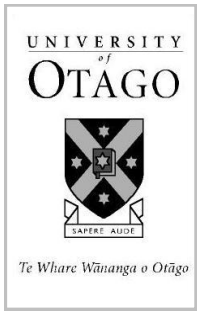


Visual Elements of Schizotypy Experiences: An Investigation of Representational Momentum and Eye-Tracking Risk Markers.

Dominic John Vettise

A thesis submitted for the degree of
Master of Science
at the University of Otago, Dunedin,
New Zealand.

2011

Abstract

Eye tracking dysfunction including smooth pursuit and voluntary eye movement are the most robust biological markers for risk of schizophrenia. Researchers suggested that eye tracking impairment may also involve higher-order functions such as errors in the prediction of an object's position, yet the relationship is unclear. Therefore, prediction of an object's position was tested through a unique phenomenon observed in schizophrenia and those at risk coined the representational momentum (RM) effect. The aim of the current study was to determine whether the prediction of an object's position is involved in eye movement anomalies and to what extent eye tracking and prediction is differently related to aspects of schizotypy. It was hypothesised that a) the eye tracking indices would be differently related to schizotypy subtypes, and b) the RM effect would significantly contribute to a model predicting risk for schizophrenia. One hundred and seventy-one participants were assessed on evidence-based eye tracking tasks that measured pursuit gain, the visual grasp reflex (VGR), and a RM task. These measures were combined to provide a model that could predict psychometric risk of schizotypy, using the Schizotypal Personality Questionnaire (SPQ). Each of the eye tracking measures were differently related to risk, but, together as a model they were not able to determine risk. More specifically, RM did not significantly contribute to the prediction of risk when it was added to a multiple regression model. Although the results were not consistent with all the current study's hypotheses, there were positive initiatives for the RM and eye tracking. It was concluded that the RM effect has the potential to improve the understanding of eye tracking dysfunction in schizophrenia. However, future research needs to be carried out to better understand the role of RM.

Acknowledgements

I have so many people I would like to acknowledge. Firstly, I would like to sincerely thank my supervisor Dr. Richard Linscott. Our countless meetings were always a pleasure, helping me to understand what anthropomorphism is, how to use the word solemnly correctly and humorous tips on wedding dress photos. Without a doubt you spent an exceptional amount of time reading my notes and developing my writing style - you taught me so much. Secondly, I would like to thank the input and time Dr. Liana Machado spent on my research. If it was not for your input the current project would have struggled to get off the ground. Thirdly, words cannot explain how appreciative I am of Jared's efforts. I can honestly say that the eye tracker would have never worked without you. Your dedication toward helping me was unrivalled and I am forever grateful. To Tobias, I appreciate your proof reading, compelling conversations and your sincere friendship throughout my project. To my family, Mum, Dad and Bianca; your kind words, reassurance and support throughout my thesis and also all five years have kept me motivated. Thank you for all the times you helped me to see the light at the end of the tunnel. Last but no means least- my wife Maria. Everyday you were there for me; every evening when I came home you greeted me with the most fantastic smile, which without doubt always made my day. You inspire me, your support was unprecedented and your love was always there- I cannot thank you enough. You had your own study, organised our wedding, did everything possible to lessen my workload and still found time to listen and give me advice about my own work. Your dedication to our relationship is an inspiration to me and thank you with all my heart.

Contents

List of Tables

List of Figures

List of Abbreviations

CCTCC	Cortico-cerebellar-thalamic cortical circuit
CNS	Central Nervous System
DLPFC	Dorsolateral prefrontal cortex
DSM-III-R	Diagnostic and Statistical Manual of Mental Disorders
PG	Pursuit Gain
RM	Representational Momentum
RMSE	Root Mean Squared Error
RT	Reaction Time
SI	Saccadic Intrusion
SPEM	Smooth Pursuit Eye Movement
SPQ	Schizotypal Personality Questionnaire
V5	Visual Area 5
VGR	Visual Grasp Reflex
WCST	Wisconsin Cart Sorting Task

Visual Elements of Schizotypy Experiences: An Investigation of Representational Momentum and Eye-Tracking Risk Markers.

We experience a world in which events occur, objects move and situations change throughout time. Our visual experience of this world appears integrated and coherent. However, it is known that there are minor systematic anomalies beneath this apparent coherence. These anomalies can be exploited to aid understanding of both normal and abnormal brain function.

A growing number of authors have proposed that lower-level perceptual anomalies such as poor smooth pursuit (the eye's tracking of a smoothly moving target), may be biological markers of risk for schizophrenia. Holzman (2000) suggested that 80% of patients with schizophrenia have abnormalities in eye movement, which can be identified through smooth pursuit and antisaccade tasks. The two visual processes disrupted in schizophrenia are the initiation and control of eye movement (Holzman, 2000). However, recent accounts have suggested the visual abnormalities associated with schizophrenia may also involve the prediction of an object's position over time (Lencer et al., 2010; Spering & Montagnini, 2010).

The prediction of an object's position has not been considered as a risk marker for schizophrenia or as part of a visual system that contributes to perceptual abnormalities. However, a phenomenon known as representational momentum (RM; Freyd & Finke, 1984) has been found to be among the anomalies associated with schizophrenia. RM is the term used to refer to a systematic misrepresentation in memory of an object's position in space. RM occurs when a stimulus, such as a still photograph of an object, includes attributes that imply motion of the object. RM is evidenced by memory performance that

suggests the memory representation for the object is moved forward in time along an implied trajectory. That is, RM can be described as a forward-in-time memory bias.

Jarrett, Phillips, Parker and Senior (2002) recently demonstrated exaggerated RM in patients with schizophrenia. A similar effect was also found in well individuals who expressed subclinical phenotypes of schizophrenia (high-risk for schizophrenia). Thus, patients with schizophrenia and high-risk individuals tend to extrapolate the motion of moving objects to a significantly greater extent than a normal control group (Jarrett et al., 2002).

The exaggerated motion functioning is fascinating because deficits in cognition are ubiquitous in schizophrenia. For example, disorganised thoughts, difficulty concentrating, lack of emotional expression and social functioning are common deficits suffered by patients with schizophrenia. Furthermore, evidence of exaggerated RM in schizotypy may add to the literature by providing a plausible reason as to why only some individuals demonstrate abnormalities in smooth pursuit and antisaccade eye movements.

Smooth pursuit eye movements (SPEM) are slow continuous eye movements that are used to track an object in motion with the individual's central vision in order to maintain a clear continuous perception of the object (Slaghuis, Hawkes, Holthouse & Bruno, 2007). Antisaccade eye movements demonstrate voluntary oculomotor movement by the suppression of a reflexive (saccadic) eye movement and by forcing the eye to voluntarily look in the opposite direction to that of a stimulus. SPEM and antisaccade eye movements are relevant because disruption of such mechanisms has been robustly demonstrated as a marker of risk for schizophrenia and schizotypy (Holzman, 2000). Researchers have further suggested that SPEM and antisaccades are both

associated with a common cognitive process such as inhibition. This in turn helps to control accuracy and speed of the eye's movement (Holzman, 2000). It may therefore be the case that people who suffer from schizophrenia demonstrate eye movement abnormalities because of the misrepresentation of an objects position, which according to Lencer et al. (2010) and Spering and Montagnini (2010) drives the movement of the eye.

Consequently, there were two primary objectives for the current study. The first of these was to identify eye movement manifestations that are related to the identification of schizotypal personality. The second objective was to investigate the contribution RM may make to eye tracking impairments demonstrated in schizotypal personality. The aim of the current study was to determine whether the prediction of an object's position is significant in influencing anomalies of eye movement associated with risk for schizophrenia. Importantly, in the current study, the SPEM task was used to measure tracking *accuracy*, the antisaccade task to measure *inhibition* and RM as a method to measure *prediction*.

I begin by describing schizophrenia and schizotypy and have identified several possible explanations for potentiated RM in schizophrenia. I then explore how RM may be related to characteristic impairments of eye tracking in schizophrenia.

Theories of Schizophrenia and Schizotypy

The exact nature and cause of schizophrenia remain highly debated. Schizophrenia was initially labelled as *dementia praecox* (Kraepelin, [1883], 1981). This label described the progressive and deteriorating nature of the illness and it implied that there is no return to pre-morbid levels of functioning (Bennett, 2006). Years later, Bleuler (1908) identified disturbance of mood and *thought* association, ambivalence and

preference for fantasy over reality as four core symptoms of schizophrenia. He also coined the term *schizophrenia*, which means *split mind* (Bleuler, 1908). The general consensus is that schizophrenia is characterised as a fundamental distortion of thinking and perception (Bennett, 2006). Both Kraeplin (1981) and Bleuler (1950) noted the existence of schizophrenia-like, but non-psychotic phenomenology in relatives of those with schizophrenia (Lenzenweger, 2006). This subclinical state was originally referred to as latent schizophrenia. Today it is often referred to as *schizotypy*, a contraction of *schizo*phrenia and pheno*type*.

Arguably one of the most interesting aspects in schizophrenia and schizotypy research is performance on tasks where those patients or individuals have demonstrated potentiated functions. Spitzer (1993) has suggested that if disinhibition is a candidate theory of schizophrenia, it should be possible to design tasks in which typically developing individuals show no potentiation, whilst schizophrenia patients demonstrate major potentiated effects. It was important to recognise theories of schizophrenia that relate to inhibition deficits. Therefore I identified three of the most relevant explanations to describe the observable occurrence of potentiated functioning in people who suffer from schizophrenia and schizotypy. These were Meehl's concept of hypokrisia and the schizotaxic brain, Andreasen's model of cognitive dysmetria and the theory of disinhibition. Through understanding the phenomenology behind schizophrenia and schizotypy, these concepts may also provide an understanding of the development of eye movement anomalies and exaggerated RM in schizotypy and patients with schizophrenia.

Hypokrisia and the schizotaxic brain. Meehl (1962, 1990) proposed that *hypokrisia*, "an insufficiency of separation, differentiation or discrimination in neural

transmission" (Lenzenweger, 2006, p. 2), is the cardinal feature of *schizotaxia*, a brain state Meehl regarded as the essential liability for schizophrenia (Lenzenweger, Maher & Manschreck, 2005). Meehl (1962, 1990) anticipated that a single gene (*schizogene*) influences the developing brain by coding for a specific functional anomaly of the synaptic control system in the central nervous system (CNS) (Lenzeweger, 2006). On the basis of this, Meehl (1962, 1990) characterised schizophrenia symptoms such as associative loosening and cognitive-affective aberrations as arising from *synaptic slippage*. Schizotaxia is defined as a "genetically determined integrative defect, predispositioned to schizophrenia which has a general population base rate of 10%" (Meehl, 1990, p. 35). That is, schizotaxia is not an observable behavioural pattern; it describes the anomalous brain functioning, involving synaptic slippage that leads to schizotypy and the liability for schizophrenia (Lenzenweger, Maher & Manschreck, 2005).

It has been proposed that hypokrisia may account for diverse effects in information processing and neurophysiological anomalies observed in schizotypal personality (Meehl, 1990). Linscott and Knight (2004) suggested that thought disorder may be explained by hypokrisia. Word stem completion tasks provide an insight into automatic memory in individuals. Linscott and Knight (2004) found that schizotypy was associated with potentiated automatic memory. The psychometrically identified schizotypy group remembered more old words than the control group. Linscott and Knight (2004) suggested that the automatic (unaware) influence of old words was greater in schizophrenia and schizotypy than in controls.

It is plausible that hypokrisia mediates neurological defects such as motor coordination, sensory integration and disinhibition (Chan & Gottesman, 2008). Furthermore, the immediate effect of hypokrisia is the comparable disorganisation of cognition and the disinhibition of automatic processes. Meehl's theory is therefore useful because it offers possible explanations of why there are some observable potentiated functions in schizophrenia and schizotypy such as automatic memory and RM. However, one disadvantage of Meehl's theory is that hypokrisia is difficult to directly test. Meehl (1990) suggested that hypokrisia (at a molecular level) is an anomaly in the synaptic control over the spiking of a neuron. Meehl (1990) also argued that the distribution of hypokrisia in the brain and the consequence of this are not equally dispersed across individuals and functions. Therefore, there may be individual differences in the way that hypokrisia affects the brain, and there are also differences in the way the same amount of hypokrisia may affect functions localised to different areas in the brain. To design an experiment that would be able to cater to this diversity makes Meehl's theory of hypokrisia challenging to test.

In sum, hypokrisia at the synaptic level characterises the schizotaxic brain. Meehl (1990) suggested that cognitive loosening associated with schizophrenia and schizotypy is a psychological process that is the result of hypokrisia. This may therefore explain the anomalies observed in the eye tracking of those at risk for schizophrenia.

Andreasen's model of cognitive dysmetria. The cognitive dysmetria model is an alternative explanation of the pathogenesis of schizophrenia. Andreasen (1996) proposed cognitive dysmetria to be the fundamental deficit in schizophrenia. Motor dysmetria refers to the deficiency of graceful coordination within the individual, which results in an

impairment of the ability to make movements exhibiting a rapid change of motion (Andreasen, Nopoulos, O'Leary, Miller, Wassink, & Flaum, 1999). Cognitive dysmetria is the cognitive or mental equivalent of motor dysmetria. Woods (1998) speculated that the aetiology and pathophysiology of schizophrenia is related to maturational and developmental brain sculpting, such as pruning or psychological experiences that affect brain plasticity.

For the cognitive system to act efficiently, the flow of information between neurons should be coordinated in a synchronised manner. However, for patients with schizophrenia, cognitive dysmetria causes a defect in the timing or sequential flow of information (Andreasen et al., 1999). Thus, Andreasen et al. (1999) argued that cognitive dysmetria causes the processing of cognitive systems (memory and attention) or the subsystems (working memory, encoding and inhibition) to be upset.

Andreasen et al. (1999) further explained that a feedback loop in the brain called the cortico-cerebellar-thalamic-cortical circuit (CCTCC), controls the flow of information between neurons. The CCTCC constantly checks, updates input and output functions and facilitates smooth execution of complex motor acts. Andreasen, Paradiso, and O'Leary (1998) hypothesised that CCTCC performs a similar function in monitoring and coordinating the execution of mental activity. A disruption in the loop leads to cognitive dysmetria and ultimately to the disordered cognition and clinical symptoms of schizophrenia (Andreasen et al., 1998, Andreasen, 1999).

Much like Meehl's theory of hypokrisia, Andreasen (1999) assumes that schizophrenia is caused by deficits in neurocogntive functioning. However, one shortcoming of cognitive dysmetria theory is that common antipsychotic drugs do not

always relieve such patients from neurocognitive symptoms such as memory and attentional deficits (Keefe, Silva, Perkins & Lieberman, 1999). Thus, according to Kaprinis, Konstantinos and Stergios (2008) if cognitive dysmetria is the fundamental deficit underlying schizophrenia, then medication should relieve neurocognitive symptoms. However, this is not the case and therefore researchers who argue against cognitive dysmetria have suggested there is more underlying schizophrenia than just cognitive dysmetria.

In sum, cognitive dysmetria has helped provide an explanation of cognitive deficits and possibly the diversity of symptoms observed in schizophrenia and schizotypy. There is evidence that the CCTCC is responsible for the rigid nature of mental activities in those with schizophrenia (Andreasen et al., 1998).

Disinhibition theory. Disinhibition in the current context is the inability to suppress irrelevant information. Disinhibition is the result of processes where there are limited or low cognitive resources. According to Andreasen (1979) the drain of cognitive resources comes from semantic intrusions constantly bombarding the brain with associations. In thinking about Andreasen's theory of cognitive dysmetria, a patient suffering with schizophrenia does not have the cognitive flexibility to deal with such a bombardment.

Disinhibition has been associated as an underlying factor involved in thought disorder in schizophrenia. An observable characteristic in schizophrenia patients is the dramatic change of semantic meaning whilst in conversation. This occurs because some words are more meaningful and have stronger charge than the original topic the patient is discussing (Beck & Rector, 2005). Patients with schizophrenia are unable to filter out or

inhibit the irrelevant words that result in disorganised speech. A comparable pattern can be observed in the antisaccadic eye movement literature. Patients with schizophrenia have trouble controlling unwanted reflexive eye movements toward a stimulus (Curtis, Calkins, Grove, Feil & Iacono, 2001). A number of researchers have suggested that patients with schizophrenia, their relatives and psychometrically identified schizotypes do not demonstrate the usual inhibitory responses (Curtis et al., 2001).

As mentioned above, if disinhibition is a candidate theory of schizophrenia, it should be possible to design tasks in which typically developing individuals show no potentiation, whilst schizophrenia patients demonstrate potentiated effects. To some extent, this has already been demonstrated in word stem tasks and automatic memory (Linscott & Knight, 2004). The literature regarding potentiated functioning in patients with schizophrenia could be made more robust by investigating performance of other inhibition tasks such as the antisaccade paradigm and RM. It is plausible that task performance could be explained by disinhibition processes. That is, not appropriately suppressing reflexive eye movements or cognitive processing of motion (respectively to the tasks mentioned above).

In summary, many researchers regard schizotypy as an expression of liability for schizophrenia. Despite being completing theories, hypokrisia, cognitive dysmetria and disinhibition are difficult to disentangle and differentiate, as they are very similar. However, all three theories are useful to understand because they propose mechanisms for increases in specific outcomes such as automatic memory and in particular, RM. Thus, the current study utilised these three closely related theories to help explain how RM is exaggerated and to what extent RM phenomena contributes to markers of risk.

Markers of Risk for Schizophrenia

There is considerable interest in studying candidate traits in populations at risk for schizophrenia but not necessarily expressing the full illness. Liabilities are demonstrated in first-degree relatives of people with schizophrenia (Tsuang, Stone, Tarbox & Faraone, 2002). The two most robust manifestations include brain abnormalities associated with perception (Seidman, Cassens, Kremen & Pepple, 1992) and eye tracking dysfunction (Levy, Holzman, Matthysse & Mendell, 1993). These are thought to be the most robust risk indicators for schizophrenia because they have demonstrated promising leads from studies of first-degree relatives (Tsuang et al., 2002).

Eye-Tracking Studies. Eye tracking deficits are one of the few widely validated behavioural markers of risk for schizophrenia. No study to date has failed to replicate the essential finding that eye tracking in schizophrenia patients is impaired in some way. Furthermore, several studies have demonstrated a relationship between cognitive control of motion and motion perception in schizophrenia (Chen et al., 1999; Stuve et al., 1997). Schizophrenia symptoms are linked with information processing impairments that underlie schizophrenia oculomotor impairments (Levy, Holzman, Matthysse, & Mendell, 1993). Patients diagnosed with schizophrenia have documented abnormalities in SPEM and antisaccadic performance (Levy et al., 1993). Disruption of SPEM occurs in 86% of individuals with schizophrenia and 50% of their first-degree relatives (Lipton, Levy, Holzman & Levin, 1983). It has been reported that 20-40% of schizophrenia patients perform well on eye tracking tasks; however, for this select group, their first-degree relatives do not (Matthysse, Holzman & Lange, 1986).

The relationship between identifying an individual at high risk for schizophrenia and poor performance on eye tracking tasks has been demonstrated through their performance on the SPEM task alone. Karoumi et al. (2001) suggested that patients with schizophrenia and their healthy biological siblings demonstrate eye movement deficits in both SPEM and antisaccade tasks. This has been further corroborated in other eye tracking studies (Matsue et al., 1994; Sereno & Holzman, 1995). Disinhibition of reflexive eye movements during smooth pursuit causes an increase in saccadic intrusions. Karoumi et al. (2001) suggested saccadic intrusions are the primary reason for poor SPEM performance in schizophrenia. Voluntary eye movements, termed antisaccades, demonstrate when there is an impairment of eye movement inhibition. There are several researchers that have provided evidence to suggest a link between schizophrenia, inhibitory oculomotor control and the antisaccade task (Clementz, McDowell and Zisook, 1994; Katsanis, Kortenkamp, Iacono and Grove, 1997).

A number of those researchers have identified the frontal lobe as being the most likely candidate for abnormal eye tracking (Holzman, 1987; Katsanis & Iacono, 1991). Specifically, frontal lobe activity is associated with smooth pursuit and antisaccade tasks, both of which require inhibition of eye movement to perform well (Levin, 1984). Holzman (2000) further elaborated that disturbance of cooperation between smooth pursuit and saccadic eye movement is the primary cause of the disrupted control of eye movements in schizophrenia, which is due to deficits of the frontal lobe.

Brain Abnormalities. Brain regions associated with eye tracking impairments is not limited to the frontal lobe. Many connecting areas have also been associated with the impairment. Several candidate brain regions have been reviewed because of the link they

have demonstrated with perceptual abnormalities in schizophrenia and at-risk populations. Researchers have linked the temporal lobe, visual area five (V5) in the parietal lobe and frontal lobe regions with eye tracking impairments.

The temporal lobe has received much attention from researchers investigating the control of eye behaviour and schizophrenia. In particular, reduction of superior temporal gyrus gray matter volume may help identify schizotypal personality disorder and patients with schizophrenia (Dickey, et al., 1999). Moreover, similar medial temporal lobe abnormalities may help differentiate which individuals will develop schizophrenia and also how severe the illness will be (Dickey et al., 1999; Dickey, McCarley & Shenton, 2002).

V5 is located in the posterior parietal cortex (dorsal stream) and is believed to play a major role in the perception of motion. David and Senior (2000) suggested that V5 controls implicit motion processing. Senior (2000) artificially lesioned area V5 through transcranial magnetic stimulation (TMS), which upset motion processing. However, V5 cannot be pinpointed specifically as having sole responsibility for the perception of motion. More recent accounts have suggested that it is likely that the motion perception deficits observed in schizophrenia only begin in area V5 (Jarrett et al., 2002; Kuperberg & Heckers, 2000). David and Senior (2000) suggested that motion processing interacts with other brain areas (such as frontal regions) that are responsible for higher cognitive roles.

Researchers have demonstrated that schizophrenia patients with eye tracking impairment were more likely to perform abnormally on neurocogntive tasks, such as the Wisconsin Card Sorting Test and Word Fluency tests, that assess frontal lobe activity

(Katsanis & Iacono, 1991; Sweeney et al., 1992). Jarrett et al. (2002) also suggested this in the first RM study that involved schizophrenia patients. This is further discussed in greater depth below. According to Park and Holzman (1992), patients with schizophrenia have a deficit in the representational processing of motion. Motion misrepresentation in schizophrenia and schizotypy was specifically related to the dorsolateral prefrontal cortex (DLPFC) - an area in the frontal lobe. For example, those who scored high on Schizotypal Personality Questionnaires (SPQ; Raine, 1991) were observed to have a subtle deficit in their DLPFC, which correlated with greater errors in spatial working memory (SWM) tasks (Park & McTigue, 1997).

There is now a large body of literature describing a robust relationship between eye tracking tasks (SPEM and antisaccade) and the frontal lobe. This literature highlights the importance of the frontal lobe's role in controlling eye behaviour and inhibiting unwanted movement. For example, Levin (1984) showed that tracking impairments are due to the frontal lobe connecting to other areas such as substantia nigra, the frontal eye fields and the superior colliculus (pertinent for eye vision). From the frontal eye fields GABA neurons (inhibitory) project onto the superior colliculus which is responsible for restraining (unwanted) saccadic movements. In animal studies that have utilised SPEM tasks, monkeys perform equally as well as humans. However, when a GABA antagonist was artificially added to monkeys' brains, they demonstrated the same performance as biological relatives of patients with schizophrenia on eye tracking tasks (Levin, 1984). This suggests that having a reduced or absent ability to inhibit unwanted saccades during the SPEM task may be related to a deficit of the frontal lobe (Friedman et al., 1992) and specifically to the frontal eye field mechanisms. Such mechanisms operate feedback

regulation of saccades and smooth pursuit during eye tracking tasks (Levin, 1984). Katsanis and Iacono (1991) suggested that patients with eye tracking impairment were also more likely to perform abnormally on the Wisconsin card sorting task (WCST) and word fluency tests that are predominantly used to assess frontal lobe activity.

Antisaccades are considered a measure of voluntary control of the eyes. Voluntary eye control needs to be carried out successfully to perform adequately on an SPEM task. The frontal eye fields (located in the frontal lobe) control performance on such tasks. Researchers have demonstrated the link among inhibitory control in specific areas of the frontal lobe, schizophrenia and the antisaccades (Clementz, McDowell & Zisook, 1994; Katsanis, Kortenkamp, Iacono & Grove, 1997; McDowell & Clementz, 1997)

In summary, findings from many studies suggest an association linking schizophrenia and schizotypy with eye behaviour control deficits. Results showed a robust link between SPEM, antisaccade tasks, schizophrenia and at-risk populations. The most prolific evidence for brain regions involved in the impairments of motion control is in area V5 and even more robustly, the frontal lobe.

Motion Control Studies in Schizotypy and Schizophrenia

Up to 80% of schizophrenia patients have abnormal eye tracking and motion control deficits (Holzman, 2000). Such deficits in schizophrenia patients and their at-risk relatives are related to smooth pursuit dysfunction (Stuve et al., 1997; Holzman, 2000). According to Levy et al. (1993) deficits of eye tracking can be explained by the disturbance in the smooth pursuit system, which is accompanied by unnecessary and increased saccadic movement – see Table 1 for eye movement terminology. Specifically, visual pursuit involves two processes: initiating the movement and being able to control

Table 1.
Definitions of Eye Movement Terminology

Eye terminology	Definition
Pursuit gain (PG)	Difference in target velocity versus eye velocity.
Root mean square error (RMSE)	Spatial discrepancy between target position and eye position.
Saccade	Rapid, jerky, reflexive eye movement.
Catch-up saccade (CS)	A saccadic intrusion that occurs when the eye falls behind the pursuit of a target.
Anticipatory saccade (AS)	Unwanted saccadic eye movement that occurs before the presentation of a stimulus.
Visual grasp reflex (VGR)	Unwanted saccadic eye movement that occurs at the time of stimulus presentation.

that movement. If one of these processes is compromised, visual pursuit becomes deviant (Holzman, 2000).

Smooth pursuit eye tracking studies. SPEM is the most robust eye tracking dysfunction associated with individuals with schizophrenia (Avila, Hong, Moates, Turano & Thaker, 2006; Holzman, 2000; Lipton et al., 1983; O'Driscoll & Callahan, 2008; Slaghuis et al., 2007). SPEM dysfunction has also been observed in their at-risk relatives (Holzman, 2000; Holahan & O'Driscoll, 2005; Lahuis, Van Engeland, Cahn, & Kemner, 2008; Levy et al., 2004; Lipton et al., 1983; O'Driscoll, Lenzenweger & Holzman, 1998).

The poor performance associated with SPEM is due to greater lag between the velocity of the eyes and the velocity of the moving target (Slaghuis et al., 2007). A number of researchers have measured what is coined *pursuit gain* in SPEM tasks. Pursuit gain is the ratio of the velocity of the eye to the velocity of the target. A pursuit gain score of 1.0 indicates a perfect match of eye and target velocity. When the eye's velocity

is slower than the target's velocity, the pursuit gain value becomes lower than 1.0. Thus, the less the eye is able to keep up with the target, the lower the value of pursuit gain. When this occurs, it is suggested that the saccadic rate has increased to compensate for the slowness of smooth eye movement (Levy et al., 1993; O'Driscoll & Callahan, 2008). Such saccades have been termed intrusion or catch-up saccades. Slaghuis et al. (2007) suggested that patients who suffer from schizophrenia experience a significant increase of intrusion saccades compared to typically developing individuals. Therefore, for patients suffering from schizophrenia the saccadic intrusions fracture and disorganise the perception of the target's motion, which prevents their ability to keep up with the moving target (Slaghuis et al., 2007). What is interesting is that schizophrenia patients display normal saccadic eye movements to peripherally presented visual cues (Nieuwenhuis, Broerse, Nielen & Jong, 2004). This may imply that the basic saccadic generation circuitry is intact, but that the ability to control intrusive saccades is functioning abnormally. According to Ettinger et al. (2003) the most reliable way to test intrusive saccadic suppression is through an antisaccadic paradigm.

Antisaccade studies. Antisaccadic tasks provide a non-invasive yet accessible means of investigating psychomotor functioning as well as higher order cognitive processes (Gooding & Basso, 2008). They demonstrate voluntary oculomotor movement by the suppression of a reflexive saccadic movement and by forcing the eye to voluntarily look in the opposite direction to that of a stimulus.

Antisaccade deficits in patients with schizophrenia are the most consistent findings in the saccadic literature (Hutton, Joyce, Barnes & Kennard, 2002). Researchers interested in antisaccadic eye movements in schizophrenia have reliably corroborated

evidence that eye-tracking deficits occur in schizophrenia (Gooding & Basso, 2008; Nkam et al., 2001; Ploner, Gaymard, Rivaud-Pechoux, Pierrot- Deseilligny, 2005; Ross, Heinlein, Zerbe & Radant, 2005). Antisaccade abnormalities are so robust that they are now considered as a marker of risk for schizophrenia (Levy et al., 2004). Impairments in the antisaccade task has been observed in first episode schizophrenia (Broerse, Crawford & den Boer, 2002), chronic schizophrenia (Boudet et al., 2005) and remitted schizophrenia (Curtis et al., 2001). Not only has antisaccade deficits been observed exclusively in the above populations, but regardless of what antisaccade paradigm is used, schizophrenia patients consistently produce fewer correct responses on a antisaccade task.

Nevertheless, it is important to be cautious of results presented in antisaccade studies. Most researchers have reported on the number of correct trials within the antisaccade task. However, this does not always suggest that inhibition alone is responsible for errors made. The errors could be attributed to blinks, head movement, anticipatory saccades and the subject's eye not being at a central fixation point at the start of a trial. For some researchers, all of these constitute a mis-administration error in the task. It is therefore essential to look at the visual grasp reflex (VGR. The VGR is a movement error in the antisaccade task. It indicates that the subject moved his or her eyes reflexively in the direction of the peripheral target opposed to the empty box, which they were instructed to look at (Machado & Rafal, 2004). When this occurs, it suggests that the higher-order inhibitory processes that were meant to override the lower-order reflexive movement were not functioning properly. The VGR is what should be investigated in antisaccade studies because it correctly demonstrates the inhibitory

processes. Researchers of schizotypy and antisaccadic eye behaviours have investigated the issue of whether VGR deficits precede the manifestation of schizophrenia. By using a standard version of an antisaccade task, nearly all schizotypes have demonstrated VGR deficits (Holahan & O'Driscoll, 2005).

For both antisaccade and smooth pursuit eye tracking tasks, the exact relationship between eye tracking performance and schizotypy symptoms is less clear. It is ambiguous whether positive aspects of schizotypy (Ettinger et al., 2005; Gooding, 1999; Holahan & O'Driscoll, 2005) or certain traits of negative schizotypy (social anhedonia-Gooding, 1999) are more strongly correlated with elevated rates of the VGR on antisaccade tasks or pursuit performance on the smooth pursuit task. The ambiguity surrounding the relationship between symptomology and performance on eye tracking tasks has motivated a heterogeneity debate.

Evidence for Eye Tracking Heterogeneity in Schizotypy and Schizophrenia

Eye tracking deficits have been replicated in well individuals who expressed subclinical phenotypes of schizophrenia. However, the relationship between such populations and eye tracking deficits are more variable compared to the identification of patients with schizophrenia. This means that eye tracking deficits can reliably distinguish schizophrenia patients from healthy individuals, however, eye tracking deficits do not always distinguish between those at-risk for schizophrenia from healthy individuals when screening a general population. The reason for the variability is not clear. Tsuang and Faraone (1995) have suggested that a heterogeneous hypothesis opposed to a homogeneous hypothesis provides the most valid explanation of the variability. Researchers that support a heterogeneity hypothesis suggest that schizophrenia can be

separated into subgroups (positive, negative and disorganised). In addition, these subgroups have different aetiologies and therefore different phenotypic expressions. These expressions are cognitive-perceptual, interpersonal and disorganised abnormalities (respective to the subgroups above). Therefore, in the general population the magnitude of eye tracking anomalies are also expressed differently between the subtypes. Because the differences in symptoms in those at risk, taken from the general population is so subtle, the anomalies that are usually able to identify risk are also subtle and difficult to distinguish. Therefore, researchers find it difficult to identify individuals as being high-risk for schizophrenia if they measure schizotypy as a homogenous risk entity.

Evidence for an etiological model of heterogeneity in at-risk populations has been robustly reported in eye tracking studies. Holahan and O'Driscoll (2005) demonstrated that antisaccade deficits are better at identifying high risk subjects with positive symptoms. In addition, researchers who looked at smooth pursuit deficits were able to identify both positive and negative symptoms. Moreover, Holahan and O'Driscoll (2005) suggested that smooth pursuit deficits were greater in those who experienced more negative symptoms. Siever et al. (1990, 1994) investigated the negative symptoms individually and demonstrated that poor smooth pursuit was associated with those who experienced more social introversion, greater anhedonia and had difficulty with interpersonal relationships. Smyris et al. (2007) demonstrated that individuals who scored high on the SPQ disorganised factor and SPQ cognitive-perceptual (positive symptoms) had a significantly lower pursuit gain than other groups and made more spatial errors on the smooth pursuit task. Both Holahan and O'Driscoll (2005) and Smyris et al. (2007) demonstrated that groups with high scores indicating largely positive

symptoms of schizotypy presented a combination of antisaccade and smooth pursuit deficits. The evidence from these studies suggest that a phenotypic group that shares positive like experiences and eye movement anomalies in both antisaccade and smooth pursuit are distinct from the general population of healthy individuals. Other researchers have demonstrated that although the three psychometrically identified subgroups perform significantly worse on eye tracking tasks, the positive and negative subtypes did not differ in smooth pursuit performance (Gooding, Miller & Kwapil, 2000; Simons and Katkins (1985). Furthermore, both Gooding, Miller and Kwapil (2000) and Simons and Katkins (1985) reported that the disorganised subtype performed worse on the smooth pursuit task than positive and negative subtype groups.

In summary, there is clear evidence of a distribution amongst schizophrenia and schizotypal subtypes on psychophysiological measures, like smooth pursuit and antisaccade eye movements (Clementz, Grove, lacono, & Sweeney, 1992). Most researchers have suggested that schizotypy and schizophrenia cannot be safely regarded as a homogeneous uniform risk entity. Instead, it can be expected that identified subgroups of schizophrenia and schizotypy have heterogeneous relationships with variables of interest such as eye tracking anomalies. Although the evidence base is large in this area, the problem (as described above) is that the evidence is mixed as to which subtypes more commonly express certain eye tracking anomalies. This presents a new challenge for researchers as to what extent they are able to reliably identify those at risk in the general population using eye tracking measures.

There are currently two presenting problems. Firstly, researchers have not investigated the extent to which eye tracking measures reliably identify subtypes of

schizotypy. Secondly, previous researchers may have limited their studies by only considering two processes that may account for eye tracking anomalies; the initiation of the eye movement and then maintaining that movement smoothly.

It is possible that individuals with schizophrenia and those at risk are not only having problems at a perceptual level, but that the problem is mediated by the functioning of a higher-order cognitive level such as prediction of a target's movement (Barnes, 2008; Lencer et al., 2010; Spering & Montagnini, 2010). This would help to explain if the two processes thought to account for poor eye tracking performance is mediated by the prediction of stimulus movement. The cognitive representation of motion is a better way of thinking about the problem because we may be able to determine the role prediction plays in eye tracking.

Representation of Motion

Representation of motion plays an important organising role in the mind. The portrayal of implicit motion is important to the arrangement and structure of cognition (Freyd, 1983). In an attempt to understand principles of cognition, many authors have hypothesised ways to test the representation of motion.

One of the first paradigms used to test the representation of motion involved two images that imply a path of motion- this is called the *freeze-frame task*. This task is depicted in Figure 1. In the freeze frame task, a photo is presented briefly. This then disappears and, after a short interval, a second photo is presented. These photos can be placed in any order (backward or forward in time), or the same photo could be shown twice. Participants must decide whether the second (last) photo was *identical* or *different*

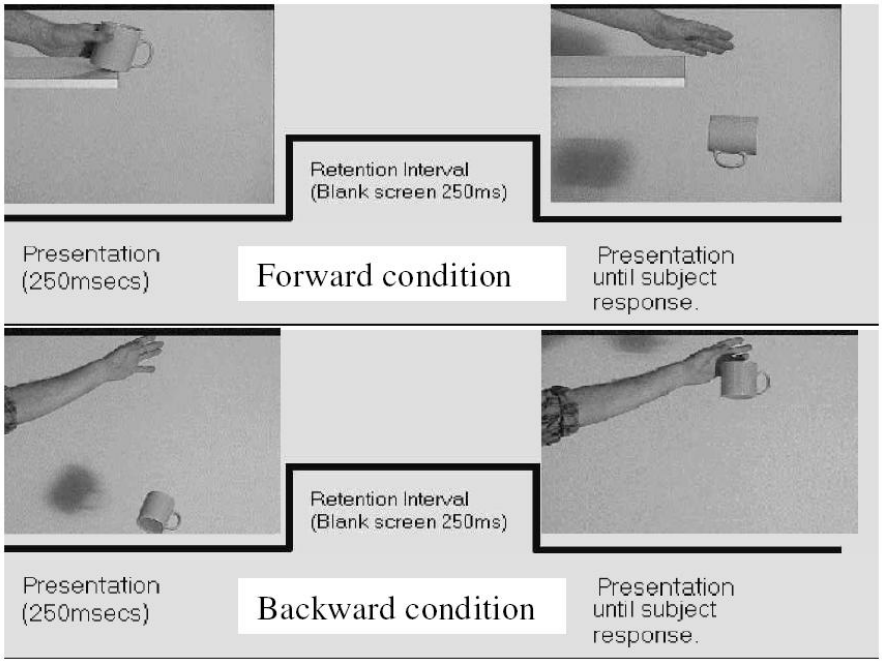


Figure 1. Procedure of the freeze-frame task commonly used to judge the representation of momentum. Extracted from Jarrett et al. (2002).

to the first photo. Subjects take more time to indicate correctly that the second image was different from the first, when the pair are in forward, temporal order, than when the images are presented backward (Freyd, 1983). Thus, Freyd (1983) concluded that it is more difficult to reject the distracter photo in the forward condition than in the backward condition. Therefore, people are able to cognitively represent implicit motion from a frozen-action scene (Freyd, 1983). More dynamic representations have been tested. One involved three temporally separated images being displayed one after the other before indicating whether the fourth, (often called the *probe*) was different or identical to the third image (Freyd & Finke, 1984). When the forth image was not as far along the trajectory plane, this was more difficult to judge in the direction of implicit motion than when it was in the opposite (backward) direction (Freyd & Finke, 1984). They suggested

that the perception of the third image's orientation was distorted in the direction of the motion due to the mental extrapolation of the object's implied trajectory, known as representational momentum (Freyd & Finke, 1984).

Representational momentum. RM is attributable to an automatic cognitive process (Jarrett et al., 2002). The term RM implies that the representation automatically acquires a momentum, analogous to the momentum in the associated object. This may be a useful metaphor (Thornton & Hubbard, 2002), although it appears that expectation plays a key role in determining the size of RM. Forward bias has been found to be greater when there are greater implied acceleration (Finke, Freyd & Shyi, 1986), greater implied velocity (Freyd & Finke, 1985), greater implied weight (Hubbard, 1997), lower implied friction (Hubbard, 1997), and more salient landmarks (Hubbard & Ruppel, 1999). RM also increases with the length of the retention interval (Freyd & Johnson, 1987). However, forward bias is also dependent on semantic knowledge about the object (Freyd & Miller, 1992; Reed & Vinson, 1996). For example, although similarly shaped, stimuli involving rockets and church steeples do not produce equivalent forward bias. The size of RM is largely dependent on the familiarity with an object's trajectory or behaviour. When the trajectory of an object is made ambiguous, this can result in a reduced or even absent forward bias (Kerzel, 2000). This bias is the result of the semantic knowledge learned or otherwise experienced about an object. Thus, evidence from RM studies has demonstrated that several factors can increase or decrease the size of RM.

Few authors have focussed on the role of divided attention in RM. Hayes and Freyd (1995) compared forward bias in two conditions. First, two objects were presented, either of which could be probed. The second condition contained only one

object. In the two-object condition, two small dots moved in a motion sequence (one vertical, the other horizontal). The task was to remember the final position the two dots were in as it was not certain which dot would be probed. A probe dot was presented in the final position as one of the other dots, either forward or backward from this final position, along the implied path of motion (Hayes & Freyd, 1995). The two-object condition produced significantly larger forward memory shifts than the single object condition (Hayes & Freyd, 1995). The divided attention makes the task more difficult for subjects to halt an automatic extrapolating process (Jarrett et al., 2002). Therefore, when less attention was paid to an object, the forward memory bias associated with the implied dynamics exaggerates RM (Finke & Freyd, 1985; Hayes & Freyd, 1995). These results suggested that RM is an automatic process and that in order to be successful in the task, the process must be consciously inhibited.

More recently, RM has been investigated in children born pre- and full-term. Taylor and Jakobson (2010) found that children born pre-term demonstrated reduced RM in comparison to full-term babies. This may be due to exaggerated inhibition causing the representational motion of an object to be halted sooner than usual (Taylor & Jakobson, 2010). These results were somewhat strange because researchers suggest that preterm babies' frontal lobe is not as developed as full term babies (Edgin et al., 2008). The frontal lobe is responsible for many processes. One example is, the more developed the frontal lobe, the less difficulties an individual has with inhibition. Inhibition is needed to halt the automatic memory process RM necessitates (Hayes and Freyd, 1995). These results are interesting because they suggest that even though pre-term babies' frontal lobes are not as fully developed as the full term babies, they display reduced RM. This

somewhat contradicts what has been observed in schizophrenia and schizotypy RM studies. As discussed in sections above, the frontal lobe in those who suffer from schizophrenia is not functioning effectively. However, Jarrett et al. (2002) observed a potentiated RM that they suggested is due to dysfunctional inhibition processes in the frontal lobe. To help clarify whether potentiated RM is specific to schizophrenia it would have been useful for researchers to have investigated RM in other impaired populations. However, this has not yet been investigated outside of schizophrenia literature.

Schizophrenia and schizotypy representational momentum studies. It was previously made clear that schizophrenia patients and healthy schizotypal individuals demonstrate motion perception deficits. RM is also a valid measure of how motion can be processed and perceived (Jarrett et al., 2002). There have been very limited studies involving RM in schizophrenia and schizotypy. Two studies have addressed the RM effect in schizophrenia (Jarrett et al., 2002) and schizotypy (Jarrett et al., 2002; Watkins, 2005). Jarrett et al. (2002) hypothesised a reduced or absent RM effect in schizotypal individuals, based on the knowledge that patients with schizophrenia show motion control deficits indicative of abnormal functioning in area V5.

In the study conducted by Jarrett et al. (2002), subjects were 50 healthy individuals and seven patients diagnosed with schizophrenia. They were tested on the freeze-frame task. The 50 healthy participants were divided into high or low schizotypy groups based on their scores on the Schizotypal Personality Questionnaire (SPQ; Raine, 1991). The study yielded contradictory results to the ones predicted. There was a potentiated RM effect in the high schizotypy group, as well as in the schizophrenia group

(Jarrett et al., 2002). Watkins (2005) has since reproduced similar results in schizotypal individuals.

Jarrett et al.'s (2002) findings are fascinating because they suggested it is the frontal lobe that is amplifying a stimulus's position forward in time, in those who are at risk for schizophrenia. Both Hayes and Freyd's (1995) RM divided attention study and Jarrett et al.'s (2002) study provide evidence that RM is an automatic process and must be halted in order to be successful in the task. To elaborate, although area V5 may be responsible for motion perception, the frontal areas account for inhibiting the automatic extrapolation process. This is consistent with antisaccadic and WCST literature, which has demonstrated that patients with schizophrenia and their at-risk relatives perform poorly on higher cognitive functioning tasks that require inhibition processing. The failure to inhibit an automatic process corresponds with several cognitive accounts of schizophrenia as well. For example, thought disorder is the failure to suppress alternative meanings of words (Frith, 1981) and perceptions are biased by contextual information (David, 1994). Jarrett et al. (2002) recognised that working memory impairments in schizophrenia may play a role in the exaggerated RM effect. In the study by Watkins (2005), spatial working memory was tested and demonstrated alongside RM. Consistent with previous literature, this was impaired in healthy schizotypal individuals although little justification was given for how SWM associated with the RM effect.

Jarrett et al. (2002) considered the neuroanatomical evidence to account for their findings. They discussed the fact that a number of studies have consistently demonstrated the role of the frontal lobe in RM through magnetoencephalography (MEG) (Amorim et al., 2000), and fMRI (Curtis et al., 1999). Furthermore, Taylor and Jakobson

(2010) suggested that the reduced RM effect in pre-term children might be due to an over-activation of the inhibition network located in the prefrontal cortex. This lead to the proposition that future studies should make use of integrated eye tracking technology, which may help explain whether the problems with smooth pursuit or saccadic inhibition play a role in the anomalies observed in RM (Taylor & Jakobson, 2010).

In sum, although it was originally suggested that V5 was of critical importance in motion perception, the frontal lobe may also be involved. This became apparent through the contradiction of Jarrett et al.'s (2002) hypothesis of a reduced or absent RM effect in patients with schizophrenia: the results demonstrated an *exaggerated* RM effect in schizophrenia and schizotypy (Jarrett et al., 2002; Watkins, 2005). Success in the freeze frame task requires that extrapolation of an object's position be inhibited along the implied trajectory plane. RM is an automatic process (Hayes & Freyd, 1995), and the evidence produced by researchers has lead to one plausible explanation for the forward memory bias observed in RM. It has been suggested that a frontal lobe anomaly such as disinhibition may facilitate schizophrenia patients and schizotypal individuals to exaggerate the RM of an object. In order to further delineate the underlying principles of potentiated functions in schizophrenia it would appear useful to demonstrate and understand the extent to how RM contributes to eye tracking predictors of schizotypy.

The Current Study

Where it is widely held that eye tracking is the most robust biological marker of risk for schizophrenia and schizotypy, the evidence suggests that there is still a considerable need to understand the processes involved in smooth pursuit. The next step for researchers is to understand the extent to which different aspects of schizophrenia and

schizotypy may be differently related to eye tracking measures. Schizotypal subgroups may have different aetiologies and therefore different phenotypic expressions. In the general population some eye tracking anomalies may only be expressed in a certain subtypes rather than being expressed as a uniform entity. Researchers have suggested that schizotypy cannot be safely regarded as a homogeneous uniform risk entity. Therefore, identifying the abnormal processes involved in schizophrenia might facilitate the exploitation of models that lead to understanding the nature of abnormal brain function in schizophrenia and schizotypy. Holzman (2000) has suggested that disruption of the initiation and control of smooth pursuit is what drives abnormalities in pursuit gain. However, more recent accounts have hypothesised that higher-order processes, such as prediction, may also be pertinent to smooth pursuit (Barnes, 2008; Lencer et al., 2010; Spering & Montagnini, 2010).

Disinhibition is one of several theories proposed to explain the pathology of schizophrenia. There is a large body of literature that suggests disinhibition theory explains the relationship between schizophrenia and the visual grasp reflex (VGR) on the antisaccade task. Researchers understand that the VGR demonstrates inhibition deficits that may cause a lack of control in pursuit gain. Moreover, Jarrett et al. (2002), suggested disinhibition can provide an explanation of forward memory bias. Thus, I suspect that the forward memory error is closely related to prediction deficits observed in pursuit gain. Therefore, the aim of the current study was to determine whether the prediction of an object's position is involved in eye movement anomalies and to what extent eye tracking and prediction is differently related to aspects of schizotypy.

The current study used the SPQ (Raine, 1991) to identify subclinical phenomenology of schizophrenia in a general population. I also used eye-tracking apparatus to investigate fundamental indices of eye movement, such as VGR and pursuit gain, along with a computer-based measure of the RM procedure. We had two hypotheses. Firstly, we expected to find different relationships between the SPQ factor scores and eye tracking tasks and predicted that (a) high SPQ cognitive-perceptual scores are associated with low pursuit gain; (b) high SPQ cognitive-perceptual scores are associated with a high percentage of antisaccade errors; and (c) high SPQ cognitive-perceptual scores are associated with the RM effect. Secondly, because the VGR and pursuit gain are considered reliable measures of perceptual performance and forward memory bias is a measure of prediction, we hypothesised that a model composed of these measures would best identify those who scored high on the SPQ cognitive-perceptual score.

Method

Participants

The participants were 171 University of Otago undergraduate psychology students. Participants were recruited as part of their psychology course to gain additional credit. Of the participants, 46 (29.9%) were male and 125 (70.1%) were female. The overall mean age of participants was 20.9 years (*SD* = 3.6); 21.2 years (*SD* = 3.9) for males and 20.7 years (*SD* = 3.4) for females. All participants reported normal or corrected-to-normal vision.

Measures

Schizotypal Personality Questionnaire (SPQ). The SPQ is a self-report questionnaire consisting of 74 yes-no items (Raine, 1991). Each *Yes* response on the SPQ scores one point. Total scores will therefore range from 0 to 74. A high score indicates high-risk for schizotypy. There are nine subscales that correspond to nine criteria included in the *DSM-III-R* definition of schizotypal personality disorder (American Psychological Association, 1994), and three factor scales labelled cognitive-perceptual, interpersonal and disorganised (SPQ-1, SPQ-2 and SPQ-3, respectively). These factor scales were made from subscale measures ideas of reference (SPQ-1), excessive social anxiety (SPQ-2), odd beliefs or magical thinking (SPQ-1), unusual perceptual experiences (SPQ-1), odd or eccentric behaviour (SPQ-3), no close friends (SPQ-2), odd speech (SPQ-3), constricted affect (SPQ-2), and suspiciousness (SPQ-1 and SPQ-2) (Raine, 1991).

The SPQ has high convergent validity (0.59 to 0.81) and criterion validity (0.63, 0.68) (Raine, 1991). Furthermore, the SPQ has high internal reliability (0.91) and test-retest reliability (0.82) (Raine, 1991). Raine (1991) demonstrated that 55% of subjects scoring in the top 10% of SPQ scores have been found to have a clinical diagnosis of schizotypal personality disorder.

Stimuli

Representational momentum task. The stimuli for the RM task were taken from Senior et al. (2000), as still frames adapted from five video clip scenes. The scenes used were of a man jumping from a ledge, a toy bus moving down a slope, water being poured from a kettle, a ball being thrown, and a cup falling from a ledge. A further five original scenes were created for the current study. These scenes consisted of a cell phone dropping from someone's hand, a man doing long jump, a ball being kicked, a man running and a frisbee being thrown. The visual angle of the images presented had a height of 13°, and a width of 16.5°.

Smooth pursuit eye movement task. The stimuli used for the SPEM task were, first, a fixation crosshair (1.5° x 1.5°), presented at the start of each trial; and second, a white filled circle 1° in diameter, which was utilised as the smooth pursuit target. The target moved in a horizontal plane at sinusoidal wave velocity across $\pm$ 15º of the centre point of the visual angle. Stimuli were presented on a black background.

Antisaccade task. Firstly, two 1° x 1° white, empty boxes marked the eye movement targets. These were placed $\pm$ 15° from the central point and were always visible throughout every trial. Each trial began with a 0.4° white fixation dot that

appeared in the centre of the screen. The fixation dot appeared for 500ms before being replaced by a neutral cue (a 0.4° x 1° white double-headed arrow, pointing both left and right) or a valid cue (a 0.4° x 0.4° white single arrowhead, pointing either left or right depending on the direction of the target). The stimuli used for the antisaccade task were presented on a black background.

Apparatus

Eye movement responses were recorded using an Applied Science Laboratories (ASL) EyeTrac6 series eye-tracker. Eye movement signals were recorded every 2ms and saved to Eyenal analysis software (ASL). Stimuli for the eye tracking tasks were presented on a 17 inch cathode ray tube (CRT) monitor. The chin rest height was changed according to each participant's height. This was so that participants' eyes were level with the centre of the CRT screen. The chin rest was used to reduce head movement, minimising the risk of mass movement and to reduce noise.

Procedure

General overview. Once informed consent was gained, participants always completed the SPQ first, calibration protocol second and then the experimental tasks followed. The order of RM, SPEM and antisaccade tasks were counterbalanced across participants. At the end of experimental tasks, the participants were fully debriefed. The entire procedure took approximately 45 minutes to complete.

Calibration protocol. Both eye tracking tasks required eye calibration. This was conducted before both eye tracking tasks began. The protocol for calibration was as

follows. Participants were seated 57cm from the monitor and their head was secured in the chin rest. The participants' eye position was calibrated using a nine-point set target system. After each block in both the antisaccade and SPEM tasks, calibration was repeated using a three-point set target system. Calibration was achieved using visual feedback from the PC. If calibration at any point was not accurate, the protocol was repeated.

Representational momentum task. Before the task began participants were given adequate instructions on what to expect and how to complete that task. The PC recorded participants' reaction time (RT) and accuracy on each trial.

In each trial in this task the first image of each pair was shown on the screen for 250ms followed by a blank screen for a further 250ms (Figure 2). The second image was then presented and remained on the monitor until the participant responded. Participants were instructed to use response keys to respond *identical* or *different* according to whether they thought the second image was identical or different to the first image. The response keys were A and L. The keys were counterbalanced across all participants. Participants were instructed to respond *identical* if they were unsure. After each response, there was a 1500ms inter-trial interval.

Altogether, participants were given four practice trials and 120 experimental trials. All experimental trials were presented in a random fashion. The RM task had two conditions. That was, the second picture was either *identical* to, or *different* to, the first picture. If the second picture was different to the first it was either forward in the plane

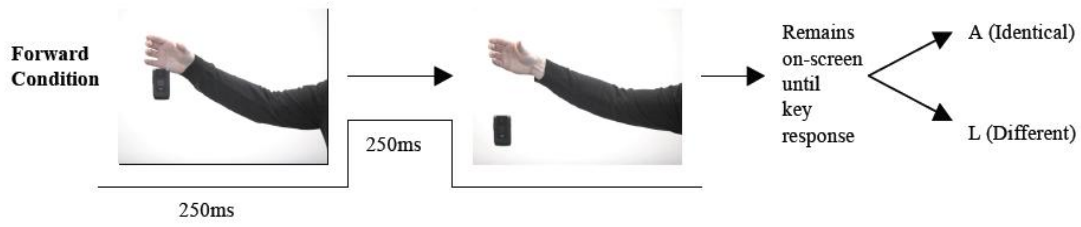


Figure 2. Schematic diagram of RM task. Depiction of cell phone dropping scene.

of motion compared to that of the first picture (*forward condition*) or backward in the plane of motion compared to that of the first picture (*backward condition*) (depicted in Figure 2). Sixty of these picture pairs were in the identical condition, 30 were in the backward condition, and 30 were in the forward condition. The RM task took approximately 10 minutes to complete.

Smooth pursuit eye movement task. Before any SPEM trials began, the equipment was calibrated. Recording began with stimulus onset. In each trial in the SPEM task a crosshair appeared in the centre of the screen for 1500ms before the smooth pursuit target replaced it. The smooth pursuit target then moved from the centre point to one extreme in horizontal plane at sinusoidal wave velocity. The target then completed five full oscillations before disappearing at the centre point. After each trial there was an inter-trial interval of 3000ms before the next trial began. This is depicted in Figure 3.

Participants were instructed to follow the stimulus with their eyes as closely as possible for the entire trial. Participants were instructed to keep their heads as still as possible throughout the eye SPEM task and to not fall behind or race ahead of the target stimulus.

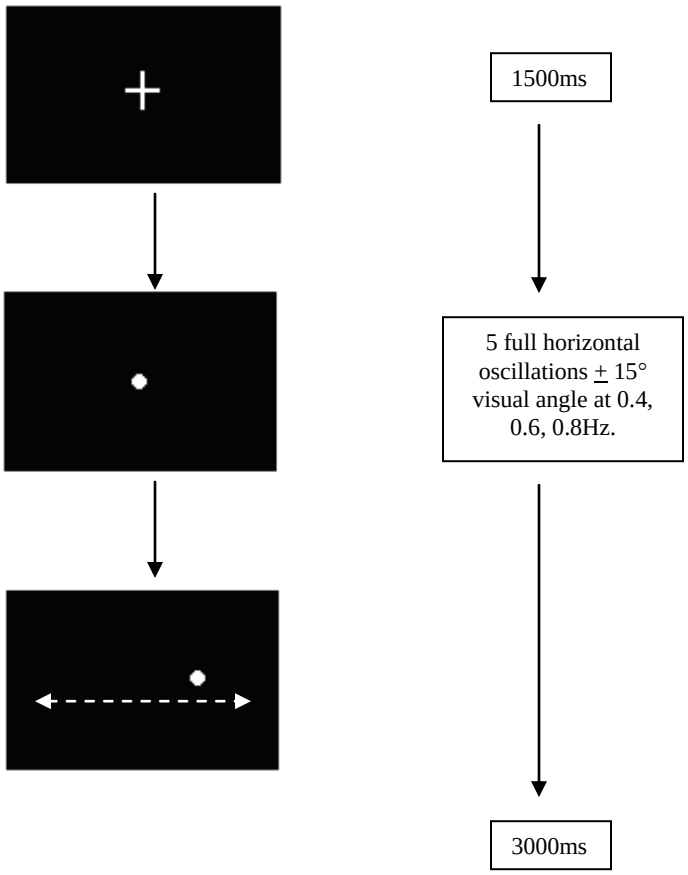


Figure 3. Schematic diagram of SPEM task.

Altogether the task consisted of two practice trials at 0.6 Hz, plus an additional four blocks of three experimental trials each. Each block differed in the velocity of the stimulus (0.4, 0.6, or 0.8Hz). At the end of each block, the participant was given the option of rest and to proceed when they felt they were ready. The SPEM task took approximately 10 minutes to complete.

Antisaccade task. Before any antisaccade trials began, the equipment was calibrated. Recording began with stimulus onset. Each trial began with a white fixation dot that appeared in the centre of the screen, along with the two target boxes. The fixation dot appeared for 500ms before being replaced by a neutral or a valid cue. Cue

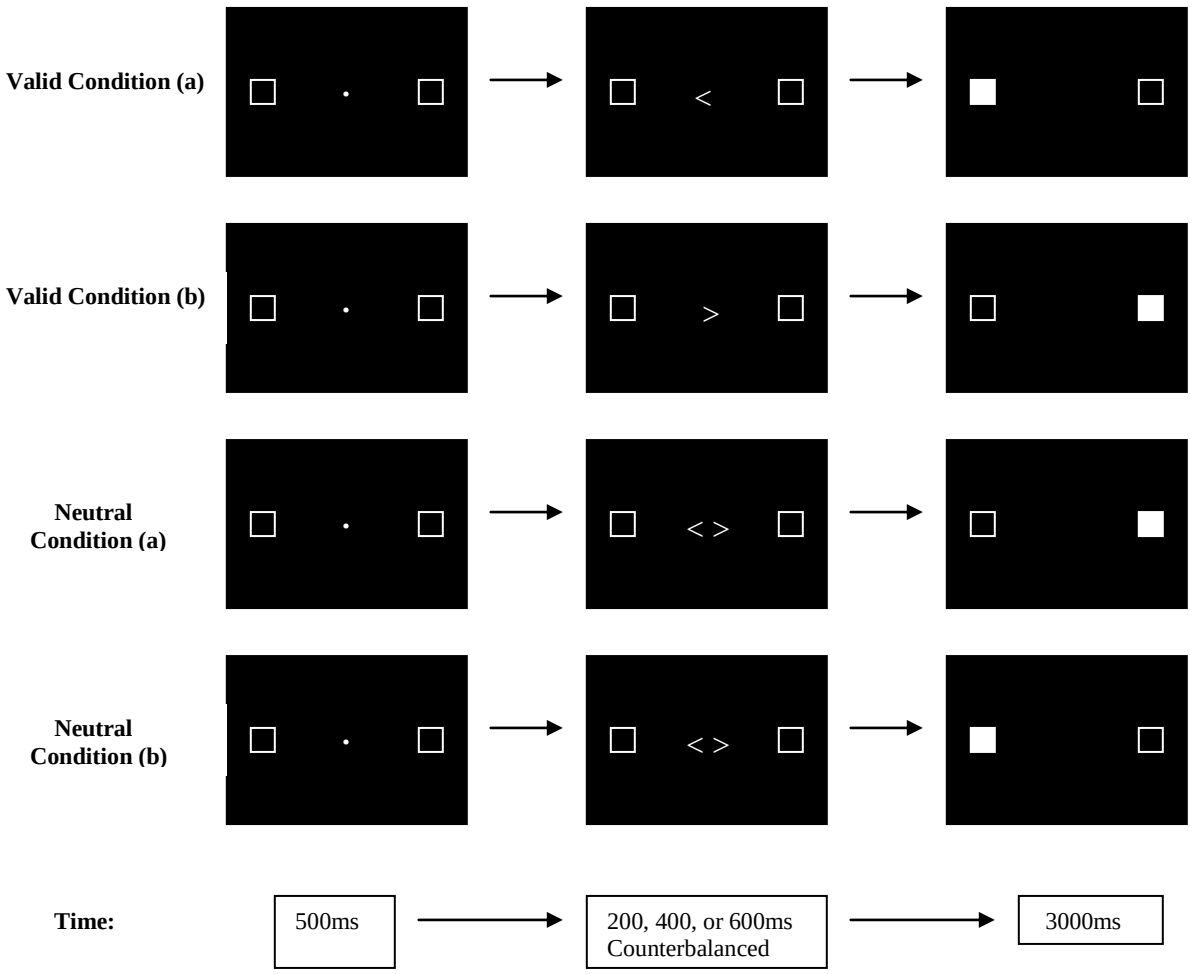


Figure 4. Schematic diagram of antisaccade task.

duration was counterbalanced across all trials (200ms, 400ms or 600ms). Following the cue, a white target stimulus filled one of the two target boxes for 3000ms. The inter-trial interval was 1500ms. This is depicted in Figure 4. Participants were instructed to fixate at the centre of the screen until a target stimulus appeared in one of the target boxes. At this point they were to move their eyes to the box opposite the target dot as soon as possible. When the target stimulus disappeared they were instructed to return to the

fixation dot in the centre of the screen in order to ready themselves for the next trial. Participants were further requested that they were not to move their eyes until the target stimulus appeared in a target box.

There were two conditions for the antisaccade task. These were valid and neutral cue conditions. If the cue was valid, the stimulus filled the box the cue pointed to. If the cue was neutral, the stimulus filled either the left or right box: The destination of the stimulus was unknown to the participant.

Altogether, the task consisted of two blocks of 16 experimental trials each; eight neutral and eight valid trials within each block. The cue condition was randomised throughout each block. Before the experimental trials began, eight practice trials were conducted to make sure participants understood the task.

Data Analysis

Dependent measures from the SPQ. The primary dependent variables for the SPQ were the total score, cognitive-perceptual, interpersonal and disorganised factor scores.

Dependent measures from the RM task. There were two measured variables in the RM task. First, the percentage of errors from the backward condition was subtracted from the percentage of errors in the forward condition to give the RM effect. A higher RM effect indicated greater forward-in-time bias. Secondly, median reaction time (RT) was recorded to identify outlying responses. As suggested by Jarrett et al. (2002) data from individual trials for which RTs (a) exceeded 3000ms, (b) were less than 100ms, or (c) were three or more standard deviations from the participant's mean, were removed from the data.

Dependent measures from the SPEM task. The measured variables for the SPEM were pursuit gain, root mean square error (RMSE) and saccadic intrusions. Pursuit gain was calculated by dividing the velocity of the eye by the velocity of the target stimulus. The extent to which the pursuit gain drops below a value of 1.0 indicated the degree of the deficit in SPEM.

To calculate pursuit gain, data from individual trials were cleaned as follows. That was, (a) all blinks were removed, (b) the outer three degrees of the stimulus pathway were removed from recording, and (c) velocities 15 degrees per second faster than the target velocity (in both the direction of the target and that opposite to the target) were removed. These were labelled as saccadic intrusions, not smooth pursuit movement.

RMSE is the measure of total spatial tracking error and can be attributed to the spatial discrepancy (see Table 1). This was calculated by first aligning the eyes and target stimulus at the beginning of the trial. As the trial commenced, the difference between the two signals was recorded and calculated. Higher scores indicated greater spatial tracking error. Both pursuit gain and RMSE were calculated for each subject and then averaged across all participants. This was calculated for each target stimulus frequency.

Dependent measures from the antisaccade task. There were three measured variables for the antisaccade task. Firstly, the percentage of correct trials across the entire task was calculated for valid and neutral conditions. A correct trial was scored when the subject moved his or her eyes toward the empty box, as instructed. Higher percent correct indicated better performance on the antisaccade task.

The second measure was the percentage of visual grasp reflexes (VGR) demonstrated across the entire task. This was calculated for both neutral and valid conditions. Higher percent VGR indicated poorer performance on the antisaccade task. Finally, the median RT for the VGR was measured.

Response outliers were discarded: in which RT was (a) less than 100ms, and (b) greater than 3000ms. Furthermore, those participants that had less than 10 correct antisaccade movements throughout the entire task, or were greater than 3 degrees from the fixation point at the start of each trial, were also excluded. This indicated that the participant did not understand the task (Machado & Rafal, 2004).

All data. Transformations were used to correct for nonnormality. The SPQ data used in the bivariate and multivariate analyses were log transformed. Kolmogorov-Smirnov test, skew and kurtosis were reported to provide evidence for the improvement of transformation of data. For skew, a larger positive value indicated the distribution had a larger skew to the left. A larger negative value indicated a larger skew to the right. For kurtosis, a higher positive value indicated the distribution had a higher peakedness and a negative value indicated a flatter distribution. Ideally both skew and kurtosis should be as close to a zero value as possible for a normal distribution of the data set. All descriptive statistics were reported as untransformed data. Analyses were conducted using SPSS and Excel. Multiple regression was used to test the association between predictor variables and schizotypy scores. Standardised β was calculated to measure how strongly each predictor variable in a two- and three-factor model influenced the prediction of schizotypy.

A group contrast (schizotypy versus non-schizotypy) was also created. Meehl

(1990) suggested that the predisposition to schizophrenia has a general population base rate of 10%. Therefore groups were determined by taking the SPQ total score and calculating Z scores as cut off points. For the schizotypy group, the cut off was a Z score greater than 1.2816. This indicated the top 10% scorers on the SPQ. For the non-schizotypy group, the cut off score was a Z score lower than 0.4307 (bottom 67% SPQ scores).

Results

Schizotypal Personality Questionnaire (SPQ)

There were no missing item responses for the SPQ. Tables 2 and 3 show the distributions of SPQ total and factor scores. The normality of SPQ total and factor scores were tested using the Kolmogorov-Smirnov statistic (KS-statistic), and by calculation of the skew and kurtosis. According to the significance of the KS-statistic, SPQ distributions were non-normal both before and after the log transformation. However, Tables 2 and 3 do provide some evidence that the skew and kurtosis of SPQ scores distribution had a small improvement once the log transformation was applied. Histogram plots of SPQ total and factor scores were also analysed. Histograms demonstrated the improvement of skew and kurtosis of SPQ total and factor score distribution. Table 4 shows the mean, standard deviation and range of SPQ total and factors scores for all participants.

Table 2
SPQ Distribution Statistics before Transformation (n = 171)

SPQ	Kolmogorov-Smirnov	Skew	Kurtosis
Total	.08	0.83	0.62
Cognitive-Perceptual	.12	0.73	-0.35
Interpersonal	.14	1.06	1.12
Disorganised	.15	0.90	0.27

Table 3
SPQ Distribution Statistics after Transformation (n = 171)

	Kolmogorov-Smirnov	Skew	Kurtosis
Total	.07	-0.08	-0.30
Cognitive-Perceptual	.11	0.05	-0.97
Interpersonal	.08	0.13	-0.41
Disorganised	.09	0.16	0.63

Representational Momentum Task and SPQ

There were no missing data from the RM task. There was no evidence of a speed-accuracy trade-off for the RM task. As shown in Figure 5, participants made errors on 17% (*SD* = 11) of forward trials and 12% (*SD* = 9) of backward trials. There was a significant difference between mean forward and backward error rates, $t(170) = 8.6$, $p < .01$. Therefore, participants made more errors in the forward condition.

Table 4
Scores on the SPQ (n =171)

	M	*SD*	Minimum	Maximum
SPQ Total	17.7	11.9	0	56
SPQ-1	5.9	5.0	0	18
SPQ-2	7.5	5.9	0	29
SPQ-3	4.3	3.5	0	15

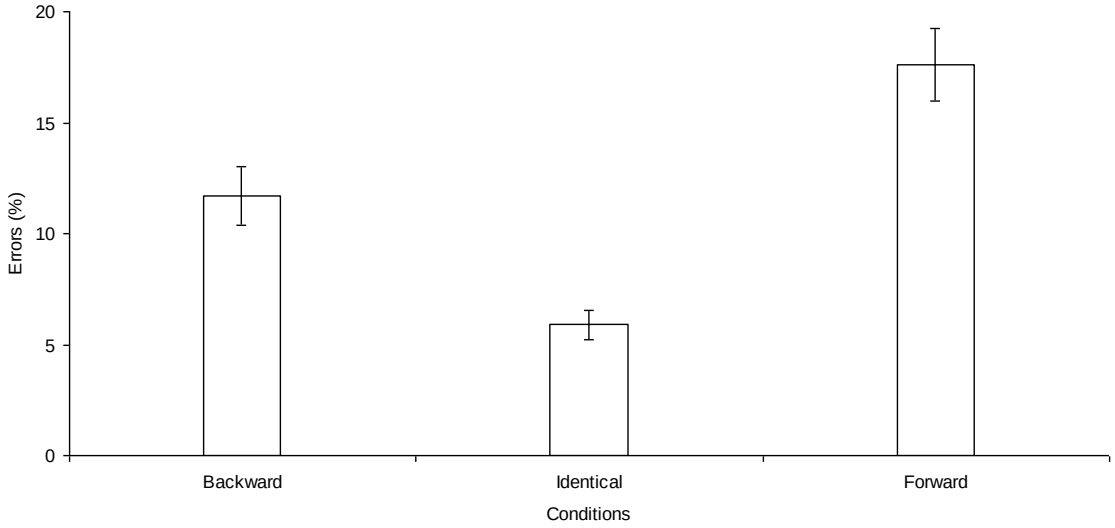


Figure 5. Mean percentage of errors made and 95% confidence intervals across all participants on the backward, forward and identical conditions in the RM task (n = 171)

Participants made errors on just under 6% (SD = 4.4) of identical trials. Two independent t-tests suggested that the number of errors made in the identical condition was significantly less than errors in the forward and backward conditions; $t(170) = 13.1$, $p < .01$ (forward); $t(170) = 8.2$, $p < .01$ (backward). The RM effect (difference in percentage errors in the forward relative to the backward condition) was 5.9 (SD = 8.9).

As shown in Table 5, across all three conditions participants had the fastest RT in the identical condition. This was demonstrated by two independent t-tests. There was a significant difference between the forward and identical condition, $t(170) = 8.3$, $p < .01$, with participants reacting faster in the identical condition.

Table 5
*RT Across Conditions of the Representational Momentum Task (n =*171)

Condition	*M*	*SD*
Forward	687.7	139.2
Backward	693.0	140.7
Identical	633.2	142.8

Furthermore, there was a significant difference in RT between the backward and identical condition, $t(170) = 9.1$, $p < .01$, with participants reacting faster in the identical condition. However, there was no significant difference between RT of the backward and forward conditions, $t(170) = 1.1$, $p > .05$.

There was a significant positive correlation between the RM effect and the SPQ score for disorganised factor ($r = .13$, $p < .05$, one-tailed; Table 6), indicating that participants who had high self-rated disorganisation had greater RM. No other significant correlations between RM indices and SPQ scores were observed.

Table 6
Correlations between SPQ Factor Scores RM Accuracy Data (n = 171)

	Backward	Identical	Forward	RM effect
Total	.00	.05	-.01	-.01
Cognitive- Perceptual	-.03	.03	.06	.10
Interpersonal	-.01	.02	-.05	-.05
Disorganised	.06	.08	-.06	.13*

Note. * = $p < 0.05$

Smooth Pursuit Eye Movement Task and SPQ

There were four participants missing information from the SPEM task. There was not enough information for these participants because of excessive blinking or head movement that created excess noise. Data from these participants were therefore removed from further analyses.

As shown in Table 7, the total pursuit gain observed was 0.60 (*SD* = 0.15). Participants demonstrated a pursuit gain of 0.70 (*SD* = 0.16) in the 0.4Hz condition, 0.68 (*SD* = 0.15) in the 0.6Hz condition and 0.42 (*SD* = 0.16) in the 0.8Hz condition. Independent *t*-tests showed that there was a significant difference in pursuit gain between the 0.6Hz and 0.8Hz condition, $t(166) = 37.2$, $p < .01$, with participants performing better in the 0.6Hz condition. Furthermore, there was also a significant difference in pursuit gain between the 0.4Hz and 0.6Hz condition, $t(166) = 4.1$, $p < .05$, with participants performing better in the 0.4Hz condition.

Across all conditions, the mean RMSE observed was 3.9 (*SD* = 1.0). According to RMSE measurement, participants performed worse on the 0.8Hz trials. This was demonstrated by independent t-tests. Firstly there was no difference between the 0.4Hz and 0.6Hz conditions. However, there was a significant difference between the 0.6Hz and 0.8Hz condition, $t(166) = 31.08$, $p < .01$, indicating that participants' accuracy was worse in the 0.8Hz condition.

Table 7
PG, RMSE and SI across Conditions of the SPEM Task (n = 167)

Variable	Condition	*M*	*SD*
PG	Total	0.60	0.15
	0.4 Hz	0.70	0.16
	0.6 Hz	0.68	0.15
	0.8 Hz	0.42	0.16
RMSE	Total	3.9	1.0
	0.4 Hz	3.2	1.2
	0.6 Hz	3.3	1.1
	0.8 Hz	5.2	0.9
SI	Total	9.4	2.0
	0.4 Hz	10.8	2.5
	0.6 Hz	11.2	2.6
	0.8 Hz	6.3	1.4

Note. PG = pursuit gain; RMSE = root mean squared error; SI = saccadic intrusion

Across all conditions, the average number of saccadic intrusions that were observed was 9.4 (*SD* = 2.0). Throughout the task conditions there were differences in number of saccadic intrusions made by participants. Firstly, there was a significant difference between saccadic intrusions in the 0.4Hz and 0.6Hz conditions, $t(166) = 4.36$, $p < .01$. Secondly, there was a significant difference between 0.4 and 0.8Hz conditions, $t(166) = 31.07$, $p < .01$. Finally, there was a significant difference between 0.6 and 0.8Hz conditions, $t(166) = 34.78$, $p < .01$. Therefore, as shown in Table 7, participants demonstrated the most saccadic intrusions in the 0.6Hz condition and the least in the 0.8Hz condition.

Table 8
Correlations between SPQ Scores and Pursuit Gain from SPEM Task (n = 167)

	Condition			Total PG
	0.4Hz	0.6Hz	0.8Hz	
Total	-.11	-.12	-.15*	-.14*
Cognitive- Perceptual	-.08	-.10	-.13*	-.11
Interpersonal	-.10	-.11	-.10	-.11
Disorganised	-.05	-.07	-.14*	-.09

Note. * = $p < 0.05$

As shown in Table 8, Pearson *r* correlation between pursuit gain scores across SPEM conditions and SPQ scores were analysed. There was a significant negative correlation between the SPQ total score and the total pursuit gain ($r = -.14$, $p < .05$, one-tailed). The SPQ total score also correlated significantly with the pursuit gain in the 0.8 Hz condition ($r = -.15$, $p < .05$, one-tailed). Furthermore, the pursuit gain measured from the 0.8 Hz condition correlated significantly with both the SPQ cognitive-perceptual factor score ($r = -.13$, $p < .05$, one-tailed) and the SPQ disorganised factor score ($r = -.14$, $p < .05$, one tailed). This indicates that those with higher self-reported cognitive-perceptual and disorganised experiences were less able to keep up with the smooth pursuit target stimulus.

Antisaccade Task and SPQ

There were 18 participants with missing data from the antisaccade task. As shown in Table 9, the participants averaged 73.5% (*SD* = 11.7) correct responses across all conditions.

Table 9
Percent Correct, VGR and RT across Conditions of the Antisaccade Task (n = 153)

	Condition	*M*	*SD*
Correct (%)	Total	73.5	11.7
	Valid	67.4	15.2
	Neutral	79.6	13.5
VGR (%)	Total	4.8	4.8
	Valid	2.1	3.6
	Neutral	7.5	8.0
RT (ms)	Total	212.5	53.4
	Valid	191.0	54.7
	Neutral	234.3	52.0

Note. VGR = visual grasp reflex.

Participants made 67.4% (*SD* = 15.2) correct responses on valid trials; that is, trials where information was given about the stimulus, prior to stimulus presentation. Participants made 79.6% (*SD* = 13.5) correct responses on neutral trials; that is, trials where there was no directional information given prior to stimulus presentation. There was a significant difference between the neutral and valid conditions, $t(150) = 8.98$, $p < .01$. Participants made more correct responses on the neutral condition.

Participants made a VGR 4.8% (*SD* = 4.8) of the time on valid trials. This figure rose to 7.5% (*SD* = 8.0) of trials with a neutral cue. A *t*-test demonstrated a significant difference between VGRs made in neutral and valid conditions, $t(150) = 8.42$, $p < .01$. As shown in Table 9 the reaction time across conditions was 212.5ms (*SD* = 53.4). An independent *t*-test on RT difference between the valid and neutral conditions demonstrated that participants had significantly faster reaction times in the valid condition ($t(150) = 19.644$, $p < .01$).

Table 10
Correlations between SPQ Scores and Antisaccade Task (n = 153)

	% Correct Valid	% Correct Neutral	% VGR Valid Condition	% VGR Neutral Condition
Total	.14*	.07	-.09	.06
Cognitive- Perceptual	.20*	.04	.00	.01
Interpersonal	.05	.08	-.11	.05
Disorganised	.11	.06	-.12	.07

Note. VGR = Visual grasp reflex
* = $p < 0.05$

As shown in Table 10, there were significant positive correlations between the percent correct in the valid condition and SPQ total score ($r = .14$, $p < .05$, one-tailed) and the SPQ cognitive-perceptual factor score ($r = .20$, $p < .05$, two-tailed).

Bivariate Correlations between Task Indices

As depicted in Table 11, the most consistent association was observed between the VGR and PG across all their conditions. The strongest correlation was demonstrated between the total VGR made in the antisaccade task and the PG in the 0.6Hz condition ($r = -.25$, $p < .01$, one-tailed). The weakest correlation between these two tasks, albeit still significant, was demonstrated between VGR made in the neutral condition and the PG in the 0.8Hz condition ($r = -.16$, $p < .05$, one-tailed).

Between SPEM and RM task indices, the PG in the 0.4Hz conditon and the forward errors made in the RM task demonstrated the strongest correlation ($r = -.17$, $p < .05$, one-tailed). The PG from the 0.8Hz condition was the only SPEM condition that did not significantly correlate with any of the RM indices. Furthermore,

Table 11
Bivariate Correlation between Task Measures

		1	2	3	4	5	6	7	8	9
1.	RM Effect									
2.	Forward Error	.63**								
3.	Backward Error	-.23**	.62**							
4.	VGR total	-.06	.12	.21**						
5.	VGR valid	.01	.11	.12						
6.	VGR neutral	-.08	.09	.20**						
7.	0.4Hz PG	-.06*	-.17*	-.15*	-.23**	-.22**	-.18*			
8.	0.6Hz PG	-.03	-.14*	-.15*	-.25**	-.23**	-.20**	.97**		
9.	0.8Hz PG	.03	-.08	-.13	-.19**	-.17*	-.16*	.83**	.86**	

Note. RM = representational momentum. VGR = visual grasp reflex.
PG = pursuit gain. * = p < .05 level one-tailed. ** = p < .01 level, one-tailed.

the RM effect did not yield significant correlations with PG on any of the SPEM conditions. Between the VGR and RM task indices, there was only one RM condition that significantly correlated with the VGR. That was the backward errors made in the RM task significantly correlated with the total VGR made in the antisaccade task (r = .21, p < .01, one-tailed). Furthermore, the backward errors made in the RM task correlated significantly with VGRs made in the neutral condition of the antisaccade task (r = .20, p < .01, one-tailed).

Multiple Regression Predictor Models of Schizotypal Personality

Multiple regression analysis was used to test if combinations of the visual elements (pursuit gain from the 0.8Hz condition, the VGR and the RM effect) significantly predicted participants SPQ total, cognitive perceptual, interpersonal and disorganised scores. Three predictor models were used to predict SPQ total and factor

Table 12
Summary of Multiple Regression Analysis for SPQ Total Score (*n* = 162)

Model	Variables	B	SE (B)	β	*t*	(*p*)
1	0.8Hz PG	-1.332	0.787	-0.143	-1.692	0.391
	Total VGR	-0.002	0.026	-0.006	-0.070	
	RM Effect	-0.012	0.014	-0.075	-0.905	
2	0.8Hz PG	-1.357	0.786	-0.145	-1.726	0.355
	Total VGR	-0.001	0.026	-0.002	-0.021	
3	0.8Hz PG	-1.380	0.707	-0.153	-1.951	0.263
	RM Effect	-0.010	0.013	-0.064	-0.820	

Note. Model 1 R^2 = .035, Model 2 R^2 = .029, Model 3 R^2 = .032. PG = pursuit gain, VGR = visual grasp reflex, RM = representational momentum, B = unstandardised coefficient.

scores. Model 1 used all three variables. Model 2 used pursuit gain from the 0.8Hz condition and the VGR as the variables. Finally, Model 3 used pursuit gain from the 0.8Hz condition and the RM effect.

As shown in Table 12, none of the models was a significant predictor of the total SPQ score. However, the best model for predicting SPQ total scores was Model 1. This included the PG from the 0.8Hz condition of the SPEM task, the total VGR made in the antisaccade task and the RM effect as the predictor variables. Moreover, these predictors only account for 3.5% of the variance, R^2 = .035, F(5, 145) = 1.05, $p > 0.05$. As shown in Table 12, the results demonstrated that the best predictor in Model 1 was the PG predictor (β = -.143, $p > 0.05$), however this itself was not significant.

As depicted in Table 12, the RM effect is a better predictor of SPQ total score than the VGR. This is demonstrated by standardised beta. Firstly, standardised beta for the RM effect (β = -.075, $p > 0.05$) is larger than VGR (β = -.006, $p > 0.05$) in Model 1. Secondly, when the two predictor variables were separated and paired with the PG predictor variable, Model 3 (R^2 = .032, F(4, 162) = 1.33, $p > 0.05$) accounts for 0.3%

Table 13
Summary of Multiple Regression for SPQ Cognitive-perceptual Score (*n* = 162)

Model	Variables	B	SE (B)	β	*t*	(*p*)
1	0.8Hz PG	-1.241	0.564	-0.105	-1.241	
	Total VGR	-0.252	0.018	0.021	-0.252	0.625
	RM Effect	0.261	0.010	0.022	0.261	
2	0.8Hz PG	-0.695	0.562	-0.105	-1.236	0.489
	Total VGR	-0.005	0.018	-0.022	-0.267	
3	0.8Hz PG	-0.815	0.509	-0.125	-1.602	0.312
	RM Effect	0.005	0.009	0.039	0.497	

Note. Model 1 R^2 = .024, Model 2 R^2 = .023, Model 3 R^2 = .029. PG = pursuit gain, VGR = visual grasp reflex, RM = representational momentum, B = unstandardised coefficient.

more variance than model 2 (R^2 = .029, F(4, 146) = 1.11, p > 0.05).

As shown in Table 13, none of the models was a significant predictor of SPQ cognitive-perceptual scores. However, the best model for predicting SPQ cognitive-perceptual scores was Model 3. This included the PG from the 0.8Hz condition of the SPEM task, and the RM effect as the predictor variables. Nevertheless, these predictors only account for approximately 3% of the variance, and this figure is not significant (R^2 = .029, F(4, 162) = 1.20, p > 0.05).

As shown in Table 13, the results demonstrated that the best predictor in Model 3 was the PG predictor (β = -.125, p > 0.05), however this itself was not significant. When the RM effect and VGR predictor variables were separated and paired with the PG predictor variable, Model 3 (R^2 = .029, F(4, 162) = 1.20, p > 0.05) accounts for 0.6% more variance than Model 2 (R^2 = .023, F(4, 146) = .86, p > 0.05).

As shown in Table 14, none of the models was a significant predictor of SPQ interpersonal scores. Even the best model for predicting SPQ interpersonal scores was Model 3, these predictors account for only 5% of the variance the result is

Table 14
Summary of Multiple Regression Analysis for SPQ Interpersonal Score (*n* = 162)

Model	Variables	B	SE (B)	β	*t*	(*p*)
1.	0.8Hz PG	-0.774	0.569	-0.144	-1.360	0.215
	Total VGR	-0.003	0.019	-0.012	-0.142	
	RM Effect	-0.013	0.010	-0.110	-1.349	
2.	0.8Hz PG	-0.800	0.570	-0.118	-1.404	0.260
	Total VGR	-0.001	0.019	-0.006	-0.069	
3.	0.8Hz PG	-0.763	0.513	-0.116	-1.488	0.180
	RM Effect	-0.012	0.009	-0.102	-1.310	

Note. Model 1 R^2 = .047, Model 2 R^2 = .035, Model 3 R^2 = .038. PG = pursuit gain, VGR = visual grasp reflex, RM = representational momentum, B = unstandardised coefficient.

not significant (R^2 = .047, F(5,145) = 1.44, p > 0.05). Furthermore, the results demonstrated that the best predictor in Model 1 was the pursuit gain variable (β = -.144, p > 0.05), however this itself was not significant. As shown in Table 14, the RM effect is a better predictor of SPQ interpersonal score than the VGR. Firstly, standardised beta for the RM effect (β = -.110, p > 0.05) is greater than the VGR (β = -.012, p > 0.05) in Model 1. Secondly, when the two predictor variables were separated and paired with the PG predictor variable, Model 3 (R^2 = .038, F(4,162) = 1.59, p > 0.05) accounts for 0.3% more variance than Model 2 (R^2 = .035, F(4,146) = 1.33, p > 0.05). Although the results are not significant, they demonstrate that the RM effect is a better predictor than the VGR of SPQ interpersonal score.

As shown in Table 15, none of the models was a significant predictor of SPQ disorganised scores. The best model for predicting SPQ disorganised scores was Model 1. However, this model only accounts for only 4% of the variance (R^2 = .041, F(5,145) = 1.24, p > 0.05). Furthermore, the results demonstrated that the best predictor in model one was again pursuit gain (β = -.155, p > 0.05), however this itself was not significant.

Table 15
Summary of Multiple Regression Analysis for SPQ Disorganised Score (*n* = 162)

Model	Variables	B	SE (B)	β	*t*	(*p*)
1.	0.8Hz PG	-0.788	0.427	-0.155	-1.844	
	Total VGR	-0.001	0.014	-0.005	-0.055	0.29
	RM Effect	-0.010	0.007	-0.122	-1.369	
2.	0.8Hz PG	-0.808	0.428	-0.159	-1.887	0.37
	Total VGR	0.000	0.014	0.002	0.019	
3.	0.8Hz PG	-0.721	0.391	-0.144	-1.844	0.19
	RM Effect	-0.011	0.007	-0.117	-1.503	

Note. Model 1 R^2 = .041, Model 2 R^2 = .029, Model 3 R^2 = .037. PG = pursuit gain, VGR = visual grasp reflex, RM = representational momentum, B = unstandardised coefficient.

As shown in Table 13, the RM effect is a better predictor of SPQ disorganised score than the VGR. Firstly, standardised beta for the RM effect (β = -.122, $p > 0.05$) is greater than the VGR (β = -.005, $p > 0.05$) in Model 1. Secondly, when the two predictor variables were separated and paired with the PG predictor variable, Model 3 (R^2 = .037, F(4,162) = 1.57, $p > 0.05$) accounts for 0.8% more variance than Model 2 (R^2 = .029, F(4,146) = 1.08, $p > 0.05$) .

In summary, the multiple regression analysis yielded no significant results. However, standardised β showed that pursuit gain from the SPEM 0.8Hz condition was the best predictor across all the models. Despite RM having a larger standardised β than the VGR, it did not improve, nor significantly contribute to any of the tested models as hypothesised.

Schizotypy versus non-Schizotypy

Meehl (1990) suggested that the predisposition to schizophrenia has a general population base rate of 10%. Therefore, two group contrasts were analysed by taking the

Table 16
Group Statistics for RM Effect

Group Membership	N	*M*	*SD*
Schizotypy	16	4.59	5.59
Non-schizotypy	112	6.65	9.13

SPQ total score and calculating Z scores as cut off points. For the schizotypy group, the top 10% of SPQ scores equalled a cut off Z score greater than 1.2816. For the non-schizotypy group, the cut off score was a Z score lower than 0.4307, which was the bottom 67% of SPQ scorers. There were no missing data from the schizotypy or non-schizotypy group for the outcome measure of the RM effect. For the outcome measure of PG there was one piece of missing information for the schizotypy group and two pieces of missing information for the non-schizotypy group. For the outcome measure of the VGR there were two pieces of missing information for the schizotypy group and nine pieces of missing information from the non-schizotypy group.

As shown in Table 16, the schizotypy group have a lower RM effect than the non-schizotypy group. However, this difference was not significant, $t(126)$, $p > 0.05$, two tailed. Secondly, as shown in Table 17 the PG is smaller for those identified in the schizotypy group, there was not a significant effect for PG, $t(123)$, $p > 0.05$, two tailed.

Table 17
Group Statistics for Pursuit Gain

Group Membership	N	*M*	*SD*
Schizotypy	15	0.60	0.14
Non-schizotypy	110	0.62	0.16

Table 18
Group Statistics for Visual Grasp Reflex

Group Membership	N	*M*	*SD*
Schizotypy	14	4.61	4.02
Non-schizotypy	103	4.89	5.13

Finally, as seen in Table 18, there was again no significant difference in the VGR between the schizotypy and non-schizotypy group, $t(115)$, $p > 0.05$, two tailed.

In summary, participants were categorised into two groups taking the SPQ total score and calculating Z scores as cut off points. There were no significant differences between the schizotypy and the non-schizotypy groups for any of the eye tracking dependent variables.

Discussion

The overall purpose of the current study was to determine whether the prediction of an object's position is involved in eye movement anomalies and to what extent eye tracking and prediction is differently related to aspects of schizotypy. I suggested that the role that prediction has in eye movement anomalies could be investigated through a) the relationship between eye tracking indices and SPQ factor scores, and b) a model that included eye tracking indices that could reliably predict schizotypy.

Summarised Results

I first hypothesised that there would be a relationship between higher SPQ cognitive-perceptual scores and a) lower pursuit gain, b) higher antisaccade errors, and c) larger RM effect. Firstly, there was evidence that those who scored higher on the SPQ cognitive-perceptual scale had a lower pursuit gain. Secondly, there was evidence to suggest that those who scored higher on the SPQ cognitive-perceptual scale, made more antisaccade errors. Both of these results provided evidence consistent with the first hypothesis. However, those who scored higher on the SPQ cognitive-perceptual scale did not have a larger RM effect. This result was not consistent with the first hypothesis.

The second hypothesis was a model that included the RM effect, pursuit gain and antisaccade. It was predicted that this model would be able to reliably predict those who scored high on the SPQ cognitive perceptual factor scale. However, the model was not significant and therefore this result was not consistent with the second hypothesis.

Comparison with Previous Literature

The results that were consistent with hypothesis one added to a long line of previous literature regarding the connection between schizotypy and eye tracking deficits.

Specifically, the current results are consistent with previous research by demonstrating that risk for schizophrenia and schizotypy is related to deficits in pursuit gain (Avila et al., 2006; Holahan &O'Driscoll, 2005; Holzman, 2000; Lahuis, Van Engeland, Cahn, & Kemner, 2008; Levy et al., 2004; Lipton et al., 1983; O'Driscoll & Callahan, 2008; O'Driscoll, Lenzenweger & Holzman, 1998; Slaghuis et al., 2007) and antisaccade accuracy (Boudet et al., 2005; Broerse, Crawford & den Boer, 2002; Curtis et al., 2001; Gooding & Basso, 2008; Hutton, Joyce, Barnes & Kennard, 2002; Levy et al., 2004; Nkam et al., 2001; O'Driscoll et al., 1998; Ploner et al., 2005; Ross et al., 2005). However, the missing relationship between schizotypy and the RM effect was not entirely consistent with previous literature. Watkins (2005) was unable to find a direct correlation between schizotypy and the RM effect. Nevertheless, when individuals were put into high or low risk groups (resulting from SPQ cut-off scores) the RM effect was associated only with those in the high-risk group (Jarrett et al., 2002; Watkins, 2005).

The current investigation of the RM effect was initiated by a considerable need to understand the processes involved in eye tracking and the extent to which these are able to identify subtypes of schizotypy. Other studies have not yet provided multivariate techniques, such as regression modelling using these predictor variables to investigate the diverse relationship between eye tracking indices and subtypes of schizotypy. However, because this was a novel approach to determine the role of RM in eye tracking anomalies to predict risk for schizotypy subtypes, there was no previous evidence that supported the current result.

Although the relationship between schizophrenia and pursuit gain deficit is the most replicated eye-tracking behaviour in schizophrenia research, the exact nature of this

relationship is less certain. Levy et al. (1993) suggested that lower pursuit gain for those at risk is characterised by increased saccadic intrusions. However it is not established that saccadic intrusions are the cause of this anomaly. Catch up saccadic intrusions can be understood as the consequence of the eye not being able to keep up with the target stimulus. In this context Holzman (2000) made the suggestion that the visual pursuit system involves two processes. One is the initiation of the movement and the other is the subsequent ability to control and monitor that movement. This was tested in the current study by the antisaccade task (initiation) and by the SPEM task (movement control). When pursuit gain decreases there has been, according to Holzman (2000), a compromise in one of these two processes. Lencer et al. (2010) have recently extended this suggestion and argued that predictive cognitions are pertinent to the smooth pursuit system. This *predictive* hypothesis was therefore tested in the current study using the RM effect.

Kelley and Bakan (1999) showed that the disrupted cooperation (between pursuit gain and saccadic inhibition) in the visual system experienced by people with schizotypy is caused by inappropriate disinhibition. Chen and Gottesman (2008) suggested that disinhibition is the result of hypokrisia or cognitive loosening. Accordingly, individuals who are at risk for schizotypy appear to have inhibition deficits and are thus less able to control the movement of their eyes. An alternative explanation is that schizotypal persons are not able to exert control over unwanted saccadic intrusions, which then disrupts the smooth pursuit system (Curtis et al., 2001; Peters et al., 2000). This notion shows similarities to other schizotypy related observations. A recent study shows that thought disorder is associated with a disinhibition of irrelevant words (Becks & Rector,

2005). The underlying mechanisms previously suggested would in summary provide a plausible explanation for both findings.

It is therefore at this point suggested that the concept of disinhibition is a relevant explanation of the underlying neuropsychological abnormalities that characterise persons with schizotypal personality traits. This theoretical framework is providing a possible explanation for the association between high scores on the SPQ cognitive-perceptual and low pursuit gain in the current study.

Further evidence for disinhibition as contributing to schizotypal phenomena is provided by the positive relationship demonstrated between the antisaccade task and SPQ cognitive-perceptual scores in this study. The implications of these findings are discussed below. Researchers believe that the antisaccade task is an observable way to test the ability or inability of an individual to inhibit eye movement. This implies that disinhibition is not only a theoretical concept, but beyond that a testable mechanism that can account for the schizotypy related anomalies observed in eye tracking.

Gooding and Basso (2008) suggest that antisaccadic tasks provide an insight into psychomotor functioning as well as higher order cognitive processes. They assess voluntary oculomotor movement through a reflexive saccadic inhibition task where the eye needs to be voluntarily forced to look in the opposite direction to a stimulus. Success or failure in the antisaccade task is determined by the suppression or failed suppression (disinhibition) of the preparatory system (Curtis & D'Esposito, 2003). According to the current results, those who were at higher risk for schizophrenia, failed to supress the preparatory system and therefore made more errors on the antisaccade task.

Various researchers show that disinhibition in persons with a high degree of schizotypal personality traits is related to hypokrisia (Lenzenweger, 2006; Maruff, Danckert, Pantelis & Currie, 1998). Hypokrisia in this context, simplified, can be seen as the cognitive loosening that according to aforementioned research, occurs in people who display schizotypal behaviours and that prohibit the control or inhibition of reflexive saccades. More specifically, some researchers have argued that this cognitive loosening has an impact on the preparatory system, which in turn affects the degree of anticipation and execution of ocular motion during an antisaccade trial (Curtis & D'Esposito, 2003; Reuter, Rakusan & Kathmanna, 2004). This argument has important implications with regard to the current study because the SPQ cognitive-perceptual score is only related to errors made in the antisaccade task in the valid cue condition and not the neutral condition. In the valid condition the advance preparation should help individuals avoid making errors. However, the preparation does not help but in fact prevents the task-performance of persons who score higher on the SPQ cognitive-perceptual scale. This shows that individuals who demonstrated a greater degree of schizotypy related cognitive-perceptual abnormalities, have greater difficulty inhibiting the reflexive saccade when given a cue that showed where the target was going to appear, than people who demonstrated lower schizotypy related cognitive-perceptual difficulties.

The aforementioned theory of disinhibition can explain the association found between errors on valid trials and scores on the SPQ (greater errors were associated with higher SPQ cognitive-perceptual scores). The current results accordingly are consistent with the approach that disinhibition is a major or may even be the primary underlying mechanism of the characteristic eye tracking performance in persons with cognitive and

perceptual schizotypal personality traits.

In summary, eye-tracking anomalies such as low pursuit gain and poor antisaccade performance are correlated with schizotypal personality. This is supported by a robust amount of literature and is corroborated in the current study. It is suggested that both of these eye-tracking anomalies occur because of a disruption of an individual's disinhibition mechanisms. However, there was no evidence found to support the second hypothesis and the predictions suggested by Lencer et al., (2010) and Spering & Montagnini, (2010), that tied all three eye tracking indices together into a predictive model. Possible explanations for these disparities are provided below.

Disparities between Current Findings and Previous Literature

Recent accounts have suggested that higher-order processes of prediction and inhibition may also be closely related to smooth pursuit anomalies (Barnes, 2008, Lencer et al., 2010; Spering & Montagnini, 2010). Other studies have not provided multivariate techniques, such as regression modelling using these predictor variables to investigate a diverse relationship between eye tracking indices and subtypes of schizotypy. Consequently, the second hypothesis expected that a model that included the RM effect and robust evidence based visual predictors (pursuit gain and VGR) could identify those who scored high on the SPQ cognitive-perceptual score. Nonetheless, the relationship that was found between RM and schizotypy will be discussed as this has implications for the results observed from the multiple regression model.

The association found between schizotypy and RM was not as hypothesised. There was no correlation between SPQ cognitive-perceptual factor scale and RM. However, a positive relationship between the SPQ disorganised factor scale and the RM

effect was found indicating that a greater RM effect is related to a higher individual SPQ disorganised score. This result is consistent with an association between representational processes and being at risk for schizophrenia. Other studies that assessed RM and psychometric indices found no relationship between RM and any SPQ factor indices (Watkins, 2005). Even when the SPQ was used as a method to divide individuals into high or low risk schizotypy groups there was still no difference in RM between the groups (Jarrett et al., 2002). The only time a difference in RM has been found was when the low risk group was compared with a group of patients with schizophrenia (Jarrett et al., 2002). The current finding is therefore interesting because it suggests that the relationship between RM and schizotypy may not be underlined by cognitive-perceptual factors, as originally hypothesised, but with anomalies of disorganisation as measured by the SPQ disorganised score.

The disorganised factor scale consists of SPQ questions generally pertaining to how individuals see themselves. For example, questions ask if they see themselves as being odd- having odd behaviour and odd speech. Therefore, current results indicate that odd behaviour and odd speech is also correlated with a disorganised or odd appearance of motion, RM effect. This could suggest that individuals who have a disorganised or fractured thought of the self (measured by the disorganised SPQ scale) might have, in analogy, also a disorganised or fractured cognitive representation of the world. Two aforementioned current theories of schizotypy underlie this explanation, namely hypokrisia (cognitive slippage) and or disinhibiton. As pointed out in the introduction, Linscott and Knight (2004) provided evidence that thought disorder may be explainable by hypokrisia since it has been related to the inability to restrain automatic memory

processes or alternatively the failure to suppress alternative meanings of words (Frith, 1981). Firstly RM has been considered an automatic process that needs to be halted in order to do well in the according tasks (Hayes &Freyd, 1995) and secondly the present results demonstrated a forward memory bias. These two findings may be understood as indications that disinhibition is responsible for the failure to inhibit the appropriate (realistic forward) momentum a motion picture conveys.

A possible process of what occurs in the RM task which is based on the concept of disinhibition, is that, the individual is unable to appropriately inhibit the cognition of momentum, and therefore fails to determine accurately how far forward in time the depicted trajectory has been in the first picture at a later point. Thus, when the second image is presented, the individual remembers the first image as being further forward in time than it actually was. Consequently in the instance when the second image is presented forward of the first image, it is more likely to be perceived as identical to the first picture. Thus, more picture pairs in the forward condition are reported incorrectly in comparison to the backward condition, which generates the calculation of the RM effect.

Meehl (1990) proposed more than twenty years ago that hypokrisia or disinhibition may be an underlying process that refines or shapes the disorganised behaviour and speech observed in persons with schizotypal behaviour. This subsequently provided the basis for an explanation as to why the reduced ability of representing momentum accurately can be a good indicator for a potentiated, high degree of disorganised and odd behaviour.

The visual indices used as predictor variables to investigate the second hypothesis were accordingly total pursuit gain, total VGR and the RM effect. The predicted

outcome variables were the SPQ total score and the cognitive-perceptual, interpersonal and disorganised SPQ factor scores. However, the proposed Model (1) (see Figure 6) was neither able to significantly predict SPQ cognitive-perceptual scores as hypothesised nor any of the other outcome variables. It may be that the visual indices were not able to predict schizotypy because the tasks were not sensitive enough to detect differences of those at risk given the current population. Possible explanations for the lack of significant prediction are provided in more detail below.

According to the current results, the VGR and the RM effect alone were not robust markers of risk. Evidence from the current study showed that the RM effect was weakly associated with the SPQ disorganised scale (not cognitive-perceptual included in the model), while the VGR was only weakly correlated with SPQ cognitive-perceptual scores. Therefore, it seemed evident that putting these visual elements together to predict schizotypy only weakens a possible association with schizotypy and any of its subtypes. To provide possible reasons for the assumed but inconsistent associations, each visual element is discussed in greater depth below.

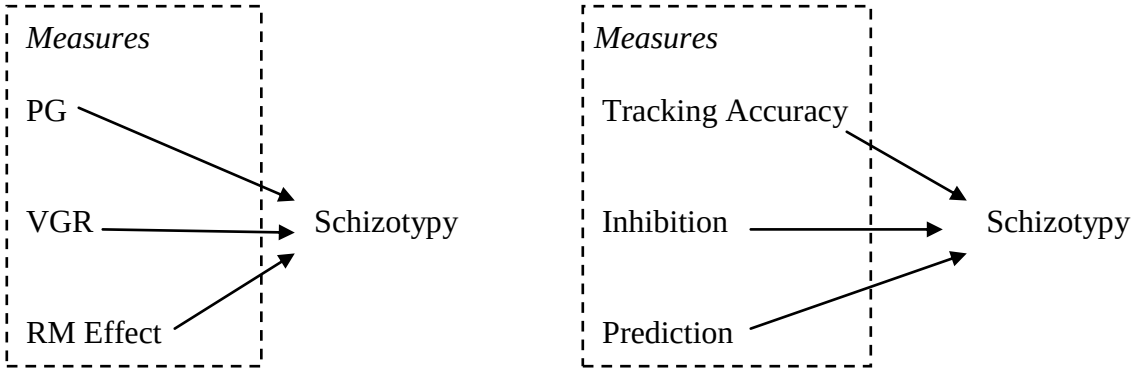


Figure 6. Proposed Model (1) for predicting schizotypy. The left model demonstrates the measured variables used to predict schizotypy scores. The right model demonstrates the umbrella terms the current study used to describe a measure that was used to predict schizotypy.

Both RM and the VGR tasks can demonstrate anomalies of inhibition. That is, in order to succeed in either task, an individual must suppress the representation of motion or a reflexive eye movement respectively. According to Holzman (2000) inhibition anomalies are vital indicators for identifying schizotypal risk. Thus, in Model (1), RM and the VGR weakened any association with schizotypy and therefore contributed to the non-significant results. However, by including these measures, it is vital that the eye tracking tasks are of the correct sensitivity. Holahan and O'Driscoll (2004) noted that it is difficult to find the correct sensitivity of antisaccade tasks for psychometrically defined schizotypal populations. Antisaccade studies have accordingly had mixed results in predicting subclinical symptoms. The cognitive demands of a task need to be optimally sensitive for the tested population so that those at risk can be reliably distinguished from those that are healthy. It therefore appears that the cognitive demands needed to be higher when testing a university population in order to detect in those at risk for schizotypy.

Additionally the RM task sensitivity may have also limited the current results. Jarrett et al. (2002) found that when individuals were categorised into high or low risk schizotype groups a significant RM effect (significant forward memory bias) was present in those identified as high risk for schizotypy but not in those identified as low risk. However, Jarrett et al. (2002) and Watkins (2005) demonstrated that the difference between high and low schizotypy groups could not be determined by the RM effect. Jarrett et al. (2002) and Watkins (2005) implied that people who have a greater risk of exhibiting schizotypical behaviour demonstrate a greater RM effect to the point where it is strongly exaggerated in those suffering from schizophrenia. The current study followed

this lead and used all participant data to determine the trend of the RM effect. However, the RM effect did not predict schizotypy as an individual predictor (albeit that it did so, though only very weakly, on the disorganised SPQ score). This consequently may be why the RM effect did not significantly contribute to the three-factor regression model.

I also investigated a categorical approach to schizotypal personality but the RM effect in the current study did not identify the differences between high schizotypy and non schizotypy. Jarrett et al. (2002) differentiated participants based on their SPQ scores into high and low risk schizotypy groups. Watkins (2005) also attempted this procedure but neither study found any significant differences in the RM effect between high and low schizotypes as indicated by the SPQ score. Since the novel approach in this study has been to research the RM effect in relation to robust biological marker for schizophrenia and schizotypy, the group effects for the RM effect were concurrently investigated. However, as with previous studies no significant differences could be found between high schizotypy and non schizotypy individuals in the RM effect.

Although no researchers have investigated both, a group/categorical and a continuous approach, the current results demonstrated that the RM effect could not identify schizotypal individuals, whether based on a continual or categorical approach to schizotypy. This provided further evidence to suggest that like the antisaccade task, the RM task was not sensitive enough to predict schizotypal behaviour or group membership. However, Jarrett et al. (2002) were able to find a quantitative difference in the RM effect when they compared a schizophrenia patient group and a low schizotypy group. This suggested, in line with the results of the current study and Watkins (2005) that the RM effect only differentiates schizophrenia populations from non-schizophrenia populations.

In summary, there are two possible explanations as to why the RM task did not contribute significantly to predicting schizotypy within the current population. Firstly, the RM task was not sensitive enough to distinguish between the subtle differences of those at high risk and those at low risk for schizotypy. Second, it appears that the RM effect phenomenon is only quantitatively different in those suffering from fully developed schizophrenia.

Heterogeneity amongst Schizotypal Eye Tracking

Previous researchers have suggested that schizotypal subgroups have different aetiologies and therefore different phenotypic expressions. Therefore, in the general population some eye tracking anomalies may only be expressed in a certain subtypes rather than being expressed similarly across all individuals at risk of schizophrenia. If this is the case then risk markers such as pursuit gain, the VGR and potentially RM should have been able to significantly predict at least one of the schizotypal subtypes. My hypothesis stated that the predictors would best identify those who scored high on the cognitive-perceptual scale. The justification for such a hypothesis was that a) the motor elements in the model were analogous to *perceptual* anomalies experienced by schizophrenia patients and, b) RM was analogous to the *cognitive* distortions that are experienced by schizophrenia patients. Although the models were unable to significantly predict any subtype of schizotypy, the results have supported a heterogeneity hypothesis of schizotypy. Each of the eye tracking measures was associated differently with each subtype of schizotypy. This is explained in greater depth below.

Task sensitivity has been associated to schizotypy symptom type (Holahan & O'Driscoll, 2004; Levy et al., 2004). Negative symptoms of schizotypy are harder to

detect through an antisaccade task than are positive symptoms (O'Driscoll et al., 1998; Larrison et al., 2000). As mentioned previously, there is clear evidence of distribution amongst schizophrenia and schizotypal subtypes on some psychophysiological measures, like eye tracking measurements (Clementz, Grove, lacono, & Sweeney, 1992). The SPEM task was the one task that demonstrated how important it was to get the sensitivity correct, dependent on the tested population. That was, there was not a significant relationship between any of the subtypes until the velocity of the stimulus was increased to 0.8Hz. It was not until this velocity that there was a relationship between those individuals who experienced greater cognitive-perceptual and disorganised distortions and poorer pursuit gain.

In response to previous findings regarding subtype symptoms I attempted to investigate whether symptom type confounded the relationship between the eye tracking tasks and the SPQ factor scores. However, the current studies sample size proved to not be sufficient to accommodate an acceptable statistical power for four mutually exclusive groups (negative, positive, disorganised symptoms and control). By neglecting differences in symptom type, the observation of the relationship included sub-groups less sensitive to the task, which contributed to the absence of a significant model. Had the current study been able to separate symptom type, a stronger association between the regression model and SPQ factor scores might have been observable. This forcefully underscores the need to develop testable disease models that take heterogeneity into account and extend the search for evidence of how robust risk markers can reliably identify subtypes of schizotypy.

Although the second hypothesis was not consistent with the current results, an objective was to investigate if RM was involved with biological markers of schizophrenia risk. Holzman (2000) suggested that the decrease in pursuit gain occurs when there is compromise of controlling and or inhibiting unwanted movement. As mentioned earlier, recent accounts suggest that the disruption in smooth pursuit is influenced by not being able to accurately predict an object's position (Lencer et al., 2010). The current study tested two alternative models. The first, (Figure 7, Model 2) was used to determine if controlling and inhibiting movement (as measured by VGR) was better at identifying schizotypy subtypes than prediction of an object's position (measured by RM- Figure 7, Model 3). Both models included tracking accuracy, because this has been the measure that researchers consider the most robust predictor of schizotypy. The results of the current study did not demonstrate any significant models that predicted schizotypy, cognitive-perceptual, interpersonal, or disorganised symptoms and therefore no subsequent comparisons were made. This finding is confusing because it was not consistent with either of the two arguments proposed in the introduction. They were a) that the prediction of movement contributes to the tracking abnormalities (Lencer et al.,

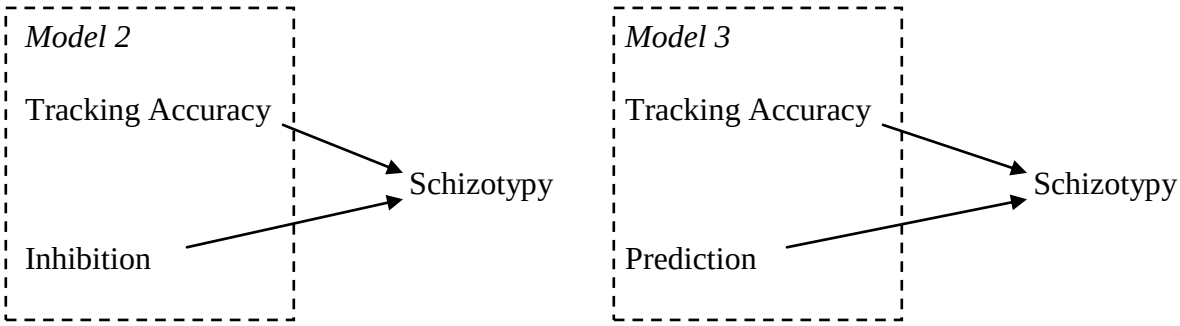


Figure 7. Models to predict schizotypy using two predictors. Both models used tracking accuracy (PG) whereas Model (2) used inhibition (VGR) and Model (3) used prediction of an objects position (RM effect).

2010), and b) that disruption of eye movement is due to initiation and control of eye movement (Holzman, 2000). Despite the current results, inhibition and prediction elements should still be acknowledged. As discussed in hypothesis one, disinhibition is suggested as one of the underlying factors that may contribute to the associations observed between task indices and schizotypy. Furthermore, RM was observed to be associated with disorganised symptoms in schizotypy and is an anomaly of schizophrenia (Jarrett et al., 2002). It is therefore advised that the true influence of both factors may be hidden by the lack of sensitivity of the corresponding tasks. This is later discussed as a limitation.

In summary, the predictors used in the tested models were unable to significantly predict SPQ cognitive-perceptual scores, or in fact any of the schizotypy scales. This and past research on the sensitivity of eye tracking tasks, provided an explanation as to why the tested indices were perhaps not able to predict a specific subtype of schizotypy. Upon further investigation of the RM effect by comparing it to the VGR, there was still no evidence that suggested that prediction has a more important role in identifying risk of schizotypy than inhibition. This was contradictory to the assumptions made by Lencer et al., (2010) and Spering & Montagnini, (2010).

Interpretation of Current Findings

The current study suggests that the prediction of an object's position is not a significant factor that influences eye movement within the current population. There are several pieces of evidence from the current study that corroborate the above statement. Firstly, there was no relationship found between RM and a) cognitive-perceptual SPQ scores, b) interpersonal SPQ scores and c) total SPQ score. Secondly,RM did not improve

any of the models for predicting schizotypy, or any subtypes of schizotypy and none of the models that included representational momentum were significant. Thirdly, beta values indicated that RM was not a significant predictor in any of the tested models. Finally, when high and low schizotypes were identified using the SPQ, there was no difference in the RM effect between the two groups.

Even though the central hypothesis was not supported there are some interesting findings in the current study. There was for example a significant association between RM and the SPQ disorganised scale. Furthermore, pursuit gain has been found to be associated to the disorganised scale, as well as to the cognitive-perceptual scale. The VGR showed a significant relation with cognitive-perceptual scale, but not the disorganised scale. As shown in Table 11, all of the aforementioned eye tracking indices are also correlated, which can be understood as supportive of the idea that there is one underlying mechanism common to all of these indices. As suggested above, this mechanism may be disinhibition or a similar process. The schizotypy specific poor performance in the antisaccade, SPEM and RM tasks would accordingly be determined by the impaired ability to halt automatic cognitive processes.

Furthermore it is noteworthy that the current results demonstrated heterogeneity. What is interesting is that none of the eye tracking indices was associated with the interpersonal (negative symptom). Each of these indices was related to positive or disorganised symptoms. This is consistent with the literature that suggests negative symptoms in schizotypy are not related to eye tracking anomalies.

Finally, it may be hypothesised that some of the eye tracking anomalies may only be fully developed and therefore observable in those who suffer from actual

schizophrenia, rather than those who demonstrate some schizotypal traits as represented by high scores on SPQ. This explanation is supported by a peculiarity observed in the SPEM task. No associations have been found between the 0.4 Hz and 0.6 Hz conditions with any of the SPQ factors or with the SPQ total score. However, the most robust association was between schizotypy and the pursuit gain of the 0.8Hz condition.

In the past, other researchers have tested several SPEM frequency conditions within and across an array of populations (Allen, 1997). However, no researchers have discussed why there is an observable difference across differing populations. The aforementioned observation suggests that the task at slower frequencies (0.4 and 0.6Hz) is insensitive to individuals with higher cognitive functioning. It has been shown that for schizophrenia patients, 0.4Hz is sufficiently sensitive to determine smooth pursuit deficits (Holzman, 2000). However, the finding in the current study indicates that this frequency may not be sufficient for differentiating between members of a population which has a more extensive educational background and most probably a higher level of cognitive functioning. It may be that the sensitivity issue had as well an impact on the RM and the antisaccade task, which might have been derogatory to the hypothesised associations. In a somewhat similar instance, Jarrett et al. (2002) only found a difference in the RM effect between a low schizotypal group and patients with schizophrenia, but not between high and low schizotypal groups. In the current study, the attempt was made to categorise individuals into high schizotypes and non schizotypes. However it appears that the disparity within the tested population is not sufficiently big to demonstrate a difference in pursuit gain performance, antisaccade performance or the RM effect.

Overall, there was no adequate evidence to support the formulated hypotheses relating to representational momentum. However, the relationship between the eye tracking indices implied areas of possibly promising research. As mentioned before the degree to which these anomalies can actually be viewed in the general population may be such an area. Although the evidence from the current study suggested that movement prediction does not influence eye tracking anomalies, it may be that it is only apparent across or within different populations, for example as a result of fully developed schizophrenia. It remains to be seen what role the representation of momentum plays in the context of schizotypal anomalies however further research will need to be done to determine whether the representation of momentum is an important piece of the puzzle.

Limitations and Issues

The interpretation of findings is constrained by elements of the study design and population related variables. A procedural limitation concerning the RM task is that the picture scenes were not identical to the picture scenes from other RM studies. Fifty per cent were taken from the Watkins (2005) study and fifty per cent were re-designed for the current study. Although our aim was to rectify some methodological limitations of previous studies through this procedure, it brought about new difficulties. Not using the exact same picture scenes as previous researchers limits the current findings regarding the RM effect because researchers in the past have indicated that the RM effect can be influenced by expectations that the participant might have of the identified target (Reed & Vinson, 1996), velocity (Freyd & Finke, 1985), target behaviour (Freyd & Finke, 1984), and implied weight and friction experienced (Hubbard, 1997). Since the current study could not entirely control for all these possible effects on the RM task, the results

should be acknowledged with a degree of caution.

Secondly, the SPEM task used five full oscillations to determine the total time of a single trial. Depending on the condition, the total amount of smooth pursuit time differed across trials. Therefore, trials with lower frequency (i.e. in the 0.4 Hz condition) spanned for longer time periods than higher frequency trials (0.8 Hz). Consequently in lower frequency trials there is a potential for more errors, more opportunity for saccadic intrusions and larger attention spans are required. Although the current study results demonstrated that the higher frequency trials were the most diagnostic, this was only for pursuit gain. The limitations above may have been responsible for the current study to not be able to make assumptions regarding measure that are more vulnerable to extended time frames- the saccadic intrusion and root mean squared error data. In addition to longer time frames, attention may have also been a problem. Rather than measuring how well the eye tracks a target, the measures may have well been corrupted by attention spans, causing laziness or the eyes to lag behind the stimulus on purpose. The current study did not control for attention.

Thirdly, the current study did not apply any exclusion criteria. This is limiting in a student population because there is no control over possible influences of psychoactive substances such as alcohol consumption the night before. Several researchers have suggested that alcohol (even at doses as low as 2ml/ kg) can disrupt SPEM (Levy, Lipton, Holzman & 1981) and saccade velocity (Baloh, Sharma, Moskowitz & Griffith, 1979) for up to 24hours post-consumption. As for the antisaccade task the effects of alcohol vary. Alcohol can either increase (Kahn, Ford & Timney, 2003) or decrease errors (Crevitis, Hanse, Tummers & Van Maele, 2000). Not controlling for influence of psychoactive

substances could have skewed the current results causing typically developing individuals to perform much closer to that of a patient with schizophrenia.

Recommendations for Future RM Research

It is important for future researchers to be aware of the above limitations within the current study. With regard to such limitations are proposed solutions that may dampen such effects. With regard to the RM stimuli, a suggestion would be to use white backgrounds, no shadows and to keep the stimuli aesthetics as similar as possible. By doing this, it gives all stimuli sets the same amount of informative cues an individual can gain. Alternatively, these factors could be tested to see if they do make a difference in results. Future studies should investigate the sensitivity of picture expectation to look at whether the mentioned factors are influenced by spatial distance or displacement in time. It will consequently be necessary to develop a standardized validated form of the RM task to control for the influence of weight, friction, semantic meaning, and target behaviour.

With attention and trial length limitations in the current study, it is suggested that future researchers determine the length of the trial by time rather than oscillations especially when using several frequency conditions. Furthermore, telling participants that there will be subtle changes in the target stimulus can facilitate attention spans. Chen, Levy, Nakayama, Matthysse, Palafox and Holzman (1999), for example, told 15 schizophrenia patients and eight controls that the stimulus would change colour and they were to count the number of times this change occurred. This type of design did not alter the results that are observed in SPEM studies and served to facilitate attention spans. Furthermore, previous researchers have used time as the prime candidate for trial length

opposed to number of oscillations. For example, Allen (1997) used 12s of continuous tracking and Holzman, O'Brian and Waternaux (1991) used 30s of 0.4 Hz sinusoidal tracking.

It is necessary for future researchers to apply exclusion criteria. Some suggestions are to exclude individuals who have consumed alcohol at least 24 hours prior to testing. Furthermore, because the current study is specifically interested in the pathology of schizophrenia, it is arguable that the exclusion of those who have a history of other mental illnesses would increase the specificity of the results.

One final piece of future research advice could be to switch the model around. I used SPQ scores as the dependent variable, and the eye tracking indices as the predictors in my model. It could be equally sound to use SPQ scores as predictors of one or more SPEM, antisaccade or representational momentum indices. One advantage of the latter is that objective measures may have less error variance. For example, no self-reporting bias and random responding are more readily identified and eliminated.

Future researchers who wish to direct their study with eye tracking and representational momentum attempt to provide directional and causal models. The current study did not attempt to argue for causality, nor did I argue a particular direction. That was, the relationship between schizotypy and eye tracking predictors may be directional. This would certainly be interesting to design a longitudinal study that would help to determine this. I advise that future researchers also us path analysis, rather than multiple regression techniques to determine the influence of representational momentum on eye tracking anomalies. It would be interesting to see whether prediction of an objects position mediates and or moderates the relationship between risk for schizophrenia and

eye tracking tasks such as the antisaccade and SPEM task. This would truly help to determine the influence prediction has in how we view the world.

Conclusion

It is widely held that smooth pursuit eye tracking is the most robust biological marker of risk for schizophrenia and schizotypy. However, the evidence suggests that there is still a considerable need to understand the processes involved in smooth pursuit eye tracking. Therefore, the current study investigated the role of three factors that may contribute to the disruption of smooth pursuit. These were tracking accuracy (pursuit gain), inhibition (visual grasp reflex) and prediction (RM effect). The current study was specifically focused on the role of the RM effect, as in the context of other visual measures this has not been explored as a predictor for schizotypy. Furthermore, the current study also investigated the role of RM and eye tracking measures to provide further evidence for the heterogeneity of schizotypy. The main findings of the current study were (a) pursuit gain was the most robust marker associated with schizotypal factor scales such as the disorganised and cognitive-perceptual scores, b) RM was only significantly associated with SPQ disorganised score, and (c) that a model comprised of pursuit gain, VGR and the RM effect was not able to significantly predict any schizotypy subtype.

The association between pursuit gain and schizotypy added to a long line of literature that supports the deficit observed in high-risk schizotypal persons in smooth pursuit eye movement. The models proposed were novel attempts to predict schizotypy but it did not achieve statistically significant predictive power and therefore was not consistent with the current hypothesis. The reason for the lack of significance may have

been due to RM and antisaccade task insensitivity for the current population. I also suggested that the anomalies in RM are only observable when schizophrenia is fully developed. The final finding suggested that prediction of an objects position was not an influential predictor of schizotypal behaviours.

To pursue the idea that representational momentum plays a part in the eye tracking anomalies future researchers first need to establish an RM task with optimised sensitivity and address the limitations previously suggested. Secondly, it was advised that future RM research should focus on a model that can be tested using path analysis to determine whether RM is a moderator or a mediator variable. Accordingly, future researchers should be able to determine whether the prediction of an object's movement is crucial when considering objective biological markers for individuals at risk for schizotypy. These minor systematic anomalies need to be exploited to aid understanding of both normal and abnormal brain function. In that way, we can begin to fully appreciate how we perceive a world in which events occur, objects move and situations change throughout time.

References

Allen, J. S. (1997). Smooth pursuit eye movements in schizophrenia in New Zealand. *Australian and New Zealand Journal of Psychiatry, 31*, 582-591.

Amorim, M. A., Lang, W., Lindinger, G., Mayer, D., Deecke, L., & Berthoz, A. (2000). Modulation of spatial orientation processing by mental imagery instructions: A MEG study of representational momentum. *Journal of Cognitive Neuroscience, 12*, 569-582.

Andreasen, N. C. (1979). Thought, language, and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry, 36*, 1325.

Andreasen, N. C. (1996). Pieces of the schizophrenia puzzle fall into place. *Neuron, 16*, 697.

Andreasen, N. C. (1999). Understanding the causes of schizophrenia (Vol. 340, pp. 645-647).

Andreasen, N. C., Nopoulos, P., O'Leary, D. S., Miller, D. D., Wassink, T., & Flaum, M. (1999). Defining the phenotype of schizophrenia: cognitive dysmetria and its neural mechanisms. *Biological Psychiatry, 46*, 908-920.

Andreasen, N. C., Paradiso, S., & O'Leary, D. S. (1998). "Cognitive dysmetria" as an integrative theory of schizophrenia: a dysfunction in cortical-subcortical-cerebellar circuitry? *Schizophrenia Bulletin, 24*, 203.

Avila, M. T., Hong, L. E., Moates, A., Turano, K. A., & Thaker, G. K. (2006). Role of anticipation in schizophrenia-related pursuit initiation deficits. *95*, 593-601.

Baloh, R. W., Sharma, S., Moskowitz, H., & Griffith, R. (1979). Effect of alcohol and marijuana on eye movements. *Aviation, space, and environmental medicine, 50*, 18.

Barnes, G. R. (2008). Cognitive processes involved in smooth pursuit eye movements. *Brain and Cognition, 68*, 309-326.

Beck, A. T., & Rector, N. A. (2005). Cognitive approaches to schizophrenia: theory and therapy. *Clinical Psychology, 1*, 577.

Bennett, P. (2006). *Abnormal and clinical psychology: an introductory textbook*: Open University Press.

Bleuler, E. (1908). Die prognose der dementia praecox (schizophreniegruppe). *Allgemeine Zeitschrift für Psychiatrie, 65*, 436-464.

Bleuler, E., & Zinkin, J. (1950). Dementia praecox or the group of schizophrenias. *New York, 199*, 211-301.

Boudet, C., Bocca, M. L., Chabot, B., Delamillieure, P., Brazo, P., Denise, P., et al. (2005). Are eye movement abnormalities indicators of genetic vulnerability to schizophrenia? *European Psychiatry, 20*, 339-345.

Broerse, A., Crawford, T. J., & Den Boer, J. A. (2002). Differential Effects of Olanzapine and Risperidone on Cognition in Schizophrenia?: A Saccadic Eye Movement Study. *Journal of Neuropsychiatry and Clinical Neurosciences, 14*, 454.

Chan, R. C. K., & Gottesman, I. I. (2008). Neurological soft signs as candidate endophenotypes for schizophrenia: A shooting star or a Northern star? *Neuroscience & Biobehavioral Reviews, 32*, 957-971.

Chen, Y., Levy, D. L., Nakayama, K., Matthysse, S., Palafox, G., & Holzman, P. S. (1999). Dependence of impaired eye tracking on deficient velocity discrimination in schizophrenia. *Archives of General Psychiatry, 56*, 155.

Clementz, B. A., Grove, W. M., Iacono, W. G., & Sweeney, J. A. (1992). Smooth-pursuit eye movement dysfunction and liability for schizophrenia: Implications for genetic modeling. *Journal of Abnormal Psychology, 101*, 117-129.

Clementz, B. A., McDowell, J. E., & Zisook, S. (1994). Saccadic system functioning among schizophrenia patients and their first-degree biological relatives. *Journal of Abnormal Psychology, 103*, 277-287.

Crevits, L., Hanse, M. C., Tummers, P., & Van Maele, G. (2000). Antisaccades and remembered saccades in mild traumatic brain injury. *Journal of neurology, 247*, 179-182.

Curtis, C. E., Calkins, M. E., Grove, W. M., Feil, K. J., & Iacono, W. G. (2001). Saccadic disinhibition in patients with acute and remitted schizophrenia and their first-degree biological relatives. *American Journal of Psychiatry, 158*, 100.

Curtis, C. E., & D'Esposito, M. (2003). Persistent activity in the prefrontal cortex during working memory. *Trends in Cognitive Sciences, 7*, 415-423.

Curtis, V. A., Bullmore, E. T., Morris, R. G., Brammer, M. J., Williams, S. C. R., Simmons, A., et al. (1999). Attenuated frontal activation in schizophrenia may be task dependent. *Schizophrenia research, 37*, 35-44.

David, A. S. (1994). Dysmodularity: a neurocognitive model for schizophrenia. *Schizophrenia Bulletin, 20*, 249.

David, A. S., & Senior, C. (2000). Implicit motion and the brain. *Trends in Cognitive Sciences, 4*, 293-295.

Dickey, C. C., McCarley, R. W., & Shenton, M. E. (2002). The brain in schizotypal personality disorder: a review of structural MRI and CT findings. *Harvard review of psychiatry, 10*, 1-15.

Dickey, C. C., McCarley, R. W., Voglmaier, M. M., Niznikiewicz, M. A., Seidman, L. J., Hirayasu, Y., et al. (1999). Schizotypal personality disorder and MRI abnormalities of temporal lobe gray matter. *Biological Psychiatry, 45*(1), 1393-1402.

Edgin, J. O., Inder, T. E., Anderson, P. J., Hood, K. M., Clark, C. A. C., & Woodward, L. J. (2008). Executive functioning in preschool children born very preterm: relationship with early white matter pathology. *Journal of the international neuropsychological society, 14*, 90-101.

Ettinger, U., Kumari, V., Crawford, T. J., Davis, R. E., Sharma, T., & Corr, P. J. (2003). Reliability of smooth pursuit, fixation, and saccadic eye movements. *Psychophysiology, 40*, 620-628.

Finke, R. A., & Freyd, J. J. (1985). Transformations of visual memory induced by implied motions of pattern elements. *Journal of experimental psychology. Learning, memory, and cognition, 11*, 780.

Finke, R. A., Freyd, J. J., & Shyi, G. C. W. (1986). Implied velocity and acceleration induce transformations of visual memory. *Journal of Experimental Psychology: General, 115*, 175-188.

Freyd, J. J. (1983). The mental representation of movement when static stimuli are viewed. *Perception & Psychophysics, 33*, 575.

Freyd, J. J., & Finke, R. A. (1984). Representational momentum. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 10*, 126-132.

Freyd, J. J., & Finke, R. A. (1985). A velocity effect for representational momentum. *Bulletin of the Psychonomic Society, 23*, 443-446.

Freyd, J. J., & Johnson, J. Q. (1987). Probing the time course of representational momentum. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 13*, 259–268.

Freyd, J. J., & Miller, G. F. (1992). *Creature motion.* Paper presented at the Thirty-third Annual Meeting of the Psychonomic Society.

Frith, C. D. (1981). Schizophrenia: An abnormality of consciousness. *Aspects of consciousness, 2*, 149-168.

Gooding, D. C. (1999). Antisaccade task performance in questionnaire-identified schizotypes. *Schizophrenia research, 35*, 157-166.

Gooding, D. C., & Basso, M. A. (2008). The tell-tale tasks: a review of saccadic research in psychiatric patient populations. *Brain and Cognition, 68*, 371-390.

Gooding, D. C., Miller, M. D., & Kwapil, T. R. (2000). Smooth pursuit eye tracking and visual fixation in psychosis-prone individuals. *Psychiatry Research, 93*, 41-54.

Haraldsson, H. M., Ettinger, U., Magnusdottir, B. B., Sigmundsson, T., Sigurdsson, E., & Petursson, H. (2008). Eye movement deficits in schizophrenia. *European archives of psychiatry and clinical neuroscience, 258*, 373-383.

Hayes, A., & Freyd, J. J. (1995). *Attention and representational momentum.* Paper presented at the Thirty-sixth Annual Meeting of the Psychonomic Society

Hayes, A. E., & Freyd, J. J. (2002). Representational momentum when attention is divided. *Visual Cognition, 9*, 8-27.

Holahan, A. L. V., & O'Driscoll, G. A. (2005). Antisaccade and smooth pursuit performance in positive-and negative-symptom schizotypy. *Schizophrenia research, 76*, 43-54.

Holzman, P. S. (1987). Recent studies of psychophysiology in schizophrenia. *Schizophrenia Bulletin, 13*, 49.

Holzman, P. S. (2000). Eye movements and the search for the essence of schizophrenia. *Brain research reviews, 31*, 350-356.

Holzman, P. S., O'Brian, C., & Waternaux, C. (1991). Effects of lithium treatment on eye movements. *Biological Psychiatry, 29*, 1001-1015.

Hubbard, T. L. (1997). Target size and displacement along the axis of implied gravitational attraction: Effects of implied weight and evidence of representational gravity. *Learning, Memory, 23*, 1484-1493.

Hubbard, T. L., & Ruppel, S. E. (1999). Representational momentum and the landmark attraction effect. *Canadian Journal of Experimental Psychology, 53*, 242-256.

Hutton, S. B., Joyce, E. M., Barnes, T. R. E., & Kennard, C. (2002). Saccadic distractibility in first-episode schizophrenia. *Neuropsychologia, 40*, 1729-1736.

Jarrett, C. B., Phillips, M., Parker, A., & Senior, C. (2002). Implicit motion perception in schizotypy and schizophrenia: A Representational Momentum study. *Cognitive neuropsychiatry, 7*, 1-14.

Kaprinis, G., Konstantinos, F., & Stergios, K. (2008). Arguments against cognitive dysmetria hypothesis of schizophrenia. *Annals of General Psychiatry, 7*, Supplement 1: S317.

Karoumi, B., Saoud, M., d'Amato, T., Rosenfeld, F., Denise, P., Gutknecht, C., et al. (2001). Poor performance in smooth pursuit and antisaccadic eye-movement tasks in healthy siblings of patients with schizophrenia. *Psychiatry Research, 101*, 209-219.

Katsanis, J., & Iacono, W. G. (1991). Clinical, neuropsychological, and brain structural correlates of smooth-pursuit eye tracking performance in chronic schizophrenia. *Journal of Abnormal Psychology, 100*, 526-534.

Katsanis, J., Kortenkamp, S., Iacono, W. G., & Grove, W. M. (1997). Antisaccade Performance in Patients With Schizophrenia and Affective Disorder. *Journal of Abnormal Psychology, 106*, 468-472.

Keefe, R. S. E., Silva, S. G., Perkins, D. O., & Lieberman, J. A. (1999). The effects of atypical antipsychotic drugs on neurocognitive impairment in schizophrenia: A review and meta-analysis. *Schizophrenia Bullitin, 25,* 201-227.

Kelley, M. P., & Bakan, P. (1999). Eye tracking in normals: SPEM asymmetries and association with schizotypy. *International Journal of Neuroscience, 98*, 27-81.

Kerzel, D. (2000). Eye movements and visible persistence explain the mislocalization of the final position of a moving target. *Vision Research, 40*, 3703-3715.

Khan, S. A., Ford, K., Timney, B., & Everling, S. (2003). Effects of ethanol on anti-saccade task performance. *Experimental Brain Research, 150*, 68-74.

Kraepelin, E. (1981). Clinical psychiatry: New York. *Scholars, Facsimiles and Reprints*.

Kuperberg, G., & Heckers, S. (2000). Schizophrenia and cognitive function. *Current Opinion in Neurobiology, 10*, 205-210.

Lahuis, B. E., Van Engeland, H., Cahn, W., & Kemner, C. (2008). Smooth pursuit eye movement (SPEM) in patients with multiple complex developmental disorder (MCDD), a subtype of the pervasive developmental disorder. *World Journal of Biological Psychiatry, 99999*, 1-8.

Larrison, A. L., Ferrante, C. F., Briand, K. A., & Sereno, A. B. (2000). Schizotypal traits, attention and eye movements. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 24*, 357-372.

Lee, K. H., & Williams, L. M. (2000). Eye movement dysfunction as a biological marker of risk for schizophrenia. *Australian and New Zealand Journal of Psychiatry, 34*, 91-100.

Lencer, R., Reilly, J. L., Harris, M. S., Sprenger, A., Keshavan, M. S., & Sweeney, J. A. (2010). Sensorimotor transformation deficits for smooth pursuit in first-episode affective psychoses and schizophrenia. *Biological Psychiatry, 67*, 217-223.

Lenzenweger, M. F. (2006). Schizotaxia, schizotypy, and schizophrenia: Paul E. Meehl's blueprint for the experimental psychopathology and genetics of schizophrenia. *Journal of Abnormal Psychology, 115*, 195.

Lenzenweger, M. F., Maher, B. A., & Manschreck, T. C. (2005). Paul E. Meehl's influence on experimental psychopathology: Fruits of the nexus of schizotypy and schizophrenia, neurology, and methodology. *Journal of clinical psychology, 61*, 1295-1315.

Levin, S. (1984). Frontal lobe dysfunctions in schizophrenia: Eye movement impairments. *Journal of psychiatric research, 18*, 27-55.

Levy, D. L., Holzman, P. S., Matthysse, S., & Mendell, N. R. (1993). Eye tracking dysfunction and schizophrenia: a critical perspective. *Schizophrenia Bulletin, 19*, 461.

Levy, D. L., Lipton, R. B., & Holzman, P. S. (1981). Smooth pursuit eye movements: effects of alcohol and chloral hydrate. *Journal of psychiatric research, 16*, 1.

Levy, D. L., O'Driscoll, G., Matthysse, S., Cook, S. R., Holzman, P. S., & Mendell, N. R. (2004). Antisaccade performance in biological relatives of schizophrenia patients: a meta-analysis. *Schizophrenia research, 71*, 113-125.

Linscott, R. J., Allardyce, J., & van Os, J. (2009). Online material supplementary to: Seeking verisimilitude in a class: a systematic review of evidence that the criterial clinical symptoms of schizophrenia are taxonic. *Schizophrenia Bulletin*.

Linscott, R. J., & Knight, R. G. (2004). Potentiated automatic memory in schizotypy. *Personality and Individual Differences, 37*, 1503-1517.

Lipton, R. B., Levy, D. L., Holzman, P. S., & Levin, S. (1983). Eye movement dysfunctions in psychiatric patients: a review. *Schizophrenia Bulletin, 9*, 13.

Machado, L., & Rafal, R. D. (2004). Control of fixation and saccades during anti-saccade task: an investigation in humans with chronic lesions of oculomotor cortex. *Experimental Brain Research, 156*, 55-63.

Maruff, P., Danckert, J., Pantelis, C., & Currie, J. (1998). Saccadic and attentional abnormalities in patients with schizophrenia. *Psychological Medicine, 28*, 1091-1100.

Matsue, Y., Osakabe, K., Saito, H., Goto, Y., Ueno, T., Matsuoka, H., et al. (1994). Smooth pursuit eye movements and express saccades in schizophrenic patients. *Schizophrenia research, 12*, 121-130.

Matthysse, S., Holzman, P. S., & Lange, K. (1986). The genetic transmission of schizophrenia: Application of Mendelian latent structure analysis to eye tracking dysfunctions in schizophrenia and affective disorder. *Journal of psychiatric research, 20*, 57.

McDowell, J. E., & Clementz, B. A. (1997). The effect of fixation condition manipulations on antisaccade performance in schizophrenia: studies of diagnostic specificity. *Experimental Brain Research, 115*, 333-344.

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist, 17*, 827-838.

Meehl, P. E. (1990). Toward an integrated theory of schizotaxia, schizotypy, and schizophrenia. *Journal of Personality Disorders, 4*, 1-99.

Nieuwenhuis, S., Broerse, A., Nielen, M., & Jong, R. (2004). A goal activation approach to the study of executive function: An application to antisaccade tasks. *Brain and Cognition, 56*, 198-214.

Nkam, I., Thibaut, F., Denise, P., Van Der Elst, A., Segard, L., Brazo, P., et al. (2001). Saccadic and smooth-pursuit eye movements in deficit and non-deficit schizophrenia. *Schizophrenia research, 48*, 145-153.

O'Driscoll, G. A., Lenzenweger, M. F., & Holzman, P. S. (1998a). Antisaccades and smooth pursuit eye tracking and schizotypy. *Archives of General Psychiatry, 55*, 837-843.

O'Driscoll, G. A., Lenzenweger, M. F., & Holzman, P. S. (1998b). Antisaccades and smooth pursuit eye tracking and schizotypy. *Archives of General Psychiatry, 55*, 837.

O'Driscoll, G. A., Strakowski, S. M., Alpert, N. M., Matthysse, S. W., Rauch, S. L., Levy, D. L., et al. (1998). Differences in cerebral activation during smooth pursuit and saccadic eye movements using positron-emission tomography. *Biological Psychiatry, 44*, 685-689.

O'Driscoll, G. A., & Callahan, B. L. (2008). Smooth pursuit in schizophrenia: a meta-analytic review of research since 1993. *Brain and Cognition, 68*, 359-370.

Park, S., & Holzman, P. S. (1992). Schizophrenics show spatial working memory deficits. *Archives of General Psychiatry, 49*, 975-982.

Park, S., & McTigue, K. (1997). Working memory and the syndromes of schizotypal personality. *Schizophrenia research, 26*, 213-220.

Ploner, C. J., Gaymard, B. M., Rivaud-Péchoux, S., & Pierrot-Deseilligny, C. (2005). The prefrontal substrate of reflexive saccade inhibition in humans. *Biological Psychiatry, 57*, 1159-1165.

Raine, A. (1991). The SPQ: A scale for the assessment of schizotypal personality based on DSM-III-R criteria. *Schizophrenia Bulletin, 17*, 556-564.

Reed, C. L., & Vinson, N. G. (1996). Conceptual effects on representational momentum. *Journal of Experimental Psychology, 22*, 839-850.

Reuter, B., Rakusan, L., & Kathmanna, N. (2005). Poor antisaccade performance in schizophrenia: An inhibition deficit? *Psychiatry Research, 135*, 1.

Ross, R. G., Heinlein, S., Zerbe, G. O., & Radant, A. (2005). Saccadic eye movement task identifies cognitive deficits in children with schizophrenia, but not in unaffected child relatives. *Journal of Child Psychology and Psychiatry, 46*, 1354-1362.

Seidman, L. J., Cassens, G. P., Kremen, W. S., & Pepple, J. R. (1992). Neuropsychology of schizophrenia. *Clinical syndromes in adult neuropsychology: The practitioner's handbook*, 213-251.

Senior, C. (2000). *A magnetic stimulation study of "representational momentum"*. Unpublished MSc thesis, University of London.

Sereno, A. B., & Holzman, P. S. (1995). Antisaccades and smooth pursuit eye movements in schizophrenia. *Biological Psychiatry, 37*, 394-401.

Siever, L., Keefe, R., Bernstein, D.P., Coccaro, E.F., Klar, H.M., Zemishlany, Z., Peterson, A.E., Davidson, M., Mahon, T., Horvath, T., & Mohs, R. (1990). Eye tracking impairment in clinically identified patients with schizotypal personality disorder. *American Journal of Psychiatry, 147*, 740-745.

Siever, L.J., Friedman, L., Moskowitz, J., Mitropoulou, V., Keefe, R., Roitman, S.L., Merhige, D., Trestman, R., Silverman, J., & Mohs, R. (1994). Eye movement impairment and schizotypal psychopathology. *American Journal of Psychiatry, 151*, 1209-1215.

Simons, R.F., & Katkin, W. (1985). Smooth pursuit eye movements in subjects reporting physical anhedonia and perceptual aberrations. *Psychiatry Research, 14*, 275-289.

Slaghuis, W. L., Holthouse, T., Hawkes, A., & Bruno, R. (2007). Eye movement and visual motion perception in schizophrenia II: global coherent motion as a function of target velocity and stimulus density. *Experimental Brain Research, 182*, 415-426.

Smyrnis, N., Evdokimidis, I., Mantas, A., Kattoulas, E., Stefanis, N., Constantinidis, T., Avramopoulos, D., & Stefanis, C. N. (2007). Smooth pursuit eye movements in 1,087 men: effects of schizotypy, anxiety, and depression. *Experimental Brain Research, 179*, 397-408.

Spering, M., & Montagnini, A. (2010). Do we track what we see? Common versus independent processing for motion perception and smooth pursuit eye movements: A review. *Vision Research*(In press).

Spitzer, M., Braun, U., Hermle, L., & Maier, S. (1993). Associative semantic network dysfunction in thought-disordered schizophrenic patients: Direct evidence from indirect semantic priming. *Biological Psychiatry, 34*, 864-877.

Stuve, T. A., Friedman, L., Jesberger, J. A., Gilmore, G. C., Strauss, M. E., & Meltzer, H. Y. (1997). The relationship between smooth pursuit performance, motion perception and sustained visual attention in patients with schizophrenia and normal controls. *Psychological Medicine, 27*, 143-152.

Sweeney, J. A., Haas, G. L., & Li, S. H. (1992). Neuropsychological and eye-movement abnormalities in 1st-episode and chronic-schizophrenia. *Psychiatry Research, 54*, 185-198.

Taylor, N. M., & Jakobson, L. S. (2010). Representational momentum in children born preterm and at term. *Brain and Cognition, 72*, 464-471.

Thornton, I. M., & Hubbard, T. L. (2002). Representational momentum: New findings, new directions. *Visual Cognition, 9*, 1-7.

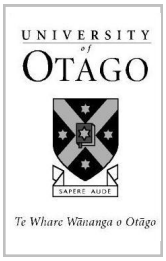
Tsuang, M. T., & Faraone, S. V. (1995). The case for heterogeneity in the etiology of schizophrenia. *Schizophrenia Research, 17*, 161-175.

Tsuang, M. T., Stone, W. S., Tarbox, S. I., & Faraone, S. V. (2002). An integration of schizophrenia with schizotypy: identification of schizotaxia and implications for research on treatment and prevention. *Schizophrenia research, 54*, 169-175.

Watkins, P. (2005). *Representational momentum in individuals at risk for schizophrenia.* Unpublished MSc thesis, University of Otago.

Woods, B. T. (1998). Is schizophrenia a progressive neurodevelopmental disorder? Toward a unitary pathogenetic mechanism. *155*, 1661-1670.

Appendix



REPRESENTATIONAL MOMENTUM AND PERSONALITY

INFORMATION SHEET FOR PARTICIPANTS

Thank you for showing an interest in this project. Please read this information sheet carefully before deciding whether or not to participate. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the Aim of the Project?

The project aims to investigate whether Representational Momentum (RM) performance can be correlated with differences in eye tracking (Antisaccade and Smooth Pursuit Eye Movement) tasks. In particular we are examining whether these effortless cognitive tasks differ between personality groups. This project is being undertaken as part of the requirements for a Master of Science Thesis.

What Type of Participants are being sought?

We are seeking undergraduate students in Psychology. People who are under the age of 17 will not be able to participate in the project.

What will Participants be Asked to Do?

Should you agree to take part in this project, you will be asked to complete a demographic questionnaire and the Raine Personality Questionnaire. Once this is completed you will then be asked to participate in the Smooth Pursuit Eye Movement (SPEM), Antisaccade and RM tasks. These are all computer-based tasks, which will take no more than 10 minutes each. The tasks are designed to be interesting and enjoyable.

Please be aware that you may decide not to take part in the project without any disadvantage to yourself of any kind.

Can Participants Change their Mind and Withdraw from the Project?

You may withdraw from participation in the project at any time and without any disadvantage to yourself of any kind.

What Data or Information will be Collected and What Use will be Made of it?
We will record your SPEM, Antisaccade and RM task performances along with your responses to the questionnaires. The responses you give will be collated with other participants' results and analysed to look at overall patterns in the data. This data may be used for presentations or publications. All of your answers will be kept confidential and you will not be identified in the research project or any publication of the research, as the personal information you provide will be kept separate from the data.

The results of the project may be published and will be available in the University of Otago Library (Dunedin, New Zealand) but every attempt will be made to preserve your anonymity.

You are most welcome to request a copy of the results of the project should you wish.

The data collected will be securely stored in such a way that only those mentioned below will be able to gain access to it. At the end of the project any personal information will be destroyed immediately except that, as required by the University's research policy, any raw data on which the results of the project depend will be retained in secure storage for five years, after which it will be destroyed.

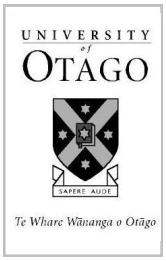
What if Participants have any Questions?
If you have any questions about our project, either now or in the future, please feel free to contact either:

Dominic Vettise,
Research Student,
Department of Psychology
Tel. 479 5681
E-mail: vetdo958@student.otago.ac.nz

Dr. Richard Linscott,
Senior Lecturer,
Department of Psychology
Tel. 479-5689
E-mail: linscott@psy.otago.ac.nz

This study has been approved by the University of Otago Human Ethics Committee. If you have any concerns about the ethical conduct of the research you may contact the Committee through the Human Ethics Committee Administrator (ph 03 479 8256). Any issues you raise will be treated in confidence and investigated and you will be informed of the outcome.

Appendix



REPRESENTATIONAL MOMENTUM AND PERSONALITY

CONSENT FORM FOR PARTICIPANTS

I have read the Information Sheet concerning this project and understand what it is about. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage.
I know that:

1. My participation in the project is entirely voluntary;

2. I am free to withdraw from the project at any time without any disadvantage;

3. Personal identifying information will be destroyed at the conclusion of the project but any raw data on which the results of the project depend will be retained in secure storage for five years, after which they will be destroyed;

4. The procedures used are not physically harmful and do not cause discomfort.

5. The results of the project may be published and will be available in the University of Otago Library (Dunedin, New Zealand) but every attempt will be made to preserve my anonymity.

I agree to take part in this project.

....
(Signature of participant) (Date)

This study has been approved by the University of Otago Human Ethics Committee. If you have any concerns about the ethical conduct of the research you may contact the Committee through the Human Ethics Committee Administrator (ph 03 479 8256). Any issues you raise will be treated in confidence and investigated and you will be informed of the outcome.