant in the embedded context. In terms of the implications on behaviour, this is thought to result in the muddling of words and disturbances in discourse characteristic of schizophrenia language dysfunction. Overall, it is apparent that in schizophrenia semantic memory functions operate via a more automatic pattern of activation compared to healthy controls. This pattern of activation can lead to deficits in speech and/or comprehension of affected individuals (e.g. Sumiyoshi et al., 2005). However it must be noted that some studies have also reported relatively intact semantic memory processing (Kiang et al., 2007; Kuperberg et al., 2006; Ruchsow et al., 2003; Sitnikova et al., 2002). These differences in findings could be attributed to task effects or sample effects including the heterogeneity of schizophrenia symptom presentation, specifically regarding thought disorder (e.g. Ober et al., 1997; Barch et al., 1996).

Certain cognitive dysfunctions are believed to be central predictors in the pathophysiology of psychotic disorders, and seem to be fairly stable across time and regardless of the presence of positive symptoms (Albus et al., 2006; Rund, 1998). UHR patients have been found to be intermediate between those with first episode psychosis and healthy controls on these enduring measures of risk (Hawkins et al., 2004; Byrne et al., 2002; Eastvold et al., 2007). Verbal fluency is one measure that has been focused on, with UHR patients demonstrating greater deficits compared to healthy controls (Eastvold et al., 2007; Hambrecht et al., 2002). Becker et al (2010) also found that the 37% of UHR patients who later transitioned to psychosis performed significantly worse at baseline on semantic verbal fluency compared to the UHR patients who did not transition. In one of the longest UHR follow-ups to-date (up to 13 years), reduced verbal fluency was found to be a major predictor of subsequent conversion to psychosis, in combination with verbal learning and memory deficits (Lin et al., 2011). Collectively, these results indicate that verbal fluency is one of the main prognostic indicators of conversion to psychosis.

Neuropsychological indicators of psychosis development for UHR patients were longitudinally studied by Lencz et al (2006). Of the 33 patients involved in the study, 12 transitioned to psychosis, with verbal working memory the only dysfunction that specifically predicted transition. Similar results were obtained in the Edinburgh High Risk Study (Cosway et al., 2000), where UHR participants performed significantly worse on measures of verbal memory and executive functioning over 2 years, indicating that the development of psychotic symptoms is preceded by a marked decline in verbal memory function. This is in line with studies demonstrating impairments in at-risk populations which are similar to those deficits observed in schizophrenia, albeit to a lesser degree (Jacquemot & Scott, 2006).

Studies of language processing abnormalities in SPD produce similar findings to those in schizophrenia. These include non-lateralisation for patients with SPD with regard to semantic processing (go/no-go task) (Asai, Sugimore & Tanno, 2009), and smaller temporal lobe volumes (e.g. Takahashi et al., 2010; 2011), which is correlated with schizotypal odd speech (Dickey et al., 2003). One of the initial studies involving males with SPD covered a variety of neuropsychological domains (Voglmaier et al., 1997). Of all the areas studied, significant deficits were observed on measures of verbal learning and abstraction, which were complimented by an overall slump in general cognitive function. These results suggest that whilst the dysfunction in SPD is not as pronounced as those observed in schizophrenia, they may still reflect a deficit in frontal and temporal lobe function. Relatives of schizophrenia patients (some of whom also met criteria for SPD) also displayed a reduction in the number of words learnt compared to controls in the California Verbal Learning test (Lyons et al., 1995). This style of learning was consistent with dysfunction in the encoding and/or retrieval of information for SPD participants, as well as impairments in the semantic organization (or 'clustering') of the words presented. Since clustering is used by healthy individuals to facilitate learning it is not surprising that related research has also documented reduced verbal learning and short term verbal retention for SPD patients (Volgaimer et al., 2000), as well as impaired verbal recall and reduced comprehension of complex grammatical structures (Caplan et al., 1990; Condray & Steinhauer, 1992; Siever, 1992). Collectively, it appears that language abnormalities in SPD exist in the early stages of verbal processing (encoding) rather than as a product of dysfunction in the organization/conceptualization of semantic information in the brain.

In regards to semantic processing in high schizotypy, individuals demonstrate similar impairments as those found in schizophrenia (for review see Tonelli, 2014). Semantic studies have produced four main findings for high schizotypal participants compared to healthy controls:

1. Hyper-activation of the semantic network: Individuals with high schizotypy have been found to categorize unrelated concepts as related significantly more often (Kiang & Kutas, 2005). This finding is supported through the reduced negativity of the N400 amplitude in response to unrelated concepts (Kiang, Prugh & Kutas, 2010; Niznikiewicz et al., 2004). The N400 is an ERP measure that reflects the relatedness of the concepts. Two words understood to be highly related trigger an N400 waveform of smaller amplitude/shorter negativity, whereas little semantic association between two words results in N400 negativity (Kutas and Hillyard, 1980). The reduced negativity of the N400 in schizotypy studies suggests these participants identify semantic relationships where there are none. Verbal fluency studies complement these findings, with significantly more atypical responses reported for high schizotypes in semantic category tasks, again providing support for an overactive semantic network (Kiang & Kutas, 2006).
2. Distortions in the use of context during the allocation of semantic meaning: Studies of semantic processing at longer SOAs are reflective of controlled semantic processing, and recruit working memory processes in order to process semantic information. Schizotypal semantic priming studies utilizing longer SOAs (750ms+) have documented impairments in controlled semantic processing. This is believed to be due to the improper use of context which arises as a consequence of working memory impairments (Wang et al., 2013; Morgan et al., 2006).
3. Failing to inhibit semantically unrelated concepts: Studies have shown that each hemisphere is responsible for the processing of different degrees of relatedness, with the left hemisphere responsible for automatic and direct processing, and the right hemisphere for indirect or ambiguous relations. Grimshaw et al (2010) was able to demonstrate that high schizotypal participants preferably utilised the right hemisphere to process meaning. This is in comparison to healthy controls that automatically process meaning in

their left hemisphere, thereby prioritizing direct concepts and inhibiting ambiguous/subordinate meanings. The consequences for high schizotypes, is the activation of distantly related and unrelated concepts, even when those concepts are not semantically correct.

4. Deficits in the access to, and/or storage of information within semantic networks: In healthy individuals, semantic concepts are organized in a multidimensional semantic space, with overlapping features closer together and more obscure concepts farther apart (Hinton, 1981). However in schizotypy, distal concepts appear to be abnormally associated, which results in false associations, or the derivation of odd meaning from otherwise innocuous stimuli (Corlett et al., 2010).

These results are significant when considering the origins of language dysfunction in schizophrenia. Studying language dysfunction in high schizotypal individuals also has the added advantage that participants are not privy to the same confounds as clinical patients; such as medicalization, and the stigma associated with a psychotic illness. Furthermore, since the language dysfunctions reported in schizotypy are qualitatively similar to those at the clinical realm of the psychosis continuum, it creates a platform for investigating the neurodevelopmental origins of these language processes, since these language dysfunctions are present prior to any clinical diagnosis.

4.2.4 Overlap between dyslexia and the psychosis continuum

It is clear that language deficits are detectable along the psychosis continuum. What is of interest now is whether these language dysfunctions are similar to other neurodevelopmental disorders such as dyslexia. In its most sweeping definition, dyslexia is understood as a specific deficit in the ability to read relative to other levels of cognitive competence (Manzo & Manzo, 1993). More comprehensively, it is conceptualized as a developmental disorder whereby major difficulties exist in the ability to decode printed information (Velluntino & Fletcher, 2005), specifically concerning the conversion of printed information into phonological representations (Hoover & Gough, 1990). An exception is acquired dyslexia, which is defined as a difficulty learning to read which develops after brain damage in previously literate individuals (Woollams, 2015). Instead of placing large emphasis on just reading impairment alone, current models outline specific deficits in recognition of words at

the orthographic (awareness of letter combinations/spelling patterns) and/or phonological (awareness of letter-sound correspondences) levels (Vellutino et al., 2004). The disturbances in phonological processing found in dyslexia (e.g. Bone et al., 2002; Pugh and McCardle, 2009), are also common to individuals with schizophrenia (e.g. Angrilli et al., 2009; Barch and Csernansky, 2007). These dysfunctions in phonological processing are thought to impact skilled reading via the letter to sound conversion process. In schizophrenia, phonological impairments are also demonstrated in research concerning; mismatch negativity generation (Javitt et al., 1995), tone matching (Javitt et al., 2000), and the ability to detect phonetic boundaries (Cienfuegos et al., 1999). Arnott and colleagues (2011) have gone one step further to link phonological processing abnormalities to reading impairments in schizophrenia, which supports shared mechanisms contributing to reading dysfunction in both schizophrenia and dyslexia.

However, the finding of normal decoding and non-word reading skills in schizophrenia (Arnott, Sali, & Copland, 2011) appears to be at odds with the phonological impairment which is associated with dyslexia (e.g. Hoover & Gough, 1990; Castles & Coltheart, 1993). Yet these schizophrenia findings are consistent with an adult manifestation of dyslexia, which is characterized by phonological impairment despite appropriate performance on reading measures (Wilson & Lesaux, 2001). Although aberrant phonological processing does appear to underlie reading dysfunction in schizophrenia, further research is required to discern whether these reading difficulties specifically reflect those present in dyslexia, or whether they reflect a more general language processing deficit.

Also common to the psychosis continuum and dyslexia are disturbances in semantic processing. In schizophrenia and schizotypy, controlled semantic processes are impacted by disinhibition, resulting in activation of contextually inappropriate responses, and subsequent impaired accuracy in semantic tasks (e.g. Tonelli et al., 2014). In dyslexia, semantic processing has been characterised by longer reaction times and less accuracy compared to controls (Schulz et al., 2008; Rüsseler et al., 2007). ERP findings have indicated these results may be due to delayed cerebral activation of areas of the brain known to process semantic information for those with dyslexia compared to controls (Schulz et al., 2008; Jednoróg et al., 2010).

Apart from language difficulties, there are other characteristics in dyslexia which are similar to the disturbances evidenced in schizophrenia, including high

rates of mixed hand preference in dyslexia (Richardson, 1994) and significantly higher levels of positive schizotypal traits relative to controls (Kim et al., 1992; Richardson, 1994). Individuals with dyslexia have also been shown to express significantly higher levels of NSS compared to controls (Roongpraiwan et al., 2013; Sadhu, 2008), which overlaps with the increased expression of NSS found in schizophrenia and schizotypy (e.g. Chan et al., 2009; 2010a). These findings could be suggestive of shared features between dyslexia and the psychosis continuum at the biological level and thus be of aetiological significance. Elevated rates of dyslexia have also been demonstrated in relatives of those diagnosed with schizophrenia (Fish, 1987; Rieder & Nichols, 1979). Within families of individuals with schizophrenia other abnormalities in language processes are also found, such as; disturbances in word perception, semantic and syntactic processing, and sentence comprehension (see Condray et al., 2002; DeLisi, 2001; Minzenberg et al., 2002 for reviews). Revheim et al. (2014) even went so far as to classify 70% of schizophrenia patients as meeting criteria for a diagnosis of acquired dyslexia.

The current thesis is concerned with the identification of possible overlapping neurodevelopmental features in schizotypy and dyslexia. The neurodevelopmental features focused on in this thesis are NSS and semantic processing. The proposition advanced here is that the psychosis continuum and developmental dyslexia share a common aetiological pathway that underlies the language disturbances evident in both phenomena. The literature highlights three hypotheses (Condray, 2005):

1. Equivalence hypothesis: common expression and underpinning aetiology where it is assumed the language disorder present in psychosis actually develops from undiagnosed developmental dyslexia.
2. Null hypothesis: phenotypic similarities between the two disorders are merely coincidental.
3. Overlapping/mixed hypothesis: the two disorders share some phenotypic similarities and aetiologies but differ in others.

At present, the current literature points towards the mixed hypothesis as being the most likely based on common findings in existing research. It is clear that dyslexia and the psychosis continuum share a phenomenology for several features at minimum, with this thesis aiming to determine whether additional commonalities

exist for known neurodevelopmental markers of psychosis risk, specifically: neurological soft signs and semantic processing. Phenotypic overlap for these additional risk markers may indicate more profound biological underpinnings, and indicate a shared vulnerability to psychopathology, which is highly relevant for prospective risk studies.

5 SPECIFIC STUDY HYPOTHESES AND PREDICTIONS

The overarching aim of this thesis is to explore the relationships between neurodevelopmental and affective risk factors for psychosis, in the context of a psychometrically at risk population in the form of schizotypal personality. To this end, five studies were conducted, using a mix of healthy university students, and individuals diagnosed with developmental dyslexia. Developmental dyslexia was investigated from a neurodevelopmental stance, with prior research pointing to possible shared neurodevelopmental origins between dyslexia and the psychosis continuum. Findings of commonalities between dyslexia and trait schizotypy for neurodevelopmental and affective risk factors for psychosis may be taken as evidence in support of that hypothesis. Schizotypy, hallucination predisposition, affective temperament, psychological distress, NSS, and semantic processing were investigated across the five studies. Generally, and in line with the continuum model of psychosis, it was hypothesised that the expression of these risk factors would be increased in those with a trait predisposition towards psychosis. Trait factors are an underutilised marker of future psychopathology (Debbané, and Barrantes-Vidal, 2015). Therefore understanding the association between schizotypy and other neurodevelopmental and affective markers along the psychosis continuum can help to inform differences between healthy schizotypal personality and the at-risk mental state. Accordingly, the specific research predictions were as follows:

5.1 Study One

Title: Does temperament mediate the relationship between schizotypy and distress?

Aim: Explore the nature of the relationship between schizotypy, temperament, distress, and hallucination predisposition.

Predictions:

1. Temperament will mediate the relationship between schizotypy and distress.
2. Hallucination predisposition will moderate the direct and indirect relationship between schizotypy and distress.

5.2 Study Two

Title: Neurological soft signs: Effects of trait schizotypy, psychological distress and auditory hallucination predisposition.

Aim: Determine whether rates of neurological soft signs differ according to expression of trait schizotypy, and if so, whether AVH predisposition has a significant effect on this difference.

Predictions:

1. Those with high levels of schizotypy will express significantly more NSS than those with low levels of schizotypy.
2. AVH predisposition will interact with schizotypy to result in the expression of significantly more NSS.

5.3 Study Three

Title: Dyslexia: Evidence for links with the psychosis continuum

Aim: Investigate whether adults with dyslexia express a significantly higher rate of neurodevelopmental risk factors for psychosis compared to healthy controls.

Predictions:

1. Those with dyslexia will express significantly more NSS than healthy controls.
2. Those with dyslexia will have higher levels of schizotypy and mixed handedness relative to controls.
3. Neurodevelopmental risk factors for psychosis (schizotypy, NSS, mixed handedness) will be predictive of dyslexia status.

5.4 Study Four

Title: Semantic processing in cognitive-perceptual schizotypy and hallucination proneness.

Aim: Investigate whether the reaction time processing and performance accuracy of individuals performing a semantic task are effected by positive schizotypy and hallucination predisposition.

Predictions:

1. Reaction time responses of those with high positive schizotypy and high hallucination predisposition will be significantly faster under ambiguous conditions (due to disinhibition), when compared to those with low positive schizotypy and low hallucination predisposition.
2. Those with high positive schizotypy and high hallucination predisposition will demonstrate atypical signal detection determinants of semantic processing

compared to those with low positive schizotypy and low hallucination predisposition.

5.5 Study Five

Title: Semantic processing in an adult dyslexia sample: interaction with schizotypy.

Aim: Explore whether schizotypy has a significant effect on individuals with dyslexia compared to those without in their reaction time processing and performance accuracy on a semantic task.

Predictions:

1. Compared to healthy controls, individuals with dyslexia will record atypical response times and signal detection determinants of semantic processing.
2. Differences between those with dyslexia and controls in response time and signal detection determinants of semantic processing will be accounted for by positive schizotypy.

6 STUDY ONE: DOES TEMPERAMENT MEDIATE THE RELATIONSHIP BETWEEN SCHIZOTYPY AND DISTRESS?

6.1 Abstract

Schizotypy is associated with heightened psychological distress. However, the factors that contribute to this relationship are somewhat unknown. A pattern of increased negative and decreased positive temperament is reported in high schizotypes; therefore temperament may be a mediator between schizotypy and psychological distress. We propose that unusual perceptual experiences may act as an additional hit in this relationship, making distress more likely in high schizotypes. Consequently, it was predicted that hallucination predisposition would moderate the relationship between schizotypy and distress, and the hypothesised indirect relationship between schizotypy, temperament and distress. Undergraduate students (N=746) completed the Schizotypal Personality Questionnaire, Launay-Slade Hallucination Scale, General Temperament Survey, and General Health Questionnaire. Results indicated higher schizotypy scores were associated with higher levels of distress. Both positive and negative temperament partially mediated the relationship between schizotypy and psychological distress, with lower levels of positive temperament and higher levels of negative temperament being associated with distress for high schizotypy scores. Hallucination predisposition did not moderate these relationships. These findings suggest that the relationship between schizotypy and distress is in part due to increased negative and decreased positive temperament. Unexpectedly, propensity to hallucinate does not appear to moderate the mediating effects of temperament on psychological distress.

6.2 Introduction

The focus on early detection and prevention efforts in schizophrenia has sparked a recent increase in studies investigating vulnerability traits along the psychosis continuum (Nelson et al., 2013). Schizotypy is a multidimensional personality trait central to many investigations given its common underlying structure with schizophrenia, sharing positive, negative, and disorganised dimensions (Fossati et al., 2003; Wuthrich and Bates, 2006). Schizotypy is believed to result from a combination of genetic, personality, and environmental factors, to produce individual differences that span across healthy, subclinical and clinical ranges (for review see Debbané and Barrantes-Vidal, 2015). Individuals with elevated levels of schizotypy display temperament and emotional functioning similar to those found in schizophrenia, albeit in an attenuated form (Chmielewski and Watson, 2008; Debbané et al., 2009). As such, schizotypy presents a valuable opportunity to study risk factors associated with psychotic disorders without the additional confounds inherent to psychiatric samples, such as medicalization, hospitalisation and chronicity of illness.

Temperament is one example of a stable, trait risk factor that has been investigated along the psychosis continuum (for review, see Horan et al., 2008). Temperament is an enduring biological variation in the tendency to experience patterns of emotions and behaviours (Rothbart, 1989). Although temperament is sometimes conceptualised as constitutional variations in reactivity and self-regulation (Rothbart, 1989), the specific focus of the current study is 'affective temperament' (also known as positive/negative affectivity (Watson and Clark, 1984). Affective temperament is concerned with the lability, range and intensity of emotions someone is predisposed to experience (Watson and Clark, 1992). In schizophrenia, the typical temperament profile is higher negative temperament and lower positive temperament in comparison to those without schizophrenia (Barch et al., 2008; Berenbaum and Fujita, 1994; Camisa et al., 2005; Gurrera et al., 2000); with similar patterns found in schizotypy (e.g. Chmielewski and Watson, 2008; Kerns, 2006; Ross et al., 2002). Individuals with high negative temperament typically perceive the world as threatening, distressing, and problematic (Watson and Clark, 1992). They are also generally dissatisfied with experiences and report elevated state negative emotions including sadness, disgust, and anger (Horan and

Blanchard, 2003a). Positive temperament reflects a person's willingness to engage in their environment, with high scorers approaching life with enthusiasm and enjoying others company (Watson et al., 1999). Studies have shown that similar to schizotypy, temperament remains stable over fluctuations in clinical state (Blanchard et al., 1998; 2001; Horan et al., 2005).

Research tracking schizotypy dimensions over time has demonstrated the predictive value of schizotypy in the development of psychotic symptoms and functional impairment throughout adolescence and adulthood (Chapman et al., 1994; Kwapil et al., 2013). In this way schizotypy has been referred to as a stable, core trait phenotypically expressed across all conditions in affected individuals (Debbané & Barrantes-Vidal, 2015). Temperament, although present from early childhood (Rothbart, 1986; Bornstein et al., 2015), is dynamic in its maturity into adulthood (Roberts & Del Vecchio, 2000). Both the environment and developmental changes in neural processes have been found to modify the trajectories and expression of reactive traits such as affective temperament (Shiner and Masten, 2012; Baker et al., 1992; Cloninger & Garcia, 2015). These findings are important in the present study given that the statistical modelling of the data requires set ordering of the variables. Based on the reviewed findings, it is hypothesised that schizotypal personality, by influencing perceptions and behaviours, is related to high negative and low positive temperament, and therefore would precede temperament in a relational model. This is not to say that schizotypy is believed to cause temperament. Rather it is suggested that they co-occur alongside each other, with the relative stability of schizotypy resulting in its position before temperament in the hypothesised model.

In those with schizotypal traits the consequences of increased negative and decreased positive temperament have not been considered in depth. However, initial findings indicate higher perceived stress and greater use of avoidant coping strategies for those with schizotypy and this co-occurring temperament pattern (Horan et al., 2007). The experience of stress has been associated with transition to psychosis (for review see Read, Bentall & Fosse, 2009). Higher subjective levels of stress, as a psychological outcome of increased negative and decreased positive temperament in schizotypy is suggested then to have relevance in transition to psychosis. Greater stress reactivity has also been demonstrated in individuals who report an increased frequency of psychotic-like experiences (Myin-Germeys & van Os, 2007). This may indicate an elevated emotional response to everyday stressors in

individuals who experience phenomena such as hallucinations. Collectively, these findings suggest that, in those with higher schizotypy scores, high negative and low positive temperament may result in higher levels of distress. Additionally, the relationship between these variables and distress may be increased by the presence of state risk factors for psychosis, such as hallucinations.

Hallucinations occur frequently in the general population (5-28%; Johns et al., 2004; Scott et al., 2006) and are not specific to the psychosis continuum (de Leede-Smith & Barkus, 2013). Yet since the co-occurrence of hallucinations with schizotypy has been linked to an increased general vulnerability to schizophrenia (Sommer et al., 2010), the interaction between hallucination predisposition and schizotypy is of particular research interest. Hallucinations are considered state phenomena given their fluctuation over time in response to mood (Delespaul, deVries, van Os, 2002). Research has also demonstrated a link between heightened negative temperament and non-clinical hallucinations (Larøi et al., 2005; Young et al., 1986). Given that anomalous experiences such as hallucinations have been shown to co-occur with schizotypal personality (Barkus et al., 2007; Sommer et al., 2010), it is possible that the co-occurrence of schizotypy and hallucination predisposition may result in heightened negative temperament, compared to those without a predisposition to hallucinatory experiences. Therefore hallucination proneness may moderate the relationship between schizotypy and negative temperament, and potentially may also combine with negative temperament to increase risk for distress in vulnerable individuals.

Prior research in non-clinical populations and help seeking samples has focused on state psychological distress as another risk factor for future development of a psychotic disorder (e.g. Yung et al., 2006; Loewy et al., 2007). In schizotypy samples, increased distress predicts transition to psychotic illness (e.g. Mason et al., 2004). Non-clinical perceptual phenomena that cause distress (such hallucinations) are also considered a prospective marker of future psychosis transition in help seeking individuals (Miller et al., 2003; Yung et al., 2005). The experience of distress has also been consistently related to increased risk of psychotic symptoms in genetically at-risk child and adolescent samples (e.g. Cella et al., 2013; Cullen et al., 2014), however the mechanisms that link schizotypy and psychological distress are not well understood. The combined effect of trait and state psychosis risk (schizotypy and hallucination proneness respectively) has been associated with

significantly greater distress when compared to state or trait factors alone (Barkus et al., 2010), suggesting that, in combination, these risk markers are a prominent contributor to distress. Therefore it is proposed that the pattern of temperament in schizotypy is a significant contributor to the relationship between schizotypy and distress, particularly in the presence of hallucination predisposition. In schizotypy, increased negative and decreased positive temperament may lead to a heightened experience of distress due to a pessimistic perception of experiences. The presence of unusual experiences such as hallucinations may increase risk for distress further.

The relationship between schizotypy and temperament has previously been identified, and separately, the link between schizotypy and psychological distress has been established. The current research sought to extend these findings by determining firstly, whether hallucination predisposition moderates the relationship between schizotypy and temperament. Next, this study sought to determine whether positive and negative temperament mediate the relationship between schizotypy and distress. Finally, this study aimed to discover whether hallucination predisposition moderates the direct and indirect relationship between schizotypy and psychological distress.

6.3 Method

6.3.1 Participants

A total of 746 students (Mean age 20.89 years (SD 5.62), age range 17 - 58 years, 73.32% female) were recruited from the University of Wollongong. An imbalanced sex ratio is common in undergraduate psychology samples (e.g. Waters et al., 2003; Paulik et al., 2006). Students participated on a voluntary basis in return for course credit. Recruitment took place throughout a 9-month time block, with the sample size reflective of student interest within this time. An initial demographic questionnaire revealed no diagnoses of schizophrenia or related psychoses in any participants.

6.3.2 Measures

All participants completed preliminary demographic questions. Following this, participants filled out a battery of questionnaires including the Schizotypal Personality Questionnaire (SPQ; Raine, 1991), Launay-Slade Hallucination Proneness Scale (LSHS; Launay and Slade, 1981), General Temperament Survey

(GTS; Clark and Watson, 1990), and the General Health Questionnaire (GHQ; Goldberg and Hiller, 1979).

The SPQ is a 74-item self-report scale requiring yes or no responses. Total score ranges between 0 and 74, and items can be divided into 3 main dimensions: Cognitive-Perceptual, Interpersonal, and Disorganised. Only the total score was reported for the purpose of the current study.

The LSHS is made up of 12-items requiring yes or no responses. Questions describe clinical and subclinical perceptual experiences, and can be used in healthy (e.g. Bentall et al., 1989; Kot et al., 2000) and clinical (e.g. Aleman et al., 1999; Kot and Serper, 2002) populations. Higher scores indicate a greater predisposition to hallucinatory experiences.

The GTS is a trait measure of Positive Temperament and Negative Temperament (the Disinhibition subscale was not used in this study) designed to measure general affective tendencies. Participants were required to respond true or false to statements describing their attitudes, interests and feelings. The Positive Temperament subscale is made up of 27 items and the Negative Temperament subscale has 28 items. The GTS was chosen as a measure of temperament due to the small number of items compared to other scales (i.e. 240 items on Temperament and Character Inventory (TCI; Cloninger et al., 1993)), which required less investment of volunteer participants' time. Further, items on the GTS appeared to tap into trait affective experience more so compared to the TCI, which is focused on temperamental motivations behind reactions to subjective experiences (i.e. novelty seeking, harm avoidance).

The GHQ is a state measure of global psychological functioning, with higher scores indicative of greater distress. Twenty-eight items rated between 0 and 3 are designed to assess how each individual's general psychological health has been over the past few weeks. Total score ranges between 0 and 84. The GHQ can also be scored with a binary scoring method; however the additive scoring method used in the present study is preferred for mediation analyses, given that psychological functioning is assessed on a continuum rather than a probabilistic likelihood of whether the respondent is in need of further psychiatric attention. The GHQ has been highly associated with psychological aspects of distress in non-clinical participants, including depression and anxiety (e.g. Cook et al., 1996; Hotopf et al., 1998).

6.3.3 Procedure

The University of Wollongong Human Research Ethics Committee approved the study. Participants were recruited via enrolment in undergraduate introductory psychology courses, and were granted course credit for their participation. Informed consent and questionnaires were completed online via a survey link.

6.3.4 Statistical Analysis

Descriptive statistics and Pearson's correlations were performed in SPSS 21 (IBM, 2012). Random missing data accounted for less than 3% of the data and was excluded case-wise for all analyses. To investigate the possibility of sex differences Independent Samples *t* tests were conducted with schizotypy, positive temperament, negative temperament, hallucination predisposition and psychological distress as the dependent variables. The possible confounding effect of age was also investigated given previous links between age and hallucination predisposition (e.g. Jardri et al., 2014). Any significant differences were controlled for in subsequent analyses.

6.3.4.1 Moderation analyses

The moderation and mediation analyses were conducted with the PROCESS macro (Hayes, 2013) in SPSS (IBM, 2012). All analyses were run with unscaled variables. The bootstrapping method (as suggested by Shrout and Bolger, 2002) was utilized with 5000 iterations and 95% bias-corrected confidence intervals. Moderation analyses are used to determine whether a specified relationship between a predictor X and an outcome Y changes as a result of another variable M (moderator). The moderating effect of hallucination predisposition was estimated with schizotypy as the independent variable and temperament (positive and negative) as the dependent variables. A significant interaction effect between schizotypy and hallucination predisposition suggests moderation has occurred.

6.3.4.2 Mediation analyses

Mediation analyses are concerned with the difference between the total effect of the treatment (X) on the outcome (Y) (*c* path), and the direct effect of X on Y after accounting for the mediating variables (M) (*c'* path). The mediation effect is calculated by multiplying specific effects of X to M (*a* path) and M to Y (*b* path). Positive and negative temperaments were investigated in separate models as

mediators of the relationship between schizotypy and distress. The mediating effects of positive and negative temperament on the relationship between schizotypy and distress was estimated with schizotypy as the independent variable, positive and negative temperament as the mediators, and psychological distress as the dependent variable. Significant mediation is indicated by a confidence interval that does not contain zero (p<0.05).

6.3.4.3 Moderated mediation analyses

To determine whether hallucination predisposition exerted an effect on the relationships between schizotypy, temperament and psychological distress moderated mediation analyses were conducted. In a moderated mediation model the strength of the mediated relationship depends upon the level of the moderator (MacKinnon et al., 2007). Two moderated mediation analyses were run, with positive and negative temperament mediators investigated separately in each model. The effects of interaction terms were tested to determine whether hallucination predisposition moderated the c' path from schizotypy to distress and the *b* paths from positive/negative temperament to distress. Hallucination predisposition moderator effects were then compared at the mean, as well as low and high levels (one standard deviation below and above the mean respectively), to evaluate the pattern of moderation. Moderated mediation is said to have occurred if the indirect effect is linearly moderated along the entire distribution. This is represented by a significant Index of Moderated Mediation (Hayes, 2015).

6.4 Results

6.4.1 Descriptive statistics

An Independent Samples *t* test to investigate sex effects revealed that females scored significantly higher on the GHQ ($t(413.902) = -5.436$, $p < .001$, Females = 25.2, Males = 20.1) and the negative temperament subscale of the GTS ($t(744) = -7.905$, $p < .001$, Females = 15.52, Males = 10.76). Therefore sex was controlled for in subsequent analyses. Descriptive statistics and correlations are presented in Table 6.1, with significant moderate to high associations between schizotypy, temperament, hallucination predisposition and psychological distress. The relationship between positive temperament and hallucination predisposition was not

significant. Age was also not significantly related to any of the investigated variables, and accordingly was not controlled for in subsequent analyses.

Table 6.1. *Descriptive statistics and Pearson's correlations for schizotypy, hallucination predisposition, temperament and distress.*

Factor	Mean (SD)	SPQ	LSHS	GTS Positive	GTS Negative	GHQ	Age
SPQ	21.85 (13.15)	-					
LSHS	2.97 (2.35)	.592***	-				
GTS Positive	17.15 (6.02)	-.278***	-.064	-			
GTS Negative	14.12 (7.55)	.613***	.377***	-.255***	-		
GHQ[a]	23.54 (11.96)	.432***	.28***	-.301***	.586***	-	
Age	20.89 (5.62)	-.008	.008	.048	-.027	-.03	-

*Mean scores for questionnaire variables are displayed with standard deviations shown in parentheses. [a] Higher scores indicate greater distress; * p < .05; ** p < .01; *** p < .001.*

6.4.2 Moderation analyses

6.4.2.1 Schizotypy and positive temperament

No significant interaction was found between schizotypy and hallucination predisposition, therefore hallucination predisposition did not moderate the relationship between schizotypy and positive temperament.

6.4.2.2 Schizotypy and negative temperament

A significant interaction effect was found between schizotypy and hallucination predisposition (b = -.014, S.E. = .0063, p = .0266), suggesting hallucination predisposition moderated the relationship between schizotypy and negative temperament. Hallucination predisposition was a significant moderator at low (.65), mean (3.5) and high (5.39) levels of hallucination predisposition. The positive relationship between schizotypy and negative temperament remained across all levels of hallucinatory predisposition. However, at low levels of schizotypy hallucinatory predisposition exerted most effect, leading to separation between the three hallucinatory predisposition groups in a rank order. At high levels of

schizotypy this order changed, with low hallucination predisposition exerting the strongest effect, followed by mean and high levels of hallucination predisposition.

6.4.3 Mediation analyses

6.4.3.1 Positive temperament mediator

The total effect (*c* path) of schizotypy on psychological distress was significant (b = .399, S.E = .031, $p < .001$).

The unstandardized estimates from the mediation model are displayed in Figure 6.1, Part I. The coefficient for the path between positive temperament and schizotypy was significant and negative (path a). The path from the positive temperament mediator to psychological distress was also negative and statistically significant (path b).

The indirect effect of schizotypy through positive temperament to psychological distress was significant (b = .052, S.E = .0115, CI = .0322 - .0789). Positive temperament explained 13% of the total association between schizotypy and psychological distress. The direct effect of schizotypy on psychological distress (path c′) remained significant in the presence of the positive temperament mediator (b = .347, S.E = .0311, $p < .001$), accounting for 87% of the total relationship.

Sex was included in the analysis as a covariate given that females had higher levels of psychological distress compared to males (reported previously). Sex was a significant covariate for the relationship between schizotypy and distress (b = 4.85, S.E. = .8983, $p < .001$). Sex was not a significant covariate for any other relationships in the model.

6.4.3.2 Negative temperament mediator

The total effect (*c* path) of schizotypy on psychological distress was significant (b = .399, S.E = .031, $p < .001$).

Figure 6.1, Part II displays the unstandardized estimates for the negative temperament mediator model. The path from schizotypy to negative temperament was positive and significant (path a). The pathway from the negative temperament mediator to psychological distress was also positive and significant (path b).

The indirect effect of schizotypy through negative temperament to psychological distress was significant (b = .279, S.E = .0256, CI = .2316 - .3321). Negative temperament explained 70% of the total association between schizotypy

and psychological distress. The direct effect of schizotypy on distress in the presence of the negative temperament mediator remained significant (b = .119, S.E. = .0357, *p* = .008). The direct effect accounted for 30% of the total association between schizotypy and distress.

Sex was a significant covariate for the relationship between schizotypy and negative temperament (b = 4.26, S.E. = .4698, *p* <.001). Sex was not a significant covariate for any other relationships in the model.

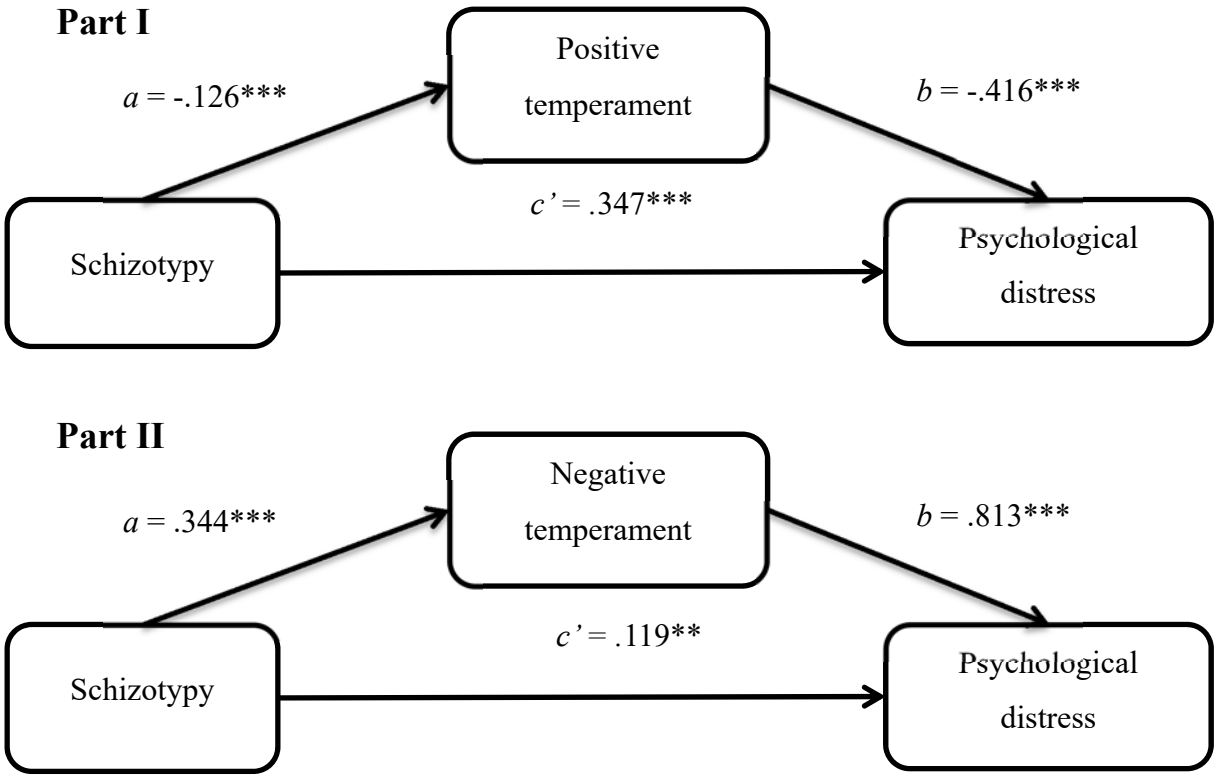

Figure 6.1. Separate path analysis of the hypothesised mediation models, with effects of positive (Part I) and negative (Part II) temperament mediators on the relationship between schizotypy and psychological distress. Values represent unstandardised OLS regression coefficients. *p <.05; **p <.01; ***p <.001.

6.4.4 Moderated mediation analyses

Next, hallucination predisposition was tested as a moderator of the mediation relationship between schizotypy, positive temperament and distress, as well as the mediation relationship between schizotypy, negative temperament and distress.

Hallucinatory predisposition was not a significant moderator of the direct relationship between schizotypy and distress. It also did not moderate the mediation models between positive temperament or negative temperament and distress.

6.5 Discussion

The primary aim of this study was to determine whether temperament mediated the relationship between schizotypy and distress. Our results indicate that although schizotypy has a direct association with distress, this relationship is partially mediated by temperament. These results extend previous findings relating temperament to schizotypy (e.g. Kerns 2005; 2006; Gooding et al., 2002) to demonstrate that temperament may influence the likelihood distress will be experienced. We also determined that hallucination predisposition only moderated the relationship between schizotypy and negative temperament. At low levels of schizotypy, those scoring highest on hallucinatory predisposition had highest scores on negative temperament with separation out from average and low hallucinatory predisposition. This rank order was seen for average schizotypes although with smaller effects. For high schizotypes this order was reversed, such that those scoring lowest on hallucination predisposition had the highest negative temperament scores. These results suggest that the relationship between schizotypy and negative temperament is affected by hallucination predisposition. Finally, results of the moderated mediation analysis indicated that hallucination predisposition did not moderate the mediation models presented.

The inclusion of negative temperament as a mediator accounted for a large proportion of the total relationship between schizotypy and distress. These results extend previous findings of an association between schizotypal traits and distress (Cella et al., 2013; Barkus et al., 2010), and schizotypy and other related psychological states, such as depression and anxiety (e.g. Lewandowski et al., 2006; Debbané et al., 2012), to demonstrate that negative temperament has a mediating role in this cascade. Furthermore, this finding supports the view that schizotypal traits do not alone lead to elevations in psychological distress and increase the risk of transition to illness (van Os et al., 2009). Previous research has shown that other genetic and environmental factors must occur in conjunction with a biological susceptibility to potentiate illness progression (for review, see Tsuang et al., 2001; Rapoport et al., 2005). The current findings are informative in understanding the mechanisms responsible for distress in schizotypy, and therefore could be useful for understanding depression and anxiety in the prodromal state (Owens et al., 2005; Rosen et al., 2006; Svirskis et al., 2005). The results of the current study suggest

negative temperament should be considered as a risk factor for exacerbating psychological distress in the presence of schizotypy.

The present study also identified positive temperament as a partial mediator in the relationship between schizotypy and distress, although to a lesser extent than negative temperament. It has been suggested that moderate levels of schizotypy enhance creative thinking; however this adaptive advantage decreases with increasing psychopathology (Nelson and Rawlings, 2010). The current results suggest that decreased positive temperament is a significant contributor to the distress observed in schizotypy. The amount of distress a person experiences in part depends on their level of schizotypy, as well as the individual's expression of protective factors, such as positive temperament.

The direct and indirect relationship between schizotypy and distress was not moderated by hallucination predisposition for either negative or positive temperament. However, hallucination predisposition did moderate the relationship between schizotypy and negative temperament. Taken together, our findings suggest that hallucination predisposition does not intensify the deleterious relationships between schizotypy, temperament and distress. These findings are in contrast to those predicted, and suggest that the effects of hallucination predisposition should not be categorized as synonymous with schizotypy in its influence on functioning (Preti et al., 2007).

There were two main limitations of the present study. The cross-sectional methodology does not permit certainty in the direction of the relationship between schizotypy, temperament and psychological distress, and as such a longitudinal study is required to more effectively justify the hypothesised associations. Knowing which variable precedes the other will assist in the identification of which early risk factors can potentially be targeted by psychological interventions to reduce the likelihood of distress occurring in young people. Furthermore, the GHQ provides only a global measure of psychological distress. Future studies should employ more specific measures separating psychological subjective distress from real world functioning.

The results of this study suggest the adverse effects of schizotypy on psychological functioning are partially dependent on the individual's temperament. Unexpectedly, hallucination predisposition does not appear to moderate the relationships between schizotypy, temperament, and psychological distress.

7 STUDY TWO: NEUROLOGICAL SOFT SIGNS: EFFECTS OF TRAIT SCHIZOTYPY, PSYCHOLOGICAL DISTRESS AND AUDITORY HALLUCINATION PREDISPOSITION

7.1 Abstract

Schizotypy is regarded as a trait vulnerability for psychotic disorders, yet alone is insufficient for development of a diagnosable disorder. Additional symptoms and psychological distress are necessary for help seeking and transition from an at risk mental state to a clinical diagnosis. The present study investigated the interaction between trait schizotypy, state auditory verbal hallucination (AVH) predisposition, distress and handedness for the expression of neurological soft signs (NSS), a neurodevelopmental vulnerability factor for psychosis. Cluster analysis formed schizotypy groups statistically across the dimensions captured by the SPQ. It was hypothesized that schizotypy and AVH predisposition would interact, resulting in significantly greater NSS. Psychological distress and handedness were hypothesized to be significant covariates, accounting for some variance in the expression of NSS between the groups. A sample of University students (n=327) completed the Schizotypal Personality Questionnaire, Launay-Slade Hallucination Scale, General Health Questionnaire and the Neurological Evaluation Scale (NES). Cluster Analysis revealed four schizotypy groups. Distress was not a significant covariate in any analysis. As expected, those with high overall schizotypy and high AVH predisposition expressed significantly greater Motor-Coordination NSS compared to those with high schizotypy and low AVH predisposition. Within the Mixed Interpersonal and Cognitive-Perceptual Schizotypy cluster, those with low AVH predisposition expressed significantly more Motor-Coordination NSS than those with high AVH predisposition. These findings suggest motor coordination NSS are detectable in schizotypy, and AVH predisposition appears to interact with these traits. This study highlights the importance of considering both trait and subclinical state risk factors when investigating risk for psychosis.

7.2 Introduction

Schizotypy is a multidimensional construct, which represents a heightened vulnerability for psychotic disorders (Kwapil et al., 2013; Salokangas et al., 2013). The schizotypal personality trait is characterized by unusual experiences of perception, oddities in speech and behavior, disorganized and disrupted thought content, paranoia/suspiciousness and flattened affect (Kwapil and Barrantes-Vidal, 2015). The multidimensional structure of schizotypy is believed to mirror that of schizophrenia, with associated phenomena grouped through factor analysis into positive, negative, and disorganized traits (Raine et al., 1994; Stefanis et al., 2004; Mason, 2015). As a result, schizotypy has become central in the investigation of psychosis risk. However, schizotypal trait is not itself sufficient for conversion to psychosis; transition to psychotic disorders requires multiple psychopathological risk factors (Barrantes-Vidal, Grant & Kwapil, 2015). Schizotypy has been found to consistently account for more than half the variance associated with subclinical psychotic phenomena, but does not account for all of it (Rössler et al., 2013). Therefore other factors must combine with schizotypal dimensions to contribute to the development of psychotic disorders. As such, research has focused on a multiple hit model for psychosis risk (e.g. Keshavan, 1999; McDonald & Murray, 2000), where neurodevelopmental and trait biological risk factors interact with state risk factors (such as psychological distress, and psychotic-like experiences (PLEs; e.g. auditory hallucinations)), to increase risk for transition. Trait factors here are perceived to be stable and reasonably consistent across time and situations. Trait and neurodevelopmental factors are often present from birth, however it may only be possible to measure or capture them at different points during development. On the other hand, state risk factors fluctuate according to internal or external factors. Trait and state factors can then be combined to gain a perspective of an individual's stable vulnerability as well as their current and transient vulnerability as a result of fluctuating experiences such as distress. Distress can be triggered by events in an individual's environment or other subjective psychological experiences. The presentation of *trait* schizotypy with *state* auditory verbal hallucination (AVH) predisposition is one combination, which may lead to the emergence of additional psychological vulnerabilities including psychological distress (Cella et al., 2008), disruptions in metacognitive processes (Barkus et al., 2010), and delusion formation

(Krabbendam et al., 2005). The greater the number of additional "hits" an individual encounters, the higher the risk of transition to psychotic disorders, with risk increasing in a dose-dependent fashion (Binbay et al., 2012; Pedersen & Mortensen, 2001). The "hit" may lead to the expression of state risk factors, or may indeed be the exacerbation or presence of compounding state risk factors operating against trait vulnerability.

It is recognized that schizotypy has neurodevelopmental origins (Raine, 2006), therefore consideration needs to be given to whether other neurodevelopmental factors are associated with schizotypy. One such neurodevelopmental factor is Neurological Soft Signs (NSS). The presence of NSS along the psychosis continuum has provided important insights into risk for psychotic illness (Bombin, Arango & Buchanan, 2005; Dazzan & Murray, 2002). NSS refer to subtle neurological irregularities that are not a component of a properly defined neurological syndrome, but rather are believed to reflect inefficiencies in the communication and processing between different brain regions (Chan and Gottesman, 2008). Recent research has linked NSS to the atrophy and abnormal activation of the cerebellum and inferior frontal gyrus, among other areas (Zhao et al., 2014). Phenotypically, NSS are observed as abnormalities in motor functions, sensory functions, disinhibition and complex motor sequencing (Heinrichs & Buchanan, 1988). The Neurological Evaluation Scale (NES; Buchanan and Heinrichs, 1989) is one of the more common measures of NSS. Factor analyses of the scale have demonstrated solutions ranging from one to five factors (e.g. Mohr et al., 1996; Emsley et al., 2005; Sanders et al., 2005). However, most analyses generally reflect a separation between motor and sensory dysfunction (e.g. Keshavan et al., 2003; Sanders et al., 2000; 2005).

There is a consensus that NSS are significantly more prevalent in schizophrenia patients compared to the general population (Zhao et al., 2013). NSS are consistently found in first episode medication-naïve patients (Mayoral et al., 2008; Zabala et al., 2006), their relatives (Gabalda et al., 2008; Mechri et al., 2009), at-risk mental state (ARMS) patients (Tamagni et al., 2013), and those with the schizotypal personality trait (Barkus et al., 2006; Barrantes-Vidal et al., 2003; Chan et al., 2010c; Kaczorowski et al., 2009). Collectively these results suggest that NSS are a neurodevelopmental marker inherent to psychosis risk (Bachmann et al., 2005; 2014). In schizophrenia NSS are related to the severity of negative symptoms and

disorganized behavior (e.g. Mohr et al., 1996; Arango et al., 2000), however are not as conclusively linked to positive symptomatology (e.g. Browne et al., 2000). Concerning schizotypy, positive correlations have been documented between Motor Coordination NSS and overall schizotypy (e.g. Chan et al., 2010c; Mechri et al., 2010); however some studies report non-significant associations (e.g. Bollini et al., 2007; Prasad et al., 2009; Theleritis et al., 2012). Likewise, positive associations have been reported between negative schizotypy and greater overall NSS (e.g. Bollini et al., 2007; Kaczorowski et al., 2009; Theleritis et al., 2012). This is similar to the association found between the negative symptoms of schizophrenia and NSS, however again this finding is not consistent across schizotypal studies (Mechri et al., 2010).

Differences in research design, including the schizotypy and NSS scales used, along with the status of participants (healthy controls versus healthy relatives of schizophrenia patients), may contribute to disparities in findings. It is also possible that NSS are related to another state component of psychosis risk such as AVH predisposition, which is conceptually separate from, but related to, schizotypy. Supporting this assertion are findings of NSS varying according to schizophrenia clinical course (e.g. Bachmann, Bottmer & Schröder, 2005; Prikryl et al., 2012), suggesting they could comprise both state and trait features (e.g. Bachmann et al., 2014). It is proposed that NSS, as neurodevelopmental markers for psychosis risk, would be present in increased levels in those with a trait risk for psychosis (i.e. those with schizotypal traits). Indeed, it is possible that NSS may contribute the expression of schizotypal traits in an individual. NSS may fluctuate around this heightened baseline depending on co-occurring state risk factors, similar to the variation in NSS seen as a result of clinical course in schizophrenia (Bachmann, Bottmer & Schröder, 2005; Prikryl et al., 2012). Those with heightened NSS may be sensitive to additional taxing from the presence of high emotional states such as distress. The distress may perturb an already taxed system to lead to increased inefficiency and expression of NSS. Those with increased levels of schizotypy also demonstrate poor emotion regulation (for review, see Giakoumaki, 2016) and consequent higher levels of depression and anxiety (e.g. Lewandowski et al., 2006). Indeed, those with schizotypal traits and co-occurring axis 1 psychiatric disorder (most frequently mood disorders and ADHD) have documented significantly greater NSS compared to schizotypy alone (Keshavan et al., 2008; Prasad et al., 2009). Therefore high levels

of distress are related to both schizotypy and heightened NSS. To account for this, it makes sense to control for general levels of distress in the current study. Distress, a state variable, is hypothesized to tax an already inefficient neurological system, to result in further disruptions in NSS. Thus state distress *may* exert a co-varying effect on the expression of neurodevelopmental risk variants for psychosis, and is hypothesised to account for some of the differences in NSS expression in schizotypy.

Another commonly reported biological marker along the psychosis continuum is reduced hemispheric symmetry, whereby the typical left hemisphere preference for language functions (e.g. Josse and Tzourio-Mazoyer, 2004)) is either reversed or absent in individuals with schizophrenia (e.g. Kawasaki et al., 2008; Bleich-Cohen et al., 2009) and schizotypy (e.g. Mohr, Bracha, and Brugger, 2003; Suzuki and Usher, 2009). In clinical studies handedness is often used as a proxy for hemispheric specialization, with right-handedness usually being indicative of left hemisphere language preference and right hemisphere visual facial processing preference (e.g. Bourne, 2006; Josse and Tzourio-Mazoyer, 2004). The observed reduction in hemispheric asymmetry for those expressing schizotypal traits has implications in the current study. Accordingly, handedness will be assessed and controlled for in order to accurately investigate differences between those expressing higher levels of schizotypal dimensions compared to those who are not.

Previous studies have made use of correlational analyses where one dimension of schizotypy is often considered to be related to one dimension of NSS. However, the dimensions of schizotypy are strongly related to one another and do not occur in isolation. Indeed there is position that an individual who scores highly on all dimensions of schizotypy could be viewed at heightened risk to those who, for example, merely express the negative dimension of schizotypy. An alternative to the previous correlational approach to schizotypy is to utilize cluster analysis to form groups statistically across the dimensions of schizotypy. This allows for individuals to be elevated on more than one schizotypy dimension simultaneously (Suhr & Spitznagel, 2001), therefore complementing correlational approaches rather than conforming to a categorical approach to psychosis risk. Cluster analysis clarifies inconsistencies evidenced by correlational approaches where individuals may have a mixed profile of positive and negative schizotypal dimensions, rather than being elevated on one dimension only (see Barrantes-Vidal et al., 2010 for further discussion). Since the current research is interested in the elevated expression of

schizotypy across the schizotypal dimensions this approach is believed to be appropriate. Previous schizotypy research has found the number of clusters to vary from three to four-group cluster solutions (e.g. Suhr and Spitznagel, 2001; Aguilera et al., 2008; Barrantes-Vidal et al., 2003; Goulding, 2005). Most often, clusters were characterized as: high overall schizotypy, positive schizotypy (with unusual perceptual experiences and cognitive disorganization characteristics), negative schizotypy (with introverted and anhedonic characteristics), and low overall schizotypy. The current study is using the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) to form clusters, and the number of clusters yielded will be based on model fit. In the context of NSS and schizotypy the cluster approach has been used once previously (Barrantes-Vidal et al., 2003). The findings of this study only reached trend level significance, which may have been due to the use of an ad hoc NSS scale which is to our knowledge, not a validated NSS measure (Obiols et al., 1999). Consequently, adopting cluster analysis in combination with a more robust measure of NSS may highlight differences attributable to the dimensions of schizotypy. The current study is using the Neurological Evaluation Scale (NES; Buchanan and Heinrichs, 1989): one of the most widely used measures of NSS within the psychosis literature (e.g. Compton et al., 2006; Chan et al., 2009; Sewell et al., 2010). Therefore the research from this study can be more easily compared with existing research in the field.

The purpose of the current study was to investigate the interaction between trait schizotypy and state AVH predisposition (i.e. multiple "hits") on NSS. It was expected that one of the clusters would be characterized by elevations in all schizotypal dimensions, whilst another would be characterized by reductions in all schizotypal dimensions. Based on previous research (e.g. Suhr and Spitznagel, 2001; Aguilera et al., 2008; Barrantes-Vidal et al., 2003; Goulding, 2005) the configuration of the other clusters was predicted to be: predominantly negative schizotypy, and predominantly positive schizotypy. Additionally, this study aimed to determine whether state psychological distress and/or atypical handedness (as a proxy for reduced hemispheric asymmetry) also accounted for the expression of NSS. Significant differences between schizotypy clusters were hypothesized for psychological distress, handedness and AVH predisposition. Concerning NSS, based on previous correlational research (e.g. Bollini et al., 2007; Chan et al., 2010c; Mechri et al., 2010; Theleritis et al., 2012) significantly greater NSS was predicted in

the cluster that is characterized by elevated scores on multiple schizotypy dimensions. We also hypothesized that distress and handedness would have co-varying effects, accounting for a significant proportion of variance between schizotypal clusters in the expression of NSS. Finally it was hypothesized that AVH predisposition, as an additional risk component under a multiple hit model, would be associated with greater NSS.

7.3 Method

7.3.1 Participants

Participants were undergraduate Psychology students who participated for course credit (n = 327, mean age = 21.5 (SD 6.8), 72% female). Participants were screened for previous head injury/neurological abnormality, history of psychotic illness, diagnosis of a learning disorder or insufficient knowledge of the English language.

7.3.2 Measures

7.3.2.1 Neurological Examination

The Neurological Evaluation Scale (NES; Buchanan & Heinrichs, 1989) comprises 26 items and was scored according to the original instructions; 0 (no abnormality), 1 (mild but definite impairment), or 2 (present), with total scores ranging between 0 and 76. Fourteen of the items are assessed bilaterally. For the purpose of this study, bilateral right and left items were summed as has been done in previous studies (Bollini et al., 2007; Theleritis et al., 2012). NSS are divided on the basis of dysfunction in three functional areas of interest: Sensory Integration (SI; audio-visual integration, stereogenesis, graphesthesia, extinction, right-left orientation), Motor-Coordination (MC; tandem walk, rapid alternating movements, finger-thumb opposition, finger-to-nose test) and the Sequencing of Complex Motor Acts (SCMA; fist-ring test, fist-edge-palm test, Ozeretski test, rhythm tapping). Other items included in the scale which contribute to the total score include: synkinesis, convergence, gaze impersistence, glabellar reflex, snout reflex, grasp reflex, suck reflex. Handedness was assessed as a standard part of the NES, with respondents asked their hand preference when performing a series of 9 different tasks (i.e. writing, opening the lid of a jar, brushing their teeth). Handedness was

determined if they indicated a preference for the same hand on 7 or more tasks. If preference for one hand was indicated for less than 7 tasks then mixed handedness was assigned. Given that non-right handedness is associated with schizotypy and the psychosis continuum in general (Somers et al., 2009), this variable is expected to impact on cluster differences and therefore will act as a covariate in analyses. All statistical analyses were conducted using the subscales as well as the total NES score.

7.3.2.2 Measures of schizotypy, AVH predisposition, psychological distress and verbal IQ

The Schizotypal Personality Questionnaire (SPQ; Raine, 1991) consists of 74 items requiring yes or no responses. Items are scored together to make a total score and three dimensions (Interpersonal Schizotypy (negative schizotypy), Cognitive-Perceptual Schizotypy (positive schizotypy), Disorganised Schizotypy). Only the dimensions were used to derive participant cluster membership.

The Launay-Slade Hallucination Scale (LSHS; Launay and Slade, 1981) is made up of 12 items measuring presence of clinical and sub-clinical hallucinatory experiences. Higher scores reflect a greater predisposition to these experiences. The LSHS is designed to be used in both clinical (e.g. Kot and Serper, 2002) and general population (e.g. Kot et al., 2000) samples. The LSHS will not be used to form cluster groupings given that it is a state measure of AVH predisposition and is changeable over time, unlike trait schizotypy.

The General Health Questionnaire (GHQ; Goldberg & Hillier, 1979) is designed to measure state psychological distress, with higher scores representative of a greater experience of distress. The scale consists of 28 items rated from 0 to 3. In non-clinical samples responses on the GHQ have been highly associated with other state measures of distress such as depression and anxiety (e.g. Hotopf et al., 1998).

Verbal intelligence was measured using the National Adult Reading Test (NART; Nelson, 1982).

7.3.3 Procedure

Ethical approval was obtained from the Human Research Ethics Committee at the University of Wollongong (approval number HE12/362). Participants were

given access to study information via a university-run research participation system. Once they signed up to the study informed consent was obtained online (with options to contact the researcher if required). Questionnaires were also completed online via a survey link. They were then invited to participate in the second stage of the study, and informed consent for this stage was obtained in writing. The NES and NART were completed during this time, with researchers unaware of participants' schizotypy cluster classification.

Four trained evaluators administered the NES and NART to participants. To assess inter-rater reliability raters jointly examined 20 participants, whereby one rater was paired up with each of the remaining raters. This procedure ensured consistency in ratings. The correlation coefficients for subscale and total scores ranged from .71 to .98.

7.3.4 Statistical Analysis

Descriptive statistics were performed in SPSS 19 (IBM, 2010). Random missing data accounted for 4.1 % of all data, and were excluded case-wise for all analyses. Normality of the data was checked using values of Skewness and Kurtosis. All values were within the +/- 2 limit, therefore parametric analyses were considered acceptable (George and Mallery, 2010). Given the similarities in the types of experiences focused on in the LSHS and Cognitive-perceptual subdomain of the SPQ, Pearson correlations were calculated initially to ensure there is some degree of distinction between these variables. SPQ subscale scores were converted into z-scores for ease of interpretation. Schizotypy clusters were derived using K-means iterative cluster analysis with Cognitive-Perceptual, Interpersonal, and Disorganised schizotypy scores. LSHS was also used initially to form clusters, however fit was poor and therefore this variable was removed. Following previous schizotypy cluster studies a 4-group cluster solution was forced (e.g. Barrantes-Vidal et al., 2003; 2010; Suhr and Spitznagel, 2001). This solution was compared to a 3-group cluster solution, however the 4-group cluster solution emerged as superior in terms of fit, as indicated by a Wilks' Lambda of .069 (4 cluster solution), versus .142 (3 cluster solution).

Demographic schizotypy group differences were investigated using Independent Samples *t* tests for continuous variables and Chi-Squared tests for categorical variables. Any significant differences at the p =.05 level (one-tailed) that

may have accounted for NSS findings were controlled in subsequent analyses as covariates. To investigate the effect of schizotypy cluster group membership and AVH predisposition on NSS, LSHS total score was split into two groups either side of the mean. Those scoring 5 or higher were in the high group (n = 109), whilst scores from 0 to 4 were considered low (n=218). Mean splitting is utilized here as an exploratory method. The goal is to determine whether the interaction between schizotypy cluster and AVH predisposition for NSS performs differently for those with high versus low AVH predisposition. Unfortunately there are not pre-existing clinical cut offs for research using the LSHS, therefore splitting at the mean is the most viable decision. Additionally, the LSHS scale used in the current study, requires dichotomous present/absent responses which is methodologically consistent with the use of categorical groupings.

A one-tailed Multivariate Analysis of Covariance (MANCOVA) was utilized to investigate group differences in the expression of NSS. In this analysis schizotypy cluster groups and LSHS mean split groups were independent variables, and NES total and subscale scores were dependent variables.

7.4 Results

7.4.1 Correlations between SPQ and LSHS

Pearson's correlations showed significant ($p < .001$) associations between LSHS and SPQ Total (r = .619), Cognitive-perceptual (r = .651), Interpersonal (r = .406) and Disorganised (r = .514) subscales. Therefore the strength of the relationship between the LSHS and SPQ Total, Cognitive-perceptual, and Disorganised subdomains is of moderate strength, whilst the association between LSHS and the Interpersonal SPQ subdomain is weak (Mukaka, 2012).

7.4.2 Schizotypy clusters

K-means iterative cluster analysis produced a four-cluster solution across the Cognitive-Perceptual, Interpersonal and Disorganised dimensions of the SPQ. A MANOVA with cluster assignment as the Independent variable and SPQ factor scores as the Dependent variables was then used to obtain a discriminative index score. Wilks' Lambda (.069) was significant ($p<.001$), which demonstrated that only 6.9% of the total variance was left unexplained. Descriptive statistics of the four

clusters are presented in Table 6.1, with names of each cluster corresponding to SPQ characteristics.

7.4.3 Demographic characteristics of Schizotypy clusters

No significant differences were found between Schizotypy clusters on sex, age, verbal IQ, living arrangements, use of health services, or presence of a diagnosed learning disorder. Significant differences did exist between clusters on handedness ($\chi^2 = 22.592$, $df = 6$, $p = .001$), AVH predisposition (F (3, 323) = 47.615, $p < .000$) and psychological distress (F (3, 323) = 22.898, $p < .001$) These differences are presented in Table 7.1. The mean SPQ total and factor scores for each cluster are also presented in Table 7.1. The cluster characteristics for the first and third clusters were straightforward, and thus were named High overall schizotypy and Low overall schizotypy respectively. The characteristics of the second and fourth clusters were more mixed. After revision, it was decided to name these clusters Disorganised schizotypy dominant and Mixed Interpersonal and Cognitive-Perceptual Schizotypy. The word ‘dominant’ is used with the Disorganised schizotypy cluster to remind the reader that this cluster is not pure in its configuration given that it also has average levels of Interpersonal and Cognitive-Perceptual schizotypy. For significant comparisons, least-significant difference post-tests were performed.

Table 7.1. *Descriptive statistics (mean, SD) and frequencies of Schizotypy clusters.*

	1. High overall Schizotypy (n=61)	**2. Disorganised Schizotypy dominant (n=90)**	**3. Low overall Schizotypy (n=117)**	**4. Mixed Interpersonal and Cognitive-Perceptual Schizotypy (n=59)**	**Test statistic and *p* value**	**Significant differences? [a]**
Sex (M:F)	15:46	31:59	28:89	17:42	$\chi^2 = 3.193$, $p = .363$	No
Age	21.59 (7.3)	21.01 (5.4)	21.88 (7.2)	21.42 (7.6)	$F = .28$, $p = .84$	No
Living arrangements (Parents:Siblings: Partner:Friends:Acquaintences:Alone)	41:3:5:7:1:4	49:5:12:12:5:7	70:4:18:11:8:6	39:1:8:5:1:5	$\chi^2 = 10.101$, $p = .813$	No
Verbal intelligence	27.44 (5.3)	27.36 (5.9)	26.84 (5.9)	27.23 (5.7)	$F = .307$, $p = .82$	No
Health service use (Y:N)	41:20	54:36	76:41	40:19	$\chi^2 = 1.283$, $p = .733$	No
Learning disorder (Y:N)	0:61	5:85	1:116	1:58	$\chi^2 = 7.32$, $p = .062$	No
SPQ Total	50.48 (7.7)	24.33 (5.8)	11.65 (5.9)	32.15 (5.4)	-	-
Cognitive-	19.33 (4.5)	7.9 (3.9)	4.84 (3.8)	12.15 (4.6)	-	-

Perceptual SPQ						
Interpersonal SPQ	20.49 (4.6)	8.11 (4.1)	4.85 (3.2)	15.98 (4.6)	-	-
Disorganised SPQ	11.49 (2.7)	8.57 (2.2)	2.03 (1.7)	4.9 (1.8)	-	-
AVH predisposition	5.95 (2.2)	3.67 (1.9)	2.1 (2.1)	3.95 (2.2)	$F = 47.615$, $p < .001$	Yes (1>2,3,4;4>3; 2>3)
Psychological distress GHQ	32.92 (13.2)	21.69 (10.9)	18.03 (11.3)	24.36 (11.1)	$F = 22.898$, $p < .001$	Yes (1>2,3,4; 4>3)
Handedness (Right:Left:Mixed)	Right = 95.1% Left = 1.6% Mixed = 3.3%	Right = 77.8% Left = 21.1% Mixed = 1.1%	Right = 88.9% Left = 7.7% Mixed = 3.4%	Right = 88.1% Left = 5.1% Mixed = 6.8%	$\chi^2 = 22.592$, $p = .001$	Yes

SD= standard deviation; N=Number of participants in group; M=Male; F=Female; Y=Yes; N=No; SPQ=Schizotypal Personality Questionnaire (Raine, 1991); AVH=Auditory Verbal Hallucination GHQ=General Health Questionnaire (Goldberg & Hillier, 1979), [a]=Post tests show which clusters differ significantly at the p=.002 level or below.

Table 7.2. *Means (standard error of the mean) of interaction effects between schizotypy clusters and AVH predisposition groups for Neurological Evaluation Scale (NES) Total and subscale scores.*

		NES Total	NES SI	NES MC	NES SCMA
High overall Schizotypy	High AVH predis.	10.45 (.59)	2.2 (.19)	**1.68 (.22)[a]**	.98 (.19)
	Low AVH predis.	9.76 (1.11)	2.65 (.31)	**.88 (.19)[ac]**	.47 (.19)
	Total	10.28 (.62)	2.48 (.22)	1.2 (1.7)	.68 (.19)
Disorganised Schizotypy dominant	High AVH predis.	9.86 (.96)	2.11 (.27)	1.25 (.16)	.68 (.27)
	Low AVH predis.	10.02 (.51)	2.34 (.19)	**1.15 (.16)[c]**	.77 (.15)
	Total	9.79 (.47)	2.22 (.17)	1.21 (.13)	.71 (.14)
Low overall Schizotypy	High AVH predis.	9.62 (1.2)	2.54 (.56)	1.46 (.42)	.77 (.26)
	Low AVH predis.	9.81 (.41)	2.51 (.14)	**1.18 (.09)[c]**	.93 (.12)
	Total	9.82 (.62)	2.49 (.22)	1.35 (.17)	.89 (.18)
Mixed Interpersonal and Cognitive-Perceptual Schizotypy	High AVH predis.	8.88 (.76)	2.17 (.27)	**1.04 (.23)[b]**	.58 (.16)
	Low AVH predis.	12.3 (.78)	2.69 (.27)	**1.83 (.25)[bc]**	1.31 (.28)
	Total	10.54 (.55)	2.44 (.19)	1.43 (.16)	.94 (.16)

Note: SI = Sensory Integration, MC = Motor Coordination, SCMA = Sequencing of Complex Motor Acts, AVH predis. = Auditory Verbal Hallucination predisposition. Significant effects (p<.05) indicated by **bold** font type. Significant differences between High and Low AVH predis. groups within the High overall schizotypy cluster denoted by [a]; Significant differences between High and Low AVH predis. Groups within the Mixed Interpersonal and Cognitive-Perceptual Schizotypy

cluster denoted by [b]; Significant differences between schizotypy clusters within the Low AVH predis. group denoted by [c].

7.4.4 Schizotypy, AVH predisposition and Neurological Soft Signs

A priori hypotheses predicted co-varying effects of handedness and psychological distress, thus these differences between clusters on handedness and psychological distress were controlled using a MANCOVA when examining group effects on NSS variables. Handedness had significant co-varying effects for NES Total score ($F(1, 317) = 17.11$, $p < .001$) and NES SCMA subscale ($F(1, 317) = 4.288$, $p = .039$). Psychological distress did not have co-varying effects for any NES variables.

No main effects were found for schizotypy or AVH predisposition on NES Total score, SI, MC or SCMA. An interaction effect was observed between schizotypy and AVH predisposition for the NES MC subscale ($F(3, 317) = 4.165$, $p = .007$; means in Table 7.2). To interpret this interaction an Independent Samples *t* test was used. Those in the High overall Schizotypy cluster with High AVH predisposition expressed significantly more MC NSS compared to those with Low AVH predisposition in the same cluster ($t(52.624) = 2.754$, $p = .008$; Table 7.2, superscript [a]; Figure 7.1).

Those in the Mixed Interpersonal and Cognitive-Perceptual Schizotypy cluster with High AVH predisposition expressed significantly less MC NSS compared to their Low AVH predisposition counterparts ($t(57) = -2.22$, $p = .03$; Table 7.2, superscript [b]; Figure 7.1).

The analysis was then rerun to determine whether differences between AVH predisposition groups were driving the significant effects. Significant differences between schizotypy clusters on NES MC were found for Low AVH predisposition ($F(3, 212) = 4.015$, $p = .008$) but not High AVH predisposition ($p = .452$). Pairwise Comparisons revealed that those low on AVH predisposition in the Mixed Interpersonal and Cognitive-Perceptual Schizotypy cluster expressed significantly more MC NSS than all other schizotypy clusters within the Low AVH predisposition group (means in Table 7.2, superscript [c]).

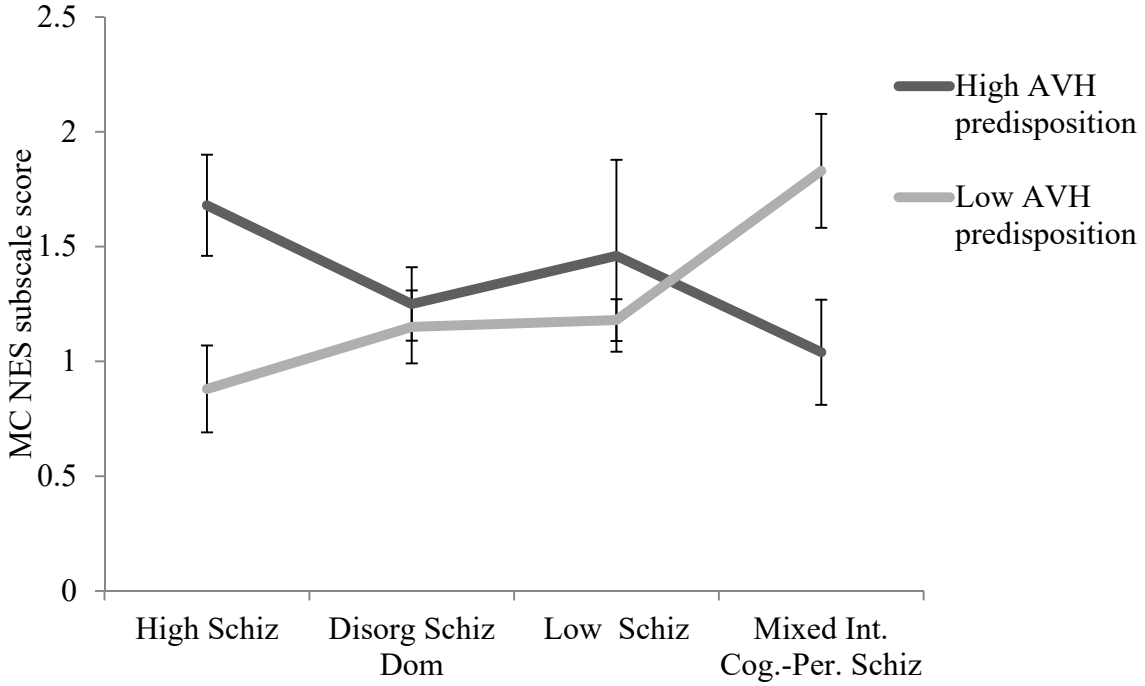

Figure 7.1. Mean Motor-Coordination (MC) subscale score (from the Neurological Evaluation Scale (NES)) for each Schizotypy cluster, with clusters split into High and Low Auditory Verbal Hallucination (AVH) predisposition. Error bars represent standard error.

7.5 Discussion

The present study investigated the effect of trait schizotypy and state AVH predisposition on the expression of NSS. In keeping with previous literature (Barrantes-Vidal et al., 2003) a four cluster solution was forced: High overall Schizotypy, Disorganised Schizotypy dominant, Mixed Interpersonal and Cognitive-Perceptual Schizotypy and Low overall Schizotypy. Those with mixed handedness were more likely to be found in the Mixed Interpersonal and Cognitive-Perceptual Schizotypy group, whilst those with left-handedness were more likely in the Disorganised Schizotypy dominant group. Handedness was a significant covariate for NES Total and SCMA scores, however no group differences were found. The data suggests there is not a simple relationship between schizotypy, AVH and NSS. Those in the High overall Schizotypy cluster with High AVH predisposition expressed significantly greater MC NSS compared to those in the same cluster with Low AVH predisposition, with this relationship reversed in the Mixed Interpersonal

and Cognitive-Perceptual Schizotypy group. Contrary to predictions there was no main effect of schizotypy clusters for NSS expression. State psychological distress did not significantly co-vary for the expression of NSS, although the schizotypy groups did report higher distress, with distress highest in the High overall Schizotypy group.

Consistent with expectations those in the High overall Schizotypy cluster with co-occurring High AVH predisposition expressed significantly greater MC NSS compared to those in the same schizotypy cluster but with Low AVH predisposition. Surprisingly this interaction was reversed for the Mixed Interpersonal and Cognitive-Perceptual Schizotypy cluster. Those in this cluster with Low AVH predisposition expressed significantly greater MC NSS compared to their High AVH predisposition counterparts. The link between motor coordination deficits and psychosis has been documented at all stages of the psychosis continuum, from prospective studies of children who go on to develop schizophrenia (Schiffman et al., 2009), adolescents with high levels of schizotypy (Mittal et al., 2008), offspring of schizophrenia patients as well as medication-naïve schizophrenia patients (Wolff & O'Driscoll, 1999). The current results extend these findings of movement abnormalities to a more specific and subtle form of motor coordination impairment in the form of neurological soft signs. Yet whilst a link appears to exist between schizotypy and motor coordination NSS, the association with state factors such as AVHs does not appear simple. MC NSS were associated with high overall schizotypy and co-occurring AVH predisposition, suggesting multiple "hits" are necessary to result in motor coordination abnormalities at the high end of schizotypy. Additionally our results indicate that higher levels of Interpersonal schizotypy when combined with moderate levels of Cognitive-Perceptual schizotypy (as in the Mixed Interpersonal and Cognitive-Perceptual Schizotypy cluster) may be sufficient in the expression of MC NSS without the additional "hit" of AVH predisposition. Gross et al (2014) reported that the Interpersonal subscale of the SPQ does not encapsulate negative schizotypy as well as the Cognitive-Perceptual subscale taps positive schizotypy. Given this limitation, the current findings highlight the utility of the cluster approach in being able to account for elevations on more than one schizotypy dimension. These results also support consistent findings in the literature linking negative symptoms of schizophrenia to elevated rates of NSS (e.g. Mohr et al., 1996; Arango et al., 2000), which have been replicated with negative schizotypy (e.g. Bollini et al.,

2007; Kaczorowski et al., 2009; Theleritis et al., 2012). Negative schizotypy has been associated with lower functional outcomes (Cohen & Davis, 2009), suggesting this schizotypy dimension in particular may be an indicator of need for care in itself (Lin et al., 2013). State risk factors alone have been reported to have low specificity in accurately predicting conversion to psychosis (Debbané & Barrantes-Vidal, 2015). Our findings demonstrate the importance of integrating both trait and state psychosis risk factors.

It was also predicted that schizotypy clusters would differ significantly in their expression of NSS, however this hypothesis went unsupported. Our predictions were based on previous correlational research (e.g. Chan et al., 2010c; Mechri et al., 2010; Theleritis et al., 2012). Since NSS are understood as neurodevelopmental markers of psychiatric risk, it follows that expression of NSS should be the result of high schizotypy in combination with other state features of risk. Other studies have demonstrated limited or no differences in neurological soft sign expression due to schizotypy alone (e.g. Obiols et al., 1999; Barrantes-Vidal et al., 2003; Bollini et al., 2007).

When psychological distress was considered in the analysis it was not a significant covariate between schizotypy and AVH predisposition for NSS expression. Previous studies utilizing community samples (healthy first-degree relatives of schizophrenia patients) have shown interview-assessed state psychopathology (axis 1 psychiatric illness) to increase NSS in those with high schizotypy (e.g. Keshavan et al., 2008; Prasad et al., 2009). Since psychopathology is by definition more severe than state distress, it may be that the degree of functional impairment focused on in the current study was not of a sufficient threshold to impact upon the expression of neurological soft signs.

Demographic characteristics may have contributed to some of the non-significant findings in this study; therefore the homogeneity of a University-educated sample is considered a limitation. Given that a large percentage of schizotypy research in this area utilizes a University-based sample (e.g. Barkus et al., 2006; Chan et al., 2010c; Kaczorowski, Barrantes-Vidal, & Kwapil, 2009), it would be extremely beneficial for future research to determine the extent to which tertiary level education impacts upon psychosis risk variables. Another factor that limits the interpretability of the present findings is the use of a cross-sectional design, given that psychosis high-risk variables are known to change over time (especially during

adolescence/early adulthood; Shah, Tandon, & Keshavan, 2013). Although the present study provides evidence that trait schizotypy and state AVH predisposition interact for the expression of motor neurodevelopmental risk, it cannot be said whether greater NSS are a result of this interaction, or whether other co-occurring variables are contributing, such as cognitive reserve (e.g. Urbanowitsch et al., 2015) or comorbidity with obsessive-compulsive symptoms (e.g. Tumkaya, Karadag, and Oguzhanoglu, 2012). Future research which tracks trait and state psychosis risk variables over time will help to disentangle more influential "hits" associated with illness transition, from less influential but co-morbid psychosis risk factors.

Although still in its infancy, research is beginning to shift from a high clinical risk approach of psychosis vulnerability to a more encompassing framework; integrating developmental traits such as schizotypy and subclinical phenomena (including AVH predisposition and distress) (Debbané & Barrantes-Vidal, 2015). The present study reports pertinent findings for the interaction between trait schizotypy and state AVH predisposition in the expression of motor NSS. When combined with previous results, the current findings provide support for the existence of abnormalities in motor coordination for individuals on the psychosis continuum. Future research which goes another step further to longitudinally investigate the interaction between trait and state psychosis risk factors may more specifically distinguish the trajectory and severity of motor NSS as individuals progress along the continuum.

8 STUDY THREE: DYSLEXIA: EVIDENCE FOR LINKS WITH THE PSYCHOSIS CONTINUUM

8.1 Abstract

Abnormalities in language processing and subtle neurodevelopmental features called Neurological Soft Signs (NSS) are common to both dyslexia and those scoring highly on psychosis proneness, or schizotypy. We investigated whether the expression of NSS and schizotypy predicted dyslexia status. Participants (N=102, 51 dyslexic) completed the Schizotypal Personality Questionnaire, Neurological Evaluation Scale, and a measure of verbal intelligence. Dyslexia status was predicted by higher NSS, lower verbal intelligence, and higher disorganised schizotypy scores. Seemingly, the schizotypal trait, NSS and dyslexia co-occur. Clinical consideration of personality vulnerabilities in dyslexia and developmental language disorders in psychosis risk require further consideration.

8.2 Introduction

Dyslexia is a neurodevelopmental disorder characterized by difficulties with fluent and accurate word recognition, poor spelling and phonological abilities (Lyon et al., 2003). The difficulties present in those with dyslexia are believed to be of neurological origin (Habib, 2000), reflecting abnormalities in neurodevelopment. There are a number of neurological factors that have been investigated in those with dyslexia, including neurological soft signs (NSS). NSS are subtle nuances in performance of behavioural tasks in areas of motor coordination, sensory integration, and the sequencing of complex motor acts (Buchanan and Heinrichs, 1989). They are thought to represent connective tract abnormalities in the brain (Mittal et al., 2013), with research implicating atrophy and abnormal activation in the cerebellum and inferior frontal gyrus, among other regions (Zhao et al., 2014). Previous research has reported that those with dyslexia express greater levels of NSS compared to those who do not have dyslexia (Roongpraiwan et al., 2013; Sadhu, 2008).

Abnormalities in neurodevelopment are not unique to language disorders such as dyslexia. NSS are found in both dyslexia and along the psychosis continuum (Dazzan and Murray, 2002; Bombin et al., 2005; Zhao et al., 2013; Barkus et al., 2006). The psychosis continuum reflects psychosis proneness or risk, spanning from the schizotypal personality trait at the non-clinical end, through schizotypal personality disorder, to first episode psychosis, and schizophrenia as the most severe manifestation of psychotic illness. There is continuity in the phenotypic experience of psychotic symptoms across the continuum, with common symptoms associated with schizophrenia (such as hallucinations and delusions) also present at sub-threshold levels in non-clinical populations (Mata et al., 2003). The expression of NSS in dyslexia and along the psychosis continuum implies overlapping neurodevelopmental aberration phenotypically detectable as NSS. Evidence of the overlap can be seen in increased positive schizotypy and mixed handedness in dyslexia (Richardson and Stein, 1993; Richardson 1994); with similar support for mixed handedness associated with schizotypy in the absence of dyslexia (Barrantes-Vidal et al., 2013c; Tsuang et al., 2013). Mixed handedness is often considered a proxy for atypical language lateralization (e.g. Szaflarski et al., 2002; however see Groen et al., 2013), with these findings suggesting abnormal lateralization may be present in both dyslexia and psychosis proneness.

There are other common factors found in dyslexia and along the psychosis continuum, including: difficulties with reading skills (Revheim et al., 2014; Lefly and Pennington, 1991); phonological skills (Bersani et al., 2006; Shaywitz et al., 1999); morphological abnormalities of the planum temporale (Shapleske et al., 1999 reviews findings in both schizophrenia and dyslexia), resulting in a reduction of the typical left cerebral asymmetry (Hori et al., 2008; Illingworth and Bishop, 2009) and reduced verbal intelligence (Khandaker et al., 2011; Shaywitz et al., 1999). Although schizophrenia has its onset in late adolescence to early adulthood, the factors that underpin it are thought to be neurodevelopmental in nature (Rapoport, Giedd and Gogtay, 2012; Weinberger, 1987). Bersani et al (2006) have suggested that schizophrenia and dyslexia share overlapping pathogenetic mechanisms specifically in relation to language disorder and neurocognitive impairment. Developmental delays have been reported to interact with environmental factors such as obstetric complications, to result in up to a five-fold increase in risk for schizophrenia (Clarke et al., 2011). Longitudinal findings indicated that children who are diagnosed with developmental language disorders had higher rates for the subsequent development of schizophrenia and schizotypal traits in adulthood, when compared to the general population and non-language disordered siblings (Clegg et al., 2005).

As mentioned previously, one factor occurring along the psychosis continuum and strongly linked to psychosis risk, is the expression of schizotypal traits or schizotypy (e.g. Nelson et al., 2013; Debbané et al., 2015; Baarrantes-Vidal, Grant and Kwapil, 2015). Schizotypal traits comprise of unusual perceptual experiences, blunted social and emotional functioning, and oddities in behaviour and language. These traits are analogous to the positive, negative and disorganized symptom clusters of schizophrenia (Claridge, 1997), however are considered non-clinical and relatively stable personality trait manifestations of schizophrenia-like symptoms. Like schizophrenia, schizotypy is believed to occur as a result of atypical development (Raine, 2006). Elevated NSS have been related to the negative (or interpersonal) schizotypal trait most commonly (Bollini et al., 2007; Kaczorowski et al., 2009; Theleritis et al., 2012). However Chan et al. (2010c) and Mechri et al. (2010) have found overall schizotypy to have a significant relationship with NSS also.

Higher rates of schizotypy, in particular, positive schizotypy, have been associated with dyslexia status (Richardson and Stein, 1993; Richardson, 1994). Schizotypy and dyslexia both have neurodevelopmental underpinnings (Raine, 2006; Thompson et al., 2015); however this study is interested in shared neurological dysfunction in the form of NSS. The increased expression of NSS in dyslexia and across the psychosis continuum suggests these observable behaviours may be indicative of shared underlying neurobiological inefficiencies. Increased rates of schizotypy and NSS in dyslexia may be indicative of possible shared neurodevelopmental trajectories between dyslexia and the psychosis continuum.

The aims of this study were two-fold. First, we aimed to investigate whether dyslexia was associated with neurodevelopmental variables of the psychosis continuum. It was expected that those with dyslexia would have increased levels of NSS, schizotypy, mixed handedness, and lower verbal intelligence relative to healthy controls. It was also expected that the relationship between schizotypal dimensions and NSS would be weaker in those with dyslexia compared to those without. The second aim was to investigate whether a collection of known neurodevelopmental characteristics associated with the psychosis continuum are predictive of dyslexia diagnostic status. It was expected that NSS, as a fundamental component of neurodevelopmental aberration, would predict dyslexia status. Based on previous research (Richardson and Stein, 1993; Richardson, 1994) the cognitive-perceptual schizotypal dimension and handedness were also expected to predict dyslexia status. Verbal intelligence is lower in those with dyslexia (van Bergen et al., 2014), therefore was expected to be strongly predictive of dyslexia status.

8.3 Method

8.3.1 Participants

One hundred and two participants took part in this study (70% female; average age: 24.47 (SD 9.69) years; range between 17 and 66 years). Participants were recruited from the undergraduate Psychology program and the wider student pool of the University of Wollongong, as well as the general community. Of the study sample, 51 participants had a diagnosis of dyslexia from a qualified psychologist. Fifty-one participants without a diagnosis of dyslexia or other learning disorder were then age and sex matched to the dyslexia sample. Participants were excluded if they reported

neurological abnormalities, psychotic illness or were not able to speak the English language fluently.

8.3.2 Measures

8.3.2.1 Neurological Soft Signs (NSS)

The Neurological Evaluation Scale (NES; Buchanan & Heinrichs, 1989) comprises 26 behavioural tasks. These items are scored on a 3-point scale; 0 (no abnormality), 1 (mild but definite impairment), or 2 (present). Total scores can range between 0 and 76. Fourteen of the items are assessed bilaterally, however in the current study bilateral items were summed as has been done previously (Compton et al., 2007; Theleritis et al., 2012).

NSS are scored on the basis of dysfunction in three areas: Sensory Integration (SI; audio-visual integration, stereogenesis, graphesthesia, extinction, right-left orientation), Motor-Coordination (MC; tandem walk, rapid alternating movements, finger-thumb opposition, finger-to-nose test) and the Sequencing of Complex Motor Acts (SCMA; fist-ring test, fist-edge-palm test, Ozeretski test, rhythm tapping). There are additional items which do not contribute to these subscales, but still contribute to the total NES score, including: synkinesis, convergence, gaze impersistence, glabellar reflex, snout reflex, grasp reflex, suck reflex. Handedness was measured by asking participants their hand preference (right/left) for 9 different activities. Hand preference was determined by summing responses: a score of 7 or higher indicates preference for that hand, however a score below 7 indicates mixed hand preference.

8.3.2.2 Schizotypy

The Schizotypal Personality Questionnaire (SPQ; Raine, 1991) is made up of 74 items requiring responses of yes or no. There is a total score as well as three dimensions (Cognitive-Perceptual, Interpersonal, Disorganised).

8.3.2.3 Verbal intelligence

The National Adult Reading Test (NART; Nelson, 1982) was used to measure verbal intelligence. This task required participants to read aloud 50 words with irregular spelling patterns. Errors were recorded when a word was mispronounced. As per the

instructions in the original manual, participants read the whole list of words and the discontinuation rule was not used. Lower scores indicate lower verbal intelligence.

8.3.2.4 Psychological distress

The General Health Questionnaire (GHQ; Goldberg and Hillier, 1979) was included as a state measure of current psychological distress. This questionnaire consists of 28 items enquiring about psychological, health, and related aspects of functioning over the previous two weeks. Items were scored from 0-3, with total scores ranging from 0-84. Higher scores represent higher levels of psychological distress. A total score of 23-24 has been indicated as the threshold for the presence of clinical distress (Sterling, 2011). Heightened levels of state distress have been reported in dyslexia (Undheim, 2003). Given that developmental disorders such as dyslexia have previously been discussed as possible risk factors for further psychiatric disorders (Remschmidt, 1996), this measure is a general indicator of current psychological functioning.

8.3.3 Procedure

The Human Research Ethics Committee at the University of Wollongong granted ethical approval for this study. Participants were approached through multiple means including the Psychology research participation program, student learning support services, flyers, word of mouth and snowballing. Participants completed the SPQ online. Individuals diagnosed with dyslexia that may have difficulties reading self-report measures were offered to complete these measures face-to-face, so that questions could be read out verbally. No participants requested this method of participation. Arrangements were made for participants to come onto campus to complete the NES, NART, and GHQ, all of which took approximately 50 minutes. Participants provided written informed consent. Individuals with dyslexia were financially compensated and those from the School of Psychology received course credit.

The NES was administered to participants by four trained evaluators. To assess inter-rater reliability 20 participants were jointly rated, whereby one rater was paired up with each of the remaining raters. Consistency in ratings was upheld, correlation coefficients for subscale and total scores ranged between .71 and .98. Raters were not blind to dyslexia status for NES scoring.

8.3.4 Statistical Analysis

Descriptive statistics were performed using SPSS 21 (IBM Corp, 2012). All variables met the +/- 2 guidelines for skewness, however the NES SCMA variable breached the +/-2 guidelines for kurtosis. Two outliers were identified using boxplot diagrams. Transformation of the NES SCMA variable was attempted however this did not improve kurtosis. Multivariate statistics are sufficiently robust to withstand violations of normality, so further investigations were continued. Independent Samples *t* tests (one tailed) were used to evaluate demographic group differences between participants with Dyslexia and Controls for continuous variables, and Chi-Squared tests for categorical variables. Correlations were computed for NSS and SPQ variables to explore the nature of the relationships between these constructs for each group (Dyslexia and Control). Both Pearson's and Spearman's correlations were conducted due to the relatively small sample size. Only those correlations which were significant across both analyses were reported as significant. A Multivariate Analysis of Variance (MANOVA) was used to investigate whether those with Dyslexia differed from Controls in the expression of NSS and schizotypy. To finish, a Binary Logistic Regression was used to determine the contributions of NSS, schizotypy, handedness, psychological distress and verbal intelligence to Dyslexia group membership.

8.4 Results

8.4.1 Demographic and clinical characteristics

Since participants with Dyslexia were matched with Controls according to age and sex, no significant differences between groups were found for these variables. All demographic data and test statistics are shown in Table 8.1. No group differences were found for living arrangements and the use of health services within the last 6 months. As expected, the Dyslexia group had a significantly higher prevalence of mixed and left-handedness reported compared to Controls. The Dyslexia group also expressed significantly higher levels of psychological distress compared to Controls. The mean level of distress for the Dyslexia group is considered above the threshold for clinically significant distress (Sterling, 2011).

Table 8.1. *Demographic, clinical statistics (mean, SD) and frequencies of Dyslexia and Control groups.*

	Dyslexia (N = 51)	**Controls (N = 51)**	**Test statistic and *p* value**
Sex (M:F)	16:35	15:36	$\chi^2 = 0.46$, $df = 1$, $p = .83$
Age	24.8 (10.37)	24.14 (9.03)	t (100)= .346, $p = .73$
Living arrangements (Parents:Siblings: Partner:Friends: Acquaintences: Alone)	22:1:9:11:5:3	26:3:10:6:4:2	$\chi^2 = 3.16$, $df = 5$, $p = .67$
Health service use within last 6 months (Y:N)	28:22	32:20	$\chi^2 = .476$, $df = 1$, $p = .49$
Handedness (Right:Left:Mixed)	Right = 80.4% Left = 7.8% Mixed = 11.8%	Right = 96.1% Left = 3.9% Mixed = 0%	$\chi^2 = 7.38$, $df = 2$, **$p = .02$**
Psychological distress	25.69 (15.47)	19.86 (12.75)	t (96.47)= *2.07*, **$p = .041$**

SD= standard deviation; N=Number of participants in group; M=Male; F=Female; Y=Yes; N=No. Significant differences between dyslexia and control groups at the p<.05 level are highlighted in ***bold.***

8.4.2 Correlations between Neurological Soft Signs and Schizotypy

Pearson's and Spearman's correlations were conducted between NSS and SPQ variables. Pearson's correlations are reported for the Dyslexia and Control groups in Table 8.2, with only those correlations which were significant across both Pearson's and Spearman's analyses reported as significant in this table.

8.4.2.1 Dyslexia Group

Concerning the Dyslexia group, a significant positive correlation was found between the Cognitive-Perceptual SPQ dimension and the NES MC subscale.

8.4.2.2 Control Group

For the Control group significant negative correlations were found between the NES SCMA subscale and the SPQ Total score and Interpersonal dimension. These correlations were repeated, removing the two outliers which were identified for the SCMA NSS variable. No differences were found, with the correlations between the NES SCMA subscale score and the SPQ Total score and Interpersonal dimension remaining negative and significant.

Table 8.2. *Correlations between SPQ and NES variables for Dyslexia and Control groups.*

	Dyslexia group (N=51)			
	SPQ Total	SPQ Cog-Per	SPQ Inter	SPQ Dis
NES Total	.178	.097	.172	.217
NES SI	.066	.059	.015	.187
NES MC	.2	.301*	.184	.074
NES SCMA	-.064	-.088	-.027	-.075
	Control Group (N=51)			
	SPQ Total	SPQ Cog-Per	SPQ Inter	SPQ Dis
NES Total	.117	.138	.084	.109
NES SI	.191	.178	.217	.085
NES MC	.128	.15	.083	.169
NES SCMA	-.29*	-.182	-.309*	-.229

Note: N = Number of participants in group; SPQ = Schizotypal Personality Questionnaire (Raine, 1991); NES = Neurological Evaluation Scale (Buchanan and Heinrichs, 1989); Cog-Per = Cognitive-Perceptual; Inter = Interpersonal; Dis = Disorganised; SI = Sensory Integration; MC = Motor Coordination; SCMA = Sequencing of Complex Motor Acts; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.000$.

8.4.2.3 Comparison of Correlations

Fisher's r-to-z transformations were used to investigate whether the Pearson's correlation coefficients for the Dyslexia and Control groups were significantly different from one another. No significant differences between the correlations were found.

8.4.3 Neurological Soft Signs, Dyslexia, and Schizotypy

The Dyslexia group scored significantly higher than the Control group on all SPQ Total and dimension scores. Those with Dyslexia were also found to express significantly more Total, SI and SCMA NSS compared to Controls. The Dyslexia group also performed significantly worse on the NART measure of verbal intelligence. Means, test statistics and effect sizes are reported in Table 8.3.

8.4.4 Dyslexia, schizotypy and psychological distress

Given that the Dyslexia group reported significantly higher levels of psychological distress and schizotypy compared to Controls, it is potentially the case that distress in dyslexia is due to schizotypy, rather than coming about as a by-product of dyslexia status. Schizotypy has been previously associated with increased psychological distress (Cella et al., 2013), and so may also contribute to the distress reported in the dyslexia sample of this study. Accordingly, an Analysis of Covariance (ANCOVA) investigated psychological distress in Dyslexia versus Control groups, with the Cognitive-Perceptual, Interpersonal and Disorganised SPQ dimensions as covariates. Results indicated that when SPQ dimensions were included as covariates, the difference in level of distress between Dyslexia and Control groups was not significant ($p = .634$). Cognitive-Perceptual schizotypy ($F(1, 97) = 8.227$, $p = .005$, $\eta^2_p = .078$) and Interpersonal schizotypy ($F(1, 97) = 10.407$, $p = .002$, $\eta^2_p = .097$) specifically were found to have significant co-varying effects.

Table 8.3. *Comparison of schizotypy, NSS and verbal intelligence in Dyslexia and Control participants (group means (SD) reported).*

	Dyslexia (N = 51)	**Controls (N = 51)**	**Test statistic, *p* value, Effect size**
SPQ Total	33.04 (16.46)	17.73 (12.53)	F (1, 100) = 27.952, **p < .001,** Cohen's d = 1.05
Cognitive-Perceptual SPQ	11.33 (7.59)	6.9 (5.93)	F (1, 100) = 10.783, **p = .001,** Cohen's d = .65
Interpersonal SPQ	13.73 (7.09)	6.94 (6.06)	F (1, 100) = 23.668, **p < .001,** Cohen's d = 1.03
Disorganised SPQ	9.45 (4.32)	4.55 (3.59)	F (1, 100) = 38.849, **p < .001,** Cohen's d = 1.23
NES Total	13.27 (4.34)	8.19 (4.46)	F (1, 100)= 33.931, **p < .001,** Cohen's d = 1.15
NES Sensory Integration	2.41 (1.29)	1.74 (1.32)	F (1, 100)= 6.588, **p = .012,** Cohen's d = .51
NES Motor Coordination	1.57 (1.25)	1.27 (1.26)	F (1, 100)= 1.39, p = .241, Cohen's d = .24
NES Sequencing of Complex Motor Acts	1.72 (2.01)	.59 (.96)	F (1, 100)= 13.271, **p < .001,** Cohen's d = .72
Verbal intelligence	21.09 (6.95)	28.51 (6.59)	F (1, 100)= 30.497, **p < .001,** Cohen's d = -1.1

SD= standard deviation; N=Number of participants in group; SPQ=Schizotypal Personality Questionnaire (Raine, 1991); NES=Neurological Evaluation Scale (Buchanan and Heinrichs, 1989). Significant differences between dyslexia and control groups at the p<.05 level are highlighted in ***bold.***

8.4.5 Predicting Dyslexia group membership

Binary Logistic Regression was used to examine the contributions of schizotypy, NSS, handedness, psychological distress and verbal intelligence to Dyslexia group membership (Table 8.4). The Enter method was used to input variables. VIF statistics for all variables remained below 10, indicating multicollinearity has not occurred. Cognitive-Perceptual SPQ, Handedness, NART (verbal intelligence) and SCMA NES were entered in the first block ($\chi^2 = 47.083$, $df = 5$, $p < .001$). Variables entered in the second block included Interpersonal SPQ, GHQ (psychological distress) and SI NES ($\chi^2 = 6.553$, $df = 3$, $p = .088$). In the third and final block Disorganised SPQ and MC NES were included in the analysis ($\chi^2 = 10.537$, $df = 2$, $p = .005$). Dyslexia group membership was predicted by NES SCMA, Disorganised SPQ and NART (verbal intelligence). In this prediction Dyslexia group membership was characterized by a greater expression of Disorganised schizotypy and SCMA NSS, as well as lower verbal intelligence. The overall model explained over 62% of the variance in Dyslexia group membership (Nagelkerke $R^2 = 0.623$), and correctly predicted 82.4% of Control participants, and 80.4% of participants with Dyslexia.

Table 8.4. *Binary Logistic Regression: Prediction of Dyslexia group membership as Outcome Variable.*

	Odds ratio	**95% C.I.**		***P* value**	**Wald Statistic**	**VIF**
		Lower	**Upper**			
Cognitive-Perceptual SPQ	.884	.761	1.027	.108	2.588	2.54
Handedness	1.249	.142	10.971	.98	.04	1.22
NART	.879	.8	.967	**.008**	7.028	1.31
Sequencing of Complex Motor Acts NES	1.707	1.026	2.841	**.04**	4.236	1.28
Interpersonal SPQ	1.062	.944	1.195	.315	1.008	2.49
Sensory Integration NES	1.336	.818	2.181	.247	1.342	1.14
GHQ	.999	.946	1.056	.973	.001	1.69
Disorganised SPQ	1.39	1.123	1.721	**.002**	9.147	2.71
Motor Coordination NES	1.036	.644	1.666	.884	.021	1.16

Note: Model fit: $\chi^2 = 64.173$, $df = 10$, $p < .001$. Enter method of variable selection was used in this analysis. For the handedness variable categorical classification right-handedness

was used as the reference point. Significant effects at p<.05 level are highlighted in ***bold****. SPQ = Schizotypal Personality Questionnaire (Raine, 1991); NES = Neurological Evaluation Scale (Buchanan and Heinrichs, 1989); NART = National Adult Reading Test (Nelson, 1982); GHQ = General Health Questionnaire (Goldberg and Williams, 1988), VIF = Variance Inflation Factor (values greater than 10 indicate multicollinearity).*

8.5 Discussion

Neurological soft signs, schizotypy and handedness were examined in a dyslexia sample compared to individuals without dyslexia. Individuals with dyslexia were found to score significantly higher on all schizotypy dimensions, and had a significantly greater rate of mixed and left-handedness compared to Controls, which confirmed results of previous studies (Richardson & Stein, 1993; Richardson, 1994). As expected those with dyslexia expressed significantly more NSS compared to Controls. Dyslexia group membership was predicted by a greater expression of SCMA NSS, higher levels of Disorganised schizotypy and lower verbal intelligence. Handedness did not contribute to predicting dyslexia status. Although not hypothesised, in our sample those with dyslexia reported significantly higher levels of distress, which were within clinical limits, as measured with the GHQ. Follow up analyses revealed that the higher level of distress in those with dyslexia may be accounted for by schizotypy, which was a novel and unexpected finding.

Significantly more SI, SCMA and Total NSS were found in the Dyslexia group compared to Controls. NSS in adults have been proposed as indicators of neurodevelopmental abnormality (Shaffer, O'Connor & Shafer, 1983; Bombin, Arango, & Buchanan, 2005). Studies investigating the neural basis of dyslexia have hypothesised that it may be a disconnection syndrome, given that compared to Controls, differences in local white matter have been reported in children and adults with dyslexia in the left inferior frontal gyrus and left temporoparietal regions (e.g. Deutsch et al., 2005; Dougherty et al., 2007; Rimrodt et al., 2010). Interestingly, abnormal activation and atrophy in the inferior frontal gyrus (among other areas) has been linked to the expression of NSS (Zhao et al., 2014). These imaging findings point to possible neurological correlates of the subtle inefficiencies detectable through the assessment of NSS.

Concerning the current study, SCMA NSS specifically was predictive of dyslexia status. Dyslexia is often comorbid with developmental coordination

disorder (DCD) (e.g. O'Hare and Khalid, 2002), which is characterized by extreme difficulties in the ability to illicit motor skills at an age appropriate level (American Psychiatric Association, 2013). Motor sequencing NSS have previously been elevated in DCD (Licari et al., 2015) and in children with coordination problems (Fellick et al., 2001). Furthermore, similar to NSS and dyslexia, the neural origins of DCD are believed to involve dysfunctions of the subcortical network, with the cerebellum-thalamus-basal ganglia circuit implicated specifically (Zwicker et al., 2011). Therefore, it is possible that the current results may be reflective of overlapping coordination difficulties in the dyslexia sample. SCMA NSS being predictive of dyslexia status seems to indicate that in dyslexia the neural pathways between sensory and complex motor brain regions are not functioning as efficiently as they otherwise should. Therefore neurodevelopmental problems underlying higher levels of motor sequencing NSS may explain the overlap between dyslexia and DCD observed in previous studies (e.g. O'Hare and Khalid, 2002).

Schizotypy was expressed in higher levels in the Dyslexia group compared to Controls. This finding is significant for two reasons: firstly, it replicates and extends previous research demonstrating increased positive schizotypy in dyslexia (Richardson and Stein, 1993; Richardson, 1994), with our results showing that all schizotypal dimensions were significantly elevated in the Dyslexia group compared to Controls. Secondly, when this finding is paired with the increased rates of NSS in dyslexia, it points towards overlapping neurodevelopmental abnormalities for dyslexia and the psychosis continuum. One of the most pervasive characteristics across the psychosis continuum is abnormalities in language processing (Kuperberg, 2010). Some researchers have gone so far as to say that the language dysfunction found in schizophrenia meets criteria for a developmental language disorder such as dyslexia (Bersani et al 2006; Condray, 2005). Neurodevelopmentally, research has shown dyslexia and psychosis to have shared genetic origins (Becker et al., 2012). The current study has added to this body of research, with both NSS and schizotypy being heightened in our Dyslexia sample compared to Controls. Unfortunately due to the cross-sectional nature of the study we are unable to comment on causation. Understanding how and when language problems, schizotypy and NSS occur along the psychosis continuum would contribute significantly to our knowledge of trait risk for psychosis, and is worthy of further research.

A significant increase in mixed handedness has previously been reported in dyslexia (Richardson, 1994; Brunswick & Rippon, 1994), with the current study replicating this result, supporting the view that abnormal structural and functional cerebral lateralisation is implicated in dyslexia (e.g. Hernandez et al., 2013; Altarelli et al., 2014). Given this body of literature, it is intriguing that handedness did not contribute unique variance to predicting dyslexia status. It is possible that with a larger sample size the predictive value of handedness may become significant, therefore replication of this research would be fruitful. Verbal intelligence was significantly lower in those with dyslexia, and also provided unique contribution to predicting dyslexia status in our study. This is perhaps unsurprising, given that the NART assesses verbal intelligence by participants reading atypically spelt vocabulary aloud. Taken together, these findings suggest that individuals with dyslexia often also present with neurodevelopmental markers that could place them in a category of risk for future psychopathology (e.g. Reichenberg et al., 2005; Tsuang et al., 2011).

There were higher levels of distress in those with dyslexia compared to those without this diagnosis. Efforts were made to recruit community-based individuals but many from a student population were included in this sample. One possibility is that those with dyslexia in a university setting may be confronted by their difficulties with greater intensity, leading to higher levels of distress than those found in the community. Indeed, a lower *perceived* IQ has been reported alongside heightened distress for adult males with dyslexia (Boetsch, Green and Pennington, 1996). It is also possible that from a clinical perspective, the cost associated with language difficulties on top of the social isolation and perceptual disturbances often linked with schizotypy may result in heightened distress. The exploratory ANCOVA results indicated that when the effects of schizotypy were controlled for those with dyslexia no longer had differences in their level of distress compared to Controls. The Cognitive-Perceptual and Interpersonal schizotypy dimensions specifically were significant covariates. These results suggest that the perceptual disturbances, flat emotionality, and unusual and asocial behaviour occurring as a result of these schizotypal dimensions, may have a role in heightening distress for those with dyslexia. Previous research has suggested that difficulties with reading and language may account for depression and low self-esteem in those with dyslexia (e.g. Riddick, 1996; Alexander-Passe, 2006). However, an alternative explanation is presented

here, whereby the schizotypal personality composition associated with dyslexia may also contribute to heightening distress.

The relationship between schizotypy dimensions and NSS within both Dyslexia and Control groups was also explored by way of correlational analyses. In dyslexia, a significant positive relationship was found between Cognitive-Perceptual SPQ and MC NES. In Controls, negative correlations were found between SCMA NES and Interpersonal as well as Total SPQ. Previous research has reported significant positive relationships between Interpersonal and Total SPQ scores and SCMA NES for healthy participants (e.g. Theletris et al., 2012; Bollini et al., 2007). Given that the current study has reported negative relationships between SCMA NSS and schizotypy variables, further research is required to tease apart these findings. The correlations between schizotypy dimensions and NSS were expected to be weaker in dyslexia compared to the same relationships in Controls. Unexpectedly no significant differences were found between Dyslexia and Control groups for the strength of the relationships between schizotypy and NSS. This finding suggests that contrary to hypotheses, the relationships between schizotypy and NSS variables for those with and without dyslexia may be similar. Further investigation with larger sample sizes is required.

Although the results of this study support and extend previous research, this research was not without its limitations. The primary limitation faced was the cross-sectional nature of the study. It would have been beneficial to have had multiple time points for the measurement of distress and schizotypy in our samples in order to determine the temporal sequence of these variables, and also to note any fluctuations over time. Another limitation of the current study was the sample size. Although the number of participants provided adequate power to perform the required analyses, it would have been beneficial to investigate whether other known state risk factors for psychosis, such as hallucination predisposition, may have impacted on the current results. Unfortunately the size of the sample did not afford us sufficient power to conduct these additional analyses. Finally, the use of self-report measures with a dyslexic sample has inherent limitations. Although the offer was made to read out measures verbally if required, no participants took up this offer. Since many of the participants diagnosed with dyslexia were currently enrolled in tertiary education, it is probable that they are not in need of assistance with reading and have developed their own compensatory strategies in this area. Yet it is also possible that questions

may have been misinterpreted as a result of the inherent deficits in reading that are associated with dyslexia.

Those diagnosed with dyslexia had higher levels of schizotypy, NSS, and psychological distress, as well as lower verbal intelligence and increased mixed/left handedness, compared to healthy controls. Unexpectedly the heightened level of distress seen in dyslexia may be related to co-occurring Interpersonal and Cognitive-Perceptual schizotypy personality traits. Dyslexia status was predicted by higher levels of Disorganised schizotypy and increased expression of SCMA NSS. These findings suggest that dyslexia shares neurodevelopmental risk variants in common with the psychosis continuum. Further research with longitudinal methods is required to understand the causal mechanisms involved in these neurodevelopmental phenomena.

9 STUDY FOUR: SEMANTIC PROCESSING IN COGNITIVE-PERCEPTUAL SCHIZOTYPY AND HALLUCINATION PRONENESS

9.1 Abstract

Introduction: Research examining semantic processing in psychosis proneness has produced mixed results. The present study aimed to elucidate potential differences in the processing of semantic relations for positive schizotypy and hallucination prone individuals compared to controls.

Method: One hundred and eighty-three participants completed the Schizotypal Personality Questionnaire, Launay-Slade Hallucination Scale, National Adult Reading Test, a handedness measure, and a computerized semantic relatedness judgement task. Participants were divided into four groups using a mean split on cognitive-perceptual schizotypy and hallucination proneness.

Results: Significant differences between groups were found for reaction time on the semantic relatedness task, with the high cognitive-perceptual schizotypy groups responding significantly slower to all word pairs compared to their low scoring counterparts. There was some evidence that high hallucination proneness was associated with significantly faster reaction times which may reflect disinhibitive processes, however additional support is required.

Conclusions: These results imply more diffuse activation of semantic information in schizotypy, which differs from the efficient semantic processing capacity demonstrated in those predisposed to hallucinations. These results have significant implications in the re-conceptualisation of hallucination proneness as distinct from positive schizotypy.

9.2 Introduction

Semantic processing refers to the processing of word meanings, where word activation stimulates other words with similar and related meanings. Semantic processing abnormalities have been proposed as central to the cognitive features of schizophrenia pathology (Goldberg et al., 1998). Schizophrenia patients exhibit semantic processing abnormalities in a variety of cognitive tasks (Brebion et al., 2004; Langdon et al., 2002; Langdon & Coltheart, 2004; Rossell et al., 2000; Tavano et al., 2008), with semantic dysfunction the result of a reduced ability to integrate context with meaning (Iakimova et al., 2005). Disorder of the inhibition and activation mechanisms necessary to facilitate the spread of activation across the semantic network are believed to be responsible for abnormal semantic processing (e.g. Kumar & Debruille, 2004; Soriano et al., 2008; Niznikiewicz et al., 2010). There are confounds in collecting these data in patients with schizophrenia including medication, substance use, the effects of diagnosis and hospitalization and the effects of chronicity of symptoms. Therefore one approach to providing more enriched information for risk factors for psychosis is to consider schizotypy as measured in the general population as an analogue or proxy for symptoms in patients.

Along with schizophrenia at the extreme end, schizotypy exists along a continuum of psychosis (Van Os, 2003), with at risk mental state and schizotypal personality disorder being intermediaries between high schizotypal individuals from the general population and diagnosed psychotic disorders (Debbané et al., 2015). Schizotypy is a normally distributed multidimensional personality trait resembling dispositional features of schizophrenia. Around 10% of the population exhibit high levels of this trait (Tien, 1991). Defining features of schizotypy include cognitive-perceptual experiences (magical ideation, ideas of reference, suspiciousness, unusual perceptual experiences), interpersonal characteristics (excessive social anxiety, no close friends, constricted affect) and disorganisation (odd speech, eccentric behaviour). Factor analytic studies have shown that the three schizotypal dimensions closely resemble the positive, negative and disorganized symptom clusters of schizophrenia (Raine et al., 1994; Stefanis et al., 2004; Mason, 2015). When schizotypy is combined with other genetic and environmental risk factors for psychosis, likelihood of transition to a diagnosable disorder is increased (e.g. van Os, Rutton & Pulton, 2008; Cannon et al., 2008). Therefore, schizotypy can be

understood as a biological and cognitive vulnerability to psychosis (Morrison et al., 2006; Woods et al., 2009; Tarbox et al., 2012; Pogue-Geile & Yokley, 2010; for review, see Tarbox & Pogue-Geile, 2011). Abnormalities in the lateralization of language processing are documented in those with positive schizotypy (Hori et al., 2008; Nunn and Peters, 2001) and those scoring highly on schizotypy in general (Mohr et al., 2005; Kravetz, Faust & Edelman, 1998; Suzuki & Usher, 2009; Weinstein & Graves, 2002). Therefore additional consideration needs to be given as to whether there are differences in semantic processing attributable to positive schizotypy and other psychotic symptoms in healthy individuals from the general population.

One of the main tasks used to evaluate activation in semantic networks is the behavioural priming paradigm. In this task a priming word is presented (e.g. ball), followed by a target word which is semantically related (e.g. soccer) or unrelated (e.g. coffee). Semantic priming occurs when the participant responds to the related word significantly faster/more accurately compared to an unrelated word. This facilitation for the related item is thought to occur because of the organisation of the semantic network into nodes. Semantically related nodes are located closer together, whilst unrelated words are farther apart. Previous experience and interaction with the related word pairs drives the priming effect. Indirect priming occurs when the prime and target are not directly related, but mediated by another concept (e.g. prime CAT and target CHEESE are mediated by MOUSE). In schizophrenia most semantic priming studies show evidence of increased indirect priming, suggesting there is less constraint on the spread of activation in the semantic system (Weisbrod et al., 1998; Zeev-Wolf et al., 2014; 2015; see Pomarol-Clotet et al., 2008 for meta-analysis).

Research investigating semantic processing in positive schizotypy is consistent with a reliance on the right hemisphere (Mohr et al., 2001; Gianotti et al., 2001; Pizzagalli et al., 2001). Semantic priming studies have documented greater indirect priming in positive schizotypy (Kerns & Berenbaum, 2000; Morgan, Bedford & Rossell, 2006). In those with high schizotypy, the activation of a broad range of distantly related associates during indirect priming results in semantic processing capabilities that are exceedingly diffuse (Grimshaw et al., 2010; Morgan, Bedford & Rossell, 2006). Yet other studies have found no relation between atypical semantic processing and schizotypy (Fisher & Weinman, 1989; Moritz et al., 1999;

Morgan et al., 2009). Therefore clarifying the nature of any differences in semantic processing in positive schizotypy is an important goal of the present study.

One mechanism proposed for the differential processing of ambiguous relations in high schizotypal individuals is reduced cognitive inhibition. A reduction in cognitive inhibitory processes has been regarded as central to high schizotypy, and is also directly related to language processing ability (Beech & Claridge, 1987). Inhibition deficits refer to dysfunctions in the ability to discriminate between factors pertinent to the current scenario and unrelated "noise". It may be that the diffuse activation of semantic associates in high schizotypy is linked to an inability to inhibit features unrelated to the task. These deficits in inhibition would explain the over-activation of right hemisphere distant and unusual meanings in both schizophrenia and schizotypy. The failure to inhibit irrelevant semantic stimuli has been supported for the most part in recent schizotypy studies (Grimshaw et al., 2010; Humphrey, Bryson & Grimshaw, 2010). Furthermore, research has shown that individuals with high schizotypy are significantly less likely to show negative priming (slower responding to a stimulus that recently had to be ignored, Moritz et al., 2000; Steel, Hemsley & Pickering, 2007), as well as having a greater propensity to endorse positive responses in many different research tasks (Reed et al., 2008; Humphrey, Bryson & Grimshaw, 2010). These findings are reflective of a reduction in cognitive inhibition, and it is this mechanism that is hypothesised to underlie atypical semantic processing in high positive schizotypy.

Positive schizotypy is a complex trait feature including unusual beliefs, thoughts and perceptual experiences. However, the processing of semantic relations has been linked more specifically to subclinical state psychotic symptoms, such as hallucinatory experiences (Vercammen & Aleman, 2010). Therefore in the current study, it is deemed necessary to distinguish between positive schizotypy as a trait-like feature and hallucinatory predisposition as a related but distinct psychotic-like experience. Auditory-verbal hallucinations (AVH) are experienced by 5- 28% of healthy individuals at some point in their lives (Johns et al., 2004, for review see de Leede-Smith & Barkus, 2013). One of the most accepted mechanisms precipitating AVH onset is a reduced ability to inhibit intrusive thoughts and memories (Badcock & Hugdahl, 2012), which are believed to occur due to impaired inhibition in the top-down processing system (Kompus et al., 2011). Since inhibitory dysfunctions exist in both trait positive schizotypy and the hallucinatory experiences, it might be

expected that lack of inhibition associated with AVH will also impact on semantic processing. If AVH proneness is a state feature reflective of psychotic illness risk, individuals with high AVH proneness should exhibit a similar pattern in semantic processing to individuals with high positive schizotypy.

In the present study, semantic processing was evaluated via the use of homographs (words with the same spelling but 2 different meanings). Disambiguating meaning in the English language requires the activation of the appropriate semantic pathway, and deactivation (inhibition) of the incongruous alternate meaning(s). In the current experiment (adapted from Grimshaw et al., 2010), participants were presented with an ambiguous word (prime), immediately followed by another word (target), which is either: related to the dominant meaning of the prime, related to the subordinate meaning of the prime, or unrelated to the prime. If the judgment of relatedness is viewed as a signal detection task, it is possible to derive measures of sensitivity (accuracy of response) and response criterion (bias in response patterns). Sensitivity to relatedness was taken to reflect differences in semantic organisation, whilst the criterion measure showed bias to report semantic pairs as related (lax decision making bias) or unrelated (conservative decision making bias) under ambiguous conditions. A stimulus onset asynchrony (SOA) of 750ms was utilised as previous research has demonstrated that this SOA is when inhibitory processes are most likely occurring (Atchley, Burgess, & Keeney, 1999; Burgess & Simpson, 1988).

It was hypothesized that the reaction time responses of high and low positive schizotypy and AVH prone groups to dominant word pairs would not differ, and would be characterized by significantly faster reaction times to dominant targets of the prime compared to unrelated targets (a task effect). For subordinate word pairs, it was expected that the low positive schizotypy and AVH prone groups would exhibit significantly slower reaction times compared to dominant pairs, due to the inhibition of subordinate meanings (which is the expected semantic function in the general population, Burgess & Simpson, 1988). Contrastingly it was hypothesized that the group factors of high positive schizotypy and AVH proneness would interact with meaning such that subordinate meanings for target words would be activated by the prime word, due to reduced inhibitory function in these groups. Therefore a smaller difference in reaction time between dominant and subordinate meanings was

hypothesized for these groups. Accuracy and decision making bias was investigated using sensitivity and criterion signal detection outcomes.

9.3 Method

9.3.1 Participants

One hundred and eighty-three undergraduate students from the University of Wollongong, NSW, Australia took part in the study to achieve credit toward their chosen first or second year course (mean age 22 years (SD 7.16), age range 17-60 years, 75.4% female). Informed verbal and written consent was obtained prior to the study commencing. Demographic characteristics of the sample are provided in Table 9.1.

9.3.2 Measures

Each participant completed an initial demographic questionnaire, requiring details such as age, sex, and any current or previously diagnosed mental illness. Handedness was determined, and following this, participants completed the Schizotypal Personality Questionnaire (SPQ; Raine, 1991), Launay-Slade Hallucination Scale (LSHS; Launay and Slade, 1981), National Adult Reading Test (NART; Nelson, 1982), and finally the computerized Semantic Ambiguity Task (adapted with permission from Grimshaw et al., 2010).

The SPQ consists of 74 items requiring either a yes or no response. These items add up to create a total score and 3 dimensions: Cognitive Perceptual (CP) Schizotypy (also referred to as the positive schizotypal dimension), made up of Ideas of Reference, Magical Thinking, Unusual Perceptual Experiences, and Suspiciousness; Interpersonal Schizotypy (negative schizotypal dimension), consisting of No Close Friends, Constricted Affect, Excessive Social Anxiety, and Suspiciousness subscales, and; Disorganized Schizotypy, with the Odd/Eccentric Behaviour and Odd Speech subscales. The overall mean score for participants on the SPQ was 27.28 (1.18 S.E.). The focus of the current study is positive schizotypy so participants were divided into groups based on their CP SPQ score. Given that no outliers were detected, the mean was considered an adequate measure of central tendency. The mean score for participants on this factor was 10.57 (0.52 S.E.).

Those with a score above the mean were the high CP schizotypy group, whilst those scoring at or below the mean were the low CP schizotypy control group.

The LSHS is a 12-item questionnaire designed to identify those predisposed to hallucinatory experiences. A LSHS total score was calculated on the basis of a positive response to each perceptual experience. The overall mean score for all participants on the LSHS was 3.43 (0.17 S.E.). Those with a score above the mean were grouped as the high AVH prone group, whilst those scoring at or below the mean were the low AVH prone group.

Handedness for each participant was determined via interview (exert question from the Neurological Evaluation Scale; Buchanan & Heinrichs, 1989). Each participant was asked which hand they prefer to use when performing 9 various tasks (i.e. writing, sweeping with a broom, unscrewing the lid of a jar). Handedness was calculated by adding up the number of times the participant used each hand. If Right/Left hand was used for 7+ activities, this determined handedness. However if the Right/Left hand was used for less than 7 activities, the individual was classified as 'Mixed' handedness.

The NART was used to estimate verbal intellectual ability. Participants were required to read aloud a list of 50 words increasing in difficulty. The number of pronunciation errors was recorded.

9.3.2.1 Semantic Ambiguity Task

This task was conducted on a laptop in a quiet room at the University of Wollongong. Participants were told that they would see one word flash up on the screen (prime), followed immediately by another word (target). Once the second word (target) disappeared they were required to make a response indicating whether both the words were related (pressing key 1) or unrelated (pressing key 2) to each other. The task consisted of a total of 144 trials, in which participants were asked to respond as accurately and quickly as possible.

The prime words were deliberately chosen to be ambiguous homographs (words with more than one meaning; first used by Burgess & Simpson, 1988). Forty different prime words were used, and matched to either a dominant related word of that prime or a subordinate related word of that prime. For example, for the prime 'ball', a dominant word pair would be 'round', whereas a subordinate word pair would be 'dancing'. Seventy-two related word pairs were used in the task (36

dominant and 36 subordinate), along with 72 completely unrelated word pairs (where a prime was pseudo-randomly paired with one dominant and one subordinate word of a different unassociated prime). Each participant saw each prime twice: paired with either a related target (half of which were dominant, half subordinate), or an unrelated target (half of which were dominant, half subordinate). Participants also saw each target on two occasions, under the same conditions as described previously. Counterbalancing occurred, such that if a participant viewed a prime paired with a dominant and related target, they would also see the same prime paired with a subordinate and unrelated target, and vice versa. Counterbalancing also occurred across pairings with the use of two word lists. These word lists were comprised of the same words, however paired differently. For example, if in the first word list the related word pair was subordinate and the unrelated word pair was dominant, this would be reversed in the second word list (so the related word pair would be dominant and the unrelated word pair would be subordinate). Participants completed the task with word list 1 *or* 2, resulting in half the participant pool completing each version of the task. The differences between the two word lists was only in the pairing of words-each word list contained the same words just paired differently. Additional details regarding the pairings of the prime and target words can be found in Grimshaw et al (2010).

Each trial was preceded by a fixation mark in the centre of the screen (1000ms), followed by a centrally presented prime word (50ms). A stimulus onset asynchrony (SOA) of 750ms then followed, after which the target was presented for 180ms. Participants were given 3000ms to make a response (Pressing key 1 for 'related' or key 2 for 'unrelated'), after which there was a further 3000ms inter-stimulus interval between their response and the beginning of the subsequent trial.

9.3.3 Procedure

Ethical approval to commence the study was obtained by the University of Wollongong Human Research Ethics Committee. Written informed consent was obtained from participants before testing commenced. Participants were reimbursed with course credit for their time.

9.3.4 Statistical Analysis

Data analysis was conducted using SPSS Version 21 (IBM, 2012). Response time analyses were based on median response times for concordant (correct) responses. To control for the random effects of both participants and items a subject (*F1*) analysis and item (*F2*) analysis were run using a Repeated Measures Analysis of Variance (ANOVA).

Since this task is a signal detection task, performance accuracy was divided into two components: sensitivity (*d´*) and criterion (*c*). Sensitivity refers to the participant's ability to accurately discriminate between related and unrelated targets (i.e. to respond correctly). The sensitivity analysis was completed using a Repeated Measures ANOVA. Criterion is related to the decision making bias of the participant. This bias is evident under conditions of uncertainty or ambiguity, under which participants will have a propensity to respond with either a lax or conservative pattern of response. A lax pattern of response would involve responding 'related' more so than 'unrelated' when uncertain, whereas in a conservative pattern of response the participant would be more likely to classify uncertain targets as 'unrelated.' For the criterion measure, positive values indicate a conservative decision making bias, whereas negative values are indicative of a lax decision making bias.

The Signal Detection variables were calculated using the Macmillan and Creelman (2005) criteria:

$d´ = z(\text{hits}) - z(\text{false alarms})$

$c = -0.5\ (z(\text{Hits}) + z(\text{false alarms}))$

To allow comparison of *c* across dominant and subordinate conditions, the *c* variable needs to be on the same scale, with the mean of the unrelated distribution as the zero point. To accomplish this, an arithmetic transformation was used, where *d´* for each condition was divided by 2, with *c* then added to it. Dominant and subordinate conditions could then be compared via a *t* test. To compare *c* between CP schizotypy and AVH prone groups *c* was then converted into relative *c´,* as suggested by Macmillan and Creelman (2005). This was done by dividing *c* by *d´*. Doing this allows the difference between the groups on *d´* to be taken into account so

that they can be compared. The *c´* for high and low CP schizotypy, and high and low AVH prone groups was then compared via the use of an ANOVA.

In cases where the number of hits or false alarms was 0 or 1, an adjustment was applied to avoid infinite values. Proportions of 0 and 1 were converted using the formula 1/(2N) and 1−1/(2N), respectively, where N symbolises the number of trials that proportion is based upon (Macmillan & Creelman, 2005). Therefore values of 0 and 1 were converted to 0.014 and 0.986 respectively. False alarms were defined as responding 'related' to an unrelated item, whereas hits were defined as the correct response (response of 'related' to a related item). Given that false alarms were universal across dominant and subordinate conditions (i.e. conditions are equal in their unrelatedness), they were summed across the conditions, making the false alarm rate out of 72.

9.4 Results

9.4.1 Participants

Demographic variables for the interaction between CP schizotypy and AVH proneness are presented in Table 9.1. There were no significant differences in sex ratios, age, handedness, SPQ total or subscale scores, or the NART measure of verbal intelligence. However, significant group differences were found for LSHS total score. Pairwise comparisons revealed that all four groups scored significantly different from each other, with those high on CP schizotypy and AVH proneness scoring highest (M = 5.898, SE = .159), followed by the low CP schizotypy, high AVH prone individuals (M = 4.333, SE = .234), then the high CP schizotypy, low AVH prone participants (M = 2.179, SE = .23), and those with low CP schizotypy and AVH prone scoring lowest for LSHS total score (M = 1.426, SE = .148).

Table 9.1. *Demographic variables for interaction between Cognitive-Perceptual (CP) schizotypy groups and Auditory-Verbal Hallucination (AVH) proneness groups.*

Variable	**High CP schizotypy, High AVH prone (n=59)**	**High CP schizotypy, Low AVH prone (n=28)**	**Low CP schizotypy, High AVH prone (n=27)**	**Low CP schizotypy, Low AVH prone (n=68)**	**Test statistic and *p* value**
Sex (Male: Female)	16:43	9:19	6:21	13:55	$\chi^2 = 2.254$, $df = 3$, N = 182, $p = 0.521$
Age	21.98 (7.85)	20.07(3.54)	21.26 (4.76)	23.01 (8.33)	F (1,178) = 2.505, MSE = 128.799, p = .115
Handedness (Right: Left: Mixed)	56:3:0	25:3:0	21:5:1	58:10:0	$\chi^2 = 10.322$, $df = 6$, N = 182, $p = 0.112$
SPQ Total score	42.75 (11.55)	32.5 (11.63)	21.48 (7.75)	14.04 (9.42)	F (1,178) = .711, MSE = 75.542, p = .4
- Interpersonal	17.61 (6.16)	14.21 (8.13)	9.26 (4.94)	6.88 (6.25)	F (1,178) = .245, MSE = 9.945, p = .621

- Disorganised	9.27 (3.57)	6.25 (3.52)	6.26 (3.72)	3.46 (3.45)	F (1,178) = .036, MSE = .454, p = .849
NART Total score	28.59 (5.38)	26.54 (5.06)	29.15 (5.16)	27.75 (5.28)	F (1,178) = .15, MSE = 4.164, p = .699

Standard deviations are shown in parentheses.

Main effects between the CP schizotypy groups revealed significant differences between high and low CP schizotypy groups for the SPQ Cognitive-Perceptual dimension ($F(1, 178) = 315.17$; MSE = 4240.7; $p < .001$; $\eta^2_p = .639$; M(high) = 16.63 (S.D. 4.6), M(low) =5.0 (S.D. 3.09)), as well as SPQ total score ($F(1, 178) = 142.272$; MSE = 15111.07; $p < .001$; $\eta^2_p = .444$; M(high) = 39.45 (S.D. 12.48), M(low) =16.16 (S.D. 9.55)), Interpersonal dimension ($F(1, 178) = 57.957$; MSE = 2355.714; $p < .001$; $\eta^2_p = .246$; M(high) = 16.52 (S.D. 6.99), M(low) = 7.56 (S.D. 5.98)) and Disorganised dimension ($F(1, 178) = 25.761$; MSE = 322.875; $p < .001$; $\eta^2_p = .126$; M(high) = 8.3 (S.D. 3.8), M(low) = 4.25 (S.D. 3.73)). Significant differences were also found between high and low CP schizotypy groups for the LSHS ($F(1, 178) = 34.654$; MSE = 51.422; $p < .001$; $\eta^2_p = .163$; M(high) = 4.7 (S.D. 2.26), M(low) = 2.25 (S.D. 1.63)). No significant differences were found between the CP schizotypy groups for sex ($\chi^2 = 2.24$, $df = 1$, N = 183, $p = 0.13$), age ($F(1, 178) = .918$; MSE = 47.182; $p = 0.339$; $\eta^2_p = .005$), handedness ($\chi^2 = 3.67$, $df = 2$, N = 183, $p = 0.16$) or verbal intelligence ($F(1, 178) = 1.083$, MSE = 29.98; $p = 0.300$; $\eta^2_p = .006$).

Significant main effects were also found between high and low AVH proneness groups. On the SPQ, the high AVH prone group reported significantly greater scores for total score ($F(1, 178) = 28.198$; MSE = 2994.96; $p < .001$; $\eta^2_p = .137$; M(high) =36.07 (S.D. 14.42), M(low) = 19.43 (S.D. 13.126)), as well as Cognitive-Perceptual ($F(1, 178) = 23.412$; MSE = 315.01; $p < .001$; $\eta^2_p = .116$; M(high) = 14.26 (S.D. 6.66), M(low) = 7.25 (S.D. 5.49)), Interpersonal ($F(1, 178) = 7.85$; MSE = 319.19; $p = .006$; $\eta^2_p = .042$; M(high) = 14.99 (S.D. 6.97), M(low) = 9.02 (S.D. 7.59)) and Disorganised dimensions ($F(1, 178) = 25.93$; MSE = 324.94; $p < .001$; $\eta^2_p = .127$; M(high) = 8.33 (S.D. 3.86), M(low) = 4.27 (S.D. 3.68)). Unsurprisingly the high AVH prone group also scored significantly higher for the LSHS ($F(1, 178) = 283.438$; MSE = 420.58; $p < .001$; $\eta^2_p = .614$; M(high) = 5.41 (S.D. 1.56), M(low) = 1.65 (S.D. 1.09)). No main effects were found between the AVH proneness groups for sex ($\chi^2 = 0.31$, $df = 1$, N = 183, $p = 0.58$), age ($F(1, 178) = .005$; MSE = .234; $p = 0.946$; $\eta^2_p = .000$), or handedness ($\chi^2 = 1.51$, $df = 2$, N = 183, $p = 0.47$). However the high AVH proneness group scored significantly worse on the NART measure of verbal intelligence compared to the low AVH proneness

group ($F(1, 178) = 4.13$, MSE = 114.38; $p = 0.044$; $\eta^2_p = .023$; M(high AVH prone) = 28.77 (S.D. 5.29), M(low AVH prone) = 27.4 (S.D. 5.22) errors).

9.4.2 Semantic Ambiguity Task Response Times

9.4.2.1 Group analysis (*F1*) for Reaction Time Data

All response time analyses were based on median concordant (correct) response times. Response times were analysed in a 2 (meaning) X 2 (relatedness) X 2 (CP schizotypy group) X 2 (AVH prone group) Repeated Measures ANOVA. In this design meaning and relatedness were the within subject variables, and CP schizotypy and AVH proneness were the between subject variables. All variables met the +/- 2 requirements for skewness and kurtosis except for the unrelated subordinate reaction time variable, which had a kurtosis value of 3.01 (S.E. 0.36). As a result box plot diagrams were used to identify possible outliers. One outlier was identified and removed, with the renewed kurtosis value subsequently meeting acceptable limits. Sphericity was not violated for this data therefore no corrections were required. When post hoc analyses were used the *p*-value was adjusted using Bonferroni corrections. Table 9.2 contains the descriptive statistics for this analysis.

Table 9.2. *Mean of the median reaction times to concordant responses in milliseconds.*

Group	**Meaning**	**Related**	**Unrelated**	**Unrelated - Related**
High CP Schizotypy,	Dominant	826 (22)	1077 (26)	251 (25)
High AVH prone	Subordinate	987 (19)	1062 (28)	75 (23)
High CP Schizotypy,	Dominant	779 (18)	1089 (32)	309 (23)
Low AVH prone	Subordinate	998 (22)	1116 (35)	118 (29)
Low CP Schizotypy,	Dominant	697 (15)	990 (24)	294 (27)
High AVH prone	Subordinate	874 (19)	958 (28)	85 (29)
Low CP Schizotypy,	Dominant	770 (19)	966 (21)	196 (20)
Low AVH prone	Subordinate	936 (18)	933 (20)	-3 (20)

Standard deviation shown in parentheses.

9.4.2.1.1 Task effects

Main effects of both meaning ($F(1, 178) = 131.607$, MSE = 1.06, $p < 0.001$, $\eta^2_p = 0.425$) and relatedness ($F(1, 178) = 86.755$, MSE = 4.2, $p < 0.001$, $\eta^2_p = 0.328$) were documented, with participants responding significantly slower to subordinate word pairs compared to dominant (M(dom) = 900ms, SE = 15, M(sub) = 983ms, SE = 16), and unrelated word pairs compared to related (M(rel) = 858ms, SE = 15, M(unrel) = 1024ms, SE = 20). A significant interaction effect was also found between meaning and relatedness ($F(1, 178) = 187.529$, MSE = 1.44, $p < 0.001$, $\eta^2_p = 0.513$, M (dom, rel) = 768ms, SE = 16, M(dom, unrel) = 1031ms, SE = 20, M(sub, rel) = 949ms, SE = 16, M(sub, unrel) = 1017ms, SE = 22).This finding is reflective of meaning impacting on reaction time responses when words are related, however when words are unrelated they are not expected to differ, as they are both the same in their 'unrelatedness' regardless of meaning.

9.4.2.1.2 Group effects

A significant main effect was found for CP schizotypy group ($F(1, 178) = 10.78$, MSE = 1.57, $p = 0.001$, $\eta^2_p = 0.057$), with the high CP schizotypy group responding slower overall compared to the low group (M(high)=992ms, SE = 22, M(low)=891ms, SE = 22). No significant interactions were found between CP schizotypy and meaning ($p = 0.055$), relatedness ($p = 0.201$), or the 3-way interaction of schizotypy with relatedness and meaning ($p = 0.478$).

No main effect was found for AVH proneness ($p = 0.633$). No significant interaction effects were documented between AVH proneness and the task factors meaning ($p = 0.137$) or relatedness ($p = 0.555$), or their interaction with each other ($p = 0.92$).

No interaction was documented between CP schizotypy and AVH proneness ($p = 0.82$). However, a significant interaction was found between CP schizotypy, AVH proneness and relatedness ($F(1, 178) = 4.06$, MSE = .197, $p = 0.045$, $\eta^2_p = 0.022$). Descriptive statistics for this analysis are in Table 9.3. To unpack this interaction the analysis was rerun with the file split by CP schizotypy. The interaction between AVH proneness and relatedness reached trend level significance for low CP schizotypy ($F(1, 93) = 3.69$, MSE = .166; $p = 0.058$, $\eta^2_p = .038$), but was not significant for high CP schizotypy ($p = 0.337$). This analysis was repeated with the file split by AVH proneness. Results showed that the interaction between CP

schizotypy groups and relatedness was significant for low AVH proneness ($F(1, 94) = 6.737$, MSE = .273; $p = 0.011$, $\eta^2_p = .067$), but not for high AVH proneness ($p = .641$). Therefore, within low AVH prone individuals, unrelated words led to larger increases in reaction time compared to related words when individuals were also high on CP schizotypy compared to low CP schizotypy.

Table 9.3. *Mean of the median reaction times (milliseconds) to related and unrelated word pairs.*

	High AVH prone		Low AVH prone	
	Related	Unrelated	Related	Unrelated
High CP schizotypy	906 (24)	1070 (33)	889 (35)	1103 (48)
Low CP schizotypy	785 (35)	974 (48)	853 (22)	950 (31)

Standard errors of the mean shown in parentheses.

9.4.2.1.3 Correlations between response time differences for dominant and subordinate related word pairs

A measure was calculated by subtracting the 'related response time' from the 'unrelated response time' for each participant, for both dominant and subordinate targets. In order to determine whether mechanisms are similar or different in our groups of interest, correlations were calculated. It was found that the correlation in response time differences between dominant and subordinate word pairs was positive, significant, and comparable in size across all four groups (High CP schizotypy, High AVH prone ($r(59) = 0.77$, $p < 0.001$); High CP schizotypy, Low AVH prone ($r(28) = 0.654$, $p < 0.001$); Low CP schizotypy, High AVH prone ($r(27) = 0.84$, $p < 0.001$); Low CP schizotypy, Low AVH prone ($r(68) = 0.656$, $p < 0.001$). This indicates that the manner in which the reaction time measure is affected by semantic relatedness is similar in both CP schizotypy and AVH prone groups, suggesting that similar mechanisms are involved in processing the meaning of the word pairs in all groups. It is also possible that the consistency in these relationships across groups is the result of similar levels of arousal affecting the cognitive efficiency of participants.

9.4.2.2 Item Analysis (*F2*) for Reaction Time Data

An analysis with items as cases was used to confirm the results obtained by the previous by-subject (*F1*) analysis. Congruity across *F1* and *F2* analyses indicates true significant differences between groups. If results are not congruent, this may indicate that a few items (or individuals) are driving the differences in reaction time performances. Median reaction times were calculated across participants for every pair of stimuli for each concordant (correct) response. Responses were analysed in a 2 (meaning) X 2 (relatedness) X 2 (CP schizotypy group) X 2 (AVH proneness group) repeated measures ANOVA. In this analysis CP schizotypy and AVH proneness group became the within-item variables, and meaning and relatedness the between-item variables.

9.4.2.2.1 Task effects

There was a significant effect of relatedness ($F(1, 281) = 37.12$, MSE = 5.62, $p < 0.001$, $\eta^2_p = 0.117$), which confirms *F1* analysis findings, (M(rel) = 887ms, SE = 16, M(unrel) = 1028ms, SE = 16). A significant effect was also found for meaning ($F(1, 281) = 14.52$, MSE = 2.19, $p < 0.001$, $\eta^2_p = 0.049$), with participants responding slower to subordinate word pairs, again supporting the *F1* analysis (M(dom) = 914ms, SE = 16, M(sub) = 1001ms, SE = 16). An interaction effect was found between meaning and relatedness ($F(1, 281) = 26.02$, MSE = 3.94, $p < 0.001$, $\eta^2_p = 0.085$, M(dom, rel) = 785ms, SE = 23, M(dom, unrel) = 1043ms, SE = 23, M(sub, rel) = 990ms, SE = 23, M(sub, unrel) = 1013ms, SE = 23). Similar to that found in the *F1* analysis, this interaction reflects the task effect where for unrelated words, meaning is not expected to influence responding, as both dominant and subordinate pairs are considered equal in their 'unrelatedness'.

9.4.2.2.2 Group effects

A significant main effect was found for CP schizotypy group ($F(1, 281) = 166.18$, MSE = 3.01, $p < 0.001$, $\eta^2_p = 0.372$) where the high group responded significantly slower compared to the low group (M(high) = 1009ms, SE = 13, M(low) = 906ms, SE = 11). CP schizotypy also interacted significantly with meaning ($F(1, 281) = 4.89$, MSE = .089, $p = 0.028$, $\eta^2_p = 0.017$). Follow-up analyses

using pairwise comparisons revealed that dominant word pairs were responded to slower in the high CP schizotypy group compared to the low group (F(1, 141) = 79.51, MSE = 1.04, $p < 0.001$, $\eta^2_p = 0.361$, M(high) = 956ms, SE = 17, M(low) = 871ms, SE = 15). Similarly subordinate word pairs were responded to slower by the high CP group compared to the low group (F(1, 140) = 88.63, MSE = 2.06, $p < 0.001$, $\eta^2_p = 0.388$, M(high) = 1062ms, SE = 20, M(low) = 941ms, SE = 16). No significant interactions were found between schizotypy groups and relatedness ($p = 0.204$), or the relatedness and meaning interaction ($p = 0.899$).

The main effect of AVH proneness was significant (F(1, 281) = 12.66, MSE = 0.25, $p < 0.001$, $\eta^2_p = 0.043$), with the high AVH prone group responding significantly faster to items compared to the low AVH prone group (M(high) =943ms, SE = 11, M(low) = 972ms, SE = 13). No significant interactions were found between AVH proneness and meaning ($p = 0.199$) or relatedness ($p = 0.261$). The 3-way interaction between AVH proneness, meaning and relatedness was also not significant ($p = 0.351$).

No interaction was found between CP schizotypy and AVH proneness ($p = 0.125$). The CP schizotypy and AVH proneness interaction effect did not interact with meaning ($p = 0.224$), however it did interact with relatedness (F(1, 281) = 5.13, MSE = 0.078, $p = 0.024$, $\eta^2_p = 0.018$). To unpack this interaction the analysis was rerun with the file split by relatedness, which revealed a significant interaction effect between CP schizotypy and AVH proneness in the related condition (F(1, 140) = 7.12, MSE = 0.109, $p = 0.009$, $\eta^2_p = 0.048$), but not in the unrelated condition ($p = 0.605$). A Paired Samples t test indicated that for those in the low CP schizotypy group, responses were significantly faster when combined with high AVH proneness as opposed to low AVH proneness (t(142) = -4.54, $p < .0001$, M(low schizotypy, high LSHS) = 811ms, SE = 17, M(low schizotypy, low LSHS) = 873ms, SE = 17, see Figure 9.1). Response times in the high CP schizotypy group did not differ as a result of AVH proneness ($p = .667$).

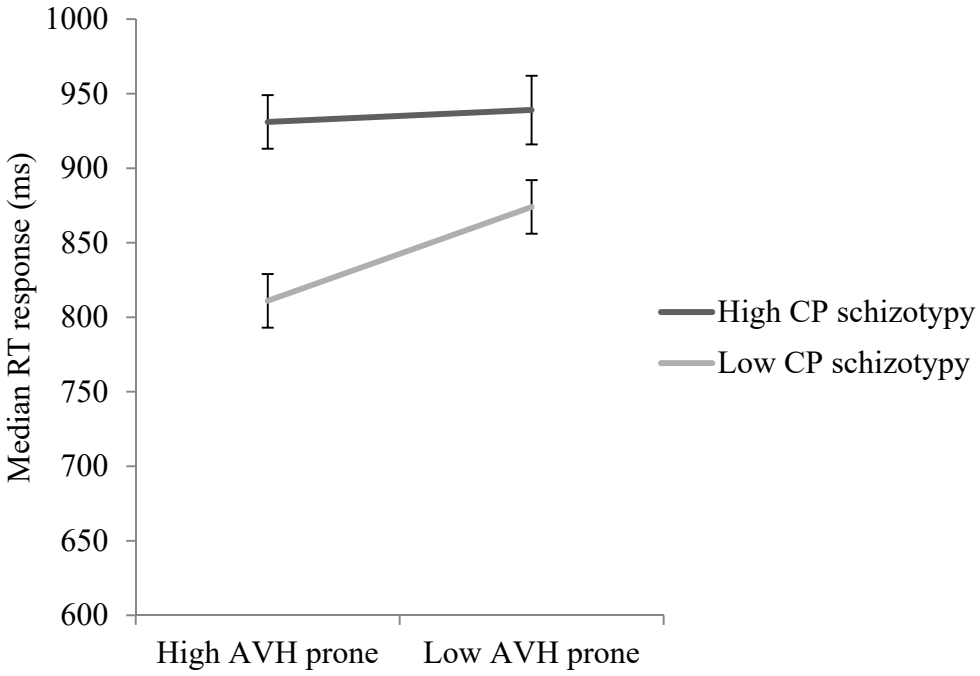

Figure 9.1. Median reaction time (RT) responses (in milliseconds) to related word pairs. Lines indicate Cognitive-Perceptual (CP) schizotypy group, with responses broken down according to Auditory Verbal Hallucination (AVH) proneness.

9.4.2.3 Consistency of results across *F1* and *F2* analyses

Congruity across *F1* and *F2* analyses is indicative of true differences in CP schizotypy and AVH proneness group effects. A comparison of these analyses revealed that task effects of meaning, relatedness, and their interaction were consistent across *F1* and *F2* analyses. Group effects of CP schizotypy were also consistent, indicating that the slower response times of those in the high CP schizotypy group are true differences. However the interaction of CP schizotypy with meaning in the *F2* analysis was not consistent in the *F1* analysis. The main effect of the high AVH proneness group responding significantly faster in the *F2* analysis also was not congruent with *F1* results. The significant interaction effect observed between CP schizotypy, AVH proneness and relatedness was consistent across *F1* and *F2* analyses, indicating a true effect driven by the faster responses of the low CP Schizotypy/High AVH prone group to related words.

9.4.3 Signal Detection Analyses

Table 9.4. *Mean values for sensitivity and relatedness judgments in Cognitive-Perceptual (CP) schizotypy and Auditory Verbal Hallucination (AVH) prone groups.*

Group	Meaning	% Hits	% False alarms	*d´*	*c´*
High CP Schiz,	Dominant	82.7 (14)	13 (10)	2.25 (.71)	.03 (.42)
High AVH prone	Subordinate	62.2 (14)		1.52 (.54)	
High CP Schiz,	Dominant	84.1 (14)	16.8 (14)	2.13 (.94)	-.17(1.04)
Low AVH prone	Subordinate	62.6 (14)		1.39 (.67)	
Low CP Schiz,	Dominant	82.9 (16)	12.3 (8)	2.35 (.78)	-.22 (1.4)
High AVH prone	Subordinate	63.9 (16)		1.59 (.59)	
Low CP Schiz,	Dominant	78.2 (19)	14.8 (12)	2.27 (.96)	.23 (1.57)
Low AVH prone	Subordinate	57.6 (15)		1.48 (.67)	

Hits are out of 36 trials each, and false alarms are out of 72 trials (the sum of both dominant and subordinate conditions). Standard deviation in parentheses. None of the reported differences between groups reached significance at the $p < .05$ level.

9.4.3.1 Sensitivity analysis (*d´*)

The sensitivity (*d´*) measure was analysed in a Repeated Measures ANOVA, with CP schizotypy and AVH proneness the between subject variables, and meaning (dominant or subordinate) the within subject variable. Descriptive statistics are found in Table 9.4.

Sensitivity analyses (*d´*) revealed a main effect of meaning for dominant and subordinate targets ($F(1, 178) = 466.333$, MSE = 43.432, $p < .001$, $\eta^2_p = 0.724$), with participants significantly more able to discriminate between unrelated and related for dominant targets (M = 2.25, SE = .07) compared to subordinate (M = 1.5, SE = .05). There were no significant main effects for CP schizotypy ($p = .402$) or AVH proneness ($p = .331$). No interaction was observed between CP schizotypy and AVH proneness groups ($p = .907$) in the sensitivity analysis.

9.4.3.2 Relative criterion analysis (*c´*)

To compare dominant and subordinate *c* within each of the group's *c* underwent an arithmetic transformation. Results indicated that dominant and

subordinate *c* in each group was the same, indicating that all groups use the same criterion regardless of whether they are responding to dominant or subordinate stimuli. Mean values were: High CP schizotypy = 1.2 (SD .52), Low CP schizotypy = 1.19 (SD .47), High AVH prone = 1.25 (SD .46), Low AVH prone = 1.16 (SD .43).

To compare *c* between CP schizotypy and AVH prone groups *c´* was used (Macmillan and Creelman, 2005). Results indicated that there were no significant main effects for CP schizotypy (p = .686) or AVH proneness (p = .544). Additionally, no interaction was found between CP schizotypy and AVH proneness (p = .093). Descriptive statistics are in Table 9.4.

9.5 Discussion

The results from this study suggest that CP schizotypy and AVH proneness differ in how they influence the processing of semantic relations, despite not being in support of initial hypotheses. Across both *F1* and *F2* analyses, the high CP schizotypy reaction time responses were characterized as slower than the low CP schizotypy group. However the high AVH prone group was found to respond to word pairs faster than the low AVH prone group. In addition, for related word pairs specifically, the low CP schizotypy group responded significantly faster when coupled with high AVH proneness, as opposed to low AVH proneness. No significant differences were found between groups in the sensitivity and criterion determinants of responding.

Unexpectedly, the effects of schizotypy AVH proneness on reaction times differed. Results indicated that those who were high on CP schizotypy responded significantly slower than those low on CP schizotypy. Contrastingly, some evidence was found for those predisposed to hallucinations to respond to word pairs faster than their respective low scoring counterparts. These findings are indicative of disparities in how state and trait psychosis risk variables influence processing of semantic relations. Given that processing speed has been shown to be intact in schizotypy samples, the slower overall response speed associated with CP schizotypy suggests increased difficulty in the processing of semantic information. It may be that in trait schizotypy, a diffuse spread of semantic activation results in more semantic nodes being activated. This increased number of activated associates is hypothesized to result in more time to reach a decision of relatedness, due to

greater difficulty identifying the specific association involved. Yet although this diffuse activation is thought to result in a slowed response time, it does not appear to compromise accuracy, which is why no differences were found in the signal detection outcomes. Findings from Gianotti et al. (2001) showed that those high on CP schizotypy were more likely to find original associations between unrelated stimuli, which is also indicative of diffuse right hemisphere activation for semantic concepts. The results of our study are consistent with this suggestion that the semantic network in schizotypy may be characterized by a more diffuse spread of activation, which results in a slower response time.

In contrast, the relatedness effects demonstrated by the high AVH prone group in one (but not both) reaction time analyses suggests disinhibitive processes may be contributing to significantly faster task completion for this group. In non-clinical AVH samples, the tendency to jump to conclusions and interpret an internally generated experience as a true sensory experience has been suggested as a central mechanism in the generation and maintenance of hallucinations (for meta-analysis, see Brookwell, Bentall and Varese, 2013). The current findings contribute tentative support to this mechanism, however given that this finding was not consistent across both reaction time analyses caution should be made when interpreting this result. Further investigation of reaction time responses to ambiguous semantic relations in AVH proneness is warranted to determine whether or not these findings are a true effect.

Although not predicted, compared to those with low CP schizotypy and low AVH proneness, those with high CP schizotypy and high AVH proneness responded to related word pairs significantly slower, whilst those with low CP schizotypy and high AVH proneness responded to related word pairs significantly faster. This interaction suggests that there may be two mechanisms work. CP schizotypy appears to result in a more diffuse spread of semantic activation, which slows response times to related word pairs. Contrastingly, AVH proneness seems to reflect disinhibitive processes, such that relationships between semantic associates are responded to significantly faster as long as schizotypy is low/normal. These findings indicate that high CP schizotypy potentially has a far more influential effect on the atypical processing of semantic relations, since the disinhibitive effects of AVH proneness were drowned out by high schizotypy, and only apparent when combined with low CP schizotypy. These findings suggest that hallucination proneness exists as a

symptom separate from positive trait schizotypy. Such a finding is in line with previous research (Daalman et al., 2011; Paulik et al., 2007; for review, see de Leede-Smith & Barkus, 2013), and points towards distinct trajectories of illness risk, where high AVH proneness is not necessarily associated with poor outcome. In relation to psychosis risk generally, it is possible that hallucinatory experiences themselves are not sufficient to confer functional impairment and risk of psychosis development. However when these experiences are combined with high positive schizotypy, the current findings suggest that any adaptive advantage conferred by AVH proneness is lost through the additional presence of all that is encompassed by the positive schizotypal trait.

A priming measure was also calculated for each participant for both dominant and subordinate words. They were then correlated for each participant group, and found to be similar in magnitude across all four groups. Although the speed of processing differs between groups, the current study suggests that the organisation of the semantic system may be the same, at least for normatively associated words. Further support for this hypothesis is offered by the lack of differences in the accuracy data. These finding suggest that scoring highly on CP schizotypy or AVH proneness has no effect on the ability to detect relationships between stimuli themselves.

No significant differences were found between the CP schizotypy and AVH prone groups for signal detection outcomes. Research has shown that in high schizotypy, the breakdown in control processes (such as inhibition) that organise semantic processing only come about when extraneous task-related demand is placed on attentional and working memory resources (Nizhikiewicz et al., 2002; 1999). Since this task was a simple judgment of relatedness, no additional demands were placed on resources, for example: integrating several contextual cues, or; processing convoluted sentences. Perhaps it is necessary to have these features imbedded in task design to lead to a less conservative decision-making style under ambiguous conditions (e.g. Grimshaw et al., 2010). The signal detection data support the conclusion that the ability to discriminate related and unrelated word pairs is not affected by CP schizotypy or AVH proneness.

There were some limitations that emerged as this study progressed. As previously mentioned, this sample consisted of reasonably high functioning university students. As a by-product of tertiary education, university samples

generally have high cognitive, social, and often financial resources compared to community samples. Consequently the failure to find significant differences in signal detection criteria may be the result of the current sample not being representative of spread of ability in the general population, although the relatively high error rates on the NART suggest that we did have a wide spread of verbal ability in the sample. Furthermore, the current study used the CP schizotypy factor to split high and low schizotypal groups. Although this has been used in previous studies testing for semantic processing abnormalities (e.g. Kostova, de Loye & Blanchett, 2011; Johnston, Rossell & Gleeson, 2008; Niznikiewicz et al., 2002), it has been suggested that the greatest differences in semantic function are observed when psychosis prone groups are characterized in terms of positive scores on language and thought deviations (Spitzer, 1997; Maher et al., 1996). Certainly schizophrenia patients with thought disorder display the greatest aberrations in semantic system functioning (see Pomarol-Clotet et al., 2008 for a meta-analysis). Perhaps splitting psychosis prone groups on a language/thought deviation measure instead would be a more viable way of investigating semantic relations, especially if participants are relatively high functioning.

In conclusion, this study considered the nature of semantic processing disturbances in both high trait CP schizotypy and high state AVH prone groups. Our findings indicate that the speed of processing ambiguous semantic relations varies according to level of trait and state psychosis risk. From these initial comparisons, it appears that the slower speed of semantic processing found in high CP schizotypy may be related to a more diffuse spread of semantic activation. Contrastingly the semantic processing capabilities associated with AVH proneness seem to be related to disinhibitive processes, resulting in an accurate and efficient speed of decision making for semantic information, but only in the context of low CP schizotypy. Previously, positive schizotypy and AVH proneness were believed to be somewhat synonymous indications of psychosis proneness, our study suggests further investigation is required to determine the separation between these two phenotypes on other psychosis-risk variables.

10 STUDY FIVE: SEMANTIC PROCESSING IN AN ADULT DYSLEXIA SAMPLE: EFFECTS OF SCHIZOTYPY

10.1 Abstract

Dyslexia refers to difficulties in reading, often accompanied by phonological processing deficits, and abnormalities in semantic processing can also be present. Abnormalities in semantic processing are typical along the psychosis continuum, and links have been made between dyslexia and the psychosis continuum, specifically with schizotypal personality trait. The current study aimed to determine whether schizotypy could account for disturbances in semantic processing in those with dyslexia. Participants (N=102), 51 of whom had a diagnosis of dyslexia, completed the Schizotypal Personality Questionnaire, a measure of verbal intelligence, and a computerised semantic ambiguity task. In the semantic task, there was evidence for those with dyslexia being significantly slower to respond than Controls. More importantly, the Dyslexia group was also less able to discriminate between related and unrelated words. Follow up analyses revealed that schizotypy was able to account for this difference in discrimination between the Dyslexia and Control groups. Further research is required to understand the mechanisms driving the association between schizotypy and dyslexia for semantic processing.

10.2 Introduction

Dyslexia is a neurodevelopmental learning disorder characterised by problems with reading, deficits in the ability to integrate letters and sounds, and phonological processing more generally (Shaywitz, 1996; Wagner and Torgesen, 1987). These problems occur despite average general intelligence, adequate educational opportunities, and no overt sensory deficits. Neural origins of dyslexia have been attributed to under-activation in posterior brain regions, and over-activation in anterior brain regions (e.g. Shaywitz et al., 1998; Georgiewa et al., 2002). Impaired semantic processing may also contribute to difficulties learning to read in dyslexia (e.g. Kronbichler et al., 2006).

Semantic processing describes the processing of general information and knowledge. In healthy individuals, semantics are believed to be stored in the brain in a conceptual network by proxy of degree of relatedness and associative links (Minzenberg, Ober, & Vinogradov, 2002). The automatic semantic activation model (Collins and Loftus, 1975) has been suggested as an explanatory model for semantic memory. In this model semantic knowledge is stored as a conceptual network, with nodes representing a piece of knowledge or concept, and the links between nodes hypothesised to represent relations between concepts. So for example, activation of the 'drink' node would also result in a spread of activation to related nodes, such as 'water' and 'juice'.

Comparisons between those with and without dyslexia have shown abnormalities in semantic processing. For instance, Torppa et al (2010) reported children with dyslexia, when aged between 2 and 5, performed more poorly than typical readers on measures of receptive and expressive language, including tasks of rapid naming and letter naming. School aged children with dyslexia performed worse on a semantic relatedness task compared to age matched controls (Chik et al., 2012). In a study by Schulz et al (2008), dyslexic and control primary school aged children performed a task which required them to indicate whether sentences were meaningful (semantically congruous) or not (semantically incongruous). Children with dyslexia were significantly slower and less accurate than controls. Similarly, adults with dyslexia performing a semantic judgement task were found to respond significantly slower, and less accurately than controls (Rüsseler et al., 2007). It has been suggested that these performances may have a neural basis, with studies

reporting delayed activation (Schulz et al., 2008; Jednoróg et al., 2010), hypoactivation or hyperactivation (Kronbichler et al., 2006; Booth et al., 2007) in brain areas known to process semantic information when those with dyslexia are compared to controls. Findings in the literature are not conclusive, with some research indicating that individuals with dyslexia perform better in formulating definitions of words (Tsesmeli and Seymour, 2006), as well as being as accurate as reading-age matched controls in synonym identification and use of target words in sentences (Chik et al., 2012).

It is also possible that the modality of stimulus presentation may have an effect on the processing of semantic associations. For those with dyslexia, stimulus presented visually necessitate evaluation in the context of impaired lexical processing, which may confound results compared to those without dyslexia. Verbally presented stimuli do not have this confounding factor. However, previous research has shown that semantic processing deficits exist in individuals with dyslexia compared to those without, independent of stimulus modality (e.g. Booth et al., 2007; Landi et al., 2010).

Over the last decade, neurodevelopmental disorders have been genetically linked to psychosis (Owen et al., 2011). The neurodevelopmental hypothesis of psychosis suggests that the development of psychotic illness comes about as a result of abnormal development of the brain interacting with adverse environmental factors (Weinberger, 1987). Longitudinal, population-based findings support this, demonstrating links between abnormal language, cognitive, motor, and social development in childhood, and the subsequent increased risk of psychosis in adulthood (e.g. Cannon et al., 2000; Cannon et al., 2002). Neurodevelopmental disorders in childhood, such as dyslexia, are shown to predict psychotic-like experiences (PLEs) in adolescence (Khandaker et al., 2014), possibly indicating shared genetic susceptibility between language problems and psychosis (Cederlöf et al., 2014; Becker et al., 2012).

Research has also broadened to focus on the relationship between dyslexia and psychosis proneness, or schizotypy (e.g. Richardson, 1994). Schizotypy exists along the psychosis continuum. The psychosis continuum refers to a spectrum of non-clinical and clinical psychosis presentations, including attenuated psychotic experiences and psychotic disorders (DeRosse and Karlsgodt, 2015). Attenuated psychotic experiences are a feature of the schizotypal personality trait, with this trait

stretching across healthy, subclinical and clinical boundaries (Claridge and Beech, 1995; Claridge, 1997), whilst clinical psychosis/diagnosed schizophrenia makes up the extreme end of the psychosis continuum. Factor analytic studies have identified three schizotypy dimensions: positive, negative, and disorganised schizotypy (Fossati et al., 2003; Reynolds et al., 2000). These dimensions are similar in composition to the three-factor model of schizophrenia symptomatology (Barrantes-Vidal et al., 2013b). Individuals with dyslexia have higher rates of positive schizotypy, as well as an increased prevalence of mixed handedness (Richardson and Stein, 1993; Richardson, 1994). Findings of mixed handedness have also been reported in high schizotypes (Barrantes-Vidal et al., 2013c; Tsuang et al., 2013), which contributes to the idea that dyslexia and the psychosis continuum may overlap (Condray, 2005).

Across the psychosis continuum abnormalities in controlled semantic processing are well documented (e.g. Rossell and Stefanovic, 2007; Tonelli, 2014). Controlled semantic processing occurs after a 750 millisecond (or more) delay in the presentation of a second word (stimulus onset asynchrony; SOA). Controlled semantic processing is named such, because it refers to the specific segment of time in which controlled processes are believed to be operating; such as expectancy effects and semantic matching (Neely and Keefe, 1989). In schizophrenia, controlled semantic processing is shown to be impaired by way of synonym identification, word association, and antonym identification, among other tasks (e.g. Rossell and David, 2006, Cacciari et al., 2015).

Irregularities in controlled semantic processing are also found in studies examining schizotypy (e.g. Kiang and Kutas, 2005; Johnston, Rossell and Gleeson, 2008; Minor and Cohen, 2012). In these studies, the positive schizotypy dimension is often linked with atypical and diffuse semantic processing (e.g. Mohr et al., 2001; Gianotti et al., 2001). Impaired inhibitory mechanisms have been hypothesised to contribute to atypical semantic functions in schizotypy (Grimshaw et al., 2010). With a breakdown in inhibitory processes, comes the increased and diffuse activation of a wider variety of semantic associates. In schizotypy, this results in a propensity to find meaning and relationships between words that may otherwise be regarded as unrelated (Grimshaw et al., 2010; Morgan, Bedford and Rossell, 2006).

Both dyslexia and schizotypy have evidenced atypicalities in semantic processing. They also share similar findings of reduced size and asymmetry in

temporal lobe and cerebellar regions, with these neuroanatomical features predicting reading and cognitive deficits in both dyslexia and schizophrenia samples (Leonard et al., 2008). Additionally, Jones et al (1994) found that children who went on to develop psychosis often reported problems with the development of language in childhood. Given the overlaps in semantic difficulties in those with dyslexia and high schizotypes, we will investigate whether schizotypy accounts for differences in semantic processing between those with and without dyslexia.

We sought to investigate semantic processing in individuals with and without dyslexia. The current study will be making use of homographs (words with two meanings). In the English language, processing a homograph correctly requires the individual to inhibit other alternative meanings of that word. Homographs usually have one word meaning that is used more frequently than the other, and thus is referred to as the dominant word meaning. The lesser used meaning of a homograph is referred to as the subordinate word meaning. The dominant word meaning is accessed more readily due to increased frequency of use, therefore it is expected that all participants will respond quicker to dominant word pairs compared to subordinate. The Dyslexia group was expected to demonstrate slower response times than Controls under ambiguous conditions, i.e. word pairs involving the subordinate meaning. It was also expected that individuals with dyslexia would exhibit atypical signal detection determinants of responding compared to controls. Given that positive schizotypy specifically has been shown to be increased in dyslexia (Richardson and Stein, 1993; Richardson, 1994), and has been associated with abnormalities in semantic processing (e.g. Kiang and Kutas, 2005; Minor and Cohen, 2012), it was expected that positive schizotypy would account for any differences in semantic processing seen between those with and without dyslexia.

10.3 Method

10.3.1 Participants

The sample was comprised of 102 participants (mean age = 24.47 (SD 9.7), age range = 17-66, 69.6% female) recruited from the School of Psychology and wider university population at the University of Wollongong, Australia. Within the sample 51 participants had a diagnosis of dyslexia from a qualified psychologist. The remaining 51 participants without a diagnosis of dyslexia or other learning disorder

were age and sex matched to the dyslexia sample from a larger participant pool who had taken part in another research study (Chapter 8; de Leede-Smith et al., submitted). Participants were screened and excluded if they were not able to speak the English language fluently, or were diagnosed with a psychotic illness or a learning disorder other than dyslexia.

10.3.2 Measures

An initial demographic questionnaire was given to all participants to determine age, sex, current living arrangements, help seeking behaviour within the past six months, primary language spoken, and presence of a learning disorder and/or mental illness. Handedness was determined using a question from the Neurological Evaluation Scale (Buchanan & Heinrichs, 1989). Participants were asked which hand they prefer when completing 9 routine tasks (i.e. writing, unscrewing the lid of a jar, brushing teeth). Handedness was determined by adding up the amount of times a participant used each hand. Dominant handedness was determined if a participant favoured one hand for seven or more activities, otherwise they were classified as mixed handedness. All participants then completed the Schizotypal Personality Questionnaire (SPQ; Raine, 1991), National Adult Reading Test (NART; Nelson, 1982), and the Semantic Ambiguity Task (adapted with permission from Grimshaw et al., 2010).

The SPQ is a 74 item Yes/No questionnaire measuring total schizotypy, and the three schizotypal dimensions: Cognitive-Perceptual (CP) Schizotypy, also known as positive schizotypy (comprising Odd Beliefs, Ideas of Reference, Unusual Perceptual Experiences and Suspiciousness subscales); Interpersonal Schizotypy, also known as negative schizotypy (made up of Constricted Affect, No Close Friends, Excessive Social Anxiety and Suspiciousness subscales), and; Disorganised Schizotypy (consisting of Odd Speech and Odd/Eccentric Behaviour subscales).

The NART was used as a measure of verbal intelligence. Participants were required to read aloud a list of 50 atypical words. Pronunciation errors were counted and recorded.

10.3.2.1 Semantic Ambiguity Task

Semantic processing was evaluated in the current study using the Semantic Ambiguity Task described by Grimshaw et al (2010). In this task, participants are

presented with an initial word, followed by a target word which is either: related to the dominant meaning of the initial word, related to the subordinate meaning of the initial word, or unrelated to the initial word. Response time was used to measure semantic processing, as well as signal detection outcomes, given that a subjective decision was required by participants when responding to each word pair. The sensitivity to relatedness measure is the ability to distinguish related pairs from unrelated pairs, and is taken to reflect differences in semantic organisation. The criterion measure evaluates biases to respond to word pairs as either related (lax decision making bias) or unrelated (conservative decision making bias). A stimulus onset asynchrony (SOA) of 750ms was used, given that previous research has found this to be the time where inhibition is most likely occurring (Atchley, Burgess, & Keeney, 1999; Burgess & Simpson, 1988).

The Semantic Ambiguity Task consisted of 144 trials, with each trial made up of a word pair. The first word in the pairing would flash up on the screen, followed immediately by the second target word. As soon as the second word disappeared from the screen, participants were required to respond, pressing key 1 if they thought the two words were related by meaning, or key 2 if they thought the two words were unrelated to each other. Participants were asked to respond as accurately and quickly as possible.

The first words in the pairings were 72 ambiguous homographs originally used by Burgess and Simpson (1988). Each homograph was then matched up with either a dominant or subordinate related word (i.e. the word 'ball' could be matched with the dominant word pair 'round' or the subordinate word pair 'dancing'). In total, 72 related word pairs (36 dominant word pairings and 36 subordinate word pairings) and 72 unrelated word pairs were used for the task. The 72 unrelated word pairs were made up by pseudo-randomly pairing a homograph with one dominant and one subordinate word of a different unassociated homograph. Each participant saw each homograph twice - once paired with a related word (either dominant or subordinate), and once paired with an unrelated word (dominant or subordinate). Participants also saw each target word twice, preceded by a related homograph and an unrelated homograph. Word pairs were counterbalanced such that if a homograph was first paired with a related word, the second time that homograph was seen it was paired with an unrelated word (and vice versa). Counter balancing also occurred via the use of two word lists across different participants such that the dominant and

subordinate meaning of each homograph was tested equally as often. Each participant saw *either* word list one or two. Additional details regarding word pairings can be found in Grimshaw et al (2010).

Each trial began with a fixation mark presented centrally on screen for a duration of 1000ms. A prime word then followed in the centre of the screen for 50ms followed by a blank screen for 700ms to produce the SOA of 750ms, which was followed by the target word that remained on screen for 180ms. Participants then had 3000ms to respond (key 1 for 'related', key 2 for 'unrelated'). After this time there was a 3000ms inter-stimulus interval between their response and the beginning of the next trial.

10.3.3 Procedure

Ethical approval was granted from the University of Wollongong Human Research Committee. Participants were given a study information sheet and written informed consent was obtained before participation commenced. Participants completed questionnaires, then began the Semantic Ambiguity Task, which was run on a laptop in a quiet room within the University of Wollongong. Participants with dyslexia who had difficulties reading were offered for questionnaires and words on the task to be read out verbally. Less than 10% of participants in the Dyslexia group requested this option. Participants with dyslexia were reimbursed with course credit or $30 cash for their time. Raters were not blind to dyslexia status due to participants with dyslexia being financially reimbursed. This strategy was used as a recruitment tool specifically for those participants with dyslexia.

10.3.4 Statistical Analysis

Data analysis was performed in SPSS 21 (IBM, 2012). Median response times for concordant (correct) responses were used as the basis of response time analyses. In an effort to control for participant and item random effects both subject (*F1*) and item (*F2*) analyses were run using a Repeated Measures Analysis of Variance (ANOVA). Following this, to determine whether positive schizotypy had an effect in the responses of those with dyslexia to the semantic task, the subject analysis (*F1*) was repeated with CP schizotypy as a covariate by way of a Repeated Measures Analysis of Covariance (ANCOVA).

For signal detection parameters performance accuracy was divided into sensitivity (*d′*) and criterion (*c*). Sensitivity is understood as the participant's ability to accurately discriminate between targets that are related, and those that are unrelated (i.e. to respond correctly). The Signal detection *d′* variable was analysed in a Repeated Measures ANOVA. This analysis was repeated with CP schizotypy as a covariate in a Repeated Measures ANCOVA, as was done in the *F1* reaction time analysis.

The criterion signal detection parameter refers to the decision making bias of the participant, where under conditions of ambiguity participants have a propensity to respond with either a conservative or lax decision making bias. A conservative response pattern would require the participant to classify more uncertain targets as 'unrelated'. Whereas a lax response pattern would see the participant more likely to classify uncertain targets as 'related'. Positive *c* values are indicative of a conservative response pattern and negative values indicate a lax response pattern. The *d′* and *c* variables were calculated via the Macmillan and Creelman (2005) criteria:

$d' = z(\text{hits}) - z(\text{false alarms})$
$c = -0.5\ (z(\text{hits}) + z(\text{false alarms}))$

False alarms were defined as a response of 'related' to an unrelated item, whilst hits are the correct response of 'related' to a related item. Given that false alarms are the same across dominant and subordinate targets (i.e. both conditions are equal in their unrelatedness), the number of false alarms was added together, giving a false alarm rate out of 72.

In order to compare the *c* for dominant and subordinate conditions, the *c* values need to be on the same scale, with the mean of the unrelated distribution as the zero point. To do this, the *d′* for each condition was divided by 2, and then *c* was added to it. This allowed dominant and subordinate *c* to be compared via a *t*-test. To compare Dyslexia and Control groups, *c* was transformed into relative *c* (*c′*) by dividing it by the *d′* value, as suggested by Macmillan and Creelman (2005). Doing so takes into account the difference in the *d′* between the two groups so that the groups can be compared. The *c′* for Dyslexia and Control groups was compared using an Independent Samples *t*-test.

For some participants, the number of hits or false alarms was equal to 0 or 1. In these cases an adjustment was made to avoid infinite values (formula 1/(2N) for the value 0 and 1-1/(2N) for the value 1; N symbolises the number of trials the proportion is based on (Macmillan & Creelman, 2005)). As a result, values of 0 and 1 were transformed into 0.014 and 0.986 respectively.

10.4 Results

10.4.1 Participants

Demographic variables for Dyslexia/Control groups are presented in Table 10.1. No significant differences were found for sex, age, living arrangements, and the use of health services over the previous six months.

For comparisons between those with and without Dyslexia, significant differences were found on Handedness (χ^2 = 7.38, *df* = 2, *p* = .025), SPQ Total (*t*(93.403) = 5.287, *p* < .001), Cognitive-Perceptual SPQ (*t*(94.44) = 3.284, *p* = .001), Interpersonal SPQ (*t*(93.705) = 4.865, *p* < .001), Disorganised SPQ (*t*(100) = 6.233, *p* < .001), and Verbal intelligence (*t*(100) = -5.522, *p* < .001).The Dyslexia group had significantly higher levels of Cognitive-Perceptual, Interpersonal, Disorganised, and Total schizotypy compared to Controls. The Dyslexia group also had significantly lower verbal intelligence, and a significantly higher rate of mixed handedness compared to Controls.

Table 10.1. *Demographic variables for Dyslexia and Control groups.*

	Dyslexia (n =51)	Controls (n = 51)
Sex (Male: Female)	16:35	15:36
Age	24.8 (1.5)	24.14 (1.3)
Living arrangements (Parents: Siblings: Partner: Friends: Acquaintances: Alone)	22:1:9:11:5:3	26:3:10:6:4:2
Health service use (Y:N)	28:22	32:19
Handedness (Right: Left: Mixed)	41:4:6	49:2:0*
Schizotypy (SPQ) Total	33.04 (2.3)	17.73 (1.7)***
Cognitive-perceptual Schizotypy	11.33 (1.06)	6.9 (.83)**

(SPQ)		
Interpersonal Schizotypy (SPQ)	13.73 (1.1)	6.94 (.84)***
Disorganised Schizotypy (SPQ)	9.45 (.61)	4.55 (.5)***
Verbal intelligence (NART)	21.1 (.97)	28.5 (.92)***

*Standard error of the mean shown in parentheses. Significant differences between groups indicated by *p < .05; **p < .01; ***p < .001*

10.4.2 Semantic Ambiguity Task Response Times

10.4.2.1 Group analysis (*F1*) for reaction time data

Analyses are based on median concordant response times and were analysed using a 2 (meaning) X 2 (relatedness) X 2 (Dyslexia/Control groups) Repeated Measures ANOVA. Meaning and relatedness were the within subject variables and Dyslexia/Control was the between subject variable. Sphericity was not violated for this data, and all variables met the +/- 2 limits for skewness and kurtosis, therefore no corrections were required. Where post hoc analyses were required Bonferroni corrections were used to adjust the *p*-value.

Main effects were found for meaning ($F(1, 100) = 48.253$, MSE = .514, $p < .001$, $\eta^2_p = .325$) and relatedness ($F(1, 100) = 88.075$, MSE = 3.522, $p < .001$, $\eta^2_p = .468$) task conditions, with participants responding significantly faster to dominant (M = 964ms, SE = 20)words over subordinate (M = 1035ms, SE = 20), and related (M = 906ms, SE = 19) words over unrelated (M = 1092ms, SE = 24). A significant interaction was found between meaning and relatedness task effects ($F(1, 100) = 142.586$, MSE = 1.717, $p < .001$, $\eta^2_p = .588$). This interaction is due to the task effect whereby for related words, participants are expected to respond quicker to dominant word pairs compared to subordinate. However for unrelated words, participant responses to dominant versus subordinate word pairs is not expected to differ as both types of word pairs are unrelated.

No main effect was found for those with Dyslexia versus Controls ($p = .262$), or for the interaction between group and meaning ($p = .263$) or relatedness ($p = .856$) task effects. A trend level effect was found for the interaction between group, meaning and relatedness ($p = .071$). Mean reaction times found in Table 10.2.

Table 10.2. *Mean of the median reaction times to concordant responses in milliseconds.*

Group	Meaning	Related	Unrelated
Dyslexia	Dominant	830 (18)	1129 (25)
	Subordinate	1022 (20)	1101 (24)
Controls	Dominant	782 (21)	1113 (27)
	Subordinate	991 (23)	1023 (23)

Standard deviation shown in parentheses.

10.4.2.1.1 *FI* analysis with CP schizotypy covariate

When CP schizotypy was added as a covariate, the task effect findings all remained unchanged. Significant effects were found for meaning ($F(1, 99) = 23.879$, MSE = .255, $p < .001$, $\eta^2_p = .194$), relatedness ($F(1, 99) = 36.327$, MSE = 1.462, $p < .001$, $\eta^2_p = .268$), and the interaction between meaning and relatedness ($F(1, 99) = 63.974$, MSE = .769, $p < .001$, $\eta^2_p = .393$). The addition of CP schizotypy as a covariate was not significant ($p = .588$), and did not alter the non-significant effects of group in the initial analysis.

10.4.2.1.2 Correlations between response time differences for dominant and subordinate related word pairs

The response times to related targets was subtracted from the response times to unrelated targets to develop a measure of response time difference for both dominant and subordinate targets. Pearson's correlations were used to check if mechanisms are similar across the Dyslexia and Control groups. The correlation in response time differences between dominant and subordinate word pairs was significant and positive for those with Dyslexia ($r(51) = .532$, $p < .001$), and Controls ($r(51) = .55$, $p < .001$). The magnitude and similarity of these correlations suggests that the response time mechanisms are the same for ambiguous and clearly related stimuli in those with and without dyslexia.

In order to see whether positive schizotypy was contributing to the mechanisms responsible for reaction time responses in the semantic task, Pearson's correlations were then conducted between CP schizotypy and response time variables for Dyslexia and Control groups. In the Dyslexia group no significant relationships were found between CP schizotypy and the dominant related ($r(51) = .022$, $p > .05$), subordinate related ($r(51) = -.271$, $p > .05$), dominant unrelated ($r(51)$

= -.24, $p > .05$) or subordinate unrelated ($r(51) = -.21$, $p > .05$) conditions. Likewise in the Control group CP schizotypy was not related to reaction time responses in the dominant related ($r(51) = .042$, $p > .05$), subordinate related ($r(51) = .149$, $p > .05$), dominant unrelated ($r(51) = .136$, $p > .05$), or subordinate unrelated ($r(51) = .128$, $p > .05$) conditions. These results suggest that positive schizotypy is not contributing to the reaction time response mechanisms for semantic processing in either group.

10.4.2.2 Item analysis (*F2*) for reaction time data

Median concordant reaction times were calculated across participants for each word pair. A 2 X (meaning) X 2 (relatedness) X 2 (Dyslexia/Control groups) Repeated Measures ANOVA was used to analyse response times. Dyslexia/Control group was the within item variable and relatedness and meaning were the between item variables.

Significant task effects were found for both meaning ($F(1, 283) = 10.331$, MSE = 1.053, $p < .001$, $\eta^2_p = .035$) and relatedness ($F(1, 283) = 43.073$, MSE = 4.392, $p < .001$, $\eta^2_p = .132$). As was found in *F1* analyses, participants responded faster to dominant targets (M = 1002ms, SE = 19) over subordinate (M = 1088ms, SE = 19), and related targets (M = 958ms, SE = 19) over unrelated (M = 1133ms, SE = 19). The interaction between meaning and relatedness was also replicated ($F(1, 283) = 22.508$, MSE = 2.295, $p < .001$, $\eta^2_p = .074$). As in the *F1* analysis, this is due to participants responding faster to dominant words compared to subordinate in the related condition. However when words are unrelated to each other, no differences in reaction time are expected as a result of meaning, as they are equivalent in their unrelatedness.

A significant main effect was found for group ($F(1, 283) = 41.494$, MSE = 1.05, $p < .001$, $\eta^2_p = .128$). Those with Dyslexia (M = 1088ms, SE = 16) responded significantly slower than those without Dyslexia (M = 1002ms, SE = 14ms). No interaction effects were found between group and meaning ($p = .741$), relatedness ($p = .413$) or the interaction between group, meaning and relatedness ($p = .958$).

10.4.2.3 Consistency in results for *F1* and *F2* main analyses

Task effects were consistent across *F1* and *F2* analyses, with participants responding significantly faster to dominant and related words. The *F2* analysis result of those with Dyslexia responding significantly slower to word pairs was not confirmed in

the *F1* analysis. This suggests the effect in the *F2* analysis may have been driven by a small number of items which may have not been as well known to the Dyslexia participants compared to Controls. Accordingly, word pairs were ordered according to reaction time difference between Dyslexia and Control groups. The word pairs LIGHT/RAIN and FILE/LETTER were detected as outliers, with subsequent reaction time differences not standing out from the distribution. It is possible that these two items contributed to the inconsistent *F1* and *F2* analysis group effects.

10.4.3 Signal Detection Analyses

10.4.3.1 Sensitivity analysis (*d′*)

A Repeated Measures ANOVA was used to analyse the sensitivity (*d'*) variables. Dyslexia/Control group was the between subject variable, and meaning was the within subject variable.

A main effect of meaning was found ($F(1, 100) = 109.051$, MSE = 14.527, $p < .001$, $\eta^2_p = 522$), with participants significantly more able to differentiate between related and unrelated targets for dominant word pairs (M = 1.065, SE = .101) compared to subordinate (M = 1.373, SE = .069).

A significant difference was also found between Dyslexia and Control groups ($F(1, 100) = 5.285$, MSE = 7.355, $p = .024$, $\eta^2_p = .05$), with the Dyslexia group (M = 1.45, SE = .117) significantly less able to differentiate between related and unrelated targets compared to the Control group (M = 1.829, SE = .117).

A significant interaction effect was also found between group and meaning ($F(1, 100) = 4.453$, MSE = 0.593, $p = .037$, $\eta^2_p = .043$). Follow up analyses revealed that meaning was significant in both Dyslexia and Control groups, with participants significantly more able to differentiate between related and unrelated targets for dominant word pairs compared to subordinate (Dyslexia group: $F(1, 50) = 41.314$, MSE = 4.624, $p < .001$, $\eta^2_p = .452$; Control group: $F(1, 50) = 67.938$, MSE = 10.495, $p < .001$, $\eta^2_p = .576$; means in Table 10.3). However differences between groups for *d′* was only significant for dominant word pairs ($t(100) = -2.414$, $p = .018$), with the *d′* for subordinate targets of Dyslexia and Control groups not significantly different ($p = .051$).

10.4.3.1.1 Sensitivity analysis with CP schizotypy covariate

When CP schizotypy was added to the analysis as a covariate the main effect of meaning remained ($F(1, 99) = 42.313$, MSE = 5.684, $p < .001$, $\eta^2_p = .299$). However, the significant difference between Dyslexia and Control groups for *d´* observed in the initial analysis no longer remained significant ($p = .116$), as well as the significant interaction between group and meaning ($p = .064$). No interaction effects were found for CP schizotypy and meaning *(*$p = .692$). However CP schizotypy did have a significant effect as a covariate in the analysis ($F(1, 99) = 4.129$, MSE = 5.572, $p = .045$, $\eta^2_p = .04$).

Table 10.3. *Mean values for hits, false alarms, and sensitivity judgments in Dyslexia and Control groups.*

Group	Meaning	% Hits	% False alarms	*d´*
Dyslexia	Dominant	74.6 (17)	21.2 (14)	1.66 (.96)
	Subordinate	61.6 (15)		1.24 (.69)
Controls	Dominant	80.4 (19)	17 (12)	2.15 (1.07)
	Subordinate	65.5 (15)		1.51 (.7)

Standard deviation in parentheses. Note: Hits are out of 36 trials each. False alarms were combined for dominant and subordinate targets, therefore are out of 72 trials.

10.4.3.2 Relative Criterion analysis (*c´*)

Due to the absolute *c* being derived from a different *d´* it was not comparable for dominant and subordinate targets. As a result, the absolute *c* value underwent arithmetic transformation, to express both in terms of distance from the distribution for the unrelated pairs, therefore allowing dominant and subordinate *c* to be compared for each group. The *c* for dominant and subordinate targets was the same for those in the Dyslexia group (M = .913, SD = .55), and those in the Control group (M = 1.08, SD = .49). This establishes that there is only a single *c* being used by each group.

In order to compare the *c* being used by the Dyslexia and Control groups, *c* was transformed into *c´* (Macmillan and Creelman, 2005). An Independent Samples

t-test indicated that no differences were found between Dyslexia (M = .256, SD = 1.5) and Control (M = .212, SD = .86) groups (p = .856) for *c´*.

10.5 Discussion

Semantic processing capabilities were examined in a Dyslexia sample compared to Controls. As expected, all participants responded faster to pairs related by the dominant meaning compared to the subordinate meaning, and to related word pairs compared to unrelated pairs. The Dyslexia group responded slower to word pairs compared to the Control group, however this finding was inconsistent across *F1* and *F2* analyses. With regards to signal detection analysis, the Dyslexia group were less able to differentiate between related and unrelated dominant word pairs, compared to the Control group. The difference in sensitivity between the groups for subordinate word pairs also approached significance. No differences were found between groups for the criterion analysis. Additionally, we investigated whether positive schizotypy was able to account for any of the differences in semantic processing between those with and without dyslexia. Positive schizotypy appeared to account for the differences between the Dyslexia and Control groups in the sensitivity analysis. This finding suggests that positive schizotypy may be responsible for the difficulties discriminating between related and unrelated word pairs observed in the Dyslexia group.

It was expected that compared to Controls, the Dyslexia group would respond significantly slower under ambiguous conditions (subordinate words). No differences between the groups were found as a result of word meaning, however in the *F1* analysis the Dyslexia group recorded significantly slower reaction times overall compared to the Control group. Previous research has demonstrated slowed response times on semantic tasks for individuals with Dyslexia (e.g. Schulz et al., 2008). When combined with EEG and ERP data, slower responses were hypothesised by Schulz et al. (2008) to reflect delayed cerebral activation in the inferior parietal region, which is known to process semantic information. Similarly, Rüsseler et al (2007) found a neural correlate of semantic processing, the N400, to persist for significantly longer in those with dyslexia compared to Controls, suggesting that semantic processing may take longer for those with dyslexia. The current results are in support of this, however given that this result was not consistent in the *F2* analysis, caution must be taken when extrapolating the meaning of these

findings. Inspection of the reaction time responses to word pairs across the groups identified 2 items which were responded to much slower in the Dyslexia group compared to Controls.

The Dyslexia group did not differ in their response pattern to the task compared to the Control group, with both groups responding with a conservative response pattern to all items and an equivalent criterion for judging relatedness. Under conditions of ambiguity, the expected semantic function is to be more cautious in response style. Participants in the task responded this way, and the lack of a group difference suggests the decision making processes of those with dyslexia is not impacted by their difficulties with language.

The Dyslexia group was less able to discriminate between unrelated and related word pairs compared to the Control group. This finding suggests that individuals with dyslexia have a greater difficulty accessing semantic information in a way that allows them to detect relationships between words. In line with this result are findings of those with dyslexia having significantly different activation patterns in areas of the brain which process semantic information, compared to those without dyslexia (e.g. Kronbichler et al., 2006; Booth et al., 2007). These atypical activation patterns could be indicative of problems activating semantic representations and keeping multiple semantic nodes active; especially given that the task in this study utilised homographs rather than words with only one meaning. Repeating the task with words with only a singular meaning may help to reveal if the reduced ability of those with dyslexia to identify the relationship between two words is due to the atypical semantic activation of words with multiple meanings, or if it is the result of having words activated without grammatical or contextual support.

The current study also reported a novel finding, in that when positive schizotypy was considered in the analysis, it seemed to explain the group differences in discrimination. This result suggests co-occurring positive schizotypy may be related to difficulties discriminating between related and unrelated words in dyslexia, rather than these difficulties occurring solely as a result of language and reading problems. Previous research has highlighted links between dyslexia and positive schizotypy (Richardson and Stein, 1993; Richardson, 1994; de Leede-Smith et al., submitted), and dyslexia and the psychosis continuum in general (e.g. Condray, 2005; Bersani et al., 2006; Revheim et al., 2014). The current study extends these findings to show that the difficulties discriminating between unrelated and related

word pairs in dyslexia may be accounted for by positive schizotypy. Under experimental conditions, compared to controls, individuals with high schizotypy have been found to identify auditory stimuli in the absence of any true stimuli significantly more often (Barkus et al., 2007; Galdos et al., 2011). Combined, these findings indicate that schizotypy is associated with deficits in the ability to distinguish between stimuli which are true and those which are not. Given that significantly higher levels of schizotypy were found in the Dyslexia group compared to Controls, this may explain why those with dyslexia had greater difficulty distinguishing whether two words were related or not.

Finally, demographic investigations indicated that the Dyslexia group reported significantly higher levels of Interpersonal, Cognitive-Perceptual, Disorganised and Total schizotypy, as well as a greater rate of mixed handedness compared to the Control group. These findings support and extend previous research, where individuals with dyslexia had higher rates of positive schizotypy and mixed handedness (Richardson and Stein, 1993; Richardson, 1994). Dyslexia has previously been associated with reductions in cerebral asymmetry (Heim et al., 2004), with the current results supporting this finding. Not only were there higher rates of schizotypy in dyslexia, but schizotypy also appeared to account for the difficulties of those with dyslexia in discriminating between related and unrelated word pairs. These findings extend previous hypotheses linking dyslexia and the psychosis continuum (e.g. Condray, 2005), to show that difficulties with semantic discrimination in dyslexia may be partially explainable by schizotypy.

There were some limitations of the current study. The lack of consistency in the *F1* and *F2* reaction time analyses is suggestive of the Dyslexia sample either having possible difficulties with some of the items in the task; which would indicate no true reaction time difference between those with and without dyslexia, or perhaps an insufficient sample size to accurately detect true differences between the groups. Accordingly, replication of this task with a larger dyslexia sample would determine whether this result is an anomaly, or if there is something inherent with these items which is difficult to process semantically for those with dyslexia. One benefit of utilising a university dyslexia sample is that they likely were familiar with the simple words used in the semantic task. As a result, the speed of word identification is unlikely to be contributing to the slowed reaction time of the Dyslexia group. Additionally, the level of functioning required of students in tertiary education

dictates that those individuals with dyslexia have likely developed effective compensatory strategies to make up for any difficulties they have reading and writing as a result of their learning disorder. As a result, the typical profile of someone with dyslexia attending university may be different compared to someone with dyslexia from the general population. Accordingly, additional research is warranted, investigating semantic processing and the possible overlap of positive schizotypy for those with dyslexia in the general population.

Individuals with dyslexia demonstrated slower reaction time responses and difficulties discriminating word pairs in terms of their relatedness. However the decision making processes of the Dyslexia group was comparable to Controls. These findings indicate that individuals with dyslexia have impaired semantic processing capabilities. Additionally, for those with dyslexia, the difficulties discriminating semantic relations seem to be partly explainable by positive schizotypy. These results indicate that schizotypy may be responsible for some of the semantic processing difficulties found in dyslexia.

11 GENERAL DISCUSSION

11.1 Summary of findings

The overall aim of this thesis was to investigate trait, state, and neurodevelopmental risk factors for psychosis, using schizotypy as a proxy for psychosis proneness. Study One examined the relationship between schizotypy, affective temperament, and psychological distress. In line with original hypotheses, decreased positive and increased negative affective temperament was found to partially mediate the relationship between schizotypy and distress, with negative temperament exerting the greatest mediating effect. Hallucination predisposition did not moderate the mediation models, however it did moderate the relationship between schizotypy and negative temperament. These results suggest that temperament contributes to the likelihood high schizotypes will experience distress.

The interaction between schizotypal trait risk and hallucination predisposition was the focus of Studies Two and Four. It was predicted that the interaction between these trait and state psychosis risk factors would lead to a greater expression of neurodevelopmental risk factors for psychosis, specifically: NSS (Study Two), and semantic processing abnormalities (Study Four). For Study two, results indicated that those with high levels of schizotypy expressed significantly more total and subscale NSS. The combination of high levels of schizotypy and high levels of hallucination predisposition also led to a significantly greater expression of Motor-Coordination NSS.

In Study Four the interaction between Cognitive-Perceptual (CP) schizotypy and hallucination predisposition was investigated for reaction time and signal detection determinants of semantic processing. CP schizotypy was used due to previous associations between positive schizotypy and abnormal semantic processing (e.g. Grimshaw et al., 2010; Morgan, Bedford and Rossell, 2006). Results indicated that those with high levels of CP schizotypy had significantly slower reaction times in a semantic task, when compared to those with low levels of CP schizotypy. Contrastingly, some evidence was found for those with high levels of hallucination predisposition to have significantly faster reaction times, compared to those with low hallucination predisposition. These findings were not in line with predictions. Instead, these results appear to suggest that CP schizotypy and hallucination predisposition impact differently on the processing of semantic information. The

results of the high CP schizotypy group appear to be in line with a more diffuse spread of activation when processing semantic associations. Contrastingly, in high hallucination predisposition, results suggest there may be a more disinhibited semantic processing capacity.

Neurological soft signs and semantic processing, as neurodevelopmental risk factors occurring along the psychosis continuum, were also investigated in a dyslexia sample. Previous research has suggested there are links between the psychosis continuum and dyslexia (e.g. Condray, 2005; Bersani et al., 2006). Accordingly, Studies Three and Five explored whether NSS and semantic processing abnormalities were expressed to a greater extent in dyslexia, compared to healthy controls. Additionally, if a greater expression of these neurodevelopmental risk factors were found, schizotypy was investigated to see if it contributed to these findings. In Study Three, results indicated that higher levels of NSS, schizotypy and mixed handedness were found in the dyslexia sample compared to controls. Higher levels of disorganised schizotypy, a greater expression of Sequencing of Complex Motor Acts (SCMA) NSS, and lower levels of verbal intelligence predicted dyslexia status. Although not expected, higher levels of psychological distress were found in those with dyslexia compared to controls. The observed differences in distress between those with dyslexia and controls seemed to be accounted for by schizotypy; a novel and unexpected finding.

Study Five examined semantic processing capabilities in those with dyslexia compared to controls, and whether schizotypy contributed to any differences found. There was some evidence that the dyslexia group responded slower in the semantic task when compared to controls. There was strong evidence that those with dyslexia were also less able to discriminate between related and unrelated words, however no differences were found between dyslexia and control groups for decision making style. Most importantly, positive schizotypy seemed to account for differences between those with dyslexia and controls in the ability to discriminate between related and unrelated words. These findings may appear to be in contrast to those reported in Study Three, where no differences were found between high and low positive schizotypy groups in terms of discrimination. However the dyslexia group in Study Five had a much higher mean schizotypy score compared to controls, resulting in a clearer distinction of schizotypy between these groups.

11.2 Implications for research in the area

Looking at this thesis holistically, one major theoretical implication seems to be the distinct characteristics of schizotypal trait and hallucinatory state psychosis risk factors. In Study One, schizotypy was found to have direct and indirect relationships with distress, however contrary to expectations, hallucination predisposition did not moderate these relationships. In Study Four, schizotypy and hallucination predisposition seemed to have differential effects on the processing of semantic information. These results indicate that schizotypy and hallucination predisposition are not synonymous in their influence on other psychological and cognitive factors (see also Preti et al., 2007). Yet in Study Two, hallucination predisposition was found to interact with schizotypy, to lead to a greater expression of Motor-Coordination NSS compared to schizotypy and low proneness to hallucinations. This result suggests that for NSS, hallucination predisposition may have additive effects when combined with schizotypy. In terms of future research in the area, these findings suggest that the effect of hallucination predisposition should be considered or controlled for in studies examining schizotypal trait risk, depending on study aims. Some of the findings of this thesis seem to suggest hallucination predisposition may not contribute to psychosis risk (Study One and Four). However, taken together, these findings point to schizotypy and hallucination predisposition being separate constructs, which have distinct effects on other psychosis risk variables.

The overlap in features between schizotypy and dyslexia may also have significance for future research. Individuals with dyslexia were found to have significantly higher rates of total and dimensional schizotypal traits, mixed handedness and NSS compared to controls (Study Three). In Study Five, positive schizotypy was also found to account for some of the semantic processing abnormalities found in those with dyslexia compared to controls. These findings suggest that there are a number of common features between dyslexia and the psychosis continuum. It seems possible that the psychosis continuum and dyslexia may have overlapping phenotypes, and by extension, share some common aetiologies (e.g. Condray, 2005). This may have implications for researchers investigating language dysfunction along the psychosis continuum, and drives further questioning regarding how dyslexia is related to the psychosis continuum. In order to fully investigate whether dyslexia and psychosis have overlapping

aetiologies, research from both literatures needs to more thoroughly control for these constructs.

11.3 Clinical implications

As discussed previously, the results of this thesis suggest that schizotypy, as a trait risk factor for psychosis, seems to act differently to hallucination predisposition in its effect on other potential psychosis risk factors. This finding suggests that schizotypy and hallucination predisposition are not synonymous in their mechanisms of influence. These results may then bring to question what component of hallucinations is relevant in terms of psychosis risk. The cognitive model of psychosis suggests that it is not necessarily PLEs per se which are related to psychopathology, but rather the cognitive appraisal of those experiences (Garety et al., 2001). Other research suggests that an individual's degree of subjective certainty in their experience of PLEs is more relevant to psychopathology compared to the frequency of PLEs (Preti et al., 2012). Clarifying which component of hallucinatory predisposition has relevance to psychosis risk will have utility in discriminating hallucinations which are benign, from those which are clinically relevant and may inform prognosis.

Schizotypy was found to be related to distress directly, and also indirectly, via increased negative and decreased positive temperament. The findings of this thesis can inform existing preventative interventions of the factors and mechanisms contributing to distress for those at psychometric risk for psychosis. For instance, high school students with high schizotypy who participated in a social skills training intervention reported improved social competence and self-esteem, and reductions in schizotypal symptoms (Liberman and Robertson, 2005). A meta-analysis of cognitive behavioural therapy for ARMS participants aimed at addressing negative appraisals of PLEs has also been shown to be effective at reducing transition to psychosis over 24 months (Hutton and Taylor, 2014). The results of this thesis suggest that clinical interventions aimed at those with heightened psychometric schizotypy may also be efficacious, particularly given the relationships with distress in this population.

Heightened levels of distress were also found in those with dyslexia (Study Three). Surprisingly, schizotypy appeared to account for this distress, which has specific clinical implications for enhancing our understanding of the psychological experiences of individuals with dyslexia. Heightened depression and low self-esteem in dyslexia have been considered to be the result of difficulties with reading and

language (e.g. Riddick, 1996; Alexander-Passe, 2006). The current findings suggest that schizotypal personality also has some relevance to the distress experienced by those with dyslexia. Having a greater understanding of the causes of distress in dyslexia is useful for targeting reductions in distress in this population, given that focused preventative strategies can be developed. In terms of what this means clinically, it may be beneficial for clinicians who are treating help-seeking individuals with dyslexia to firstly be aware of the links between dyslexia and schizotypy, and be mindful of the possibility that their clients' distress may be contributed to by schizotypal traits. Further, if indicated, psychological interventions aimed at reducing the distress associated with schizotypal traits may be a beneficial line of future clinical research and intervention in those affected individuals.

Aside from distress, the overlap between schizotypy and dyslexia was a common theme in Studies Three and Five. It is possible that language difficulties in childhood in the presence of other psychosis risk factors, such as trait schizotypy or PLEs, may be used as a clinical marker for heightened risk for psychosis (e.g. Bearden et al., 2000). Certainly more research exploring these links is needed to understand the potential significance of these phenomena from a young age. However the findings of this thesis hopefully encourage investigations into the co-occurrence of these phenomena, and what these overlapping trajectories mean in the context of psychosis risk.

Finally, an increased expression of NSS was found in both dyslexia and high schizotypy samples, and additionally hallucination predisposition was found to have additive effects with schizotypy in the expression of these neurodevelopmental aberrations. NSS have been the focus of many investigations along the psychosis continuum (e.g. Chan et al., 2016). The current results indicate that with the addition of hallucination propensity, the expression of NSS in schizotypy was even greater, suggesting the expression of NSS may be sensitive to the number of psychosis risk factors a person has. These findings point to the potential of NSS as a marker of heightened risk.

11.4 Limitations of the present research

One of the biggest limitations of the research in this thesis was the cross-sectional design of the studies. This design was chosen given that the research questions were largely exploratory in nature. However as a result, the findings from this thesis can only be interpreted as associational, and the temporal relationships between variables cannot be commented on.

The use of self-rating scales, as opposed to structured interview-based assessment measures is another limitation. Measurement of schizotypy and PLEs can be confounded by numerous factors, including:

- Misunderstanding the nature of the questions (i.e. AVH questions could be interpreted as relating to hearing ability) (see Kessler et al., 2005).
- Normalising the experiences (i.e. paranoia could be interpreted as actual intended harm).
- Poor insight may distort responses, especially those concerning emotion, wellbeing and delusions (i.e. by way of jumping to conclusions; Van Dael et al., 2006).
- Perceived stigma associated with PLEs may result in them being falsely denied (e.g. Hanssen et al., 2003).

Despite these limitations, self-report measures of sub-clinical psychotic experiences have been shown to be highly accurate in detecting these experiences in the general population (e.g. Kelleher et al., 2011). Additionally, self-report measures were chosen due to the flexibility it allowed volunteer participants, who were able to complete most self-report scales in their own time.

The participants who took part in this study were (for the most part) students enrolled in tertiary education. Epidemiological research has found that students enrolled in university differ from those in the general population, with higher rates of mental health problems and psychological distress reported in university students (Stallman, 2010). As a result, the findings from this thesis, although perhaps representing an enriched sample, may not be generalisable to the general population. This limitation has specific relevance to Studies Three and Five, where a dyslexia sample was the focus of these investigations. Given that individuals with dyslexia by definition struggle with language and reading tasks, it is not surprising that individuals with dyslexia only make up 0.2 to 0.4% of tertiary student populations

(Richardson and Wydell, 2003; Stampoltzis and Polychronopoulou, 2008). Accordingly, it is possible that individuals with dyslexia who attend university differ from those with dyslexia who are not enrolled in tertiary education. For example, it may be expected that those attending university have developed strategies to better manage their disabilities, and limit the impact of their dyslexia on day-to-day tasks. Alternatively, it is possible that those with dyslexia experience higher levels of distress compared to those in the general population, as a result of confronting their difficulties with language and reading on an every day basis. Until thorough comparison studies have been conducted investigating the characteristics of individuals with dyslexia who attend university versus those who do not, the representativeness of the current dyslexia sample to those in the general population is unknown.

The current thesis utilised a university-based sample for all research studies. Contrary to findings by Stallman (2010) discussed previously, poor mental health has been related to substantially lower educational achievements (Patel et al., 2007). Therefore it may be that university students have a lower risk for psychopathology due to their inherent protective factors (education, social support), as well as the higher level of functioning required to successfully progress through university. As a result, it could be conceived that psychosis risk research conducted with university samples has limited utility. However the individual differences approach to schizotypy implies that there is meaningful variation associated with schizotypy, and that these differing expressions should be evident in student samples. Accordingly, student samples may represent a conservative group, given that they are expected to have protective factors and relatively good premorbid adjustment. Therefore, any significant findings related to schizotypy in university samples encourage the extension of those research methods to broader community samples. Furthermore, high schizotypes who remain functioning are of just as much scientific importance as those who decompensate to psychotic illness, as they are able to inform us of protective factors and the potential significance of these factors in preventing possible transition to psychosis.

Another limitation was the use of similar scales for the measurement of trait and state psychosis risk. Both the SPQ and the LSHS were used across Studies One, Two and Four. Although Cognitive Perceptual Schizotypy and AVH predisposition (as measured in the LSHS) are distinct constructs, there was some item content that

was undoubtedly shared between the two scales. Indeed, correlation analyses presented in the Introduction illustrated moderate associations between the constructs. In order to maximise construct validity, future studies should aim to eliminate shared item content between these two scales..

One final limitation was the use of the NART as a measure of verbal IQ across all studies. Whilst the NART is a widely used measure of verbal IQ, there are obvious limitations in it's use with individuals with dyslexia, given that the task requires participants to read the words out loud. The individuals with dyslexia who participated in studies Three and Five were however enrolled in tertiary education, suggesting that their specific learning disorder is not *as* likely to impact on their day-to-day functional reading capacity compared to others in the community with the same diagnosis. Furthermore, the NART has been used as a measure of verbal IQ for individuals with dyslexia in previous peer-reviewed research (e.g. McCrory et al., 2000; Johnston et al., 2008), suggesting that it's inclusion in studies Three and Five of this thesis is not an isolated occurrence. Nonetheless, in order to maximise variability control, it is recommended that future research interested in measuring or controlling for verbal IQ in those with dyslexia do so using spoken measures of verbal IQ rather than those which require the participant to read stimuli.

11.5 Directions for future research

Given that many of the limitations associated with this thesis are related to the cross-sectional design of the studies, it follows that future research would benefit from employing a longitudinal research design. Specific longitudinal research questions that may come out of this thesis include:

1. An analysis from childhood through to adulthood, which investigates the development of schizotypy and affective temperament over time. This may go part way in understanding how schizotypal personality develops, and identify how affective temperament interacts with schizotypy over time. Given that this thesis identified affective temperament (particularly negative temperament) as relevant in the relationship between schizotypy and distress, following these traits over time will also be useful in understanding when and how distress develops as a result of these traits, as well as identifying other contributors to these relationships.
2. An analysis of the relationship between schizotypy, affective temperament and distress over time, which also takes into account fluctuations in PLEs such as hallucinations. Doing so can help in determining whether the additive effect of trait and state psychosis risk impacts on the level of distress experienced by an individual. Although Study One of this thesis aimed to explore this link, given that the research was cross sectional it only provided a snap shot of this relationship at one point in time. Having a propensity to hallucinate was not found to contribute to this relationship statically, however this result may change over time, especially in periods of high risk for psychosis (late adolescence/early adulthood). Following individuals longitudinally can determine whether the relationship between schizotypy, affective temperament and distress changes as a result of PLEs such as hallucinations.
3. An analysis of NSS alongside schizotypy and hallucination predisposition psychosis risk factors over time. The current thesis found that hallucination predisposition exacerbated the Motor Coordination NSS found in those with high levels of schizotypy. This finding suggests that when both trait and state psychosis risk factors are present, the expression of subtle neurodevelopmental abnormalities is increased. A longitudinal analysis,

which is able to account for fluctuations in state risk factors such as hallucinations, will provide additional support for this finding. Additionally, if support for this finding does prevail, NSS could potentially then be researched as a clinical marker of psychosis risk which is changeable depending on the number of risk factors the individual presents with.

4. An analysis of the development of schizotypy and NSS in individuals with language difficulties from early childhood through to adulthood. If schizotypal traits are associated with NSS from childhood, and if this association is stable and persists over time, this may point to a shared aetiology between dyslexia and schizotypy. Genetic analyses could also be used to explore the possible genetic overlap between the psychosis continuum and dyslexia, and clarify the nature of the relationship between these two disorders to determine whether or not they share genetic origins.

The current thesis sheds light on the previously mixed findings of semantic processing in schizotypy. In Study Four, schizotypy appeared to be associated with a more diffuse spread of semantic activation, whereas hallucination predisposition seemed to be in line with a more disinhibited style of semantic activation. These findings suggest that future studies should take into account participant's levels of both schizotypy and hallucination predisposition. This may produce a more interpretable pattern of results for those areas of schizotypy research where mixed findings have prevailed, such as is the case with semantic processing.

11.6 Conclusions

This thesis examined associations between schizotypy, affective and neurodevelopmental risk factors for psychosis in the form of: affective temperament, psychological distress, NSS, and semantic processing. Results indicated that schizotypy is associated with a heightened expression of NSS and abnormal semantic processing, with hallucination predisposition also contributing to these findings. Individuals with dyslexia shared features with the psychosis continuum, including: heightened levels of schizotypy, mixed handedness, increased expression of neurological soft signs, and deficits in the ability to discriminate semantic information. Combined, these findings point to associations between psychometric schizotypy and known risk factors for psychosis, providing additional evidence for the hypothesised aetiological continuity between schizotypy and schizophrenia. Given that this thesis was specific to schizotypy in its investigations, these findings also highlight the relevance of schizotypal trait as a contributor to affective, neurodevelopmental, and language functioning in the non-clinical population. Consideration of the relationship between schizotypy and other trait and state psychosis risk factors over time may clarify understanding of the developmental trajectories that may result in psychotic illness.

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13 APPENDICES

13.1 Appendix A: Participant information sheets and consent forms

Stage 1: Online questionnaire research component

University of Wollongong 

Participation Information Sheet

Unusual perceptions and the personality trait schizotypy

This is an invitation for you to participate in a study conducted by researchers at the University of Wollongong. The research is called "Unusual perceptions and the personality trait schizotypy". The purpose of the research is to investigate the factors associated with sleep related unusual experiences in the general population. We will tell you a little about the factors we are interested in but you will be free to ask further questions of the researcher.

The personality trait we are interested in is called schizotypy. This sounds a little like a mental health disorder called schizophrenia but in fact the two are different. Much like other personality traits such as extraversion (how out-going you are), schizotypy is normally distributed in the general population. This means most people score around the average but as many people score extremely high as extremely low on the personality trait. Scoring particularly high or low on schizotypy does not necessarily carry any negative connotations it is merely part of the interesting complexities which make up people's personalities! However if you do have any concerns please feel free to discuss them with us. We are interested in how schizotypal personality, emotional processing styles and behaviours may be related to the experience of different sleep related unusual perceptions. It is common for some people to hear perceptions such as voices, music, and other indistinguishable noises when they are not actually there. These perceptions can occur when taking part in day-to-day activities, and can also occur in the drowsy state experienced just before falling asleep, or right when you wake up. We are interested in the details and characteristics of these experiences in different people. If you have any questions regarding our research we will be happy to answer them. We will not be able to offer individual feedback on your responses to the questionnaires. However, we will be able to provide you with a summary of the findings of the study so if you are interested please let us know.

WHAT WE WOULD LIKE YOU TO DO

In this study we ask that you complete a number of questionnaires relating to your personality, behavior, perceptions and how you react in different scenarios. By

providing this type of information we are able to gain an understanding of the way you perceive situations, and this helps us in determining your personality traits and emotional appraisals. It is expected that this section should take 100-120 minutes of your time. Examples of some of Yes/No statements we will ask are:

- People sometimes find me aloof and distant.
- No matter how hard I try to concentrate unrelated thoughts always creep into my mind.
- Sometimes I suddenly feel scared for no good reason.
- I trick myself into believing something is okay when it's not.

We do appreciate this may seem like a long time to be committing to taking part in a study. However people often find the process informative and you will be helping to forward research into personality and mental health. If you or someone close to you has experienced problems associated with mental health difficulties we will provide contact details for a range of health and support services which are available to assist you, including:

- *Lifeline: 13 11 14*
This is a 24-hour confidential support line which is able to provide individuals with both information and support, and if necessary refer you on to appropriate mental health networks.

- *Life Resolutions: 1300 3249 32*
This is a network of trained professional psychologists within Australia. They cater for a wide variety of mental health areas and concerns, and are located in a multitude of locations across Sydney and Australia-wide.

- *Northfields Clinic:* (02) 4221 3747
Based at the University of Wollongong, this clinic provides high quality psychological services at a heavily discounted rate. They offer a range of clinical assistance, ranging from initial assessments through to group therapy and highly specialized individualized sessions.

Your involvement in this study is voluntary and you may withdraw your participation from the study at any time and any data that has been gathered to that point will be withdrawn and destroyed. If you do choose to withdraw your consent your withdrawal will not have any adverse effect and will in no way affect your treatment, studies or relationship with the University of Wollongong. Once we analyse the data obtained from this study there is a possibility we may want to contact you for participation in an additional stage of research. If you do not wish to be contacted for any additional participation please indicate this on the consent form below.

The questionnaire responses obtained in the study will be stored in a password protected computer file. This will guarantee that your information remains confidential. Findings from this study will be published in a thesis and possibly an academic journal. The data obtained will also be combined with findings from other related research studies. Your anonymity will be maintained by immediately separating the cover sheet (with possible identifying information) from your questionnaires. Confidentiality will also be preserved by assigning numbers rather than names to the written records, as well as only reporting on grouped data, not individual cases.

If you have any questions or concerns associated with this study and the experimental procedures please feel free to contact the researchers associated with the study:

Saskia de Leede-Smith	Emma Barkus
Faculty of Psychology	Faculty of Psychology
(02) 4221 4513	(02) 4221 8134
saskia@uow.edu.au	ebarkus@uow.edu.au

Alternatively, if you have any concerns or complaints regarding the way this research has been conducted, you can contact the University Ethics Officer, on (02) 4221 4457 or by email at rso-ethics@uow.edu.au

Thank you for your interest in this study.

CONSENT FORM

Unusual perceptions and the personality trait schizotypy

I have been given information about "*Unusual perceptions and the personality trait schizotypy"* and discussed the research project with Saskia de Leede-Smith who is conducting this research as part of a Doctor of Philosophy supervised by Emma Barkus in the department of Psychology at the University of Wollongong.

I have been advised of the potential risks and burdens associated with this research, which include the possibility of unpleasant memories and/or feelings being revived if myself or someone close to me has suffered a mental health issue, and have had an opportunity to ask Saskia any questions I may have about the research and my participation.

I understand that my participation in this research is voluntary, I am free to refuse to participate and I am free to withdraw from the research at any time. My refusal to participate or withdrawal of consent will not affect my treatment in any way or my relationship with the University of Wollongong.

If I have any enquiries about the research, I can contact Saskia (saskia@uow.edu.au) or Emma (ebarkus@uow.edu.au). If I have any concerns or complaints regarding the way the research is or has been conducted, I can contact the Ethics Officer, Human Research Ethics Committee, Office of Research, University of Wollongong on 4221 4457.

By ticking the boxes below I am indicating my consent to:

☐ Filling out a battery of forms in relation to my personality, behavior, perceptions and how I react emotionally in different scenarios.

☐ I understand that the data collected from my participation will be combined with other existing data and used for a thesis and publication in academic journals, and I consent for it to be used in that manner.

☐ I am happy to be contacted to take part in studies of a similar nature.

Signed: Date:

..../...../.....

Email to participants of stage 1 inviting further participation in stage 2 of research study (involving NSS and semantic processing task).

Hello!

You recently took part in an online study (Unusual perceptions and the personality trait schizotypy) through the Psychology research participation scheme. In your responses you indicated that you wouldn't mind being contacted for studies of a similar nature. We would like to invite you to take part in a related study that is involved in evaluating the different behavioural nuances that each person possesses. If you choose to participate we will ask you to complete a range of activities that people often find enjoyable! These will include: touching your finger to your nose with your eyes closed, hand co-ordination tasks, identifying objects purely through touch, and listening and repeating sound patterns. All of these tasks are formulated to measure differences in people's sensory integration, motor co-ordination, and sequencing of complex motor acts. It is expected that this will only take around 45 minutes of your time, and you will be compensated for this with the allocation of one credit point through the research participation scheme.

If you are interested in participating please log onto the Psychology Research Participation System and click on the study entitled 'Invited study: schizotypy'. In order to sign up for a timeslot you need to enter the access code for this study which is given below.

Access code: summer

Thank you for your time and hoping to see you in the near future,

Saskia de Leede-Smith

University of Wollongong 

Participation Information Sheet

Unusual perceptions and the personality trait schizotypy (stage 2)

This is an invitation for you to participate in the second stage of a study conducted by researchers at the University of Wollongong. The research is called "Unusual perceptions and the personality trait schizotypy". The purpose of the research is to investigate the factors associated with the experience of auditory perceptions in the general population. We will tell you a little about the factors we are interested in but you will be free to ask further questions of the researcher.

The personality trait we are interested in is called schizotypy. This sounds a little like a mental health disorder called schizophrenia but in fact the two are different. Much like other personality traits such as extraversion (how out going you are), schizotypy is normally distributed in the general population. This means most people score around the average but as many people score extremely high as extremely low on the personality trait. Scoring particularly high or low on schizotypy does not necessarily carry any negative connotations it is merely part of the interesting complexities which make up people's personalities! However if you do have any concerns please feel free to discuss them with us. We are interested in how schizotypal personality, emotional processing styles and behaviours may be related to the experience of different auditory and unusual perceptions. It is common for some people to hear perceptions such as voices, music, and other indistinguishable noises when they are not actually there. These perceptions can occur when taking part in day-to-day activities, and can also occur in the drowsy state experienced just before falling asleep, or right when you wake up. We are interested in the details and characteristics of these experiences in different people. If you have any questions regarding our research we will be happy to answer them. We will not be able to offer individual feedback on your responses to the questionnaires. However, we will be able to provide you with a summary of the findings of the study so if you are interested please let us know.

WHAT WE WOULD LIKE YOU TO DO

This stage of the research project is concerned with examining your responses to some behavioural tasks. These tasks assess your sensory integration, motor co-ordination, sequencing of complex tasks, and memory. It is expected that this section of the experiment will take between 30 and 40 minutes. Although this sounds like a long time it will pass quickly since you will be completing different tasks which

people quite often enjoy! Some of the tasks you will be asked to complete include: touching you finger to your nose with your eyes closed; a tandem walk; standing on one leg; illustrating your hand preference through holding different stationary products, and; remembering different words.

We do appreciate this may seem like a long time to be committing to taking part in a study. However people often find the process informative and you will be helping to forward research into personality and mental health. If you or someone close to you has experienced problems associated with mental health difficulties we will provide contact details for a range of health and support services which are available to assist you, including:

- *Lifeline: 13 11 14*
This is a 24-hour confidential support line which is able to provide individuals with both information and support, and if necessary refer you on to appropriate mental health networks.

- *Life Resolutions: 1300 3249 32*
This is a network of trained professional psychologists within Australia. They cater for a wide variety of mental health areas and concerns, and are located in a multitude of locations across Sydney and Australia-wide.

- *Northfields Clinic:* (02) 4221 3747
Based at the University of Wollongong, this clinic provides high quality psychological services at a heavily discounted rate. They offer a range of clinical assistance, ranging from initial assessments through to group therapy and highly specialized individualized sessions.

Your involvement in this study is voluntary and you may withdraw your participation from the study at any time and any data that has been gathered to that point will be withdrawn and destroyed. If you do choose to withdraw your consent your withdrawal will not have any adverse affects and will in no way affect your treatment, studies or relationship with the University of Wollongong.

The information collected from the study will be stored in a locked filing cabinet, and the data entered onto the computer will be stored in a password protected computer file. Both these procedures will guarantee that your information remains confidential. Findings from this study will be published in a thesis and possibly an academic journal. Your anonymity will be maintained by immediately separating the cover sheet (with possible identifying information) from your questionnaires. Confidentiality will also be preserved by assigning numbers rather than names to the written records, as well as only reporting on grouped data, not individual cases.

If you have any questions or concerns associated with this study and the experimental procedures please feel free to contact the researchers associated with the study:

Saskia de Leede-Smith	Emma Barkus
Faculty of Psychology	Faculty of Psychology
(02) 4221 4513	(02) 4221 8134
saskia@uow.edu.au	ebarkus@uow.edu.au

Alternatively, if you have any concerns or complaints regarding the way this research has been conducted, you can contact the University Ethics Officer, on (02) 4221 4457 or by email at rso-ethics@uow.edu.au

Thank you for your interest in this study.

CONSENT FORM

Unusual perceptions and the personality trait schizotypy (stage 2)

I have been given information about "*Unusual perceptions and the personality trait schizotypy"* and discussed the research project with Saskia de Leede-Smith who is conducting this research as part of a Doctor of Philosophy supervised by Emma Barkus in the department of Psychology at the University of Wollongong.

I have been advised of the potential risks and burdens associated with this research, which include the possibility of unpleasant memories and/or feelings being revived if myself or someone close to me has suffered a mental health issue, and have had an opportunity to ask Saskia any questions I may have about the research and my participation.

I understand that my participation in this research is voluntary, I am free to refuse to participate and I am free to withdraw from the research at any time. My refusal to participate or withdrawal of consent will not affect my treatment in any way or my relationship with the University of Wollongong.

If I have any enquiries about the research, I can contact Saskia (saskia@uow.edu.au) or Emma (ebarkus@uow.edu.au). If I have any concerns or complaints regarding the way the research is or has been conducted, I can contact the Ethics Officer, Human Research Ethics Committee, Office of Research, University of Wollongong on 4221 4457.

By ticking the boxes below I am indicating my consent to:

☐ Taking part in a variety of behavioural tasks, of which will assess my sensory integration, motor co-ordination, sequencing of complex motor acts, and memory.

☐ I understand that the data collected from my participation will be used for a thesis and publication in academic journals, and I consent for it to be used in that manner.

☐ I am happy to be contacted to take part in studies of a similar nature.

Signed: Date:

..../...../.....

University of Wollongong 

Participation Information Sheet – Dyslexia and factors associated with schizotypy

This is an invitation for you to participate in a study conducted by researchers at the University of Wollongong. The research is called "Dyslexia and factors associated with schizotypy". The purpose of the research is to investigate whether certain behaviours and emotional styles are associated with dyslexia. We will tell you a little about the factors we are interested in but you will be free to ask further questions of the researcher.

The personality trait we are interested in is called schizotypy. This sounds a little like a mental health disorder called schizophrenia but in fact the two are different. Much like other personality traits such as extraversion (how out going you are), schizotypy is normally distributed in the general population. This means most people score around the average but as many people score extremely high as extremely low on the personality trait. Scoring particularly high or low on schizotypy does not necessarily carry any negative connotations it is merely part of the interesting complexities which make up people's personalities! However if you do have any concerns please feel free to discuss them with us. We are interested in how certain personality, emotional processing styles and behaviours may be related to dyslexia. We will be happy to answer any questions you have. We will not be able to offer individual feedback on your responses to the questionnaires. However, we will be able to provide you with a summary of the findings of the study if you are interested please let us know.

WHAT WE WOULD LIKE YOU TO DO

In the first section of the study we ask that you complete a number of questionnaires relating to your personality, behaviour and how you react in different scenarios. By providing this type of information we are able to gain an understanding of the way you perceive situations, and this helps us in determining your personality traits and emotional appraisals. It is expected that this section should take 20-30 minutes of your time. Examples of some of Yes/No statements we will ask are:

- People sometimes find me aloof and distant.
- No matter how hard I try to concentrate unrelated thoughts always creep into my mind.
- Sometimes I suddenly feel scared for no good reason.
- I trick myself into believing something is okay when it's not.

The second section of the study is concerned with testing your reading ability and thinking processes. In this phase you will be asked to complete a number of different tasks, all of which will together assess your sensory integration, motor co-ordination, sequencing of complex tasks, memory, learning style, reading ability, and how well you are able to switch between tasks. It is expected that this section of the experiment takes between 1 ½ and 2 ½ hours. Although this sounds like a long time it will pass quickly since you will be completing different tasks which people often quite enjoy! Some of the tasks you will be asked to complete include: touching you finger to your nose with your eyes closed; a tandem walk; standing on one leg; illustrating your hand preference through holding different stationary products; reading different words and non-words out loud, and; naming words with similar meanings to some of the stimuli presented.

We do appreciate this may seem like a long time to be committing to taking part in a study. However people often find the process informative and you will be helping to forward research into personality and mental health. If you or someone close to you has experienced problems associated with mental health difficulties we will provide contact details for a range of health and support services which are available to assist you, including:

- *Lifeline: 13 11 14*
This is a 24-hour confidential support line which is able to provide individuals with both information and support, and if necessary refer you on to appropriate mental health networks.

- *Life Resolutions: 1300 3249 32*
This is a network of trained professional psychologists within Australia. They cater for a wide variety of mental health areas and concerns, and are located in a multitude of locations across Sydney and Australia-wide.

- Northfields Clinic: (02) 4221 3747
Based at the University of Wollongong, this clinic provides high quality psychological services at a heavily discounted rate. They offer a range of clinical assistance, ranging from initial assessments through to group therapy and highly specialized individualized sessions.

Your involvement in this study is voluntary and you may withdraw your participation from the study at any time and any data that has been gathered to that point will be withdrawn and destroyed. If you do choose to withdraw your consent your withdrawal will not have any adverse affects and will in no way affect your treatment, studies or relationship with the University of Wollongong.

The questionnaires collected from the study will be stored in a locked filing cabinet, and the neurocognitive data gathered will be stored in a password protected

computer file. Both these procedures will guarantee that your information remains confidential. Findings from this study will be published in a thesis and possibly an academic journal. Your anonymity will be maintained by immediately separating the cover sheet (with possible identifying information) from your questionnaires. Confidentiality will also be preserved by assigning numbers rather than names to the written records, as well as only reporting on grouped data, not individual cases.

If you have any questions or concerns associated with this study and the experimental procedures please feel free to contact the researchers associated with the study:

Saskia de Leede-Smith	Emma Barkus
Faculty of Psychology	Faculty of Psychology
(02) 4221 4513	(02) 4221 8134
saskia@uow.edu.au	ebarkus@uow.edu.au

Alternatively, if you have any concerns or complaints regarding the way this research has been conducted, you can contact the University Ethics Officer, on (02) 4221 4457 or by email at rso-ethics@uow.edu.au

Thank you for your interest in this study.

CONSENT FORM

Dyslexia and factors identified with schizotypy

I have been given information about "*Dyslexia and factors identified with schizotypy"* and discussed the research project with Saskia de Leede-Smith who is conducting this research as part of a Doctor of Philosophy supervised by Emma Barkus in the department of Psychology at the University of Wollongong.

I have been advised of the potential risks and burdens associated with this research, which include the possibility of unpleasant memories and/or feelings being revived if myself or someone close to me has suffered a mental health issue, and have had an opportunity to ask Saskia any questions I may have about the research and my participation.

I understand that my participation in this research is voluntary, I am free to refuse to participate and I am free to withdraw from the research at any time. My refusal to participate or withdrawal of consent will not affect my treatment in any way or my relationship with the University of Wollongong.

If I have any enquiries about the research, I can contact Saskia (saskia@uow.edu.au) or Emma (ebarkus@uow.edu.au). If I have any concerns or complaints regarding the way the research is or has been conducted, I can contact the Ethics Officer, Human Research Ethics Committee, Office of Research, University of Wollongong on 4221 4457.

By ticking the boxes below I am indicating my consent to:

☐ Filling out a battery of forms in relation to my personality, behaviour and how I react emotionally in different scenarios.

☐ Taking part in a series of neurocognitive tests which will assess my sensory integration, motor co-ordination, sequencing of complex tasks, memory, learning style, reading ability, and how well I am able to switch between tasks.

☐I understand that the data collected from my participation will be used for a thesis and publication in academic journals, and I consent for it to be used in that manner.

☐ I am happy to be contacted to take part in studies of a similar nature.

Signed: Date:

..../...../.....

13.2 Appendix B: General information sheet

General Information Sheet

The following questions are interested in a general overview of yourself. Please circle or answer questions as appropriate.

1. Sex: Male / Female

2. Age: _____

3. What is your current living arrangement?

At home with parents
With sibling(s) or other family/extended family member(s)
With an intimate partner (husband/wife/fiancée/boyfriend/girlfriend)
With friend(s)
With acquaintance(s)
Alone

4. Have you sought help from a medical practitioner or medical services within the past year?

Yes / No

5. If yes, do you receive regular care from a health/medical service provider?

Yes / No

6. If yes, please list what type of service you use and why (i.e. doctor for heart palpitations, psychologist for anxiety).

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7. Have you been diagnosed with a learning disability?

Yes / No

8. If yes, what was the diagnosis?

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9. Have you ever suffered from any neurological problem?

Yes / No

10. When you are in the drowsy state right before you fall asleep or upon waking, have you ever had any unusual perceptual experiences?

Yes / No

11. If yes, how often do these unusual perceptual experiences occur?

Very infrequently (once a year or less)
Infrequently (once every 6 months or more)
Sometimes (every 3 – 6 months)
Frequently (once a month)
Very frequently (more than once a month).

12. Do you/ have you ever had an imaginary friend?

Yes / No

13. If yes, over what ages was this friend in your life?_____

13.3 Appendix C: Schizotypal Personality Questionnaire (Raine, 1991)

Please answer each item by ticking (Yes) or the (No) following the question. There are no right or wrong answers, and no trick questions. Work quickly and do not think too long about the exact meaning of the questions.

		YES	NO
1	Do you sometimes feel that things you see on the TV or read in the newspaper have a special meaning for you?		
2	I sometimes avoid going to places where there will be many people because I will get anxious.		
3	Have you had experiences with the supernatural?		
4	Have you often mistaken objects or shadows for people, or noises for voices?		
5	Other people see me as slightly eccentric (odd).		
6	I have little interest in getting to know other people.		
7	People sometimes find it hard to understand what I am saying.		
8	People sometimes find me aloof and distant.		
9	I am sure I am being talked about behind my back.		
10	I am aware that people notice me when I go out for a meal or to see a film.		
11	I get very nervous when I have to make polite conversation.		
12	Do you believe in telepathy (mind-reading)?		
13	Have you ever had the sense that some person or force is around you, even though you cannot see anyone?		
14	People sometimes comment on my unusual mannerisms and habits		
15	I prefer to keep to myself.		
16	I sometimes jump quickly from one topic to another when speaking.		
17	I am poor at expressing my true feelings by the way I talk and look.		
18	Do you often feel that other people have got it in for you?		
19	Do some people drop hints about you or say things with a double meaning?		
20	Do you ever get nervous when someone is walking behind you?		
21	Are you sometimes sure that other people can tell what you are thinking?		
22	When you look at a person, or yourself in a mirror, have you ever seen the face change right before your eyes?		
23	Sometimes other people think that I am a little strange.		
24	I am mostly quiet when with other people.		
25	I sometimes forget what I am trying to say.		
26	I rarely laugh and smile.		
27	Do you sometimes get concerned that friends or co-workers are not really loyal or trustworthy?		
28	Have you ever noticed a common event or object that seemed to be a special sign for you?		
29	I get anxious when meeting people for the first time.		
30	Do you believe in clairvoyancy (psychic forces, fortune telling)?		
31	I often hear a voice speaking my thoughts aloud.		
32	Some people think that I am a very bizarre person.		

		YES	NO
33	I find it hard to be emotionally close to other people.		
34	I often ramble on too much when speaking.		
35	My "non-verbal" communication (smiling and nodding during a Y N conversation) is poor		
36	I feel I have to be on my guard even with friends.		
37	Do you sometimes see special meanings in advertisements, shop windows, or in the way things are arranged around you?		
38	Do you often feel nervous when you are in a group of unfamiliar people?		
39	Can other people feel your feelings when they are not there?		
40	Have you ever seen things invisible to other people?		
41	Do you feel that there is no-one you are really close to outside of your immediate family or people you can confide in or talk to about personal problems?		
42	Some people find me a bit vague and elusive during a conversation.		
43	I am poor at returning social courtesies and gestures.		
44	Do you often pick up hidden threats or put-downs from what people say or do?		
45	When shopping do you get the feeling that other people are taking notice of you?		
46	I feel very uncomfortable in social situations involving unfamiliar people.		
47	Have you had experiences with astrology, seeing the future, UFOs, ESP or a sixth sense?		
48	Do everyday things seem unusually large or small?		
49	Writing letters to friends is more trouble than it is worth.		
50	I sometimes use words in unusual ways.		
51	I tend to avoid eye contact when conversing with others.		
52	Have you found that it is best not to let other people know too much about you?		
53	When you see people talking to each other, do you often wonder if they are talking about you?		
54	I would feel very anxious if I had to give a speech in front of a large group of people.		
55	Have you ever felt that you are communicating with another person telepathically (by mind-reading)?		
56	Does your sense of smell sometimes become unusually strong?		
57	I tend to keep in the background on social occasions.		
58	Do you tend to wander off the topic when having a conversation?		
59	I often feel that others have it in for me.		
60	Do you sometimes feel that other people are watching you?		
61	Do you ever suddenly feel distracted by distant sounds that you are not normally aware of?		
62	I attach little importance to having close friends.		
63	Do you sometimes feel that people are talking about you?		
64	Are your thoughts sometimes so strong that you can almost hear them?		
65	Do you often have to keep an eye out to stop people from taking advantage of you?		
66	Do you feel that you are unable to get "close" to people?		

67	I am an odd, unusual person.		
68	I do not have an expressive and lively way of speaking.		
69	I find it hard to communicate clearly what I want to say to people.		
70	I have some eccentric (odd) habits.		
71	I feel very uneasy talking to people I do not know well.		
72	People occasionally comment that my conversation is confusing.		
73	I tend to keep my feelings to myself.		
74	People sometimes stare at me because of my odd appearance.		

PLEASE CHECK THAT YOU HAVE ANSWERED ALL OF THE QUESTIONS

13.3.1 Appendix D: Launay-Slade Hallucination Scale (Launay & Slade, 1981)

The questions below describe a number of experiences which you may have had. Some of these seem unusual however previous research has demonstrated that a significant number of people have had them. Please indicate either Yes or No as appropriate.

	Experience	
1. No matter how hard I try to concentrate unrelated thoughts always creep into my mind.	Yes	No
2. In my daydreams I can hear the sound of a tune almost as clearly as if I were actually listening to it.	Yes	No
3. Sometimes my thoughts seem as real as actual events in my life.	Yes	No
4. Sometimes a passing thought seems so real that it frightens me.	Yes	No
5. The sounds I hear in my daydreams are usually clear and distinct.	Yes	No
6. The people in my daydreams seem so true to life that sometimes I think they are.	Yes	No
7. I often hear a voice speaking my thoughts aloud.	Yes	No
8. In the past I have had the experience of hearing a person's voice when in fact no one was there.	Yes	No
9. On occasion I have seen a person's face in front of me when no one was in fact there.	Yes	No
10. I have heard the voice of the Devil.	Yes	No
11. In the past I have heard the voice of God speaking to me.	Yes	No
12. I have been troubled by hearing voices in my head.	Yes	No

13.4 Appendix E: Neurological Evaluation Scale (Buchanan & Heinrichs, 1989)

Neurological Evaluation Scale

1. Tandem Walk

Instructions: Subject to walk, in a straight line, 12 feet, heel to toe.

Assessment:

0 = no missteps after subject has completed first full step

I = one or two missteps after completion of first full step

2 = 3 or more missteps, grabbing, or falling.

2. Romberg Test

Instructions: Subject to stand with his/her feet together, eyes closed, his/her arms held parallel to the floor, and fingers spread apart. The subject is to maintain this position for 1 min.

Assessment:

0 =relatively stable, minimal swaying

1 =marked swaying

2 =subject steps to maintain balance or falls.

3. Adventitious Overflow

Instructions: Same as Romberg Test.

Assessment:

0 = absence of movement of fingers, hands, or arms

1 = irregular fluttering movement of fingers only

2 =irregular fluttering movement extended to hands and; or arms.

4. Tremor

Instructions: Same as Romberg Test.

Assessment:

0 = no tremor

1 = mild, fine tremor

2 = marked, fine or coarse tremor.

5 & 6. Cerebral Dominance

a. Handedness

Instructions: Ask subject to demonstrate how he/she would write, throw a ball, use a tennis racket, strike a match, use scissors, thread a needle, use a broom, use a shovel, deal cards, use a hammer, brush teeth, and unscrew the lid of a jar.

Assessment:

R-Subject writes with right hand and performs at least seven other activities with right hand

M-Subject writes with right/ left hand but performs less than seven other activities with right/left hand

L-Subject writes with left hand and performs at least seven other activities with left hand

b. Footedness

Instructions: Ask subject to demonstrate how he/she would kick a ball.

Assessment:

R-Subject kicks ball with right foot

L-Subject kicks ball with left foot.

c. Eyedness

Instructions: Ask subject, with both eyes open, to look at a distant object through a hole in the center of a 3-inch x 5-inch index card that is held with both hands 18 inches in front of the subject. The subject is to close one eye at a time and tell the examiner with which eye closed did he/she lose sight of the object.

Assessment:

R-Subject loses sight of object with right eye closed

L-Subject loses sight of object with left eye closed.

7. Audio-Visual Integration

Instructions: The subject is asked to match a set of tapping sounds with one of three sets of dots presented on a 5-inch x 7-inch index card. The subject is instructed to close his/her eyes during the tapping. Three practice trials are performed first to ensure that the subject under- stands the directions.

Assessment:

0 = no error

1 = one error

2 = two or more errors.

8. Stereognosis

Instructions: Subject, with eyes closed, is asked to identify an object placed in his/ her hand. Subject is instructed to feel the object with one hand and to take as much time as needed. If subject cannot name the object, he/ she is asked to describe for what purpose the object is used. The subject starts with the dominant hand, based on the prior evaluation of handedness, or the hand with which he/ she writes, if there is mixed hand dominance. The instructions are repeated at the beginning of the second trial.

Assessment:

0 = no errors

1 = one error

2 = more than one error.

9. Graphesthesia

Instructions: Subject, with eyes closed, is asked to identify the number written on the tip of his/her forefinger. The order of hands is determined as with stereognosis.

Assessment:

0 = no errors

1 = one error

2 = more than one error.

10. Fist-Ring Test

Instructions: The subject is asked to alternate placing his/her hand on the table, in the position of a fist, with the thumb placed either over the knuckles or over the middle phalanges and placing his/ her hand, on the table, in the position of a ring, with the tips of the thumb and forefinger touching and the remaining three fingers extended. The subject is to bring his/her arm into the upright position between each change in hand position. If the subject does not perform the movement accurately or in a manner that can be appropriately assessed, he/ she is to be stopped, to be

reinstructed, and to start the test again. The subject is to repeat each set of hand position changes 15 times.

Assessment:

0 = *no major disruption of motion after first repetition;* errors limited to incomplete extension of fingers in ring position and no more than two hesitancies in the transition from fist to ring or vice versa and no more than one fist/ ring confusion

1 =*no major disruption of motion after first repetition or complete breakdown of motion;* more than two hesitancies in the transition from fist to ring, difficulty in developing and maintaining a smooth, steady flow of movement, three to four fist/ ring confusions, or any total of three but not more than four errors.

2 =*major disruption of movement or complete breakdown of motion, or more than four fist/ ring hesitations or confusions.*

11. Fist-Edge-Palm Test

Instructions: Ask the subject, using a smooth and steady rhythmic pattern, to touch the table with the side of his*I* her fist, the edge of his*I* her hand, and the palm of his*I* her hand. The subject is to break contact with the surface of the table between each change in hand position, but not to bring the arm back in full flexion. The subject is to repeat this sequence of position changes 15 times.

Assessment:

0 =*no major disruption of motion after first repetition;* errors limited to no more than two hesitancies in the transition from one position to the next and no more than one mistake in hand position.

1 = *no major disruption of motion after first repetition or complete breakdown of motion;* more than two hesitancies in the transition from one position to another, difficulty in developing and maintaining a smooth, steady flow of movement, three to four position confusions, or any total of three or four errors.

2 = *major disruption of movement or complete breakdown of motion, or more than four hesitations or position confusions.*

12. Ozeretski Test

Instructions: The subject is to place both hands on the table, one hand palm down and the other hand in the shape of a fist. The subject is then asked simultaneously to alternate the position of his/her hands in a smooth and steady motion. The subject is asked to repeat this motion 15 times.

Assessment:

0 = *no major disruption of motion after first repetition;* errors limited to no more than two hesitancies in the transition from one position to the next and no more than one mistake in hand position.

1 =*no major disruption of motion after first repetition or complete breakdown of motion;* more than two hesitancies in the transition from one position to another, difficulty in developing and maintaining a smooth, steady flow of movement, three to four position confusions, or any total of three, but no more than four errors.

2 =*major disruption of movement or complete breakdown of motion, or more than four hesitations or position confusions.*

13. Memory

Instructions: Subject is told four words and is asked to repeat them immediately after they are all presented. If the subject is unable to repeat the four words correctly, they are represented. If the subject still cannot repeat the four words after a total of three presentations of the words, the test is terminated and the subject is given a score of 2 for both parts of the item. If the subject is able to repeat the four words after the initial or two subsequent presentations, he*1* she is then asked to remember the words as well as possible and told that he/ she will be asked to repeat the words twice later on during the interview. The subject is then asked to recall the four words at *5* and 10 min.

Assessment:

0 = Subject remembers all words

1 = Subject remembers three words

2 = Subject remembers fewer than three words.

14. Rhythm Tapping Test

Part A

Instructions: Ask the subject to reproduce exactly the series of taps heard while the subject has eyes closed. The subject may have eyes open while reproducing series of taps.

Assessment:

0 = no errors

1 = one error of either non-discrimination between soft and hard sounds, rhythm, or error in number of taps

2 = more than one error.

Part B

Instructions: Ask the subject to produce a series of taps as instructed.

Assessment:

0 = no errors

l = one error

2 = more than one error.

15. Rapid Alternating Movements

Instructions: Ask the subject to place his/ her hands palm down on legs. The subject is to start with his/ her dominant hand and is to slap his/ her leg distinctly with the palm and the back of his (her hand in an alternating motion. The determination of dominance is as described above (see item 8). The subject is to perform the task 20 times, with both hands, one hand at a time.

Assessment:

0 = no major disruption of motion, hesitation, or mistake in hand placement

1= no major disruption of motion or one to two hesitations or mistakes in hand placement

2 =major disruption of motion or three or more hesitations or mistakes in hand placement.

16. Finger-Thumb Opposition

Instructions: Ask the subject to place both hands palm up with fingers fully extended on his*I* her legs. The subject is to start with his/ her dominant hand and is to touch the tip of his/ her fingers with the tip of his/her thumb, from forefinger to pinky, returning to forefinger, for a total of I 0 repetitions.

Assessment:

0 =no major disruption of motion and no more than one mistake

1 =no major disruption of motion or two to three mistakes

2 =major disruption of motion or four or more mistakes.

17. Mirror Movements

Instructions: The subject's hand, which is not performing the Finger-Thumb Opposition

Test, is observed for parallel movements of the fingers and thumb.

Assessment:

0 = no observable movements of the fingers

1 = minor, inconsistent, or repetitive movements of the fingers

2 = consistent, distinctive movements of the fingers.

18. Extinction (Face-Hand Test)

Instructions: The subject is seated, with hands resting palm down, on his/ her knees and with eyes closed. The subject is told that he/ she will be touched on either the cheek, hand, or both, and is to say where he/she has been touched. If the subject names just one touch, he/she is asked-the first time this occurs only-if he/she felt a touch anywhere else. The simultaneous touching is done in the following order: right cheek-left hand, left cheek-right hand, right cheek-right hand, left cheek-left hand, both hands, and both cheeks.

Assessment:

0 = no errors

1 = one error

2 = more than one error.

19. Right/Left Confusion

Instructions: Subject is asked to point to his/her right foot, left hand; place his/ her right hand to left shoulder, left hand to right ear; point to examiner's left knee, right elbow; with examiner's arms crossed, point to examiner's left hand with his/ her right hand, and with examiner recrossing arms, point to examiner's right hand with his/ her left hand.

Assessment:

0 = no errors

1 = one error

2 = two or more errors.

20. Synkinesis

Instructions: Subject is instructed to follow the cap of a pen with his*1* her eyes only as it is moved between extremes of horizontal gaze. If the subject moves his/ her head, the subject is asked to keep his/ her head still and follow the cap of a pen with the eyes only.

Assessment:

0 = no movement of the head

1 = movement of the head on first trial but not when specifically told to keep head still

2 = movement of the head even when told to keep head still.

21. Convergence

Instructions: Subject is instructed to follow the cap of a pen with his/ her eyes as it is moved toward the subject's nose.

Assessment:

0 =both eyes converge on object

1 =one or both eyes are unable to converge completely, but can converge more than halfway

2 =one or both eyes fail to converge more than halfway.

22. Gaze lmpersistence

Instructions: Subject is instructed to fix his/her gaze on the cap of a pen at a 45 ° angle in the horizontal plane of the right and left visual fields for 30 sec.

Assessment:

0 = no deviation from fixation

1 = deviation from fixation after 20 sec

2 = deviation from fixation before 20 sec.

23. Finger to Nose Test

Instructions: The subject is instructed to close eyes and touch the tip of his/her nose with the tip of his/her index finger.

Assessment:

0 = no intention tremor or pass-pointing

1 = mild intention tremor or pass-pointing

2 = marked intention tremor or pass-pointing.

24. Glabellar Reflex

Instructions: Subject is instructed to fix his/her gaze on a point across the room. The subject is approached from above the forehead outside of the visual field, and the examiner taps the glabellar region 10 times with the index finger.

Assessment:

0 =three or fewer blinks

1 =four or five full blinks, or more than six partial or full blinks

2 = six or more full blinks.

25. Snout Reflex

Instructions: Subject is instructed to relax, and the examiner presses his finger against the subject's philtrum.

Assessment:

0 =no contraction of the orbicularis orris (or puckering of the lips)

2 =any contraction of the orbicularis orris (or puckering of the lips).

26. Grasp Reflex

Instructions: The subject is instructed not to grab, and the examiner strokes the inside of the subject's palm between the index finger and thumb. This procedure is repeated a second time with the subject being asked to spell the word "help" backwards.

Assessment:

0 = no flexion of the subject's fingers

I =mild flexion of the subject's fingers on first trial or flexion of any kind on second trial

2 =marked flexion of the subject's fingers on first trial.

27. Suck Reflex

Instructions: The examiner places the knuckle of a flexed index finger or tongue depressor between the subject's lips.

Assessment:

0 = no movement

2 = any pursing or sucking motion by the subject's lips.

13.5 Appendix F: Semantic Ambiguity Task word lists

Word list one

Prime	Target	Meaning	Relatedness
mass	church	subordinate	related
scale	climb	subordinate	related
fan	club	subordinate	related
palm	coconuts	subordinate	related
ball	dancing	subordinate	related
pitch	dark	subordinate	related
count	dracula	subordinate	related
tie	draw	subordinate	related
toast	drink	subordinate	related
trunk	elephant	subordinate	related
light	feather	subordinate	related
post	fence	subordinate	related
drill	fire	subordinate	related
perch	fish	subordinate	related
plain	flat	subordinate	related
fawn	flatter	subordinate	related
corn	foot	subordinate	related
stamp	foot	subordinate	related
foul	football	subordinate	related
mole	freckle	subordinate	related
match	game	subordinate	related
nursery	garden	subordinate	related
shower	gifts	subordinate	related
miss	girl	subordinate	related
box	gloves	subordinate	related
green	golf	subordinate	related
blow	hit	subordinate	related
stand	holder	subordinate	related

stage	horses	subordinate	related
horn	ivory	subordinate	related
log	journal	subordinate	related
bound	leap	subordinate	related
right	left	subordinate	related
bolt	lightning	subordinate	related
yard	metre	subordinate	related
cabinet	minister	subordinate	related
cast	play	dominant	related
calf	moo	dominant	related
racket	tennis	dominant	related
hound	dog	dominant	related
bug	insect	dominant	related
pen	pencil	dominant	related
court	jury	dominant	related
force	physics	dominant	related
train	travel	dominant	related
draw	paint	dominant	related
drop	fall	dominant	related
break	smash	dominant	related
gin	tonic	dominant	related
watch	time	dominant	related
pot	lid	dominant	related
field	grass	dominant	related
block	wood	dominant	related
file	papers	dominant	related
brush	comb	dominant	related
jam	berry	dominant	related
sight	eyes	dominant	related
wax	candle	dominant	related
race	colour	dominant	related
port	boat	dominant	related

sage	spice	dominant	related
foil	tin	dominant	related
pool	wet	dominant	related
march	april	dominant	related
straw	plastic	dominant	related
draft	cold	dominant	related
coast	ocean	dominant	related
magazine	articles	dominant	related
rich	money	dominant	related
coach	trainer	dominant	related
mate	friend	dominant	related
cricket	bat	dominant	related
mass	play	dominant	unrelated
scale	moo	dominant	unrelated
fan	tennis	dominant	unrelated
palm	dog	dominant	unrelated
ball	insect	dominant	unrelated
pitch	pencil	dominant	unrelated
count	jury	dominant	unrelated
tie	physics	dominant	unrelated
toast	travel	dominant	unrelated
trunk	paint	dominant	unrelated
light	fall	dominant	unrelated
post	smash	dominant	unrelated
drill	tonic	dominant	unrelated
perch	time	dominant	unrelated
plain	lid	dominant	unrelated
fawn	grass	dominant	unrelated
corn	wood	dominant	unrelated
stamp	papers	dominant	unrelated
foul	comb	dominant	unrelated
mole	berry	dominant	unrelated

match	eyes	dominant	unrelated
nursery	candle	dominant	unrelated
shower	colour	dominant	unrelated
miss	boat	dominant	unrelated
box	spice	dominant	unrelated
green	tin	dominant	unrelated
blow	wet	dominant	unrelated
stand	april	dominant	unrelated
stage	plastic	dominant	unrelated
horn	cold	dominant	unrelated
log	ocean	dominant	unrelated
bound	articles	dominant	unrelated
right	money	dominant	unrelated
bolt	trainer	dominant	unrelated
yard	friend	dominant	unrelated
cabinet	bat	dominant	unrelated
cast	church	subordinate	unrelated
calf	climb	subordinate	unrelated
racket	club	subordinate	unrelated
hound	coconuts	subordinate	unrelated
bug	dancing	subordinate	unrelated
pen	dark	subordinate	unrelated
court	dracula	subordinate	unrelated
force	draw	subordinate	unrelated
train	drink	subordinate	unrelated
draw	elephant	subordinate	unrelated
drop	feather	subordinate	unrelated
break	fence	subordinate	unrelated
gin	fire	subordinate	unrelated
watch	fish	subordinate	unrelated
pot	flat	subordinate	unrelated
field	flatter	subordinate	unrelated

block	foot	subordinate	unrelated
file	foot	subordinate	unrelated
brush	football	subordinate	unrelated
jam	freckle	subordinate	unrelated
sight	game	subordinate	unrelated
wax	garden	subordinate	unrelated
race	gifts	subordinate	unrelated
port	girl	subordinate	unrelated
sage	gloves	subordinate	unrelated
foil	golf	subordinate	unrelated
pool	hit	subordinate	unrelated
march	holder	subordinate	unrelated
straw	horses	subordinate	unrelated
draft	ivory	subordinate	unrelated
coast	journal	subordinate	unrelated
magazine	leap	subordinate	unrelated
rich	left	subordinate	unrelated
coach	lightning	subordinate	unrelated
mate	metre	subordinate	unrelated
cricket	minister	subordinate	unrelated

Word list two

Prime	Target	Meaning	Relatedness
mass	weight	dominant	related
scale	weigh	dominant	related
fan	air	dominant	related
palm	sweaty	dominant	related
ball	round	dominant	related
pitch	ball	dominant	related
count	number	dominant	related
tie	knot	dominant	related
toast	bread	dominant	related
trunk	roots	dominant	related
light	sun	dominant	related
post	letter	dominant	related
drill	bit	dominant	related
perch	bird	dominant	related
plain	simple	dominant	related
fawn	deer	dominant	related
corn	grain	dominant	related
stamp	letter	dominant	related
foul	smell	dominant	related
mole	tunnel	dominant	related
match	light	dominant	related
nursery	baby	dominant	related
shower	soap	dominant	related
miss	hit	dominant	related
box	square	dominant	related
green	grass	dominant	related
blow	air	dominant	related
stand	erect	dominant	related
stage	actors	dominant	related
horn	brass	dominant	related

log	brown	dominant	related
bound	tied	dominant	related
right	wrong	dominant	related
bolt	nut	dominant	related
yard	grass	dominant	related
cabinet	dishes	dominant	related
cast	mould	subordinate	related
calf	muscle	subordinate	related
racket	noise	subordinate	related
hound	pester	subordinate	related
bug	phone	subordinate	related
pen	pig	subordinate	related
court	players	subordinate	related
force	police	subordinate	related
train	practise	subordinate	related
draw	prize	subordinate	related
drop	rain	subordinate	related
break	rest	subordinate	related
gin	rummy	subordinate	related
watch	see	subordinate	related
pot	smoke	subordinate	related
field	study	subordinate	related
block	tackle	subordinate	related
file	tool	subordinate	related
brush	tooth	subordinate	related
jam	traffic	subordinate	related
sight	view	subordinate	related
wax	wane	subordinate	related
race	win	subordinate	related
port	wine	subordinate	related
sage	wise	subordinate	related
foil	again	subordinate	related

pool	balls	subordinate	related
march	band	subordinate	related
straw	barn	subordinate	related
draft	beer	subordinate	related
coast	bicycle	subordinate	related
magazine	bullets	subordinate	related
rich	cake	subordinate	related
coach	carriage	subordinate	related
mate	chess	subordinate	related
cricket	chirps	subordinate	related
mass	mould	subordinate	unrelated
scale	muscle	subordinate	unrelated
fan	noise	subordinate	unrelated
palm	pester	subordinate	unrelated
ball	phone	subordinate	unrelated
pitch	pig	subordinate	unrelated
count	players	subordinate	unrelated
tie	police	subordinate	unrelated
toast	practise	subordinate	unrelated
trunk	prize	subordinate	unrelated
light	rain	subordinate	unrelated
post	rest	subordinate	unrelated
drill	rummy	subordinate	unrelated
perch	see	subordinate	unrelated
plain	smoke	subordinate	unrelated
fawn	study	subordinate	unrelated
corn	tackle	subordinate	unrelated
stamp	tool	subordinate	unrelated
foul	tooth	subordinate	unrelated
mole	traffic	subordinate	unrelated
match	view	subordinate	unrelated
nursery	wane	subordinate	unrelated

shower	win	subordinate	unrelated
miss	wine	subordinate	unrelated
box	wise	subordinate	unrelated
green	again	subordinate	unrelated
blow	balls	subordinate	unrelated
stand	band	subordinate	unrelated
stage	barn	subordinate	unrelated
horn	beer	subordinate	unrelated
log	bicycle	subordinate	unrelated
bound	bullets	subordinate	unrelated
right	cake	subordinate	unrelated
bolt	carriage	subordinate	unrelated
yard	chess	subordinate	unrelated
cabinet	chirps	subordinate	unrelated
cast	weight	dominant	unrelated
calf	weigh	dominant	unrelated
racket	air	dominant	unrelated
hound	sweaty	dominant	unrelated
bug	round	dominant	unrelated
pen	ball	dominant	unrelated
court	number	dominant	unrelated
force	knot	dominant	unrelated
train	bread	dominant	unrelated
draw	roots	dominant	unrelated
drop	sun	dominant	unrelated
break	letter	dominant	unrelated
gin	bit	dominant	unrelated
watch	bird	dominant	unrelated
pot	simple	dominant	unrelated
field	deer	dominant	unrelated
block	grain	dominant	unrelated
file	letter	dominant	unrelated

brush	smell	dominant	unrelated
jam	tunnel	dominant	unrelated
sight	light	dominant	unrelated
wax	baby	dominant	unrelated
race	soap	dominant	unrelated
port	hit	dominant	unrelated
sage	square	dominant	unrelated
foil	grass	dominant	unrelated
pool	air	dominant	unrelated
march	erect	dominant	unrelated
straw	actors	dominant	unrelated
draft	brass	dominant	unrelated
coast	brown	dominant	unrelated
magazine	tied	dominant	unrelated
rich	wrong	dominant	unrelated
coach	nut	dominant	unrelated
mate	grass	dominant	unrelated
cricket	dishes	dominant	unrelated

13.6 Appendix G: General Health Questionnaire (Goldberg & Hillier, 1979)

The following questions are concerned with how your health has been in general, ***over the past few weeks***. Please answer all the questions on the following pages simply by circling the answer which you think most applies to you. Remember this is about present and recent complaints not those which you have had in the past.

Have you recently:

1. Been feeling well and in perfectly good health?	Better than usual	Same as usual	Worse than usual	Much worse than usual
2. Been feeling in need of a good tonic?	Not at all	No more than usual	Rather more than usual	Much more than usual
3. Been feeling run down and out of sorts?	Not at all	No more than usual	Rather more than usual	Much more than usual
4. Felt that you are ill?	Not at all	No more than usual	Rather more than usual	Much more than usual
5. Been getting any pains in your head?	Not at all	No more than usual	Rather more than usual	Much more than usual
6. Been getting a feeling of tightness or pressure in your head?	Not at all	No more than usual	Rather more than usual	Much more than usual
7. Have been having hot or cold spells?	Not at all	No more than usual	Rather more than usual	Much more than usual
8. Lost much sleep over worry?	Not at all	No more than usual	Rather more than usual	Much more than usual
9. Had difficulty in staying asleep once you are off?	Not at all	No more than usual	Rather more than usual	Much more than usual
10. Been managing to keep yourself busy and occupied?	More so than usual	Same as usual	Rather less than usual	Much less than usual

11. Been taking longer over things you do?	Quicker than usual	Same as usual	Longer than usual	Much longer than usual
12. Felt on the whole that you were doing things well?	Better than usual	About the same	Less well than usual	Much less well
13. Been satisfied with the way you've carried out your task?	More satisfied	About the same as usual	Less satisfied than usual	Much less satisfied than usual
14. Felt that you are playing a useful part in things?	More so than usual	Same as usual	Less useful than usual	Much less useful
15. Felt capable of making decisions about things?	More so than usual	Same as usual	Less so than usual	Much less capable
16. Felt constantly under strain?	Not at all	No more than usual	Rather more than usual	Much more than usual
17. Been able to enjoy your normal day-to-day activities?	More so than usual	Same as usual	Less so than usual	Much less than usual
18. Been getting edgy and bad tempered?	Not at all	No more than usual	Rather more than usual	Much more than usual
19. Been getting scared or panicky for no good reason?	Not at all	No more than usual	Rather more than usual	Much more than usual
20. Found everything getting on top of you?	Not at all	No more than usual	Rather more than usual	Much more than usual
21. Been thinking of yourself as a worthless person?	Not at all	No more than usual	Rather more than usual	Much more than usual
22. Felt that life is entirely hopeless?	Not at all	No more than usual	Rather more than usual	Much more than usual

23. Been feeling nervous and strung up all the time?	Not at all	No more than usual	Rather more than usual	Much more than usual
24. Felt that life isn't worth living?	Not at all	No more than usual	Rather more than usual	Much more than usual
25. Thought of the possibility that you might make away with yourself?	Definitely not	I don't think so	Has crossed my mind	Definitely have
26. Found at times that you couldn't do anything because your nerves were too bad?	Not at all	No more than usual	Rather more than usual	Much more than usual
27. Found yourself wishing you were dead and away from it all?	Not at all	No more than usual	Rather more than usual	Much more than usual
28. Found that the idea of taking your own life kept coming into your mind?	Definitely not	I don't think so	Has crossed my mind	Definitely has

13.7 Appendix H: National Adult Reading Test (Nelson, 1982)

Instructions for NART: These words are pronounced differently to how they read. Please speak each word out loud and clearly so that the experimenter can hear. Please try to pronounce each word as best you can even though some of the words may be quite difficult.

List of words

CHORD
ACHE
DEPOT
AISLE
BOUQUET
PSALM
CAPON
DENY
NAUSEA
DEBT
COURTEOUS
RAREFY
EQUIVOCAL
NAIVE
CATACOMB
GAOLED
THYME
HEIR
RADIX
ASSIGNATE
HIATUS
SUBTLE
PROCREATE
GIST
GOUGE

SUPERFLUOUS
SIMILE
BANAL
QUADRUPED
CELLIST
FACADE
ZEALOT
DRACHM
AEON
PLACEBO
ABSTEMIOUS
DETENTE
IDYLL
PUERPERAL
AVER
GAUCHE
TOPIARY
LEVIATHAN
BEATIFY
PRELATE
SIDEREAL
DEMESNE
SYNCOPE
LABILE
CAMPANILE

13.8 Appendix I: General Temperament Survey (Clark & Watson, 1990)

The original General Temperament Survey is made up of 3 dimensions-positive temperament, negative temperament, and disinhibition. This thesis only used the positive and negative temperament dimensions. Questions making up the disinhibition dimension were removed.

Listed below are a series of statements a person might use to describe his/her attitudes, feelings, interests, and other characteristics. Read each statement and decide how well it describes you. If the statement is TRUE or MOSTLY TRUE, fill in the circle in the first column (under the T) in front of that item. If it is FALSE or MOSTLY FALSE, fill in the circle in the second column (under the F). There are no right or wrong answers, and no trick questions.

Please answer every statement, even if you are not completely sure of your answer. Read each statement carefully, but don't spend too much time deciding on the answer.

T	F	
O	O	1. I am able to approach tasks in such a way that they become interesting or fun.
O	O	2. I sometimes rush from one activity to another without stopping to rest.
O	O	3. I often have strong feelings such as anxiety or anger without really knowing why.
O	O	4. I lead an active life.
O	O	5. I sometimes get too upset by minor setbacks.
O	O	6. My mood sometimes changes (for example, from happy to sad, or vice versa) without good reason.
O	O	7. Sometimes I feel "on edge" all day.
O	O	8. I lead a very interesting life.
O	O	9. I frequently find myself worrying about things.
O	O	10. My anger frequently gets the best of me.
O	O	11. I get excited when I think about the future.
O	O	12. People would describe me as a pretty enthusiastic person.
O	O	13. I can easily find ways to liven up a dull day.
O	O	14. Small annoyances often irritate me.
O	O	15. Sometimes I suddenly feel scared for no good reason.
O	O	16. In my life, interesting and exciting things happen every day.

T F

O O 17. I sometimes get all worked up as I think about things that happened during the day.
O O 18. Other people sometimes have trouble keeping up with the pace I set.
O O 19. I can get very upset when little things don't go my way.
O O 20. I live a very full life.
O O 21. I am often nervous for no reason.
O O 22. I often take my anger out on those around me.
O O 23. I am usually alert and attentive.
O O 24. I would describe myself as a tense person.
O O 25. I put a lot of energy into everything I do.
O O 26. I often worry about things I have done or said.
O O 27. I can make a game out of some things that others consider work.
O O 28. It takes a lot to get me excited.
O O 29. Sometimes life seems pretty confusing to me.
O O 30. I can work hard, and for a long time, without feeling tired.
O O 31. I am sometimes troubled by thoughts or ideas that I can't get out of my mind.
O O 32. My pace is usually quick and lively.
O O 33. I often have trouble sleeping because of my worries.
O O 34. Most days I have a lot of "pep" or vigor.
O O 35. I don't get very upset when things go wrong.
O O 36. People would describe me as a pretty energetic person.
O O 37. I often feel nervous and "stressed."
O O 38. I have days that I'm very irritable.
O O 39. In my life, I would rather try to do too much than too little.
O O 40. I get pretty excited when I'm starting a new project.
O O 41. Little things upset me too much.
O O 42. I am often troubled by guilt feelings.
O O 43. I seem to be able to remain calm in almost any situation.
O O 44. I worry about terrible things that might happen.
O O 45. I like to stir up some excitement when things are getting dull.
O O 46. I am often playful around other people.
O O 47. I worry too much about things that don't really matter.
O O 48. I am sometimes "on the go" so much that I wear myself out.
O O 49. Often life feels like a big struggle.
O O 50. I have more energy than most of the people I know.
O O 51. Things seem to bother me less than most other people.

O O 52. I sometimes feel angry for no good reason.

O O 53. I often feel lively and cheerful for no good reason.

O O 54. People sometimes tell me to slow down and "take it easy."

O O 55. I am usually enthusiastic about the things that I do.

13.9 Appendix J: Published manuscripts

de Leede-Smith, S. & Barkus, E. (2013). A comprehensive review of auditory verbal hallucinations: lifetime prevalence, correlates and mechanisms in healthy and clinical individuals. *Frontiers in Human Neuroscience, 7*, 1–25.

de Leede-Smith, S., Roodenrys, S., Horsley, L., Matrini, S., Mison, E., & Barkus, E. (2017). Neurological soft signs: Effects of trait schizotypy, psychological distress and auditory hallucination predisposition. *Schizophrenia Research: Cognition, 7,* 1-7.