Getting a grip on Schizotypy:

Elucidating dorsal stream deficits in subclinical Schizophrenia spectrum using visually guided movement

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Declaration

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university. To the best of the author's knowledge, it contains no material previously published or written by another person, except where due reference is made in the text.

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Abstract

Schizophrenia Spectrum disorders (SDD) are associated with disproportionate dorsal visual stream dysfunction, relative to ventral visual stream function. However, there remains a gap in our understanding of the neuropsychology and functional implications of this deficit. In particular, the balance of evidence implicating dorsal stream deficits is from visual perception tasks (e.g., visual masking, contrast thresholds) and neglects another well-validated model of dorsal stream function; notably, the Perception-Action Model (PAM; Goodale & Milner, 1992). Within this framework, the dorsal visual stream performs the necessary transformations on visual information to guide movement (e.g., reaching, grasping). Accordingly, disorders with known dorsal stream impairment (e.g., dorsal lesions following stroke) have been empirically associated with selective deficits in visually guided movement. However, whether such deficits also characterise SSD remains unclear. Moreover, recent neuroimaging research has identified two sub-pathways within the dorsal stream and the differential impact of SSD across these sub-pathways has not been studied. Furthermore, most of these studies have focussed on persons with Schizophrenia. Whilst this population is likely to exhibit the most pronounced deficits, little is known about whether these deficits occur across the entire SSD spectrum and are, therefore, core to the condition or a result of confounding factors of increased severity (e.g., medication use, institutionalisation). Accordingly, this thesis aimed to investigate dorsal stream impairments in sub-clinical SSD (i.e., trait-level Schizotypy) using an experimental task measuring visually guided movement. In addition, following unexpected results from this first experiment, the thesis also undertook a comprehensive psychometric evaluation for the Oxford- Liverpool Inventory of Feelings and Experiences (OLIFE; Mason et al., 1995) to clarify its measurement of Schizotypy.

Study 1 examined the nature of SSD-related dorsal stream deficits using the PAM as an explanatory framework. It extended previous research by (1) utilising visuomotor tasks with known neurobiological correlates to dorsal sub-pathways (i.e., dorsomedial and dorsolateral) and (2) recruiting a subclinical sample varying on trait-level Schizotypy. It was hypothesised that increased Schizotypy would correlate with specific differences in the kinematic parameters of visually guided hand movements. Furthermore, these differences were hypothesised to provide insight into differential impairment of the dorsomedial and dorsolateral sub-pathways based on whether they appeared across movement type (i.e., pointing versus grasping), and/or target type (i.e., stationary versus perturbed). Results supported dorsal-specific deficits in SSD; however, conclusions suggesting the differential impairment of the sub-pathways were less clear. Given the unexpected results from Study 1, coupled with an ongoing debate about the measurement of Schizotypy and its latent structure, Study 2 sought to assess the dimensional architecture of the measure used to quantify Schizotypy in the previous study, namely, the OLIFE. Past research has supported two-, three-, and four-factor models of Schizotypy, with little consensus over which architecture is valid. Specific criticisms of the OLIFE also include the validity of its item content and overall length. These shortcomings may have undermined the feasibility of finding the differential kinematic patterns of interest across level of Schizotypy hypothesised in Study 1. As such, Study 2 employed both Classical Test Theory and Item Response Theory to validate and revise the OLIFE. This process resulted in a final 16-item short form, the OLIFE16. Study 3 applied this new structure of the OLIFE to the results from Study 1 to reassess whether increased Schizotypy is associated with alterations in visually guided movement. However, once again, no differences in the kinematics of visually guided movement as a function of Schizotypy or any of its latent dimensions were revealed. These results suggest that dorsal visual stream dysfunction in SSD may only be observed in clinical populations, which, in turn, questions the validity of these deficits as core to the disorder. Nevertheless, the new OLIFE16 provides a valid and reliable short measure of Schizotypy and support for the three-factor architecture of SSD. Future directions in differentiating dorsal sub-pathway deficits in SSD and further validation of the OLIFE-16 are discussed.

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List of Abbreviations

AIP	Anterior intraparietal (region)
BOLD	Blood-oxygen Level Dependent
BPD	Borderline Personality Disorder
CogDis	Cognitive Disorganisation subscale
CTT	Classical Test Theory
DTT	Dual Trends Theory
GA	Grip Aperture
ImpNon	Impulsive Nonconformity subscale
IntAnh	Introvertive Anhedonia subscale
IPS	Intraparietal sulcus
IRT	Item Response Theory
LOC	Lateral occipital complex
MIP	Medial intraparietal (region)
MSS	Multidimensional Schizotypy Scale
MT	Movement Time
OLIFE	Oxford Liverpool Inventory of Feelings and Experiences
OLIFE16	OLIFE 16-item short form
$OLIFE_{Total}$	OLIFE Total Score
PAM	Perception Action Model
PGA	Peak Grip Aperture
$PGA_{t\%}$	Time of Peak Grip Aperture as proportion of overall movement time
PHS	Peak Hand Speed
$PHS_{t\%}$	Time of Peak Hand Speed as proportion of overall movement time
PMd	Dorsal premotor cortex

PMv	Ventral premotor cortex
PPC	Posterior parietal cortex
SGA	Stable Grip Aperture
SPD	Schizotypal Personality Disorder
SPQ	Schizotypal Personality Questionnaire
SSD	Schizophrenia Spectrum Disorders
TCVS	Two Cortical Visual Systems
UnuExp	Unusual Experiences subscale

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Thesis Overview and Structure

Schizophrenia Spectrum disorders (SSD) are associated with disproportionate dorsal visual stream dysfunction, relative to ventral visual stream function. However, there remain several gaps in our understanding of the neuropsychology and functional implications of this deficit. First, the balance of evidence implicating dorsal stream deficits is from visual-perception tasks (e.g., visual masking, visual contrast thresholds) and neglects another well-validated model of dorsal stream function – namely, the Perception-Action Model (PAM; Goodale & Milner, 1992). Within this model, the dorsal visual stream performs the necessary transformations on visual information to guide movement (e.g., reaching, grasping). Accordingly, disorders with known dorsal stream impairment (e.g., brain lesions) manifest selective deficits in visually guided movement. However, whether such deficits also characterise, SSD remains unclear. Second, recent neuroimaging experiments have identified two sub-pathways within the dorsal stream, and the differential impact of SSD across these sub-pathways has not been studied. Third, research in this area has focussed on patients with Schizophrenia *per se*. While this population is likely to exhibit the most profound deficits, little is known about whether these deficits occur across the entire Schizophrenia spectrum and are, therefore, core to the condition or a result of factors likely to be confounded with illness severity (e.g., medication use, institutionalisation). To address these gaps, the current research program investigated dorsal stream impairments, using visual-movement tasks known to differentially activate dorsal stream sub-pathways, in a sub-clinical SSD sample.

A graphical overview of the structure of this thesis can be found in Figure 0.1. The layout of this thesis is such that Chapters 1 and 2 contain comprehensive literature reviews which form the foundation for subsequent empirical studies. This is followed by Chapters 3, 4, and 5, which detail three empirical studies that are written as manuscripts for submission to peer-reviewed journal articles. As such, some of the information included in the literature review (Chapters 1 and 2) may be repeated in the empirical chapters. Finally, Chapter 6 provides a general discussion which integrates the main findings and broader research implications. The following is a detailed review of each chapter.

Chapter 1 reviews an evolutionary model of functional brain organisation as a framework for understanding the development of dorsal stream impairments in SSD. It contextualises the proceeding studies by reviewing the primary theories upon which the experimental studies are based; namely, the Dual Trends Theory (DTT) and the Perception Action Model (PAM). This chapter reviews the extant evidence for dorsal stream impairment among persons with SSD and the literature explicating the anatomically and functionally distinct sub-pathways of the dorsal stream responsible for visually guided movement. This chapter concludes with the first research aims of this thesis; (1) to assess SSD-related impairment of the dorsal visual stream using predictions generated within the PAM framework and a novel visuomotor task; and (2) to investigate the contribution of each of the two major sub-pathways of the dorsal visual stream underlying this impairment.

Chapter 2 reviews Schizophrenia as a spectrum and gives evidence for this approach. It also explores subclinical SSD, labelled widely (but not exclusively) as Schizotypy. Discussion of one of the most widely used psychometric measures of Schizotypy, the Oxford Liverpool Inventory of Feelings and Experiences (OLIFE), is also provided. This chapter also introduces the third and fourth research aims of this thesis, which emerged directly from unexpected findings from the first study. These aims were to (3) investigate the validity and factor structure of the OLIFE in an Australian sample and (4) refine and validate the OLIFE using both Classical Test Theory (CTT) and Item Response Theory (IRT) approaches.

Chapter 3 reports on Study 1, which was conducted to examine the nature of SSD-related visuomotor (i.e., dorsal stream) deficits using the PAM as an explanatory framework and a novel experimental task. The study extends previous research by including a visuomotor task tapping visuomotor processes known to differentially correlate with each of the dorsal sub-pathways (i.e., dorsomedial and dorsolateral sub-pathways) and sampling from a nonclinical sample varying on trait-level Schizotypy. It was hypothesised that Schizotypy would correlate with specific kinematic parameters of visually guided hand movements. Furthermore, these differences were hypothesised to provide insight into differential impairment of the dorsomedial and dorsolateral sub-pathways based on whether they appeared across movement type (i.e., reaching versus stationary estimating), hand position (i.e., pointing versus grasping), and/or target type (i.e., stationary versus moving). However, results failed to reveal the hypothesised kinematic differences as a function of Schizotypy. Because of these unexpected results, the thesis then undertook a comprehensive psychometric evaluation for the Oxford-Liverpool Inventory of Feelings and Experiences (OLIFE) to clarify its measurement properties.

Chapter 4 reports on the psychometric evaluation of the OLIFE (Study 2), with a particular focus on factorial and content validity. Specific criticisms of the OLIFE include the validity of its proposed four-factor structure (which is counter to most theoretical accounts of Schizotypy as constituted by three underlying dimensions), item content/coverage, and overall length. While the accurate measurement of Schizotypy is important generally, the OLIFE's psychometric shortcomings may have specifically accounted, at least in part, for the lack of differential patterns across kinematic measures in Study 1. As such, Study 2 employed both CTT and IRT methods to validate and revise the OLIFE. This process resulted in a final 16-item short form, the OLIFE16.

Chapter 5 reports on Study 3, where kinematic results from Study 1 were re-analysed using the OLIFE16 to assess whether more psychometrically-sound indicators of Schizotypy symptoms were indeed associated with alterations in visually guided movement kinematics. However, results revealed no meaningful change from the original outcomes; that is, no difference across kinematic measures were observed as a function of Schizotypy or any of its latent dimensions.

Chapter 6 provides a general discussion that situates the results from the three studies in this thesis within the context of the broader research literature. Possible explanations for the unexpected results from Study 1 and 3 are reviewed in greater depth, including a discussion of Study 1's methodology and the possibility that dorsal visual stream dysfunction may only be observed in clinical populations only and, therefore, not *necessarily* core to SSD. Discussion on the utility of the OLIFE16 as a valid and reliable brief measure of Schizotypy is also provided. Future directions investigating the integrity and function of the dorsal visual sub-pathway deficits in SSD and further validation of the OLIFE16 are recommended.

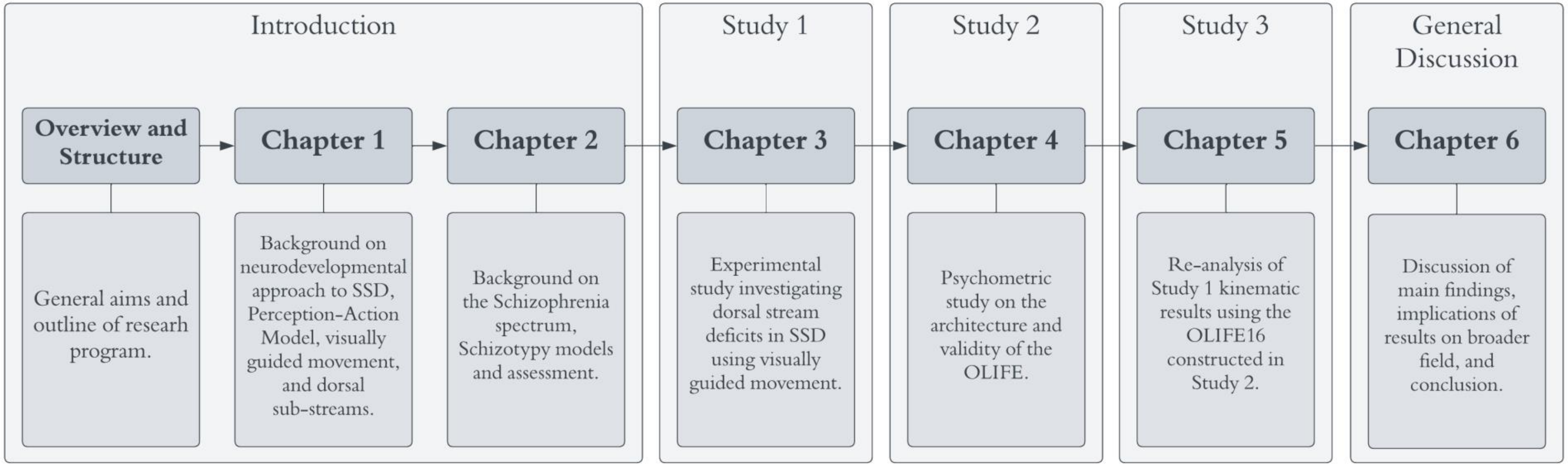

Figure 0.1 Thesis overview.

CHAPTER 1

Contextualising Dorsal Stream Deficits in Schizophrenia Spectrum Disorders

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1.1 Functional Brain Organisation

1.1.1 Classical Lesion-Behaviour Approaches

Since John Harlow's 1848 seminal paper documenting the behavioural changes experienced by railroad worker Phineas Gage after surviving an iron spike penetrating his frontal lobes, our understanding of human brain function and organisation has relied principally on the inferences drawn from lesion-behaviour models (Broca, 2006; Harlow, 1848; Jakobson et al., 1991; Wernicke, 1970). These models posit that behavioural changes observed following injury to specific brain regions/structures, *de facto*, implicate said regions/structures as the substrate generating and/or guiding those behaviours in the context of healthy brain function (Huey & Lieberman, 2012). Despite their success, the application of lesion-behaviour models of brain function and organisation often fall short in their ability to offer parsimonious and valid models of psychopathology, such as Schizophrenia Spectrum Disorders (SSD). Moreover, there are fundamental discrepancies between the assumptions underlying lesion-behaviour models and the clinicopathological characteristics of SSD. First, observations of SSD brain structure have failed to reveal the macroscopic lesions that would be expected in acquired brain pathology. Instead, SSD are associated with subtle, microscopic abnormalities (Arnold & Trojanowski, 1996; Del Fabro et al., 2021; Kong et al., 2020). Second, SSD is associated with impairments in widespread neural networks across the whole brain (Hoffman & McGlashan, 2001; Price & Drevets, 2012). Third, SSD, among other psychopathologies, has been suggested to be neurodevelopmental in nature (i.e., the interaction between early neurobiological abnormalities and developmental experience is what produces psychopathology) (Insel, 2010; Jaaro-Peled & Sawa, 2020; Rund, 2018). Consequently, there is need for alternative models of brain function and organisation that can accommodate the diversity of symptoms, widespread neural differences, and developmental variances that are associated with SSD.

1.1.2 Cytoarchitectonics

Evolutionary cytoarchitectonic theories offer a framework that can accommodate many of these observations. Specifically, the Dual Trends Theory (DTT; Pandya & Yeterian, 1985; Sanides, 1969), a neurobiological framework outlining the phylogenetic progression of the mammalian cortex, has been used to understand a variety of cognitive, perceptual, and behavioural impairments observed in SSD (Christensen & Bilder, 2000; Giaccio, 2006). The DTT posits that, across phylogeny, all cortical regions have evolved from two primary neural origins (i.e., prime moieties): the archiocortex (hippocampus) and paleocortex (pyriform cortex) (Christensen & Bilder, 2000; Giaccio, 2006). Cortical expansion and elaboration have emanated from these origins to form two highly interconnected, but independent, cortical systems: the archicortical trend and the paleocortical trend. These systems can be delineated by their anatomy, cytoarchitecture and myeloarchitecture, patterns of neural connectivity, and function (Goldberg, 1985; Pandya & Barnes, 1987; Sanides, 1969).

Anatomically, the archicortical, or dorsal-medial, trend includes the medial and dorsolateral regions of the frontal, parietal, and occipital lobes, the hippocampus, and the adjacent parahippocampal gyrus (Giaccio, 2006; Sanides, 1969). Conversely, the paleocortical, or ventral-lateral, trend includes the ventral portions of the front, parietal, and occipital lobes, most of the temporal lobe, and the insular lobe (Giaccio, 2006; Sanides 1969). Cytoarchitectonic and myeloarchitectonic analyses of the cerebral cortex offer strong support for DTT by revealing cortical layers to be distinguishable by appearance, successive thickness, neuronal density, and overall myelination, with each successive layer bringing increasing laminar complexity (Abbie, 2004; Pandya et al., 1986; Pandya & Yeterian, 1985). Moreover, the division of the cortex into dual trends is also reflected in the organisation of cortico-cortical connections such that cortical areas associated with one trend are preferentially networked with other cortical areas within the same trend (Pandya & Yeterian, 1985). Taken together, these foundational studies suggest that the widely used dorsal/ventral dichotomy within human and primate sensory systems are likely to reflect fundamental cytoarchitecture that has evolved across phylogeny in response to evolutionary principles and environmental demands. A chief instantiation of the dual architecture of the primate cortex is the dual visual processing streams (e.g., (Allman & Kaas, 1971; Goodale & Milner, 1992; Ungerleider & Mishkin, 1982; Zeki et al., 1991).

1.2 Dorsal and Ventral Visual Processing Streams

Primate visual processing systems are characterised by well-defined differences between the dorsal and ventral visual processing streams; these differences exist across cellular/laminar organisation, structure, connectivity, and function (Goodale et al., 1994; Mishkin et al., 1983; Ungerleider & Haxby, 1994; Van Essen & Gallant, 1994). The dorsal visual processing stream is specialised for processing coarse spatial information and movement, whereas the ventral visual processing stream processes detailed spatial information and related perceptual characteristics necessary for object recognition (e.g., colour, texture) (Goldberg, 1985; Ungerleider & Mishkin, 1982). This dissociation in function was first popularized by Ungerleider and Miskin's (1982) as the Two Cortical Visual Systems (TCVS) Model, which also labelled the ventral stream the 'what' system and the dorsal stream the 'where' system. Evidence for this model initially came from non-human primate lesion studies (Ungerleider & Mishkin, 1982), with subsequent evidence drawn from the visual performance of human patients with acquired brain lesions (Prather et al., 2004;

Ungerleider & Haxby, 1994; Wang et al., 1999) and neuroimaging studies (Beauchamp et al., 2002; Courtney & Ungerleider, 1997; Ellison & Cowey, 2006; Haxby et al., 1991; McIntosh et al., 1994). Importantly, however, Goodale and Milner (1992) proposed a variation of the TCVS Model. Rather than focussing on the type of information processed by the respective visual streams, they emphasise the transformations of perceptual information necessary for behaviour. Their Perception-Action Model (PAM) specifies that the dorsal stream subserves vision for action (e.g., visually guided reach and grasping), while the ventral stream mediates vision for perception (e.g., object recognition) (Goodale & Milner, 1992). Using Ungerleider and Mishkin's (1982) TCVS Model, investigators have examined the relative integrity of the dorsal versus ventral visual processing streams in SSD.

This research has shown that patients with SSD are disproportionately impaired on tasks tapping visual dorsal stream function. Such experimentation has found deficits in a range of tasks known to be mediated by dorsal stream function, including impaired motion processing deficits (Chen, Levy, et al., 1999; Chen, Palafox, et al., 1999), reduced attentional spotlight modulation (Cornblatt & Keilp, 1994; Elahipanah, 2013; Elahipanah et al., 2011; Slaghuis & Thompson, 2003), attenuated attention in peripheral vision (Elahipanah et al., 2011; Slaghuis & Thompson, 2003), poor target location discrimination (Cadenhead et al., 1998; O'Donnell et al., 1996), lower contrast sensitivity (Kéri et al., 2002), and greater masking deficits (Butler et al., 2001; Butler & Javitt, 2005; Rassovsky et al., 2004, for a commentary on SSD and visual masking please see Appendix A). Similar, albeit attenuated, patterns of poor performance have also been recorded in persons with subclinical traits along the Schizophrenia spectrum (e.g., Schizotypy) (Bedwell et al., 2013; Cappe et al., 2012; Koychev et al., 2010). However, as noted above, theoretical and empirical developments have expanded the role of the dorsal stream to include visuomotor action as specified by the PAM. Despite these conceptual advances, little research into whether these functions are also impaired across the Schizophrenia spectrum has been conducted. Before elucidating in more detail on the use of visually guided movement as a window into ventral and dorsal visual processing streams, the next section will review the fundamental tenets of the PAM and the associated experimental methods used in its validation.

1.2.1 Perception-Action Model of Vision

The PAM hypothesises that the ventral stream permits the formation of perceptual representations, which contain information about the enduring characteristics of an object in conjunction with its spatial relations (Goodale & Milner, 1992). That is, the ventral stream operates on relative metrics, processing target objects in the environment relative to other objects and surfaces. Conversely, the dorsal stream is associated with mediation of visual control and skilled action (e.g., reaching and grasping). Thus, the dorsal stream acts as an online feedback loop, providing information about target objects in real-time. Consequently, the dorsal stream operates using absolute and veridical metrics to allow for accurate interactions (Goodale & Milner, 1992). The two streams are also known to link with differing higher-order brain functions. Specifically, the ventral stream is connected to long-term memory, while the dorsal stream governs motor function (Westwood & Goodale, 2003). For these reasons, the ventral pathway is referred to as the vision-for-perception system, while the dorsal pathway is referred to as the vision-for-action system (Goodale & Milner, 1992).

Evidence for the dissociation of pathways for perception and action was first garnered through the study of patients, such as patient D.F., who developed visual form agnosia from damage to occipitotemporal areas (i.e., ventral stream) (Goodale et al., 1991). Most notably, D.F. demonstrated significant deficits in object recognition and representation, such that she was unable to distinguish if two objects were identical in form; however, her ability to differentially grasp the very same shapes remained intact (Goodale et al., 1991). Conversely, patients with optic ataxia resulting from parietotemporal (dorsal) damage show clear signs of grasping impairments (e.g., difficulties in calibrating reaching movements with respect to target location and inaccurate hand pre-shaping), but no impairments in their ability to perceptually discriminate between targets (Cavina-Pratesi et al., 2013; Jakobson et al., 1991). The unique differences in abilities across visual form agnosia and optic ataxia demonstrates the dissociation between ventral/perceptual function and dorsal/action function. As such, behavioural vision and visually guided movement paradigms can be used to differentiate the functional integrity of dorsal and ventral streams (Aglioti et al., 1995; King et al., 2008).

1.2.2 Visually Guided Movement

As noted above, commonly used methods to differentiate dorsal and ventral stream processing in the context of PAM are comparisons between visual perception and visually guided movements (see Budisavljevic et al., 2018; Busan et al., 2009; Ciavarro et al., 2013; Culham et al., 2003; Galletti et al., 2003; Króliczak et al., 2007). In these tasks, participants are typically required to make either perceptual judgements about an object (e.g., orientation) or direct reach-to-grasp movements towards the same object (Budisavljevic et al., 2018; Culham et al., 2003; Tunik et al., 2005). Research has shown that reach-to-grasp movements are associated with greater dorsal stream activity (or preferentially disrupted by dorsal stream lesions), while perceptual judgements are associated with ventral stream activity (or preferentially disrupted by ventral stream lesions) (Budisavljevic et al., 2018; Culham et al., 2003). In particular, fMRI studies have verified blood-oxygen-level-dependent (BOLD) signal increases in the anterior intraparietal (AIP) area and intraparietal sulcus (IPS) (both part of the dorsal stream) during reach-to-grasp movements, but in the lateral occipital complex (LOC) (part of the ventral stream) during object perception tasks (Culham et al., 2003; Króliczak et al., 2007).

Other experimental manipulations have also been used to dissociate ventral and dorsal stream function in the context of PAM. These include changes to the stimuli characteristics (e.g., target perturbation, visual illusions; see Bruno & Franz, 2009; Desmurget et al., 1999), immediate and delayed movement execution (Cohen et al., 2009), visually-versus memory-guided movement (Heath et al., 2005; Vaillancourt et al., 2003), and adopting different hand grips (e.g., precision versus power grip, see Begliomini et al., 2007; Desmurget et al., 1999; Ehrsson et al., 2000; Tunik et al., 2005). Target perturbation involves the introduction of small changes in object size, orientation, or location during grasping movements. It has been shown that movement corrections in this context are dependent on dorsal stream function (Tunik et al., 2005). In these tasks, target perturbations are often synchronised with saccades to capitalize on saccadic suppression, which prevents the participant from consciously perceiving the change (Desmurget et al., 1999; Goodale et al., 1986). Alternatively, processing of object characteristics when displayed under illusion conditions (e.g., Ebbinghaus or Muller-Lyer illusions) affects the vision-for-perception (ventral) system but not vision-for-action (dorsal) system (Franz & Gegenfurtner, 2008). Immediate and delayed visually guided movement entails constraining the initiation of visually guided movement responses. These studies show that during immediate grasping conditions, input is mostly dorsally driven; however, once a delayed response is introduced, the same actions become more ventrally driven (Milner et al., 2001). These results are consistent with the assumption that visuomotor computations are carried out in 'real time'. That is, the vision-for-action system computes object characteristics immediately before and during movement execution and does not use short-term or working memory. Conversely, the vision-for-perception system focuses on identity and meaning of the object rather than position and size, hence, possesses robust memory to represent stable and enduring characteristics of the environment (Goodale & Milner, 1992). Therefore, grasping objects immediately after they appear is associated with dorsal stream activation, while delayed grasping is correlated with ventral stream processing (Cohen et al., 2009). Similarly, visually guided movement, compared to memory-guided movement, is consistent with research showing that the dorsal stream provides online feedback during movement execution, which can only be utilised through visually guided (closed-loop) movements (Heath et al., 2005). When vision is disrupted during movement (e.g., memory-guided or open-loop), the primary pathway recruited becomes the ventral stream as the only source of visual information to guide movement comes from memory (Heath et al., 2005).

These manipulations can be used in conjunction with each other, or as standalone instantiations, to differentially activate either dorsal or ventral visual streams. However, research shows that perceptual versus movement, target perturbation (with consequent movement correction), and handgrip manipulations render the most consistent and robust dissociations (Budisavljevic et al., 2018; Cavina-Pratesi et al., 2018; Tunik et al., 2005). In contrast, visual illusions show inconsistent dissociations. A common finding from previous visual illusion studies is that, while perceptual estimates of objects are modulated by illusion conditions, reach-to-grasp movements are immune to illusion effects (Aglioti et al., 1995; Danckert et al., 2002, 2002; King et al., 2008). However, several studies have also found that the Ebbinghaus/Titchner illusion alters reach-to-grasp movements to a similar degree as perception (Bruno et al., 2008; Bruno & Franz, 2009; Franz & Gegenfurtner, 2008; Smeets & Brenner, 2006). The lack of differentiation between action and perception using visual illusions may be the result of subtle variations in the specific measures employed. For example, the difference between how perceptual estimates of target size are made in these studies varies widely from: (1) direct comparisons, where the Ebbinghaus/Titchner illusion is viewed in its entirety and comparisons are made between two illusory stimuli (see Figure 1.1.A and also Aglioti et al., 1995); (2) separate comparisons, where size comparisons are made using a comparison disc and only part of the whole Ebbinghaus/Titchner illusion (see Figure 1.1.B. and also (Franz et al., 2000, 2003); and, (3) manual estimations, where comparisons are made by measuring aperture size between thumb and index finger (see Figure 1.1.C. and also Haffenden & Goodale, 1998; King et al., 2008)

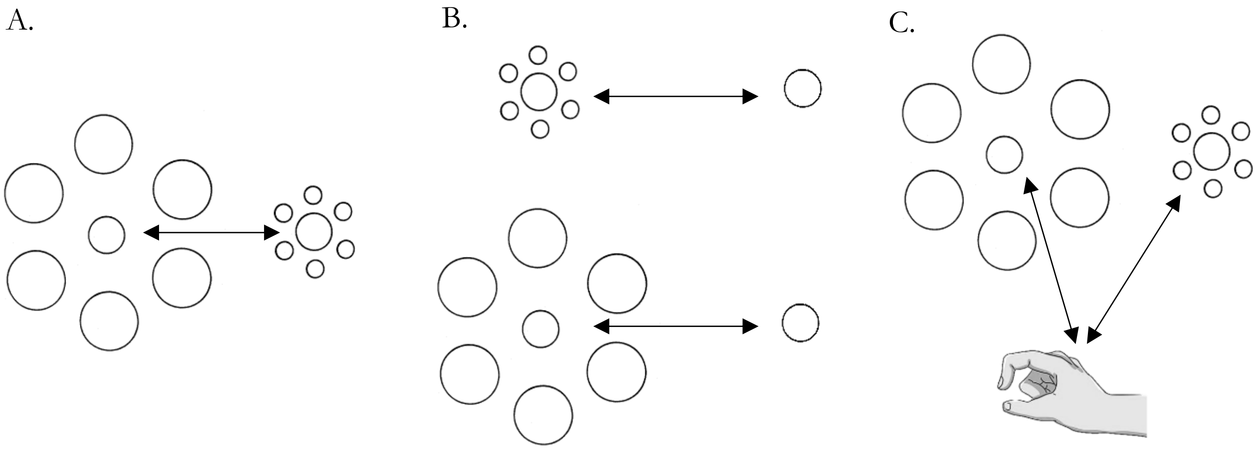

Figure 1.1 Examples of size comparisons in Muller-Lyer illusion paradigms. A. Direct comparison. B. Separate comparison. C. Manual estimation.

These subtle task variations can result in large variations in the illusory effect. For example, direct comparisons led to a 50% increase under the illusion compared to separate comparisons (Franz & Gegenfurtner, 2008). In general, manual estimations demonstrate the largest illusion effects followed by direct, and then separate, comparisons. As a result, an alternative explanation for the observed results is that an artefact of mismatched task demands has contributed to the observed effect (i.e., larger discrepancies between action versus perception-oriented tasks are not reflective of differences in these two systems, but rather differences in the power of the illusion, as driven by perceptual task characteristics). More importantly, if the power of the illusion is equated across these various perceptual measures, differences between grasp and perception tasks frequently disappear (Franz & Gegenfurtner, 2008). Other commonly used illusions (e.g., the Muller-Lyer illusion) have also generated similar criticisms (see Bruno & Franz, 2009; Weidner & Fink, 2007 for review). Therefore, the use of visual illusions as a tool for dissociating the vision-for-action from vision-for-perception systems is undermined by the above-reviewed methodological shortcomings.

1.3 Dorsomedial and Dorsolateral Sub-pathways

Advances in neuroimaging and brain stimulation have allowed for the differentiation of sub-pathways within the dorsal stream. Specifically, research has identified two anatomically segregated parieto-frontal circuits: (1) a dorsomedial circuit, consisting of the posterior parietal cortex (PPC) and the caudal dorsal premotor cortex (PMd); and, (2) a dorsolateral circuit, consisting of the anterior intraparietal (AIP) area and the ventral premotor cortex (PMv) (Galletti et al., 2003; Grol et al., 2007). Both pathways have been linked to independent components of manual prehension - a reach component and a grasp component (Grol et al., 2007; Jeannerod, 1988; see Figure 1.2).

The dorsomedial circuit has been linked to manual prehension (Andersen & Buneo, 2002); specifically, the coding of reach information for planning and controlling arm position by integrating somatosensory and visual input (Rizzolatti et al., 1996). Anatomically, the dorsomedial circuit connects two regions within the PPC; the anterior portion of the occipioparietal sulcus (area V6A; Bosco et al., 2010) and the medial intraparietal (MIP; Johnson et al., 1996) area, to the PMd. On the other hand, the dorsolateral circuit has been linked to the grasp component of manual prehension, which is largely used in the transformation of intrinsic properties of the object to be grasped into motor commands for hand pre-shaping (Brochier & Umiltà, 2007). The circuitry of this pathway is the connection between the AIP, within the inferior parietal lobe (IPL), to area F5 within PMv (Culham et al., 2003; Fluet et al., 2010; Turella & Lingnau, 2014). 'Virtual lesion' studies, using Transcranial Magnetic Stimulation (TMS), have supported the identification of specific brain regions involved in reaching and grasping. For example, Desmurget and colleagues (1999) found that stimulation of PPC (i.e., in the dorsomedial sub-pathway), resulted in overreaching and an inability to correct errors in trajectory. Conversely, Begliomini and colleagues (2007), found the association to be specifically linked to precision grip (as opposed to whole-hand grip) reach-to-grasp movements. Other dorsolateral regions, including the PMv, have also been implicated in coding grasp-related information (Olivier et al., 2007) while the PMd has been associated with object lifting (Davare et al., 2006).

The proposed independence of these two dorsal stream sub-pathways remains vigorously debated. Some studies suggest significant overlap and integration between structures of the dorsolateral and dorsomedial circuits, with some areas simultaneously coding for elements of both reach and grasp movements (see Figure 1.2; Cavina-Pratesi et al., 2018; Monaco et al., 2015; Turella & Lingnau, 2014). Studies of macaque monkeys show that area V6A in the dorsomedial stream is sensitive to wrist orientation and grip formation (Breveglieri et al., 2016, 2018; Galletti & Fattori, 2018). The human homologue of V6A, the superior parietal occipital cortex (SPOC), also encodes for both hand transport and grip formation (Vesia et al., 2017). These results suggest that the dorsolateral circuit mediates grasp movements, whereas the dorsomedial circuit is directly involved in the interaction between the reach and grasp movements during manual prehension (Turella & Lingnau, 2014). Nevertheless, the use of reaching and grasping movements to further understand the dorsal visual processing stream is well validated.

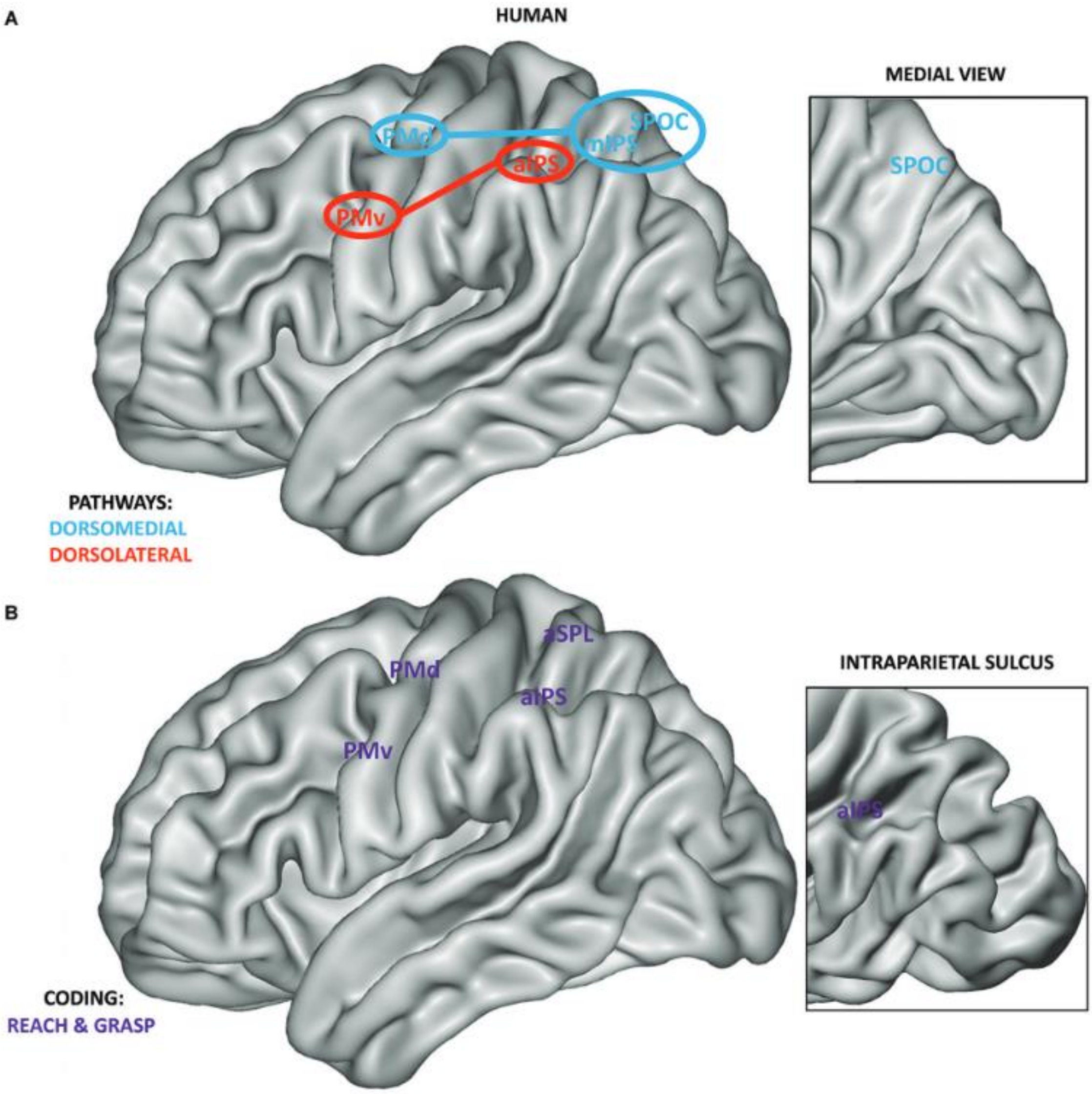

Figure 1.2 Neural correlates of dorsomedial and dorsolateral sub-pathways. A. Anatomical localisation of human grasping regions within the dorsomedial (blue) and dorsolateral (red) pathways. B. Definition of regions within the PPC and premotor cortices showing grasp and reach coding (purple). Copied from Turella & Lingnau, 2014.

1.4 Conclusion

This chapter reviewed the DTT and PAM models for understanding brain development and human visual processing, respectively. These models provide explanatory frameworks for research into SSD concerned with elucidating dorsal-specific deficits. Specifically, the PAM provides clear behavioural predictions and measures to differentially test ventral and dorsal visual processing stream function. While the use of visual illusions has been a popular paradigm to test such pathways, concerns around the validity of results due to methodological disparities were revealed. An alternative method of manipulation using visually guided movement under differing task demands (perceptual estimation versus reaching) was presented as a superior approach. Furthermore, the presence of functionally distinct sub-pathways within the dorsal stream (dorsomedial and dorsolateral sub-pathways) is presented along with behavioural means of assessing the integrity of these circuits by manipulating movement type (reach-to-point versus reach-to-grasp) and target type (stationary versus perturbed). The relative integrity of these sub-pathways in SSD is unknown. Given the identification of two separate sub-pathways within the dorsal stream, research investigating the differential contribution of these sub-pathways to SSD-related dorsal stream dysfunction is warranted. Therefore, the first two aims of this thesis are (1) to assess SSD-related impairment of the dorsal visual stream using predictions generated within the PAM framework and a novel visuomotor task; and (2) to investigate the contribution of each of the two major sub-pathways of the dorsal visual stream underlying this impairment. Before reviewing Study 1, another central aspect of the current thesis is reviewed – that is, the conceptualisation of, and empirical support for the organisation of SSD along a spectrum, as opposed to discrete categories of disorders.

CHAPTER 2

The Schizophrenia Spectrum

2.1 Dimensionality of Schizophrenia

Traditionally, Schizophrenia has been conceptualised as an independent category of psychopathology, discrete from other psychiatric illnesses (e.g., Major Depressive Disorder, Generalised Anxiety Disorder). This conceptualisation has been the cornerstone of most current diagnostic taxonomies, including the Diagnostic and Statistical Manual 5 (DSM-5; American Psychiatric Association, 2013). According to the DSM-5, Schizophrenia is exemplified by the presence of key clinical/behavioural criteria including (but not limited to) hallucinations, delusions, disorganised thinking, bizarre behaviours, and impaired social and other functional skills. The constellation of symptoms typifying Schizophrenia can be empirically categorised into three related, but separate, dimensions: positive symptoms (e.g., hallucinations), negative symptoms (e.g., anhedonia), and disorganised symptoms (e.g., tangentiality). Although the categorical diagnostic approach has been dominant in mental health research and dates back to Kraepelin (1919/1971), the field has been long-troubled by observations of considerable heterogeneity in the clinical expression of Schizophrenia, as well as high rates of comorbidity, blurred category boundaries, mixed clinical presentations, and overly stringent diagnostic criteria (Bleuler, 1950; Bromet et al., 2011; Kotov et al., 2020, 2022; Regier et al., 2013; Wing & Agrawal, 2003). These vagaries have spawned much research into the validity of diagnostic categories, including robust measurement evaluations of the latent structure of psychopathology (Cuesta et al., 2007; Haslam et al., 2012; Kotov et al., 2017, 2018; Watts et al., 2019). These studies have largely failed to reveal categorical distinctions between disorders, including Schizophrenia and related disorders. Instead, they reveal robust evidence that diagnostic categories are better conceptualised as existing across a set of continua (i.e., dimensional) and better distinguished by magnitude, as opposed to quality (Cuesta et al., 2007; Kotov et al., 2022). This research has also demonstrated that the boundaries between clinical disorders and healthy functioning is also dimensional (Haslam et al., 2012; Kotov et al., 2017; Watts et al., 2019). As such, we have witnessed a paradigm shift in understanding SSD, and their relationship to healthy functioning, as existing on a spectrum (which is also conceptually acknowledged in the organisation and presentation of psychotic disorders, moving from least to most severe, in the DSM-5).

Despite the recent popularity and empirical support of dimensional models, it is important to note that these ideas have a much longer history. One of the first mainstream dimensional approaches to understanding Schizophrenia was a quasi-dimensional one (Rado, 1953). This model traverses both categorical and dimensional frameworks and proposes that Schizotypy, a personality organisation associated with Schizophrenia, presents in approximately 10% of individuals in the population, and is referred to in this scheme as Schizotypes (Rado, 1953). This group contrasts with the rest of the population (approximately 90%) who do not present with Schizotypy and are, therefore, at low risk for developing a psychotic disorder. This idea was further advanced by Meehl (1969), who proposed the presence of a dominant autosomal *schizogene* which manifests as aberrant synaptic functioning and leads to defective neurointegrative processes. Meehl called this genetic abnormality *schizotaxia* (Meehl, 1962). According to Meehl, schizotaxia is necessary but insufficient in the development of Schizophrenia. More specifically, Meehl suggested that the interaction between schizotaxia and various environmental influences determines the level of subsequent impairment and development of Schizophrenia and related disorders (Meehl, 1962). Therefore, the genetic vulnerability in this model is taxonic in nature, while levels of disease expression are considered dimensional; hence a quasi-dimensional framework.

Experimental support for the quasi-dimensional approach comes from studies using taxometric analyses (Rawlings et al., 2008b; Waller & Meehl, 1998). Taxometric analyses are a group of statistical procedures used to determine whether heterogeneity among observed variables manifest from the presence of latent taxons (i.e., categories) or different positions along a common dimension. A review by Rawlings et al., (2008) found that 15 out of 19 taxometric studies on Schizotypy supported a categorical structure, with taxonic base rates ranging from 0.03 to 0.13. These results directly support Meehl's (1990) theory that approximately 10% of the population are schizotypes. Furthermore, a large-scale quantitative review of taxometric research including 177 articles with 311 distinct findings showed Schizotypy, along with substance use disorders and autism, to be the only psychopathology domains tested that demonstrated promising evidence for psychological taxa (Haslam et al., 2012). Although compelling, significant methodological concerns have since been raised regarding these studies. First, poor sampling methods which result in clinical samples with insufficient power or large unrepresentative samples are common in these studies. Second, despite Schizotypy being a multidimensional construct, these studies often only investigated one aspect of Schizotypy (e.g., either positive or negative symptomatology), and with only one taxometric procedure (most commonly MAXCOV). Lastly, Rawlings and colleagues (2008) contend that results from studies that supported a categorical model with a base rate of 10% may be due to positively skewed sample distributions rather than true reflections of underlying taxonic structure. To address these shortcomings, Rawlings and colleagues (2008) conducted a taxometric study using a large diverse adult sample, multiple measures of Schizotypy, and conducted analyses with multiple taxometric procedures (MAXEIG and MAMBEC) using a simulation approach developed by Ruscio and colleagues (2007) which takes into account indicator skew. Their results supported a dimensional latent structure of Schizotypy (Rawlings et al., 2008). Haslam and colleagues (2012) also noted that evidence of taxa was less likely to be observed in recent and methodologically stronger studies, specifically, those that employed comparative fit indices based on simulated comparison data.

Further criticism of the quasi-dimensional approach includes the high prevalence of unusual experiences reported within the general population. Several studies examining unusual experiences in the general population have found endorsement rates to vary widely, from 5% to 77% (Hanssen et al., 2005; Scott et al., 2009; Verdoux et al., 1998). In Verdoux and colleagues' (1998) study, they observed 46.9% of people believed in the existence of telepathic communication, 23.4% believed in witchcraft, voodoo, and/or occult, and 16% reported hearing voices. The presence of significantly higher rates of unusual experiences in the general population than was estimated by Meehl indicates that Schizotypy may not be a strictly taxonic entity that affects only a small portion of the population. Instead, Schizotypy may be distributed as a multidimensional trait that varies across the entire population, where low levels of Schizotypy show little to no functional impairment and high levels of Schizotypy are associated with greater functional impairment and a greater likelihood of meeting diagnosis for Schizophrenia or related disorders (Bentall et al., 1989). This variation would also include a continuum of psychotic-like experiences ranging from unusual experiences (e.g., subclinical symptoms) to symptomatology consistent with full-blown psychosis (e.g., frank delusions and/or hallucination).

The fully dimensional approach proposed by Claridge and colleagues accounts for the high prevalence of unusual experiences observed within the normative population and is consistent with more recent models of Schizophrenia that highlight continuity and overlap between clinical and non-clinical psychosis (Claridge & Beech, 1995; Claridge & Davis, 2002; Linscott & van Os, 2010). For example, it has been found that people with a diagnosis of psychotic disorders tend to score high on measures of Schizotypy (Camisa et al., 2005; Lenzenweger, 1994). However, it should be noted that the opposite is not true; that is, high scores on Schizotypy measures do not necessarily correlate with meeting diagnostic criteria for a clinical disorder. In fact, most people who score highly on measures of Schizotypy report good subjective well-being (Goulding, 2004) and exhibit strengths in creativity (Batey & Furnham, 2008; Nelson & Rawlings, 2010). The non-commutative nature of Schizotypy and Schizophrenia supports the notion that Schizotypy is related to, but not sufficient for, developing Schizophrenia. Similarities in the latent structure (i.e., three-factor models) between Schizotypy and Schizophrenia indicators also support a dimensional model (Rossi & Daneluzzo, 2002; Wuthrich & Bates, 2006). More recent taxometric analyses on Schizophrenia and related conditions have also shown support for dimensionality (Adjorlolo et al., 2021; Haslam et al., 2020 (review)). A fully dimensional approach proposes psychosis to have a multifactorial aetiology, where multiple genetic, epigenetic, and environmental/experiential factors interact to determine outcomes and phenotypic expressions, all of which lie on a continuum from healthy functioning to clinical psychosis (Allardyce et al., 2007; International Schizophrenia Consortium, 2009). Here, we will refer to this continuum as the Schizophrenia spectrum. Adoption of the Schizophrenia spectrum and dimensionality of psychosis implies the importance of Schizotypy research as a direct window into understanding the aetiology and breadth of SSD. Moreover, these samples offer the distinct advantage of potentially evading the confounds present in clinical research and associated with disorder severity (e.g., illness chronicity, medication use, institutionalisation). For these reasons, the samples recruited in this research program were drawn from the nonclinical, community population expressing varying levels of Schizotypy.

2.2 Schizotypy Measurement

As previously discussed, Schizotypy is a multidimensional construct associated with SSD (Meehl, 1962, 1989, 1990). It is theorised to be widely distributed throughout the general population and, therefore, its valid and reliable measurement is essential, especially as it relates to SSD. An important aspect of construct validity is its latent architecture (Meehl &

Cronbach, 1955), usually measured through multivariate methods such as factor analysis and latent-trait modelling. Structural and theoretical models of Schizotypy have varied and reflect both the dominant theories of the period and the specific measurements available for analysis. While some researchers suggest a two-factor model of positive and negative dimensions (e.g., Chapman Psychosis Proneness Scales; Chapman et al., 1980), others have argued for a three-factor model with the addition of a disorganisation factor, such as that found in the Schizotypal Personality Questionnaire (SPQ; Raine, 1991). Further still, research has also alluded to the presence of a fourth, impulsivity factor, as reflected in the Oxford Liverpool Inventory of Feelings and Experiences (OLIFE; Mason et al., 1995; Mason & Claridge, 2006).

The heterogeneity of proposed and observed factor structures across Schizotypy measures has also been influenced by the approach used in their development. Generally, measures of Schizotypy fall into one of three types: (1) measures that seek to quantify single dimensions within Schizotypy (e.g., the Chapman and Chapman Magical Ideation scale (MgI; Eckblad & Chapman, 1983); (2) measures which mirror DSM criteria for various SSD (e.g., Schizotypal Personality Questionnaire (SPQ; Raine, 1991), which directly assesses for the criteria listed under Schizotypal Personality Disorder; and, (3) measures that adopt a multidimensional trait lens and seek capture the entire spectrum of Schizotypy across the general population (e.g., Oxford Liverpool Inventory of Feelings and Experiences; OLIFE; Mason et al., 1995; and, more recently, the Multidimensional Schizotypy Scale; MSS; Kwapil et al., 2018). For the purposes of the current research program, the third category of Schizotypy measurement is most consistent with a fully-dimensional approach to SSD, as such, the OLIFE was used as the primary measure of trait-level Schizotypy.

2.2.1 *The Oxford Liverpool Inventory of Feelings and Experiences (OLIFE; Mason et al., 1995)*

Since its conception in 1995, the OLIFE has been used extensively in a variety of research domains to measure Schizotypy in relation to other mental/brain processes, including perception and attention (Granger et al., 2012; Jolley et al., 1999; Mason et al., 2004; Steel et al., 2002; Tsakanikos & Reed, 2003), reasoning and learning (Burch, Hemsley, et al., 2006; Moran et al., 2003; Sellen et al., 2005; Tsakanikos et al., 2003), language (Carlin & Lindell, 2015; Nunn & Peters, 2001), face processing (Batty et al., 2014; Bell & Halligan, 2015), and handedness (Barrantes-Vidal et al., 2013; Shaw et al., 2001). The OLIFE has also been used to demonstrate relationships between Schizotypy and dissociation (Holmes & Steel, 2004), childhood abuse (Startup, 1999), religious group membership (Farias et al., 2013; Smith et al., 2009), paranormal beliefs (Dagnall et al., 2016; Goulding, 2004), and the heritability of personality traits (Jang et al., 2005; Linney et al., 2003). Translations of the OLIFE have been completed in over eight different languages, including German (Grant et al., 2013), Brazilian Portuguese (Alminhana et al., 2020), French (Sierro et al., 2016), Persian (Yaghoubi & Mohammadzadeh, 2012), Hungarian (Kocsis-Bogár et al., 2016), and Romanian (Stanciu & Papasteri, 2017). Moreover, alternate forms were constructed and validated, including a short form based on a twin study (OLIFE-S; Mason et al., 2005) and a child version (CO-LIFE; Evans et al., 2018).

The original OLIFE was constructed by factor analysing the Combined Schizotypal Traits Questionnaire (CSTQ; Bentall et al., 1989); a 420-item scale consisting of 15 scales measuring psychotic-experiences and personality within the normative population. An initial Exploratory Factor Analysis (EFA) was conducted using the maximum likelihood estimator and varimax rotation. A correlation matrix was constructed from biserial correlations of each item with factor scores regressed from the factor matrix. Item selection was then carried out following three criteria. First, size of loading; items with factor loadings greater than 0.4 for the first factor or 0.3 for subsequent factors, were retained. Those with cross-loadings greater than 0.3 were removed. Second, endorsement rate; items with endorsement rates between 0.8 and 0.2 were retained; however, these benchmarks were relaxed for highly relevant items with endorsement rates just shy of the criteria. Finally, repetition avoidance; where two items of highly similar content were identified, the one of higher loading was retained. Following item selection, Cronbach's alpha for each factor was calculated, which demonstrated adequate internal consistency ($\alpha > 0.7$). The final measure consists of 104 true/false items which disaggregated into four factors, labelled Unusual Experiences, Cognitive Disorganisation, Introvertive Anhedonia, and Impulsive Non-conformity. The Unusual Experiences (UnuExp) factor contains 30 items which focus on hallucinations, perceptual disturbances, and magical thinking. These items largely reflect the 'positive' symptoms of psychosis. The Cognitive Disorganisation (CogDis) factor consists of 24 items enquiring about difficulties in concentration, attention, and decision-making, as well as elements of anxiety and purposelessness. The Introvertive Anhedonia (IntAnh) factor consists of 27 items with 15 reverse-worded items. The items in this subscale largely reflect the 'negative' symptoms of psychosis including lack of enjoyment and dislike of emotional and/or physical intimacy with a preference for independence and solitude. The final factor, Impulsive Nonconformity (ImpNon) consists of 23 items with six reverse-worded items. These items appeal to violent, self-abusive, and reckless behaviours.

Despite its widespread use, the validity of the OLIFE's factor structure and item content have come under significant scrutiny. The emerging concerns can be summarised as follows: (1) the validity of the ImpNon factor as reflecting a core feature of Schizotypy, (2) validity of the CogDis factor as an accurate representation of cognitive disorganisation within the Schizophrenia spectrum, and (3) whether the subscales and/or items of the OLIFE coalesce into a meaningful indicator of general Schizotypy. Each of these concerns will now be reviewed in turn.

Research has raised questions about the face validity of the ImpNon items. Rationally, the items appear to reflect constructs closer to impulsive, aggressive, anti-social, and sensation-seeking traits, much akin to those indicative of Cluster B Personality Disorders, such as Borderline Personality Disorder (BPD) or Anti-social Personality Disorder (ASPD) (Pickering, 2004). Examples of items reflecting this content include '*Do you often have an urge to hit someone?*', '*Have you ever taken advantage of someone?*', and '*Do you ever have the urge to break or smash things?*' (Mason et al., 1995). It should be noted that, historically, BPD and Schizotypal traits have been conceived as falling under the rubric of 'borderline states'; descriptors for patients residing at the boundary between neurosis and psychosis (Gunderson & Singer, 1975; Knight, 1953); however, they were separated and have been treated as independent constructs since the DSM-III (APA, 1980). Despite this shift in nosology, links between cluster B personality traits, especially BPD and SSD traits remain. For example, BPD and SSD are frequently comorbid; however, it is also noted that BPD exhibits high comorbidity with a range of psychopathologies, including ADHD (Philipsen, 2006), anxiety (Grant et al., 2008; Kaess et al., 2013; Tomko et al., 2014), depression (Grant et al., 2008; Kaess et al., 2013), antisocial personality (Becker et al., 2000; Loas et al., 2013), and substance use disorders (Tomko et al., 2014; Trull et al., 2018). Still, other researchers point out the overlap in both aetiology and symptomatology of BPD and SSD (see Zandersen & Parnas, 2019, 2020). Specifically, Zandersen and Parnas (2019a) highlighted the presence of 'identity disturbance' and 'feelings of emptiness' in both BPD and SSD and showed in a small sample of 30 BPD patients, that 70% also met criteria for either Schizotypal Personality Disorder (SPD) or Schizophrenia. Nevertheless, contemporary models of SSD and Schizotypy do not reference impulsivity and BPD as core features (see Barrantes-Vidal et al., 2015; Debbané & Barrantes-Vidal, 2015; Grant et al., 2018). As such, the inclusion of 13 (out of 23) items on the OLIFE ImpNon scale from the Borderline Personality Scale (*n*=7) (STB; Claridge & Broks, 1984), the Eysenck's Personality Questionnaire (EPQ) Lie scale (*n*=4), and the EPQ

Addiction and Criminality scale (*n*=2) (Eysenck & Eysenck, 1975) are not aligned with the current theoretical understanding of Schizotypy. Other researchers have come to similar conclusions and suggest that a disorganisation-like eccentricity dimension may be worth consideration should a fourth factor in Schizotypy be warranted (Kwapil, Gross, Silvia, et al., 2018; Oezgen & Grant, 2018). Although exhibition of borderline and anti-social behaviours in psychosis is sometimes observed (Orjiakor et al., 2019), the inclusion of these items has resulted in poor predictive and discriminative validity. For example, elevated scores on ImpNon are not characteristic of first-degree relatives of people with Schizophrenia (Claridge et al., 1983), nor do high scores on this scale significantly predict increased risk of SSD in the normative population (Chapman et al., 1994). While patients score significantly higher than controls on UnuExp, CogDis, and IntAnh, ImpNon scores did not distinguish between clinical and non-clinical groups (Cochrane et al., 2010; Thomas et al., 2019). Factor analytic studies on the OLIFE short form (OLIFE-SF; Mason et al., 2005) have also raised concerns about the validity of the ImpNon factor with both three- (without ImpNon) and four-factor (with ImpNon) models found to adequately fit the data (Fonseca-Pedrero, Ortuño-Sierra, Mason, et al., 2015; Lin et al., 2013; Sierro et al., 2016). In summary, poor predictive and discriminant validity and structural instability undermine the validity of the ImpNon factor.

Similarly, items that make up the CogDis subscale of the OLIFE have also been criticised (Kwapil, Gross, Silvia, et al., 2018). Although disordered thinking is a well-established dimension of Schizotypy, and SSD more broadly, whether the CogDis subscale validly reflects this dimension is questionable. Indeed, CogDis has frequently been known to correlate with social anxiety and low self-esteem (Fonseca-Pedrero, Ortuño-Sierra, Sierro, et al., 2015; Kwapil, Gross, Silvia, et al., 2018; Mason, 2015). While socially anxious individuals are likely to endorse the items within CogDis, Oezgen and Grant (2018) argue that the reasons for endorsement is fundamentally different for those with social anxiety compared to those high on Schizotypy. They suggest that a socially anxious person may avoid large groups due to feeling anxious about judgements in social situations, while an individual high in disorganized Schizotypy may avoid the same situation because socialising in groups can make it difficult to follow a conversation or train of thought (Oezgen & Grant, 2018). The current CogDis items have been criticised for reflecting social anxiety, rather than true disorganised thinking, which may lead to poor discriminant validity (Kwapil, Gross, Silvia, et al., 2018). As is the case with borderline features, social anxiety is also not considered a core characteristic of Schizotypy and SSD. Therefore, refinement of the current CogDis items to validly reflect disorganised thinking is warranted.

The third, and final, concern surrounding the OLIFE is the absence of a total or global score. The scale's original authors recommended against the use of composite scores which combine two or more of the OLIFE subscales. They argue, from both theoretical and empirical grounds, that there is significant evidence to suggest Schizotypy to be a multidimensional construct with separable components (Mason & Claridge, 2006). This approach has also been retained in newer scales of Schizotypy, such as the Multidimensional Schizotypy Scale (MSS; Kwapil, Gross, Silvia, et al., 2018), where again, computation and use of a 'total' score is not recommended. Nevertheless, from a pragmatic and research point of view, investigating the overall latent structure of the OLIFE and comparing models which include a total score (e.g., hierarchical or bifactor models), would only further our understanding of the construct and measurement of Schizotypy.

Despite the shortcomings reviewed above, the popularity of the original OLIFE led to creation of a short form consisting of 43 items, which also preserve its four-factor structure (OLIFE-SF; Mason et al., 2005). This short form was created by retaining items that maximised the discrepancy concordances between monozygotic (MZ) and dizygotic (DZ) twins. This approach to item reduction exploits genotypic variance; however, it risks the loss of elements of Schizotypy that may not be heritable. Indeed, research has shown that, whilst there are genetic underpinnings of SSD, the presence of sub-clinical and clinical experiences of psychosis are impacted by many determinants including prenatal and postnatal factors, environmental stressors, adverse life events, and drug use (Walker et al., 2004). Therefore, the OLIFE-SF may only be capturing a certain type of Schizotypy; those traits that are most heritable. To build a measure which captures the multidimensional nature of Schizotypy, and not just the heritable elements, application of classical test theory (CTT) and item response theory (IRT) to the refinement of the OLIFE would be beneficial.

2.2.2 Other Measures of Schizotypy

While the current research program used the OLIFE as a measure of Schizotypy, it is acknowledged that there are other measures of Schizotypy available. The Schizotypal Personality Questionnaire (SPQ; Raine, 1991) and the Multidimensional Schizotypy Scale (MSS; Kwapil, Gross, Silvia, et al., 2018) are two other popular inventories that seek to measure Schizotypy and its latent dimensions. As these two measures have also significantly impacted the history and trajectory of the construct of Schizotypy, they will now be reviewed.

Schizotypal Personality Questionnaire (SPQ; Raine, 1991)

The SPQ is a 74-item, dichotomous response (yes/no) questionnaire used to measure schizotypal personality based on the diagnostic criteria found in the Diagnostic and Statistical Manual 3rd edition, revised (DSM-III-R; American Psychiatric Association, 1987). It operationalises these traits into nine subscales: Odd beliefs or Magical thinking, Unusual perceptual experiences, Ideas of reference, Paranoid ideation/suspiciousness, Excessive social anxiety, No close friends, Constricted affect, Odd or eccentric behaviour, and Odd speech. According to Raine (1994), these subscales reflect the three dimensions of Schizotypy; positive, negative, and disorganised symptoms. The Paranoid ideation, Magical thinking, Ideas of reference, and Unusual perceptual experiences subscales are associated with positive Schizotypy, while Constricted affect, No close friends, and Social anxiety subscales measure negative Schizotypy, and Odd eccentric behaviours and Odd speech subscales reflect disorganized Schizotypy (Raine, 1991).

Construction of the SPQ was born from need for a comprehensive measure of Schizotypal Personality Disorder (SPD) which encompassed all associated traits. In this vein, a primary aim of the SPQ was to identify individuals at high risk of developing Schizophrenia, but who were not yet experiencing clinical psychotic symptomology. Therefore, the SPQ is based on the quasi-dimensional model of Schizophrenia, as opposed to the fully dimensional model (Lenzenweger, 2011). These origins of the SPQ are important to consider as they have significant impacts on its utility. Most notably, while there are similarities between Schizotypy and SPD, these concepts are distinct and are not interchangeable (Lenzenweger, 2011). As SPD is viewed as more severe within the Schizophrenia spectrum perspective, the experiences quantified by the SPQ may be too severe to adequately capture Schizotypy (Lenzenweger, 2011). Therefore, use of the SPQ as a measure of pure Schizotypy in the general population may lead to floor effects and an inaccurate representation of the breadth of Schizotypy in the population. While the SPQ was considered for use in the current research program, it was ultimately not chosen for the reasons outlined above.

Multidimensional Schizotypy Scale (MSS; Kwapil et al., 2018)

The MSS is a relatively new measure of Schizotypy which was developed in close consort with the fully dimensional model of Schizophrenia and the three latent dimensions of Schizotypy; positive, negative, and disorganised (Kwapil, Gross, Silvia, et al., 2018). It comprises 77 dichotomous (yes/no) items which load onto the three factors of Schizotypy. According to Kwapil and colleagues (2018), the positive scale assesses experiences related to magical ideation, superstitions, supernatural beliefs, odd somatic and perceptual experiences, suspiciousness, and unusual thought processes, while the negative scale reflects constricted affect, lack of interest in social situations, anhedonia, lack of energy, diminished verbal communication, and avolition, and the disorganised scale measures slow thought processes, difficulty concentrating, loose associations, confusion, racing thoughts, and lack of flow in conversations. The aim in constructing the MSS was four-fold. First, and most importantly, Kwapil and colleagues observed that the available measures for Schizotypy did not reflect contemporary multidimensional models of Schizotypy and SSD, with measures either addressing only one dimension (e.g. magical ideation; Eckblad & Chapman, 1983) or including dimensions with no empirical evidence as a core feature of Schizotypy (e.g., Impulsive Nonconformity from the OLIFE; Mason et al., 1995). Second, all previous measures were constructed using CTT alone. With the advent of newer statistical methods such as IRT and Differential Item Functioning (DIF), the MSS was constructed using all three approaches to maximise psychometric strength. Third, the MSS utilised a much larger and more diverse sample from both university students and Amazon Mechanical Turk (MTurk) during test construction. And finally, the MSS sought to create a measure of Schizotypy with updated and modern language compared to its counterparts. Since its construction in 2018, the MSS has demonstrated strong internal consistency and validity (see Kwapil et al., 2018a; Kwapil et al., 2018b). As data collection for Study 1 was completed when the MSS was published, it was not used as the measure of Schizotypy. Nevertheless, the importance of the MSS in relation to this research program is discussed further in Chapter 6.

2.3 Conclusion

Contemporary models of SSD specify a fully dimensional approach which spans from sub-clinical levels of Schizotypy in the normative population to full-blown psychotic experiences in patient populations, here referred to as the Schizophrenia spectrum. This approach has received significant empirical support (e.g., taxometric studies). The adoption of the Schizophrenia spectrum in psychological research implies the importance and value of understanding Schizotypy; a multidimensional construct associated with Schizophrenia. As such, the studies in this research program will recruit a nonclinical, community sample varying on levels of Schizotypy. Concurrently, the construct of Schizotypy possesses a complex history which has led to heterogeneity in the conceptualisation of its latent dimensions and subsequent measurement. The OLIFE, a popular measure of Schizotypy, has come under significant scrutiny regarding its latent structure and item content. Due to some unexpected results from Study 1, coupled with the criticisms surrounding the OLIFE reviewed above, the third and fourth aims of this thesis were to (3) investigate the validity and factor structure of the OLIFE in an Australian sample and (4) to create and valid and efficient version of the OLIFE using both CTT and IRT.

CHAPTER 3

Study 1: Investigating Dorsal Stream Deficits in SSD Using Visually Guided Movement

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Research shows disproportionate dorsal stream dysfunction, relative to ventral stream, among those with SSD (Doniger et al., 2002; King et al., 2008; Sehatpour et al., 2010). However, there remains a gap in our understanding of the neuropsychology and functional implications of this deficit. In particular, this research neglects another robust and well-validated model of dorsal stream function – the Perception-Action Model (PAM; Goodale & Milner, 1992). Within this framework, the dorsal visual processing stream performs the necessary transformations on visual information to guide movement (e.g., reaching, grasping). Accordingly, disorders with known dorsal stream impairment (e.g., acquired dorsal stream lesions) are associated with selective deficits in visually guided movement (Faugier-Grimaud et al., 1978; Rossetti et al., 2005). However, to date, only one previous study has directly tested whether patients with SSD manifest visuomotor deficits consistent with dorsal stream impairment (King et al., 2008). Although this study supports the hypothesised SSD-related dorsal stream deficit, it tested prehension under conditions of visual illusion, which has been methodologically criticised. Furthermore, most previous studies on dorsal stream deficits in SSD have focussed on patients with diagnosed Schizophrenia. Whilst this population likely exhibits the most pronounced deficits, little is known about whether these deficits manifest across the entire spectrum of SSD or result from confounding factors associated with illness severity (e.g., long-term medication, institutionalisation).

Additionally, research has recently identified two specific sub-pathways of the dorsal stream that control unique visuomotor processes: the dorsomedial and dorsolateral sub-pathways. Whether these pathways are differentially impacted by SSD and, therefore, give rise to discrete visuomotor deficits is unknown. Given that the dorsal stream occupies a large area of posterior cortex, with many cytoarchitectonically distinct regions, a more localised understanding of the SSD-associated impairment is essential. As such, this study aimed to: (1) replicate dorsal visual stream deficits in the broader Schizophrenia spectrum and within the context of PAM using experimental methods that improve on past studies and (2) use behavioural methods to test the integrity of specific dorsal stream sub-pathways.

Briefly, the PAM posits that the ventral stream permits the formation of perceptual representations which contain information about the enduring characteristics of an object in conjunction with its spatial relations (Goodale & Milner, 1992). Conversely, the dorsal stream is associated with mediation of visual control and skilled action (e.g., reaching and grasping). Thus, the dorsal stream acts as an online feedback loop, providing real-time information about the target objects (Budisavljevic et al., 2018; Culham et al., 2003; Tunik et al., 2005). For these reasons, the ventral pathway is often called the vision-for-perception system, while the dorsal pathway is called the vision-for-action system (Goodale & Milner, 1992). Given these functional dissociations, behavioural vision and visually guided movement tasks have been used to differentiate the functional integrity of these visual processing streams.

To date, only one previous study has examined dorsal deficits in people with Schizophrenia through a PAM lens (King et al., 2008). This study used visual illusions as an experimental manipulation by employing a variation of the Ebbinghaus/Titchner illusion to differentiate dorsal and ventral visual processing stream functioning. Two groups of participants, those with diagnosed Schizophrenia and healthy controls, were required to either manually estimate (from a static, home position) or reach towards and grasp a standardised target block next to variably sized flankers. In each condition, the size of participants' grip aperture (i.e., the 3-dimensional (3D) distance between the thumb and index finger opening) was measured via a magnetic motion tracker (Flock of Birds™). Results showed that manual estimation varied as a function of flanker size in both groups. However, only the patient group were influenced by the illusion during the reach-to-grasp condition, supporting a specific, Schizophrenia-related dorsal stream deficit. Since this initial study, no follow-up studies have been conducted to validate these findings. Moreover, given criticisms surrounding the use of visual illusions to elucidate vision-for-action and vision-for-perception systems (see Chapter 1), replication of these findings using alternative methods is justified.

In addition, neuroimaging studies have differentiated two anatomically segregated parietofrontal circuits (i.e., sub-pathways) within the dorsal stream: (1) the dorsomedial circuit, consisting of the posterior parietal cortex (PPC) and the caudal dorsal premotor cortex (PMd); and, (2) the dorsolateral circuit, consisting of the anterior intraparietal (AIP) area and the ventral premotor cortex (PMv) (Galletti et al., 2003; Grol et al., 2007). These sub-pathways have been linked to independent components of manual prehension, a reach component and a grasp component, respectively (Grol et al., 2007; Jeannerod, 1988). Furthermore, these sub-pathways are also differentially recruited based on target characteristics. Specifically, during prehension toward stationary targets, the dorsomedial circuit is recruited, while for perturbed targets, the dorsolateral circuit (Tunik et al., 2005). Taken together, these associations provide well-defined behavioural methods for dissociating ventral and dorsal visual stream integrity and dorsomedial and dorsolateral sub-pathway integrity through different manual prehension tasks (see Figure 3.1). Importantly, these sub-pathways and their functional integrity have yet to be investigated in SSD.

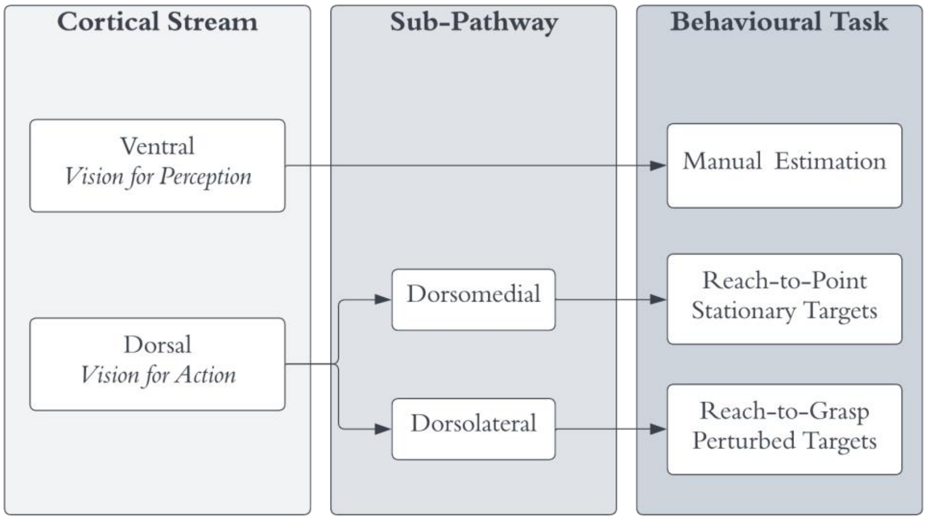

Figure 3.1. Dissociation of dorsal/ventral and dorsomedial/dorsolateral pathways through behavioural measures.

3.1 Kinematics of Prehension

The operationalisation of manual prehension tasks as correlates of underlying neural circuitry lies in the measurement of kinematics, a subfield of physics concerned with the geometry of motion. For manual estimation, Stable Grip Aperture (SGA), the stabilised 3D distance between the thumb and index finger, is most often used to quantify prehension during perception as it allows for more direct comparison with kinematic variables as opposed to other measures such as verbal reports of estimated size (Franz, 2003). Furthermore, it provides task validation such that participants with intact ventral stream functioning should produce SGA measures that differ as a function of target size, i.e., larger apertures for larger targets and vice versa.

The kinematics of reach-to-point and reach-to-grasp include a range of measures quantifying the time and space parameters of prehension. Basic examples of these include movement time (the difference in time between movement onset to movement offset), grip aperture (the vector distance between the index finger and thumb finger), and hand speed (the velocity of the hand at any one point during movement). Used in combination, these variables provide rich information about the characteristics of manual prehension. Research on prehension in normative populations has established that the kinematics of reaching movements consists of two phases: an initial acceleration phase and a subsequent deceleration phase (Elliott et al., 2001; Lin et al., 2007, 2010). These phases are demarcated by the time of Peak Hand Speed (PHS) (i.e., the time at which the maximum velocity of the hand during the movement is reached). The proportion (%) of movement time spent reaching PHS ($PHS_{t\%}$) (i.e., the acceleration phase) reflects pre-planned, feedforward control (Lin et al., 2007, 2010). Conversely, the remaining proportion reflects visual or proprioceptive feedback to correct movement trajectories. Therefore, greater reliance on pre-planned, feedforward control is evidenced by relatively greater $PHS_{t\%}$, while greater reliance on feedback and corrective control is reflected in relatively lower $PHS_{t\%}$ (Wang et al., 2011; Wang et al., 2018; Wu et al., 2001). Greater PHS is also associated with reach-to-grasp versus reach-only tasks (Grol et al., 2007). Based on this framework, PHS and $PHS_{t\%}$ are the key kinematic variables of interest for understanding reach movements.

Similarly, during grasping the Peak Grip Aperture (PGA), the maximum vector distance between the thumb and index finger throughout the movement, is known to correlate with target size, such that larger targets elicit larger PGAs (Breveglieri et al., 2022; Tunik et al., 2005). Furthermore, perturbed targets also elicit larger PGAs compared to unperturbed/stationary targets (Breveglieri et al., 2022). Like reach, the grasping component of prehension is also understood to consist of two phases: an initial pre-programmed grip formation phase, followed by a feedback and corrective phase (Erickson & van Kan, 2005). These phases are demarcated by the occurrence of PGA. The proportion of time spent to achieve PGA, denoted as $PGA_{t\%}$, reflects the pre-programmed grasping component of prehension, with the remainder denoting feedback and grip correction. Therefore, greater $PGA_{t\%}$ signals greater reliance on feedforward, pre-planned processing with the converse, suggesting greater reliance on feedback, corrective processing. Based on these relationships, PGA and $PGA_{t\%}$ are key variables of interest during grasping movements.

3.2 Present Study

The purpose of the present study was to examine SSD-related dorsal stream (and sub-pathway) deficits using the PAM as an explanatory framework. To achieve this goal, a novel task was developed that combines target (i.e., stationary versus perturbed) and movement (i.e., reach-to-point versus reach-to-grasp) manipulations in ways that allow for discrete functional measurement of dorsal stream sub-pathways (i.e., dorsomedial and dorsolateral pathways). Potential confounds of illness severity were mitigated by recruiting a normative population sample varying on levels of trait-level Schizotypy.

Based on the literature review and the aims of this study, the following hypotheses are presented:

1. Level of Schizotypy will not be associated with changes in SGA during manual size estimation. This will confirm intact ventral visual stream functioning across the sample.
2. Greater Schizotypy will be associated with greater proportion of overall movement time taken to reach peak hand speed ($PHS_{t\%}$) during reach-to-point and reach-to-grasp. If this relationship is observed, it will demonstrate Schizotypy traits to be linked to a stronger reliance on pre-planned movement and implicate dorsomedial sub-pathway dysfunction.
3. Greater Schizotypy will also be associated with greater PGA and proportion of overall movement time taken to reach PGA ($PGA_{t\%}$) during reach-to-grasp. This will show impairment in target scaling and correction during grasp, respectively. This pattern of performance would implicate dorsolateral dysfunction in Schizotypy.

3.3 Method

3.3.1 Participants

One hundred and fourteen (81 females, 32 males, and 1 nonbinary) undergraduate students from the Australian National University completed the study for course credit or monetary remuneration. All participants reported normal or corrected-to-normal vision, which was confirmed with a Snellen test during the experiment. One participant withdrew partway through the study, and another failed to achieve 20/20 on the Snellen test; both were excluded. Twelve participants were excluded for being left-handed as the task required wrist rotation in a clockwise direction. Three participants were excluded because either their Negative Impression Management ($T \geq 92$) or Positive Impression Management ($T \geq 68$) scores from the Personality Assessment Inventory (Morey, 1991) suggested biased or distorted responding. Participants also completed a general health questionnaire which queried their own mental health diagnoses as well as first-degree relatives. Participants were not excluded based on mental health history, as previous studies have demonstrated high levels of Schizotypy to be associated with a broad range of mild psychopathology (Barrantes-Vidal et al., 2009; Neill, 2014). However, if they reported an SSD diagnosis for either themselves or a first-degree relative, they were removed in order to keep the sample within the bounds of subclinical SSD. Overall, no participants were removed based on this exclusion criteria. Follow data extraction, three additional participants were excluded because their motion-tracking data could not be extracted. The final sample consisted of 94 participants. Table 3.1 summarises the final sample's demographic characteristics, and Table 3.2 summarises clinical and health characteristics. Refer to Appendix B for extended OLIFE descriptives and distribution of the final sample.

Table 3.1 Summary of demographic characteristics of sample.

Variable	Mean (SD)/Proportion
Age (years)	21.1 (SD=3.98) (range 18 to 44)
Gender	67 female (26 male, 1 undisclosed)
Ethnicity	32 Caucasian; 62 Asian
Handedness	94 right-handed

Table 3.2 Summary of health demographics, Schizotypy level (OLIFE), general psychopathology (PAI) scores for the sample.

Measure	Mean (SD)/Proportion	
Estimated FSIQ	108 (11.67)	
Health demographics		
Mental Illness or Psychiatric Dx	10.6%	(primarily depression and anxiety)
Relative with Mental Illness or Psychiatric Dx	16.0%	(primarily depression and anxiety)
Neurological Dx or episode	6.4%	(primarily concussion)
Perceptual or Cognitive Dx or disturbance	1.1%	
Learning Disorder Dx or learning difficulties	2.1%	
ADHD Dx or school related problems	3.2%	(all ADHD Dx)
OLIFE (Raw Score)		
Unusual Experiences (UnuExp)	10.5 (6.0)	
Introvertive Anhedonia (IntAnh)	8.8 (5.0)	
Cognitive Disorganisation (CogDis)	14.3 (5.0)	
Impulsive Nonconformity (ImpNon;	7.4 (3.1)	
Total Sore	41.0 (12.7)	
PAI-SF (T-Score)		
Infrequency (INF)	54 (10.4)	
Negative Impression Management (NIM)	53 (9.5)	
Positive Impression Management (PIM)	46 (10.0)	
Somatic Complaints (SOM)	50 (7.1)	
Anxiety (ANX)	60 (12.2)	
Anxiety-Related Disorders (ARD)	59 (12.0)	
Depression (DEP)	58 (11.6)	
Mania (MAN)	53 (10.3)	
Paranoia (PAR)	52 (9.1)	
Schizophrenia (SCZ)	57 (11.9)	
Borderline Features (BOR)	58 (9.4)	
Antisocial Features (ANT)	54 (8.4)	
Alcohol Problems (ALC)	50 (8.6)	
Drug Problems (DRG)	49 (6.9)	
Aggression (AGG)	49 (8.4)	
Suicidal Ideation (SUI)	53 (11.1)	
Nonsupport (NON)	55 (11.5)	
Treatment Rejection (RXR)	46 (9.6)	
Dominance (DOM)	48 (10.3)	
Warmth (WRM)	44 (10.8)	

Note: Unless otherwise stated, values are means (and standard deviations). Estimated full scale IQ from the Matrix Reasoning and Coding subtest of the Wechsler Adult Intelligence Scale—Fourth Edition (validity of .881 for the dyad selected; (Girard et al., 2015)). Dx = diagnosis. ADHD = Attention Deficit Hyperactivity Disorder. OLIFE = Oxford Liverpool Inventory of Feelings and Experiences. PAI-SF = Personality Assessment Inventory Short Form. All PAI-SF subscale scores are T scores.

3.3.2 Materials

Clinical and Cognitive Instruments.

A *Demographic and Health Questionnaire* collected participants' age, sex, gender identity, handedness, ethnicity, educational level, occupation, and whether English was their first language. Health related information included history of mental, psychiatric, neurological, perceptual, learning, and school-related behavioural problems, as well as whether participants had first degree relatives with mental or psychiatric disorder.

The *Oxford Liverpool Inventory of Feelings and Experiences (OLIFE; Mason et al., 1995)* quantified Schizotypy through 104 true/false items which measure Schizotypy. The items fall into four factors: Unusual Experiences (UnuExp), Introvertive Anhedonia (IntAnh), Cognitive Disorganisation (CogDis), and Impulsive Nonconformity (ImpNon). A total score was calculated by summing all factor scores; $\text{OLIFE}_{\text{Total}}$. A brief inconsistency scale was developed by taking four items from the first half of the OLIFE, negatively wording them, and presenting them again in the second half of the OLIFE. This scale was scored by summing the eight items (four original items and four negatively items). As the negatively worded items would cancel out the original items, scores ranged from -4 to 4 with a score of 0 denoting perfect consistency and greater deviations demonstrating greater inconsistency. This measure was only used as source of response validation and was not included when scoring the OLIFE.

The *Personality Assessment Inventory Short Form (PAI-SF; Morey, 2014)* is a 160-item measure of adult personality and psychopathology. These items fall into 19 subscales. This included two validity scales: Positive Impression Management (PIM), Negative Impression Management (NIM), eleven clinical scales: Somatic Complaints (SOM), Anxiety (ANX), Anxiety-Related Disorders (ARD), Depression (DEP), Mania (MAN), Schizophrenia (SCZ), Paranoia (PAR), Borderline Features (BOR), Antisocial Features (ANT), Alcohol Problems (ALC), Drug Problems (DRG), four treatment scales: Aggression (AGG), Suicidal Ideation

(SUI), Nonsupport (NON), Treatment Rejection (RXR), and two interpersonal scales: Dominance (DOM), and Warmth (WRM). Participants responded on a four-point scale ranging from False, Somewhat True, Mainly True, and Very True.

The *Matrix Reasoning and Coding subtests from the Wechsler Adult Intelligence Scale 4th edition (WAIS-IV; Wechsler, 2008)* were used to estimate global intelligence and chosen based on research verifying Matrix Reasoning and Coding as a psychometrically robust two-point estimate of Full-Scale IQ (FSIQ; as measured by the WAIS-IV) (Girard et al., 2015). FSIQ estimates were calculated using the formula from (Tellegen & Briggs, 1967).

The *Snellen Eye Test (Snellen, 1873)* is a simple visual acuity test. Where appropriate, participants were asked to wear their corrective lenses throughout the experiment.

Experimental Apparatus.

Index finger, thumb, and wrist positions were tracked during the experiment with a magnetic motion-tracking system (3DGuidance trakSTAR™; Acension Technologies Corporation, Milton, VT). Three-dimensional positioning data including x, y, z coordinates and three measures of angular rotation (i.e., elevation, azimuth, and roll) were sampled at 134Hz for the duration of experimental conditions. These coordinates were in reference to a magnet that was placed 200mm to the left of the receiver/amplifier. A custom-built unit controlled the conditions under which participants completed reaching and grasping movements. This unit was a 300mm x 300mm x 300mm black box, constructed from laminated medium density fibreboard, with wheels on its base that were placed on 35mm tracks to allow for horizontal movement. Inside, it was fitted with a rotary actuator motor (Kollmorgen model no. S6MH4) connected to a horizontal axel, which extended through a 10mm x 10mm hole in the centre of the front panel onto which target objects could be attached. The front facing edge of the unit was placed 420mm from the participant. Target objects were attached at a centre height of 190mm from desk level. The motor was controlled through a USB Digital I/O (USB-1024 Series from Measurement Computing) which was programmed using custom written code in LabView (Bitter et al., 2006). The axel was synced with a home button (100mm x 170mm) placed 30mm in front of the participant. Depression of the home button was used as a starting point of movement for participants for all trials. On perturbation trials, releasing the home button would initiate target rotation. Target objects were opaque, white resin rectangular prisms with a width and depth of 10mm but varied in length from 50mm, 70mm, and 100mm. One end of the target was marked with a red dot as a reference position for reach-to-point movements. Refer to Figure 3.2 for a schematic representation of the experimental setup.

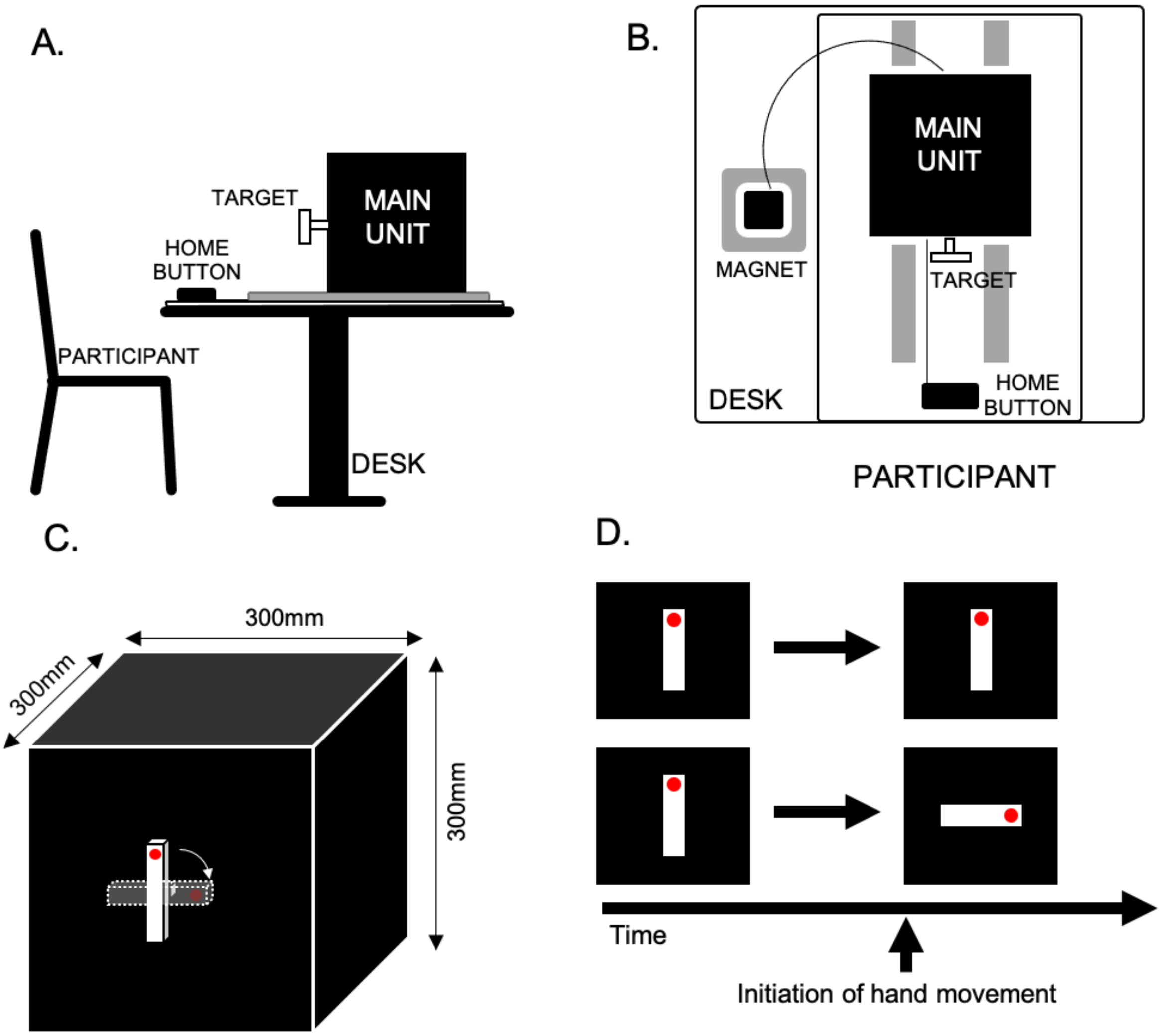

Figure 3.2 Schematic of experimental setup. A. Side view of experimental setup. B. Top view of experimental set up. C. Main unit and schematic of target object rotation. D. Schematic representation of trial type where the top row shows stationary trials across time and bottom row shows perturbed/rotation trials across time. The red dot denotes target index finger position on reach-to-point trials.

3.3.3 Procedure

All participants reviewed an information sheet and provided informed, written consent prior to commencing the study. Participants then completed all questionnaires on an iPad whilst the experimenter was out of the room. Refer to Appendix B for copies of these documents. Upon completion, the participant was administered the Snellen test and Matrix Reasoning and Coding subtests from the WAIS-IV. Participants then completed three experimental tasks (size estimation, reach-to-point, and reach-to-grasp). The order of tasks was such that size estimation was completed first, followed by the movement tasks, which were counterbalanced between participants. This sequence was chosen as to avoid any contamination by movement tasks on size estimation, e.g., from muscle memory.

For the size estimation condition, participants were required to manually estimate, using their dominant thumb and index finger, the length of three targets (5cm, 7cm, and 10cm). The prism was attached to the axel extending from the front of the encasement box, such that the length of the prism was clearly visible and directly in front of the participant. Participants were instructed to adjust their index finger and thumb aperture to indicate the length of the target object, while keeping their hand over the home button (and away from the target). Participants completed three manual estimations for each of the three targets.

For reaching tasks, participants were asked to start in a 'home' position, which entailed pressing down a 100x170mm home button (with their dominant hand) that was positioned 30mm in front of the edge of the desk where they were seated. The start of each trial was signalled by an audio beep, at which time participants initiated the pre-instructed movement (i.e., either reach-to-point or reach-to-grasp). On reach-to-point trials, participants released the home-button and moved their index finger until it contacted the red dot (8mm in diameter) placed 1mm from the top, front-facing edge of the target object (see Figure 3.2. D). On reach-to-grasp trials, participants moved their index and thumb to either end of the long edge of the target and grasped the target, such that their index finger was on the same end as the red dot. After successfully contacting the target object, participants were instructed to return to the 'home' position and depress the home-button to signal the end of a trial and wait for the next audio beep to start the next trial. On perturbation trials, release of the home-button initiated target rotation which entailed a 90° clockwise rotation.

All participants completed four experimental conditions with a 2 (movement type; point or grasp) x 2 (target type; stationary or perturbed) x 2 (target size; 50mm or 70mm) design. Each condition consisted of ten trials. Movement type and target size were kept consistent within conditions; however, target type was pseudo-randomised to achieve half stationary and half perturbed trials within each condition. All aspects of this project were approved by the ANU Human Ethics Committee.

Motion Tracking Data Processing and Extraction

Participant data files were split into individual movement files and then processed through a custom Python program (Python v2.7.18; Van Rossum & Drake, 1995) which extracted the kinematic variables listed in Table 3.3 for each individual movement parameters across the eight experimental conditions. During data extraction, data for 14 participants was unable to be extracted for at least one of the eight experimental conditions, leaving incomplete data sets for those participants. The data that was extracted for these participants were still included in the analyses (refer to Appendix B for details of missing data).

Table 3.3 Summary of kinematic variables extracted from raw motion tracking data.

Variables Measured	Variable Description
Movement Time (MT)	Total time from movement onset to movement offset.
Movement onset (M_{on})	The onset of reach and grasp movements was determined as exceeds 5% of the respective peak wrist velocity.
Movement offset (M_{off})	The moment when the velocity of the wrist sensor in the x axis direction falls and remains below 5% of peak velocity, i.e., the target is grasped.
Grip Aperture (GA)	The vector distance between the index finger and thumb finger sensors, measured at 10% intervals of MT from 0% (movement onset) to 100% (movement offset). GA reflects the opening and closing of the fingers that occurs when reaching to grasp an object.
Peak Grip Aperture (PGA)	The maximum three-dimensional distance between the index and thumb markers between movement onset & offset.
Time to Peak Grip Aperture (PGA_t)	Difference in time between the movement onset and when PGA is achieved.
Peak Hand Speed (PHS)	Maximum velocity reached during movement as measured by the wrist marker.
Time to Peak Hand Speed (PHS_t)	Difference in time between the movement onset and when PHS is achieved.
Stable Grip Aperture (SGA)	Averaged grip aperture across duration of each individual target size estimation.

3.4 Results

The data was analysed using repeated measures analysis of covariance (RM ANCOVA), with conditions (target size, movement type, and target type) as the independent variables, $OLIFE_{Total}$ as a covariate, and kinematic variables as dependent variables. Additional RM ANCOVAs were also run with individual OLIFE factors as covariates. Where appropriate, the Bayesian counterpart to RM ANCOVA was calculated to further verify effects. Main effects were assessed using BF_{10}. To isolate interaction effects, the $BF_{inclusion}$ (BF_{incl}) factor was used, which compares models with the effect to equivalent models without the effect, excluding higher order interactions (JASP Team, 2022). The size of BF_{incl} can be interpreted in the same way as BF_{10}, such that, values below 1 indicate support for the null hypothesis, values between 1 and 3 suggest a weak effect, values between 3 and 10 suggest a moderate effect, and values greater than 10 suggesting a strong effect (van den Bergh et al., 2020). For continuity, and ease of comparison, the frequentist and Bayesian results are reported together. For extended details on data analysis including corrections for non-normality, see Appendix B.

3.4.1 Control Variables

Prior to reviewing the main results, analyses of control variables were completed to validate experimental manipulations and confirm that participants were responsive to these manipulations.

Target Size

Target size was varied to control for potential artefacts arising from using a single target size. Across seven kinematic variables, three revealed significant main effects for target size. As expected, these occurred in SGA, GA, and PGA where larger targets elicited larger grip apertures. A total of 117 interaction effects were tested with target size and only four were significant. Two of the four were observed in PGA and GA analysis, which were expected. The remaining two were unexpected and occurred with PHS and PHS_t. For PHS, a suppressor effect between Target Size x Movement Type x ImpNon was observed ($F(1, 75) = 2334.3$, $p = .006$, $\eta_p^2 = .10$). Similarly, a small suppressor effect in PHS_t between Target Size x Movement Type x UnuExp interaction was revealed ($F(1, 75) = 6.4$, $p = .014$, $\eta_p^2 = .08$). Given their very small effect sizes and the number of familywise comparisons conducted across these analyses (i.e., increasing the probability that these effects are spurious), further interpretation of these was withheld. Therefore, the subsequent results are not likely to be due to an artefact of target size.

Movement Time (MT)

Differences in MT across all conditions as a function of Schizotypy were tested. Since many of the kinematic variables of interest are time sensitive (e.g., peak hand speed time), it is important to demonstrate that any differences observed in key kinematic variables are due to differences in Schizotypy or other experimental manipulations and not from underlying variations in movement time. As is common with time data, raw MT data across all conditions demonstrated non-normality (Shapiro-Wilk test *p* ranged from <.001 to .087). A log transformation improved normality (Shapiro-Wilk test *p* ranged from .016 to .733). See Appendix B for summary of corrections. Log(MT) was then submitted to a 2 (Target Size) x 2 (Movement Type) x 2 (Target Type) RM ANCOVA. No significant effects were observed between MT and level of Schizotypy, suggesting that participants did not differ in movement time as a function of Schizotypy level. Significant main effects were observed for Movement Type (point, grasp), $F(1, 75) = 43.6$, $p<.001$, $\eta_p^2 = .37$, $BF_{10} = 3.1e^6$, and Target Type (stationary, perturbed), $F(1,75) = 24.5$, $p<.001$, $\eta_p^2 = .25$, $BF_{10} = 3283.7$. The interaction between Movement Type and Target Type was also significant, $F(1,75) = 7.8$, $p = .007$, $\eta_p^2 = .09$, $BF_{incl} = 5.3$. These results show that grasping movements towards perturbed targets took longer to complete than pointing movements towards stationary targets. This pattern of results is consistent with previous manual prehension research in normative populations (Wang et al., 2018).

3.4.2 Hypothesis 1: Manual Estimation

Stable Grip Aperture (SGA)

Participants' perception of target size was assessed by analysing SGA across three manual estimation conditions (5cm, 7cm, and 10cm). Due to violations of sphericity (Mauchly's test of sphericity $p<.01$), Greenhouse-Geisser corrected results are reported, where appropriate (see Appendix B for full results). A main effect for Target Size was observed, $F(1.8, 158.9) = 867.9$, $p<.001$, $\eta_p^2 = .91$, $BF_{10} = 7.14e^{90}$. These results show that perceived target size differs as function of actual target size (i.e., shorter targets were estimated as shorter, and longer targets estimated as longer; see Figure 3.3, where, for illustrative purposes, participants have been grouped into high and low OLIFE scores around the median). A significant, albeit small covariate effect, was observed with CogDis, $F(1.8, 158.9) = 3.6, p<.034, \eta_p^2 = .04, BF_{incl} = 6.70$, such that, while at small target sizes (5cm) SGA did not differ as a function of CogDis, at larger target sizes (7cm and 10cm) CogDis was positively associated overestimation of target sizes. No other measures of Schizotypy; UnuExp, IntAnh, ImpNon, or $OLIFE_{Total}$ were associated with SGA. These results support the first hypothesis, that scores on the OLIFE would not correlate with perceptual, manual estimations (i.e., the ventral stream task in this experiment).

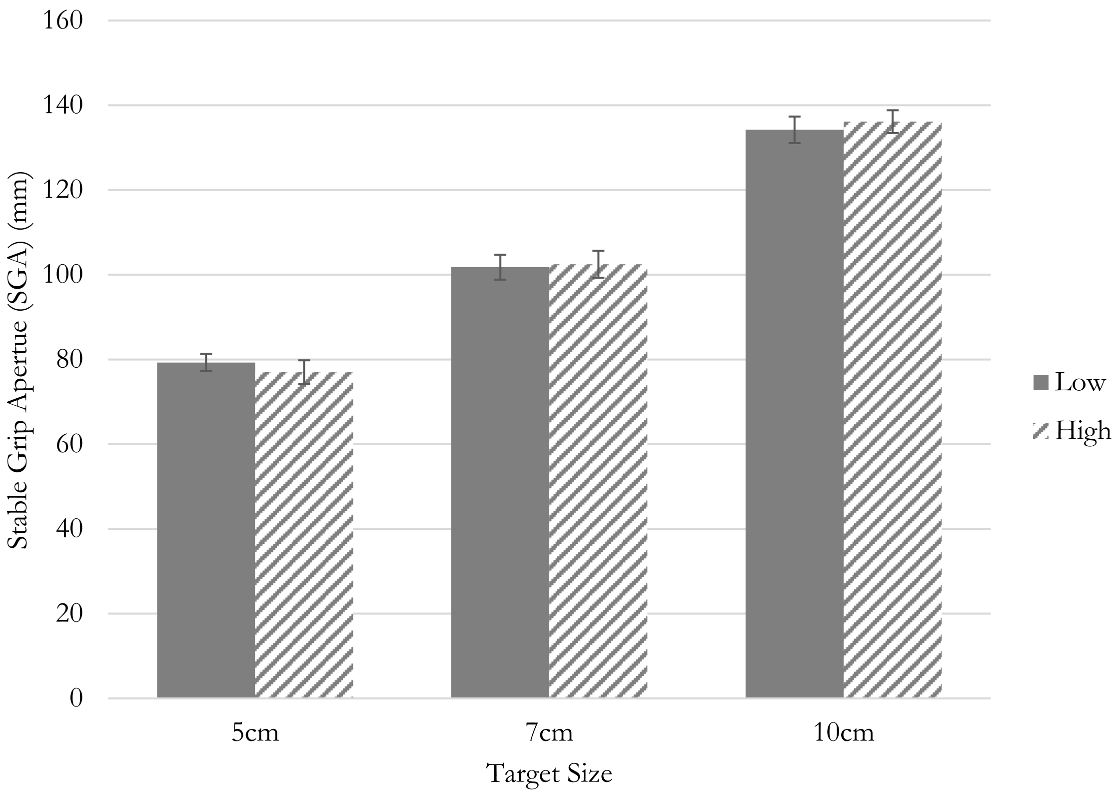

Figure 3.3 Comparison of SGA at each target size with a median split applied to level of Schizotypy (OLIFE Total Score). Low Schizotypy = solid bars, high Schizotypy = striped bars. Error bars represent standard error.

3.4.3 Hypothesis 2: Reliance on pre-planned movement

To test whether increased Schizotypy is associated with a stronger reliance on pre-planned movement, 2 (Target Size) x 2 (Movement Type) x 2 (Target Type) RM ANCOVAs were computed for Peak Hand Speed (PHS) and proportion of overall MT taken to reach PHS ($PHS_{t\%}$), with $OLIFE_{Total}$ and factor scores as covariates. Observing larger $PHS_{t\%}$ with increased OLIFE scores would support this hypothesis.

Peak Hand Speed (PHS).

A significant main effect of Movement Type (point vs. grasp), $F(1, 75) = 76.9$, $p<.001$, $\eta_p^2 = .51$, $BF_{10} = 2.37e^{10}$. This shows PHS was greater in reach-to-grasp compared with reach-to-point movements. This effect is consistent with previous studies of prehension comparing grasp versus reach (Grol et al., 2007). No significant effects were found as a function of Schizotypy or its latent dimensions (see Figure 3.4).

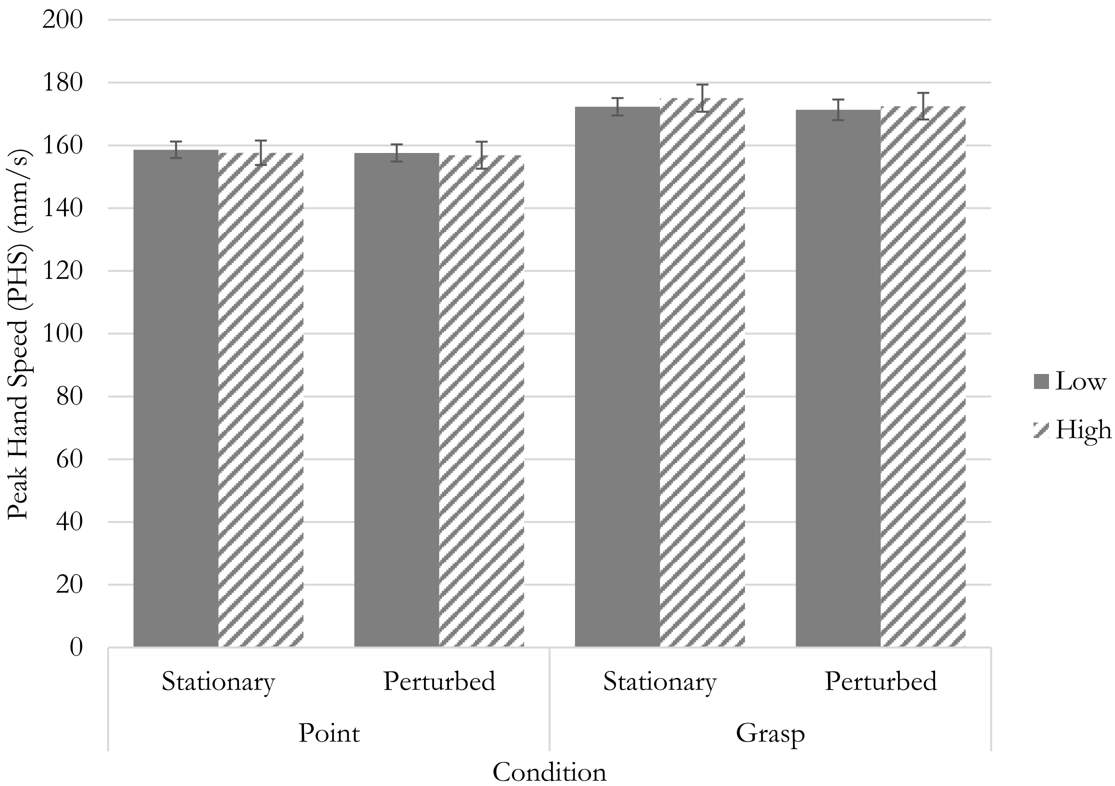

Figure 3.4 Results are collapsed across target size.

Peak Hand Speed Time (PHS_t).

To standardise PHS_t across total MT, PHS_t data were transformed to a percentage of MT and is denoted as $PHS_{t\%}$. A significant main effect of Target Type (stationary vs. perturbed) was revealed, $F(1, 75) = 31.3$, $p<.001$, $\eta_p^2 = .30$, $BF_{10} = 21687.6$, whereby $PHS_{t\%}$ was significantly smaller (i.e., PHS occurred earlier in the movement) during perturbed compared to stationary trials). This is consistent with previous research showing that more time is spent in corrective feedback during perturbed compared to stationary trials (Dubrowski et al., 2002). Significant, albeit small, interactions were observed between Target Type x Target Size (η_p^2=.09) and Target Type x Movement Type (η_p^2=.08). These showed that PHS occurred later (larger $PHS_{t\%}$) on stationary trials for larger targets (7cm) but earlier on perturbed trials for larger targets (7cm) compared to smaller targets (5cm) and that on perturbed trials, PHS occurred later (larger $PHS_{t\%}$) during reach-to-point compared to reach-to-grasp movements, respectively.

The relationship to Schizotypy was observed in the Movement Type (point, grasp) x $OLIFE_{Total}$ covariate interaction, $F(1, 78) = 9.67$, $p = .003$, $\eta_p^2 = .06$, $BF_{10} = 9.73$ (see Figure 3.5). This interaction showed that increased Schizotypy is associated with greater $PHS_{t\%}$ (i.e., more time spent in pre-planned movement); however, this was only seen in the reach-to-point condition. During reach-to-grasp, $PHS_{t\%}$ did not differ as a function of Schizotypy. Target Type (stationary, perturbed) x IntAnh interaction was also significant, $F(1, 75) = 4.689$, $p = .034$, $\eta_p^2 = .059$, however, was not supported in the Bayes analysis, $BF_{incl} = 0.73$.

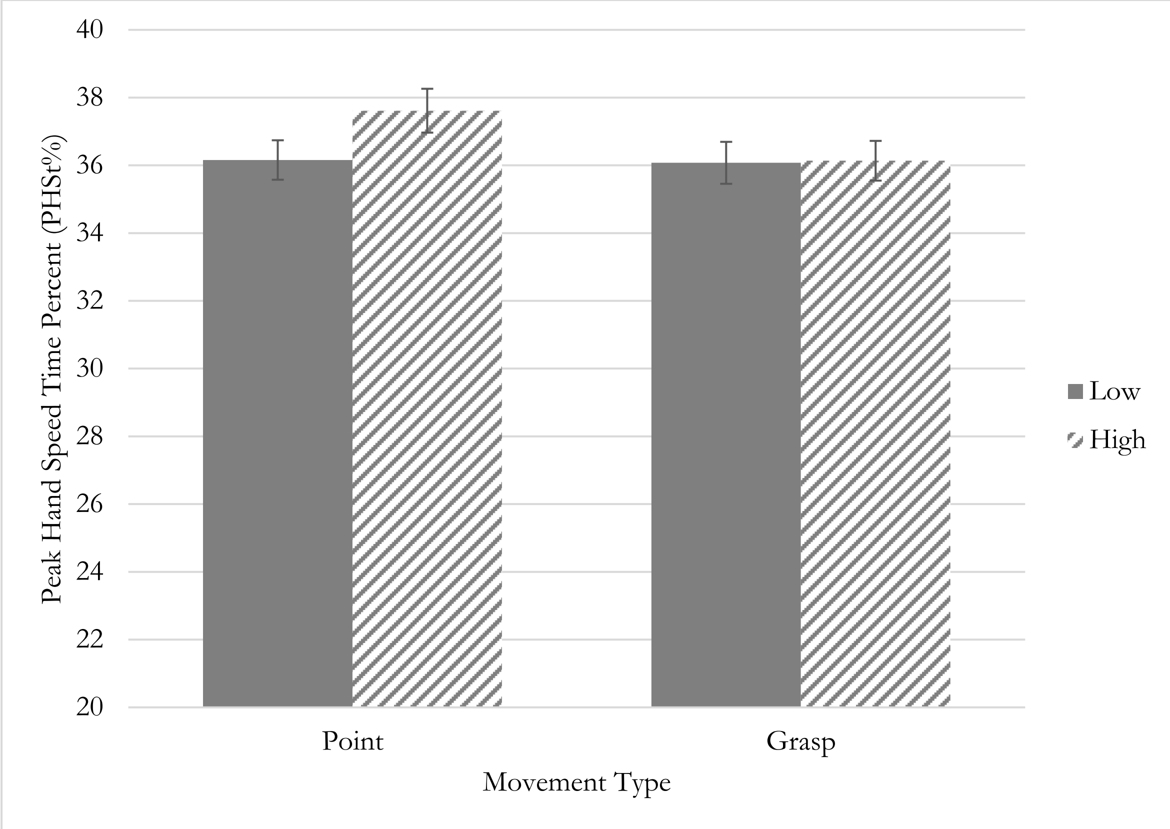

Figure 3.5 Peak Hand Speed Time Percent as a function of Movement Type (point, grasp) and Schizotypy (OLIFE total score) with a median split applied. Values are averaged across unused RM factors. Error bars represent standard error.

Overall, these results partially support the second hypothesis. Whilst increased Schizotypy was associated with larger $PHS_{t\%}$, indicating great time spent in the pre-programmed phase of movement, this was only seen during reach-to-point movements.

3.4.4 Hypothesis 3: Target size scaling and correction during grasping

To test whether increased Schizotypy is associated with impairments in target scaling and correction, 2 (Target Size) x 2 (Movement Type) x 2 (Target Type) RM ANCOVAs were computed for Peak Grip Aperture (PGA) and proportion of overall MT taken to reach PGA ($PGA_{t\%}$), with $OLIFE_{Total}$ and factor scores as covariates. Observing larger PGA and $PGA_{t\%}$ with increased OLIFE scores would support this hypothesis.

Peak Grip Aperture (PGA).

A significant main effect of Target Size (5cm, 7cm), $F(1, 81) = 24.4$, $p<.001$, $\eta_p^2 = .23$, $BF_{10} = 3.40e^{32}$ was revealed. This confirms that participants correctly scaled larger grip apertures for larger targets. A main effect of Target Type (stationary vs. perturbed), $F(1, 81) = 7.89$, $p = .006$, $\eta_p^2 = .09$, $BF_{10} = 8.07e^{8}$, showed participants also produced larger apertures on perturbed compared to stationary trials. A significant main effect of $OLIFE_{Total}$ was also observed, $F(1, 84) = 4.2$, $p = .043$, $\eta_p^2 = .048$, $BF_{10} = 1.70$, which showed a small effect such that Schizotypy was positively correlated with PGA across all conditions, see Figure 3.6. Although the interaction between Target Type x Target Size was insignificant, when UnuExp was added as a covariate, a small effect was observed, $F(1, 81) = 5.47$, $p = .022$, $\eta_p^2 = .063$, $BF_{incl} = 1.87$. This showed that, for perturbed targets, higher UnuExp was associated with larger PGA for smaller targets (5cm) but not larger targets (7cm), where PGA was comparable across the spectrum of UnuExp scores.

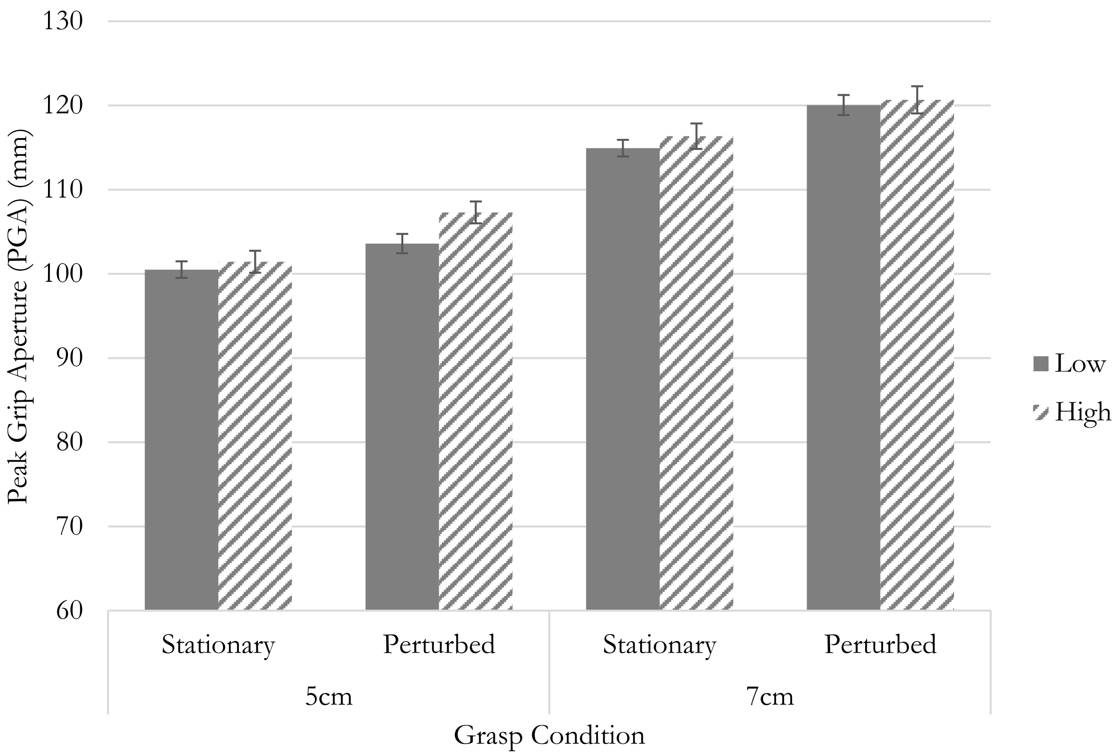

Figure 3.6 Peak Grip Aperture (mm) as a function of Target Type (stationary, perturbed), Target Size (5cm, 7cm) and Schizotypy (OLIFE total score) with a median split applied on reach-to-grasp trials. Values are averaged across unused RM factors. Error bars represent standard error.

Peak Grip Aperture Time (PGA_t).

To standardise PGA_t across total MT, PGA_t data was transformed to a percentage of MT and is denoted as $PGA_{t\%}$. $PGA_{t\%}$ analysis revealed a significant main effect of Target Type (stationary, perturbed), $F(1, 81) = 20.6$, $p<.001$, $\eta_p^2 = .20$, $BF_{incl} = 391.08$, and Target Size (5cm, 7cm), $F(1, 81) = 8.4$, $p<.005$, $\eta_p^2 = .09$, $BF_{incl} = 6.65$. No significant effects were observed with level of Schizotypy as a covariate. These results show the proportion of overall movement time taken to reach PGA is only impacted by target type and size, such that $PGA_{t\%}$ is delayed in perturbed compared to stationary trials and in larger (7cm) compared to smaller (5cm) targets, see Figure 3.7.

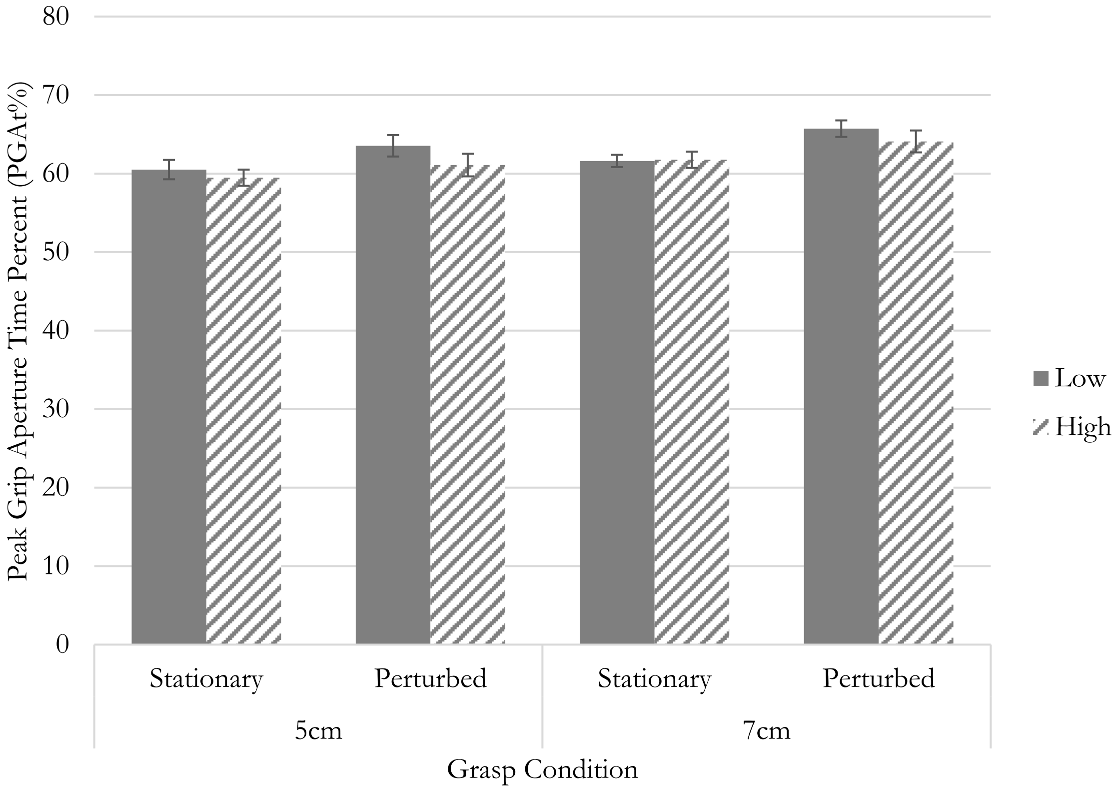

Figure 3.7 Proportion of overall movement time taken to reach Peak Grip Aperture ($PGA_{t\%}$) as a function of Target Type (stationary, perturbed), Target Size (5cm, 7cm) and Schizotypy (OLIFE total score) with a median split applied on reach-to-grasp trials. Values are averaged across unused RM factors. Error bars represent standard error.

Overall, these results partially support the third hypothesis, such that increased Schizotypy was associated with greater PGA across all conditions suggesting less accurate target scaling. However, the proportion of overall movement time taken to reach PGA ($PGA_{t\%}$) was not associated with Schizotypy and, therefore, increased Schizotypy does not correlate with greater reliance on pre-planned movement.

Grip Aperture (GA)

Given the coarseness of using PGA, grip aperture (GA) across the entire movement was analysed in 10% increments from 0% (movement onset) to 100% (movement offset at the target) to further investigate whether Schizotypy is associated with changes in target scaling across the entire course of reach-to-grasp movements. GA over 11 Intervals of Movement (0% to 100%) were submitted to a repeated measures ANCOVA with Target Size (5cm, 7cm) and Target Type (stationary, perturbed) for reach-to-grasp conditions. Significant main effects of Interval of Movement, Target Size, and Target Type were observed, $F(2.4, 191.7) = 757.6$, $p<.001$, $\eta_p^2 = .90$, $F(1, 81) = 219.9$, $p<.001$, $\eta_p^2 = .73$, and $F(1, 81)= 56.1$, $p<.001$, $\eta_p^2=.41$, respectively. These results suggest that grip aperture changed across the movement, was greater for the 7cm compared to the 5cm target, and greater during perturbed compared to stationary trials. A significant interaction effect of Target Type x Interval of Movement was observed, $F(3.4, 274.4) = 15.8$, $p<.001$, $\eta_p^2 = .16$. The Target Size x Interval of Movement interaction was also significant, F(2.8, 229.6)=77.3, p<.001, , $\eta_p^2=.488$.

A significant, although small, between subjects main effect for OLIFE total was observed, $F(1,84) = 4.9$, $p= .03$, $\eta_p^2=.06$. This showed that higher Schizotypy was associated larger GA across the whole movement. However, pairwise comparisons conducted at each Interval of Movement (0% to 100%) between low and high $OLIFE_{Total}$ (median split) did not reveal any significant differences. See Appendix B for more details.

3.5 Discussion

The current study measured visually guided movement under differing task conditions to assess the integrity of ventral and dorsal streams, and the dorsolateral and dorsomedial sub-pathways, in a subclinical sample varying on trait-level Schizotypy. Previous research has established the presence of dorsal-specific impairments in SSD; however, limited research has investigated this deficit using the PAM model or using visually guided movement in sub-clinical SSD. It was expected that Schizotypy level would not impact performance on the ventral task (manual estimation) but would be associated with differences on dorsal tasks (reach-to-point and reach-to-grasp). Specifically, it was hypothesised that higher OLIFE scores would be associated with larger $PHS_{t\%}$, PGA, and $PGA_{t\%}$. Consistent with previous findings, level of Schizotypy did not impact visual size estimation (ventral); however, it was associated with differences in reaching and grasping (dorsal stream) (see Table 3.4 for summary).

The dorsal-specific deficits were observed differentially across temporal epochs of reaching ($PHS_{t\%}$) and the scaling component of grasping (PGA). For the reaching component of prehension, differences due to level of Schizotypy were observed in $PHS_{t\%}$ such that higher Schizotypy was associated with more time spent in the acceleration phase of reaching (larger $PHS_{t\%}$) compared to lower Schizotypy, but only during reach-to-point trials. As previously reviewed, the time of PHS demarcates the proportion of overall movement time devoted to pre-planned movement (acceleration phase) and corrective movement (deceleration phase). Therefore, these results indicate that increased Schizotypy is associated with a stronger reliance on pre-planned movement than lower Schizotypy. Furthermore, this effect was only observed in the reach-to-point condition and not in the reach-to-grasp condition, suggesting that the reach component of prehension is differentially impacted by level of Schizotypy across different movement types. For reach-to-grasp conditions, increased Schizotypy was associated with greater PGA across all conditions. This shows increased Schizotypy is related to overestimations in target scaling during grip formation. Although hypothesised, no differences in $PGA_{t\%}$ were observed as a function of level of Schizotypy suggesting that the relative proportion of time dedicated to pre-programmed versus online feedback control during grasping is not impacted by Schizotypy.

Table 3.4 Summary of hypotheses and results.

Hypothesis	Outcome	Results
H1: Level of Schizotypy will not be associated with changes in Stable Grip Aperture (SGA) during manual size estimation.	Supported	SGA not associated with OLIFE score.
H2: Greater Schizotypy will be associated with greater proportion of overall movement time taken to reach peak hand speed ($PHS_{t\%}$) during reach-to-point and reach-to-grasp movements.	Partially supported	During reach-to-point, increased OLIFE score associated with larger $PHS_{t\%}$. No association during reach-to-grasp.
H3: Greater Schizotypy will also be associated with greater peak grip aperture (PGA) and proportion of overall movement time taken to reach PGA ($PGA_{t\%}$) during reach-to-grasp.	Partially supported	Increased OLIFE score associated with larger PGA (and GA across the entire movement). No association between $PGA_{t\%}$ and OLIFE score.

3.5.1 Experimental Validation

Before interpreting the broader implications of these results, evidence of experimental validation is provided to bolster the subsequent discussion. Experimental validation is important in kinematic studies as they strengthen the interpretation of the experimental effects observed by demonstrating that participants responded to the experimental conditions in ways that are consistent with established kinematic patterns of performance in human prehension. Consistent with previous research, SGA was observed to vary as a function of target size, as did PGA and GA across the duration of the movement (Franz, 2003). On the reach component of prehension, PHS was greater for grasping compared to pointing conditions and time of PHS occurred earlier for perturbed compared to stationary trials, consistent with Jakobson and Goodale (1991). For the grasping component, PGA was observed to be greater for perturbed compared to stationary trials and time of PGA was delayed on perturbation and larger target trials, as seen by Breveglieri and colleagues (2022). These patterns between kinematic measures and experimental conditions provide validation that participants were adequately responding to experimental manipulations and further support the validity of the Schizotypy-related differences reported above.

Additionally, no association with Schizotypy was observed with MT. This is important as some of the kinematic variables of interest are time sensitive (e.g., the time of PHS and time of PGA). If differences in overall MT were present, then any relationships observed with time of PHS and time of PGA may be an artefact of differences in overall MT rather than the kinematic landmarks of interest. This demonstrates that the significant results reported above between Schizotypy and $PHS_{t\%}$ can be attributed to Schizotypy and not to underlying differences in overall MT. Similarly, no differences in PHS as a function of Schizotypy were observed, suggesting that the increased time spent reaching PHS in high Schizotypy is not attributable to those participants reaching a faster PHS. Taken together, this validation evidence strengthens the interpretation of significant effects between kinematic variables and level of Schizotypy as valid.

3.5.2 Implications for Dorsal Stream Function

The goal of the current study was to interpret the findings as reflective of neural function within specific dorsal stream pathways. Based on previous research, differences in the temporal aspects of reaching would implicate the dorsomedial sub-pathway, while differences arising in grip formation would involve the dorsolateral sub-pathway (Grol et al., 2007; Jeannerod, 1988). The results demonstrated partial support for deficits in both dorsal sub-pathways through two significant findings. First, increased Schizotypy was associated with increased $PHS_{t\%}$ (i.e., participants with higher Schizotypy scores spent proportionally more time in the 'acceleration' phase of reaching compared to those with lower Schizotypy). This suggests a stronger reliance on pre-planned movement. However, this was only observed in reach-to-point movements and not during reach-to-grasp. These results suggest that the reach component of prehension is differentially impacted by level of Schizotypy across different movement types. Since the dorsomedial sub-pathway is implicated in subserving the reach component, this evidence suggests that this sub-pathway may respond differently during reach-to-point and reach-to-grasp movements. If this is the case, the dorsomedial sub-pathway may not be as discreetly bound to the reach component as initially proposed. Alternatively, it is also possible that differences in $PHS_{t\%}$ as a function of Schizotypy were not observed during reach-to-grasp movements due to compensation from the dorsolateral sub-pathway. These results demonstrate that reaching alone and reaching to grasp do not constitute a linear adage of 'reach' and 'grasp', but rather an interaction between these components. Furthermore, research has also supported this idea by demonstrating activation from both dorsomedial and dorsolateral regions during reaching and grasping movements (Filimon, 2010).

Second, we also observed increased Schizotypy to be associated with greater PGA across all conditions (i.e., participants with higher Schizotypy scores scaled larger index and thumb openings during reach-to-grasp compared to those with lower Schizotypy). This suggests a tendency to overscale target size. This finding is consistent with King and colleagues (2008), who found Schizophrenia patients to exhibit larger PGAs during grasping compared to normative controls. They concluded this result as evidence of dorsal-specific deficit in Schizophrenia. We extend this interpretation and suggest that since target scaling is a core part of the 'grasp' component of manual prehension, the observed differences implicate the dorsolateral sub-pathway specifically. Overall, the pattern of results supports selective SSD deficit in dorsal stream functioning, relative to intact ventral stream functioning. However, it should be noted that the effects observed in both $PHS_{t\%}$ and PGA in this study were small (η_p^2=.059 and .048, respectively). Comparatively, the PGA effect size observed in King and colleagues' (2008) study was large (η_p^2=.258). Such differences in effect size may be secondary to the fact that the current study examined participants with considerably less severe psychotic disturbances than those tested by King et al. (2008).

3.5.3 Limitations

There are two primary limitations to the current study which should be highlighted. First, while there is evidence to suggest that participants were responding appropriately to the manipulations, the validity of these specific manipulations in their ability to tap dorsal sub-pathways are inferential. Previous research has shown associations between the neural regions of the dorsomedial sub-pathway (e.g., area V6A), and reach-related kinematics with disruptions leading to reaching errors (Battaglini et al., 2002; Karnath & Perenin, 2005). Concurrently, disruptions to dorsomedial areas such as the anterior intraparietal (AIP) have led to significant impairments in hand pre-shaping during grasp (Gallese et al., 1994; Tunik et al., 2005). Although the dissociation of dorsomedial and dorsolateral sub-pathways being responsible for different components of manual prehension has garnered much support, without the use of simultaneous neuroimaging, it is unknown whether the specific tasks used in the current study directly dissociate these visuomotor channels. It is possible that the tasks themselves inadequately activated the relevant sub-pathways of interest resulting, therefore, in attenuated results. Second, comparisons with the only previous study which has specifically investigated dorsal stream deficits in SSD using PAM and visually guided movement (King et al., 2008) are constrained due to differences in both methodology and sampling. Specifically, King and colleagues (2008) employed visual illusions to differentially stimulate dorsal and ventral visual processing streams across two groups: patients with SSD diagnosis and normative controls. The decision to improve the methodological approach in this study from that of King and colleagues (2008) was based on more recent research demonstrating methodological flaws in the use of visual illusions to differentiate dorsal and ventral visual processing streams. Furthermore, the use of normative population varying on level of Schizotypy in the current study was grounded in the empirical and theoretical support for Schizophrenia and related conditions as lying on a continuum rather than discreet categories. While both changes were substantiated by advances in empirical evidence, they sacrifice the ability to directly compare the current results to King and colleagues' (2008) study and draw causal conclusions about the factors that may be leading to these unexpected results. As such, it may have been pertinent to change only one factor; either method or sampling, to allow for clearer comparison.

3.5.4 Future Directions

While there are many possible explanations for the significant reduction in effect size in the current study, three are particularly salient. These include: (1) methodological considerations, (2) sampling considerations, and (3) measurement considerations. First, differences in the methodology used between the current study and that of King and colleagues (2008) may have impacted the relationships observed between level of SSD and kinematic measures. Given the pitfalls of visual illusion paradigms, as discussed in the introduction, it is possible that the observed effects in King and colleagues' (2008) study may be due to their use of visual illusion which may have inadvertently magnified the differences seen between patients and controls (Bruno & Franz, 2009). Second, visually guided movement may only be clearly observable at the severe/high end of SSD (e.g., patients with a Schizophrenia diagnosis). Indeed, previous research has shown deficits to attenuate moving from higher to lower levels on the Schizophrenia spectrum (Cochrane et al., 2012). Given the subtly of the deficits observed in King and colleagues (2008) study which involved patients with Schizophrenia diagnosis, deficits in visually guided movement may not be readily observed in kinematic measures at low to mid-levels along the Schizophrenia spectrum. If this is the case, this brings on questions about whether these deficits are truly core to the condition and thus, are still present in subclinical SSD, but merely undetectable, perhaps due to compensation from other neural networks (da Cruz et al., 2020) or if they are specific to clinical levels of SSD. If the latter, then the potential that these deficits are an artefact of the population, e.g., antipsychotic use, would warrant further inquiry. Third, the construct of Schizotypy and its latent structure are still actively debated. Past research has supported two-, three-, and four-factor models of Schizotypy, with little consensus over which architecture is valid (Fonseca-Pedrero et al., 2018; Fonseca-Pedrero, Ortuño-Sierra, Mason, et al., 2015; Lin et al., 2013; Sierro et al., 2016). Lack of clarity regarding the latent structure of Schizotypy is reflected in the numerous psychometric measures purporting to measure this construct. For example, the Chapman Psychosis Proneness Scales (CPPS; Chapman et al., 1976) reflect a two-factor model of Schizotypy, the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) yields a three-factor model, whilst the OLIFE manifests a four-factor structure. Moreover, researchers have questioned the validity of factor structure and content validity of the OLIFE; specifically, the construct validity of the Impulsive Nonconformity and Cognitive Disorganisation factors (Kwapil, Gross, Silvia, et al., 2018; Lin et al., 2013; Pickering, 2004). The implications of poor construct validity of the OLIFE in this study may have significant impacts on the ultimate patterns and relationships observed between kinematic variables and level of Schizotypy.

3.6 Conclusion

The current study used a relatively novel approach to investigate dorsal-specific deficits in SSD using PAM and visually guided movement and to test the differential functional integrity of two dorsal sub-pathways. It is also the first study to test these deficits in subclinical SSD measured as trait-level Schizotypy. The results showed small effects for increased Schizotypy to be associated with impairments in both the reaching and grasping components of manual prehension. While results lend further support for dorsal-specific deficits in SSD, conclusions around the implications for the dorsomedial and dorsolateral sub-pathways remain inconclusive. From the three possible explanations offered above, it is the problematic construct validity if the OLIFE that seems most pressing. Therefore, we propose that an investigation into the architecture of the OLIFE is necessary, with any new structures to be used to re-analyse the kinematic data from the current experiment as this would provide an opportunity to reassess the relationship between Schizotypy and kinematic measures with improved psychometric clarity.

CHAPTER 4

Study 2: A Comprehensive Psychometric Investigation of the OLIFE Using an Australian Sample

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Over the last several decades, much research has focussed on developing self-report scales for measuring Schizotypy, a multi-dimensional personality trait that varies across normative and pathological populations. These scales have varied in their coverage and item content. Although some (e.g., Schizotypal Personality Questionnaire; SPQ; Raine, 1991) were created to reflect the DSM diagnostic criteria for Schizotypal Personality Disorder, others have focussed on specific dimensions of Schizotypy (e.g., the Chapman and Chapman Scale of Perceptual Aberration; Chapman et al., 1980). These scales differ considerable in their theoretical approach and psychometric properties, creating significant heterogeneity in the measurement and understanding of Schizotypy. To address this, a comprehensive measure with a defined latent structure of Schizotypy, the Oxford Liverpool Inventory of Feelings and Experiences (OLIFE; Mason et al., 1995), was created and has become one of the most widely used Schizotypy questionnaires. Unlike its diagnostically informed counterparts, the OLIFE conceptualises Schizotypy traits as distributed across the whole population, with higher scores indicating greater psychosis proneness and increased likelihood of more serious psychopathology. The OLIFE was initially distilled from the Combined Schizotypal Traits Questionnaire (CSTQ; Bentall et al., 1989), a 420-item scale amalgamated from a broad array of smaller scales and measuring approximately fifteen psychotic and related personality traits (all CSTQ scales of origin are listed in Table 4.1).

Table 4.1 Scales used to form the Combined Schizotypal Traits Questionnaire (CSTQ; Bentall et al., 1989)

Questionnaire	Reference
Eysenck Personality Questionnaire	(Eysenck & Eysenck, 1975)
Extraversion (E)	
Neuroticism (N)	
Psychoticism (P)	
Lie (L)	
Schizotypal Personality Scale (STA)	(Claridge & Broks, 1984)
Borderline Personality Scale (STB)	
Chapman & Chapman's scales	
Physical (PhA) and Social (SoA) Anhedonia	(Chapman et al., 1976)
Perceptual aberration (PAb)	(Chapman et al., 1980)
Magical ideation (MgI)	(Eckblad & Chapman, 1983)
Hypomanic personality (HoP)	(Eckblad & Chapman, 1986)
Launay and Slade's Hallucination scales (LSHS)	(Launay & Slade, 1981)
Nielsen and Petersen's Schizophrenism scale (NP)	(Nielsen & Petersen, 1976)
Seven item Schizoidia scale from the MMPI	(Golden & Meehl, 1979)
Delusions Symptoms States Inventory (DSSI)	(Foulds & Bedford, 1975)
Delusions of Contrition (dC)	
Delusions of Persecution (dP)	
Delusions of Grandeur (dG)	
Delusions of Disintegration (dD)	

Classical Test Theory approaches were applied to the 420 items of the CSTQ, which resulted in the OLIFE, a 104 true/false item, four-factor model including (1) Unusual Experiences, (2) Cognitive Disorganisation, (3) Introvertive Anhedonia, and (4) Impulsive Nonconformity factors (Mason et al., 1995). The Unusual Experiences (UnuExp) factor focuses on hallucinations, perceptual disturbances, and magical thinking. These items largely reflect the 'positive' symptoms of psychosis. The Cognitive Disorganisation (CogDis) factor enquires about difficulties in concentration, attention, and decision-making, as well as elements of anxiety and purposelessness. The Introvertive Anhedonia (IntAnh) factor reflects the 'negative' symptoms of psychosis, including lack of enjoyment and dislike of emotional and/or physical intimacy with a preference for independence and solitude. The final factor, Impulsive Nonconformity (ImpNon), canvasses violent, self-abusive, and reckless behaviours.

Since its conception, the OLIFE has also garnered several criticisms. The most notable concerns the validity of the ImpNon factor. From a theoretical perspective, the

ImpNon factor does not appear in traditional or contemporary models of Schizotypy. Early theoretical conceptualisations of the multidimensional nature of Schizotypy, such as that put forward by Meehl (1973), specified three features of Schizotypy: cognitive slippage, anhedonia, and magical ideation and does not include features of violent, self-abusive, or reckless behaviours. Similarly, recent investigations of the multidimensionality of Schizotypy have also supported the notion of a positive, negative, and disorganised structure, with no references to impulsive behaviours (Bentall et al., 1989; Raine et al., 1994; Vollema & van den Bosch, 1995). Moreover, the ImpNon items appear to reflect anti-social and sensation-seeking traits, similar to those described in personality disorders, such as Borderline and Antisocial Personality Disorder (Pickering, 2004). Arguably, these differences are consistent with the factor's poor ability to predict subsequent development of Schizophrenia or discriminate groups along the Schizophrenia spectrum. For example, high ImpNon were absent among relatives of persons with Schizophrenia (Claridge et al., 1983), a group known to produce elevated scores on measures of Schizotypy. Furthermore, high ImpNon scores in the normative population do not significantly predict increased risk for the future development of SSDs (Chapman et al., 1994). When comparing nonclinical controls to persons diagnosed with Schizophrenia, ImpNon scores could not distinguish between groups despite patients scoring significantly higher across UnuExp, CogDis, and IntAnh factors (Cochrane et al., 2010). Similarly, factor analytic studies of the OLIFE have revealed that three-factor models (i.e., without the ImpNon factor) fit the data just as well as four-factor models (Fonseca-Pedrero, Ortuño-Sierra, Mason, et al., 2015; Fonseca-Pedrero, Ortuño-Sierra, Sierro, et al., 2015; A. Lin et al., 2013; Sierro et al., 2016). Poor predictive, discriminative, and factorial validity collectively undermine the psychometric appropriateness of ImpNon and the OLIFE architecture more broadly.

To a lesser extent, concerns about the content validity of items in the CogDis factor have also been raised. In general, psychometric measurement studies of Schizotypy have had trouble in assessing cognitive disorganisation in Schizotypy for two main reasons. First, these symptoms may be due to related conditions (e.g., anxiety and depression), which can cloud the measurement of disordered thinking specific to Schizotypy. Second, the experience of disorganised thinking itself may impair a respondent's ability to identify and report such experiences accurately. Additionally, some researchers have suggested that the CogDis items in the OLIFE reflect concepts closer to social anxiety and low self-esteem (Kwapil, Gross, Silvia, et al., 2018). Other criticisms of the OLIFE include limited psychometric validation with modern statistical analyses such as IRT (Kwapil, Gross, Silvia, et al., 2018; Kwapil & Barrantes-Vidal, 2015), lack of a total score (Mason et al., 2005), and participant burden due to the long questionnaire length (Gross et al., 2018).

4.1 The Present Study

The current study seeks to address these shortcomings by investigating the construct validity and factor structure of the OLIFE in an Australian sample and, in the process, refine the scale using psychometric techniques to create a short form based on robust statistic- and content-driven methods. From this overarching aim are three specific objectives. First, to re-examine the latent structure of the OLIFE using Exploratory Factor Analysis (EFA) then Confirmatory Factor Analysis (CFA), bifactor modelling, and hierarchical factor modelling. Second, to follow rigorous methodological guidelines for item refinement by using content review, Classical Test Theory (CTT), then Item Response Theory (IRT) approaches (Coste et al., 1997; Goetz et al., 2013). Finally, validate the improved scale by testing model fit, reliability, and concurrent validity in a new sample. Based on previous studies on the OLIFE and the dimensional nature of Schizotypy, it is hypothesised that a single-level, three-factor model would produce the strongest fit, despite other models demonstrating adequate fit. The new OLIFE scale is also hypothesised to demonstrate strong convergent validity with existing measures of Schizotypal traits and divergent validity with constructs unrelated to Schizotypy (e.g., extraversion and agreeableness).

4.2 Method

4.2.1 Participants

Data were collected through three recruitment rounds as part of a broader research program into Schizotypy from 2018 to 2022. This resulted in 1,085 complete responses from 826 online and 259 face-to-face participants. Online participants were sourced from across Australia (see Figure 4.1). Face-to-face participants primarily consisted of undergraduate psychology students at the Australian National University. Inclusion criteria involved current residency in Australia, age between 18 to 60 years at the time of participation and reported (online) or revealed via testing (in-person) normal or corrected-to-normal vision (due to the visual nature of some associated experimental tasks). Following data screening and cleaning, the final sample consisted of 985 participants (604 females, 357 males, 24 others) with a mean age of 30.2 years (*SD* = 10.9); see Table 4.2 for a full summary of sample characteristics. The ANU Human Research Ethics Committee approved all aspects of the research.

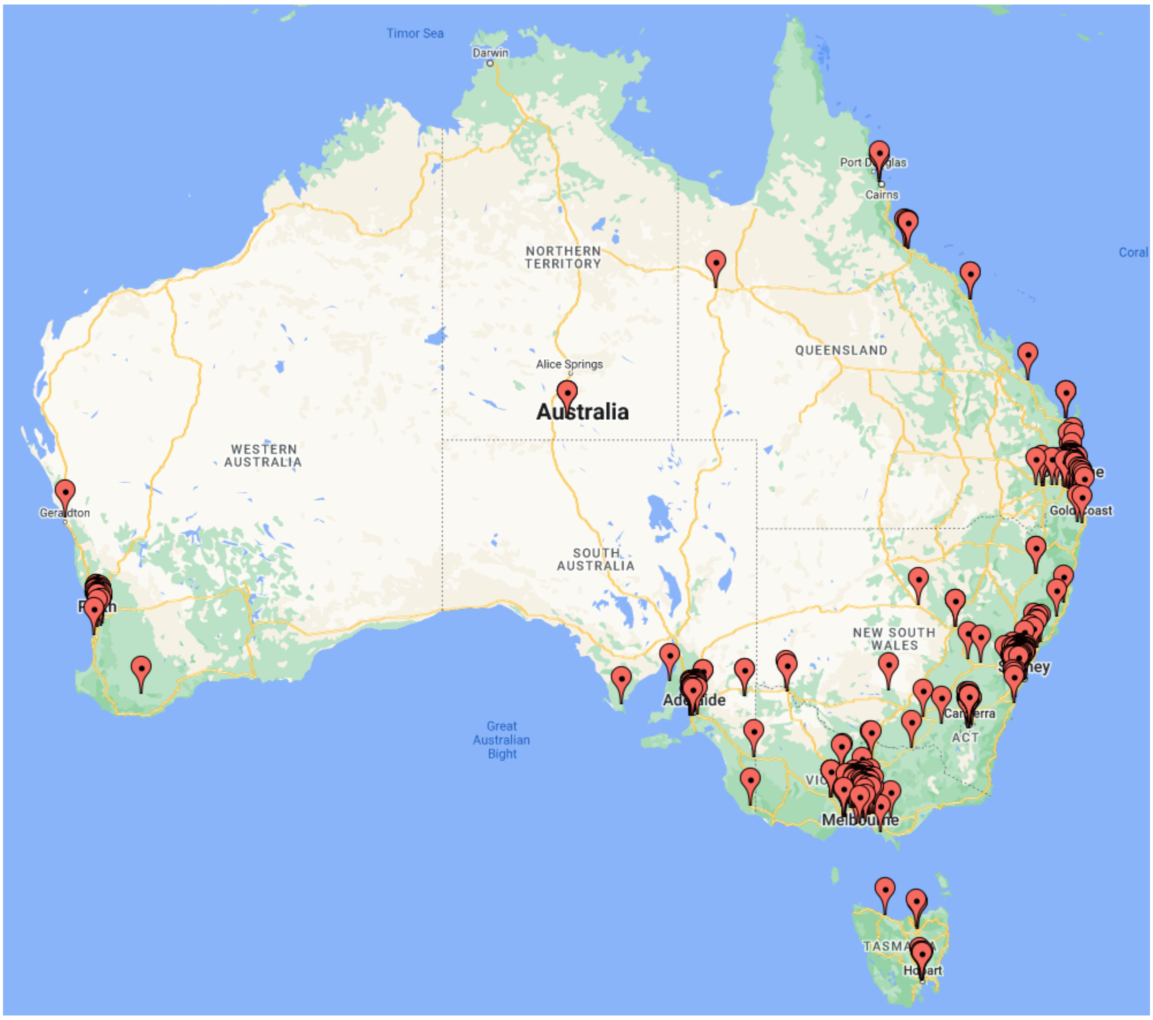


Figure 4.1 Participant location across Australia, collected through postcode information.

4.2.2 Procedure

An online survey was created in Qualtrics™ and distributed via Prolific™. Refer to Appendix C for a copy of the questionnaire. As the aim of the study was to validate and refine the OLIFE for measurement of Schizotypy in the normative population, measures were taken to collect a sample that was relatively representative of the Australian population, i.e., census-matched. As such, recruitment for the online sample was staggered to allow for small adjustments to regulate the demographics of the final online sample. A total of 11 blocks of recruitment were completed, each ranging from 5 to 100 responses per block. Primarily age and gender were adjusted between blocks as there was significant skew toward younger (18-35 years) and female-identifying participants. The survey was estimated to take 30 minutes to complete and included the following questionnaires:

A *Demographic and Health questionnaire* solicited demographic and health variables including age, gender identity, postcode, religiosity (as measured by the Centrality of Religiosity 5 item scale; CRS-5, Huber & Huber, 2012), quality and quantity of adverse life events, education, occupation, sleep quality, occupation and work hours, English as first language, and a brief vocabulary test for English proficiency. Health-related information included a self-reported history of mental, psychiatric, neurological, perception, learning, and school-related behavioural problems, as well as first-degree relatives with a mental health disorder.

The *Oxford Liverpool Inventory of Feelings and Experiences (OLIFE; Mason et al., 1995)* measured Schizotypy across 104 true/false items constituting four factors: Unusual Experiences (UnuExp; 30 items), Introvertive Anhedonia (IntAnh; 27 items), Cognitive Disorganisation (CogDis; 24 items), and Impulsive Nonconformity (ImpNon; 21 items).

The *Hagen Matrices Test Short form (HMT-S; Heydasch et al., 2013)* estimated IQ through six matrices items. In this test participants were shown incomplete, visually abstract matrices and asked to choose the correct piece to complete the matrix from eight options. Participants were given a maximum of two minutes per item and presented with two untimed sample items with instructions and feedback prior to the six test items.

Personality Assessment Inventory Short Form (PAI-SF; Morey, 2014) subscales measuring Anxiety (ANX), Depression (DEP), Paranoia (PAR), Schizophrenia (SCZ), Alcohol Abuse (ALC), and Drug Abuse (DRG) were used to gauge general psychopathology. Negative Impression Management (NIM) and Positive Impression Management (PIM) scales were also included to assess response distortion. Participants were excluded during data screening and cleaning if their scores on these scales exceeded recommended cut-offs (NIM T-score > 92 and/or PIM T-score > 68; Morey, 1991; Sellbom & Bagby, 2008). A total of 67 PAI-SF items were used, and participants responded on a four-point Likert from *false* (0) to *very true* (3).

The *Community Assessment for Psychic Experiences (CAPE-15; Capra et al., 2017)* measured the frequency of positive psychotic-like experiences through 15 items on three subscales: Persecutory Ideation (PI; 5 items), Bizarre Experiences (BE; 7 items), and Perceptual Abnormalities (PA; 3 items). Items were answered on a 4-point Likert scale from *never* (1) to *nearly always* (4).

The *Personality Inventory for DSM-5 Faceted Brief Form Psychoticism Scale (PID-5-FBF Psychoticism; American Psychiatric Association, 2013)* measured trait psychoticism through 12 items across three factors: Unusual Beliefs and Experiences (UnBE; 4 items), Eccentricity (Ecc; 4 items), and Perceptual Dysregulation (PeDy; 4 items). Items were answered on a four-point Likert scale from *false* (1) to *often true* (4).

The *Mini International Personality Item Pool (Mini-IPIP; Donnellan et al., 2006)* measured the Big 5 personality traits: Neuroticism (NE), Extraversion (EX), Openness (OP), Agreeableness (AG), and Conscientiousness (CO) using 20 items answered on a 5-point Likert scale from *strongly disagree* (0) to *strongly agree* (4).

All scales listed above possessed acceptable levels of internal consistency (see Table 4.3 for summary). *Embedded response validation checks* were added to ensure data quality and were measured in four ways. First, participants with response times greater than two standard deviations below the median response time were removed due to insufficient time to ensure adequate attention and processing. Second, an embedded inconsistency scale within the OLIFE was created by negatively wording four items from the first half and placing them in the second half of the OLIFE. This was used to assess inconsistency in responding within this scale. Scores ranged from -4 to 4 with a score of 0 meaning no inconsistency and larger deviations meaning greater inconsistency. Third, four attention checks were randomly allocated within the second half of the questionnaire. These consisted of items prescribing a discrete response (e.g., 'Please select False for this question.'). Scores ranged from -4 to 4 with a score of 0 indicating no errors in responding. OLIFE inconsistency and attention validation checks were assessed together. Note that the combined scored had a range of -8 to 8 with a score of 0 meaning no inconsistency or attention errors. Participants with a total inconsistency and attention score outside of -4 to +4 were removed, as this suggested failure of at least 50% of the consistency and attention checks. Finally, the last item on the questionnaire asked participants if they believed their data was valid and useable. It was made clear to participants that, regardless of their response to this item, they would still be compensated for their time and effort as outlined in the information sheet. Any participants who reported their data to be invalid or unusable were removed from subsequent analysis. The use of these validation checks was guided by Meade and Craig (2012).

Data from face-to-face participants only included responses from a subset of the questionnaires listed above. Namely, the demographic and health questionnaire, OLIFE, and the full PAI-SF. An IQ estimate for these participants was measured using a two-subtest dyad of the *Weschler Adult Intelligence Scale Fourth Edition (WAIS-IV; Wechsler, 2008)*. Specifically, Matrix Reasoning and Coding subtests were used as these have been identified to provide a strong estimate of overall Full-Scale IQ (FSIQ; Girard et al., 2015).

4.2.3 Data Analytic Plan

Screening and cleaning of the 1,085 complete responses involved removing participants based on (1) failure to meet attention or consistency validation checks, (2) duplication of participants, and (3) evidence of response distortion as measured by PIM and NIM scales. The data were then randomly split into Exploratory (Sample 1, n = 437) and Confirmatory (Sample 2, n = 548) halves. Data analysis was carried out in two phases: development and confirmation. The development phase included exploration of factor structure and item refinement using the exploratory sample (Sample 1; *n*=437). The confirmation phase included validation of the final model from the development phase in the confirmatory sample (Sample 2; *n*=548). The steps used in each of these phases were informed by methodological guidelines for item reduction and scale shortening put forth by Coste and colleagues (1997) and updated by Goetz and colleagues (2013). All analyses were completed through JASP (v0.16.3; Team JASP, 2022) and R (v4.2.1; R Core Team, 2022) via RStudio (v4.1.2; RStudio Team, 2020). Descriptive statistics were compared across Samples 1 and 2 to ensure there were no significant differences between them (see Table 4.2 for a summary).

Phase 1: Development

A flowchart summary of the development phase can be found in Appendix C. This phase consisted of four parts: (1) assessment of dimensionality, (2) expert item content review, (3) statistical item refinement, and (4) assessment of latent structure. Assessment of dimensional architecture comprised the following protocol: CFA of the current OLIFE structure followed by EFAs with varying factor structures. In Model 1, we tested the original factor structure of the OLIFE. In Model 2, based on the extant research literature, we tested a CFA with a three-factor structure of the OLIFE, which removed items constituting the ImpNon scale. In Model 3a. we used EFA to identify the best fitting model by maximising common variance using a parallel analysis and scree plot approach. Models 3b. to 3f. use EFA to systematically specific factor structures from one to five factors. Model 4 estimated the potential for method effects, by using bifactor modelling to estimate shared variance among items worded in the same direction (i.e., protrait and contrait items). Models were estimated using Diagonally Weighted Least Squares (DWLS) to account for the dichotomous indicators (Muthén et al., 1997), and model fit was estimated based on the following indices: non-significant chi-square ($\chi 2$), Comparative Fit Index (CFI) and Tucker-Lewis Index (TFI) > .950, Root Mean Square Error of Approximation (RMSEA) (including 90% confidence interval) and Standardised Root Mean Square Residual (SRMR) greater than .08 and .06, respectively (Hu & Bentler, 1999).

Following outcomes from these models, item content for each factor was reviewed. As recommended by Coste and colleagues (1997), when the original composite measurement scale, in this case the OLIFE, is not considered to be the gold-standard in its construct of measurement, expert-based approaches to item refinement are recommended, followed by statistical considerations. Reliance on statistical measures, such as factor loadings, communalities, and Cronbach's alpha alone, when the parent scale is not considered the gold standard, results in retention of items that are highly correlated and, thereby, threaten adequate content coverage in the new scale (i.e., content validity; Coste et al., 1995; Stevens, 1946). Based on these considerations, the item content of the four factors of the OLIFE were assessed by one expert in the field of psychosis and SSD research. Only items which were deemed to adequately reflect the underlying dimension of Schizotypy were retained.

Further item refinement was achieved by examining factor loadings from a four-factor CFA followed by submitting the retained items into a four-factor EFA to assess communalities. A factor loading cut-off of 0.32 (Tabachnick et al., 2019) and a communality cut-off of 0.20 (Child, 2006; Yong & Pearce, 2013) was used to further refine items. A four-factor CFA was completed on the retained items (i.e., those that survived the evaluation of factor loadings and communalities), which confirmed a robust model fit. These items were then submitted to a unidimensional IRT model for each factor. For comprehensiveness, all removed items were also entered into unidimensional IRTs with their respective factor to assess whether any removed items provided unique breadth to the dimension beyond that of retained items. Items were refined based on item information, item content, and test information functions.

The items retained following the above processes were then assessed for their latent structure. Three competing structures were compared: (1) a single-level factor structure, (2) a hierarchical model with higher-order general factor, and (3) a bifactor model.

Phase 2: Confirmation

The final model from the development phase was tested in the confirmatory sample (n=548). Model fit was assessed using a CFA of the structure specified from the development phase. Internal consistency was measured using McDonald's omega (ω). Spearman's rho (ρ) correlations were used to assess convergent and discriminant validity between the new model and the original OLIFE, CAPE15, PID Psychoticism, Mini IPIP, and PAI-SF subscales.

4.3 Results

4.3.1 Data Screening and Demographic and Clinical Characteristics of the Samples

From the 1,085 complete responses, 100 responses were removed during screening and cleaning: 13 responses due to failure to meet attention validation checks, 43 duplicate responses, and 44 responses with evidence of response distortion (7 due to high PIM; 37 due to high NIM). The final sample used for analysis consisted of 985 participants. Table 4.2 and Table 4.3 convey the demographic and clinical characteristics of Sample 1, respectively.

Table 4.2 Summary of demographics for overall sample and exploratory and confirmatory split.

Demographic	Overall Sample	Sample 1 Exploratory Sample	Sample 2 Confirmatory Sample
Size	985	437	548
Age	30.2 (10.9)	30.41 (10.92)	29.99 (10.87)
Gender (%)			
Female	61.3	62.3	60.6
Male	36.3	35.2	37.0
Trans*	0.7	0.9	0.5
Non-binary	1.5	1.6	1.5
Did not disclose	0.2	0.0	0.4
Ethnicity (%)			
Caucasian/White	61.9	62.2	61.5
Asian	32.5	31.3	33.4
African	0.5	0.5	0.5
Aboriginal or Torres Strait Islander	0.4	0.5	0.4
Pacific Islander	0.4	0.9	0.0
Hispanic or Latino	1.2	1.6	0.9
Other (text response)	3.1	3.0	3.3
State (%)			
ACT	27.7	29.3	26.5
NSW	21.0	23.8	18.8
VIC	21.0	19.2	22.6
QLD	13.7	12.8	14.4
SA	5.3	4.1	6.2
WA	6.0	6.9	5.3
NT	0.1	0.0	0.2
TAS	1.9	1.1	2.4
Did not disclose	3.3	2.8	3.6
Urbanicity (%)			
Urban/Major Regional Area	84.7	84.0	85.2
Regional/Remote Area	12.1	13.3	11.1
Did not disclose	3.2	2.7	3.7
Education (%)			
< Year 10 cert	0.3	0.2	0.4
Year 10 cert	1.2	1.1	1.3
Year 12 cert	34.1	33.9	34.3
Apprenticeship	11.5	12.1	10.9
Bachelor	35.0	34.8	35.2
Masters	14.0	13.3	14.6
Doctoral	3.9	4.6	3.3
Intelligence			
DQ (FSIQ estimate)	109 (12.6)	110 (12.90)	109 (12.32)
HMT-S score (max score 6)	3.7 (1.6)	3.6 (1.6)	3.7 (1.6)

DQ=deviation quotient; FSIQ=Full Scale Intelligence Quotient; HMT-S=Hagen Matrices Test Short form.

Table 4.3. Summary of descriptives for Sample 1 (exploratory sample).

Measure	*n*	Mean	*SD*	Scale Range	Internal Consistency
OLIFE					(O. Mason et al., 1995)
Unusual Experiences	437	10.79	7.09	0-29	α=0.89
Cognitive Disorganisation	437	13.68	6.42	0-24	α=0.87
Introvertive Anhedonia	437	9.64	5.75	0-25	α=0.82
Impulsive Nonconformity	437	7.10	3.52	0-19	α=0.77
CAPE-15	318	5.92	5.06	0-32	α=0.84
Persecutory Ideation	318	3.73	2.63	0-15	(Capra et al., 2017)
Bizarre Experiences	318	1.62	2.38	0-15	
Perceptual Abnormalities	318	0.57	1.12	0-7	
PID-5-FBF Psychoticism	318	0.62	0.52	0.00-2.83	α=0.78
Eccentricity	318	1.13	0.88	0.00-3.00	(Anderson et al., 2018)
Unusual Beliefs	318	0.46	0.60	0.00-3.00	
Perceptual Dysregulation	318	0.26	0.48	0.00-3.67	
Mini IPIP					(Cooper et al., 2010)
Extraversion	318	2.52	1.01	1.00-5.00	α=0.81
Agreeableness	318	3.76	0.81	1.00-5.00	α=0.70
Conscientiousness	318	3.37	0.87	1.00-5.00	α=0.68
Neuroticism	318	3.08	0.94	1.00-5.00	α=0.72
Openness to Experience	318	3.85	0.80	1.75-5.00	α=0.70
PAI Subscales					(Sinclair et al., 2009)
Anxiety	437	60.97	15.13	39-105	α=0.91
Depression	437	63.12	16.02	38-109	α=0.90
Schizophrenia	437	57.66	13.68	38-115	α=0.82
Paranoia	437	55.93	10.62	33-92	α=0.82
Alcohol Abuse	437	50.80	10.44	41-106	α=0.89
Drug Abuse	437	51.95	10.89	42-102	α=0.85
Positive Impression Management	437	46.32	10.33	15-67	
Negative Impression Management	437	54.61	10.96	46-85	

OLIFE = Oxford Liverpool Inventory of Feelings and Experiences, CAPE-15 = Community Assessment of Psychic Experiences 15 item, PID-5-FBF Psychoticism = Personality Inventory for DSM-5 Faceted Brief, Mini IPIP = Mini International Personality Item Pool, PAI = Personality Assessment Inventory, %tile = percentile. a = Cronbach's alpha.

4.3.2 Development Phase

A flowchart detailing all steps of the development phase can be found in Appendix C.

Assessment of Dimensionality

CFA of the OLIFE (Model 1) revealed mediocre fit, with clear room for improvement. The 3-Factor solution (Model 2), with ImpNon items removed, showed minor improvement on the original model fit. These results supported the need for further investigation of the latent factor structure. Results from models tested in the initial dimensionality assessment are summarised in Table 4.4.

Table 4.4. Summary of models tested in assessment of dimensionality.

Model	χ^2	df	CFI	TLI	RMSEA (CI_{90})	SRMR
1. CFA OLIFE 4 Factor	11851.74	5144	.869	.867	.055 (.054-.056)	.072
2. CFA OLIFE 3 Factor	7432.76	3077	.890	.887	.057 (.056-.059)	.073
3a. EFA unspecified factors	5950.84	4649	--	.858	.025 (.023-.027)	--
3b. EFA 5 Factor	6591.39	4846	--	.818	.029 (.027-.030)	--
3c. EFA 4 Factor	7019.86	4946	--	.789	.031 (.029-.033)	--
3d. EFA 3 Factor	7969.95	5047	--	.709	.036 (.035-.038)	--
3e. EFA 2 Factor	9456.92	5149	--	.574	.044 (.042-.045)	--
3f. EFA 1 Factor	10916.25	5252	--	.453	.050 (.048-.051)	--
4. Bifactor (OLIFE + Methods)	8522.11	5124	.934	.932	.039 (.038-.041)	.061

All CFA models used the DWLS estimator and factor variance was used to scale factors. All EFA models used DWLS estimator with a Promax rotation. $\chi 2$ = chi-square, CFI = Comparative Fit Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Square Error of Approximation, CI_{90} = 90% confidence interval, SRMR = Standardised Root Mean Square Residual.

EFAs (Models 3a. to 3f.), with varying numbers of specified factors, revealed the presence of a methods factor that consisted of only negatively worded items from both the IntAnh and ImpNon scales. This factor was reliable such that it appeared in all EFA models, except the unidimensional model. To test whether negatively worded item variance was better explained by a methods factor or their respective substantive factor, a bifactor model (Model 4), which specified the four original OLIFE factors and a general methods factor, was conducted. Negatively worded ImpNon item factor loadings ranged from -0.019 to .240, while those for the methods factor ranged between .263 to .765 (refer to Table 4.5 for a summary). Negatively worded IntAnh item factor loadings ranged from -0.138 to .397, while those for the methods factor were from -0.007 to .946. On closer inspection, all negatively worded ImpNon items loaded with greater magnitude onto the methods factor than the ImpNon factor, except item 41, which cross-loaded at 0.263 (Methods) and 0.240 (ImpNon). Nine of the 15 negatively worded IntAnh items loaded better on the methods factor than the IntAnh factor. A further four items were cross-loaded, and only two items (39 and 42) loaded better onto the IntAnh factor. Based on these findings, all items with stronger methods factor and cross-loading items (19 in total) were removed; all positively worded items and IntAnh negatively worded items 39 and 42 were retained for expert item content review.

Table 4.5. Reverse coded item factor loadings on the Methods and substantive factors from Model 4.

			Std. Factor Loading	
Scale	**Item**	**Question**	**Methods**	**Substantive**
Impulsive Nonconformity	5	Are you usually in an average sort of mood, not too high and not too low?	**0.602**	-0.019
	16	Do you stop to think things over before doing anything?	**0.672**	0.140
	22	Would being in debt worry you?	**0.765**	0.139
	41	Would it make you nervous to play the clown in front of other people?	**<u>0.263</u>**	**<u>0.240</u>**
	82	When in a group of people do you usually prefer to let someone else be the centre of attention?	**0.621**	0.203
	91	Do you consider yourself to be pretty much an average kind of person?	**0.449**	0.001
Introvertive Anhedonia	1	Do you find the bright lights of a city exciting to look at?	**0.730**	-0.138
	14	Can just being with friends make you feel really good?	**0.865**	0.048
	26	Can you usually let yourself go and enjoy yourself at a lively party?	**<u>0.305</u>**	**<u>0.367</u>**
	39	Do you like going out a lot?	-0.007	**0.363**
	42	Do you have many friends?	0.209	**0.397**
	45	Are you rather lively?	**<u>0.270</u>**	**<u>0.273</u>**
	49	Do you like mixing with people?	**<u>0.414</u>**	**<u>0.461</u>**
	59	Do you enjoy many different kinds of play and recreation?	**0.611**	0.275
	64	Does it often feel good to massage your muscles when they are tired or sore?	**0.946**	-0.038
	69	On seeing a soft, thick carpet have you sometimes had the impulse to take off your shoes and walk barefoot on it?	**0.638**	-0.188
	77	Is it fun to sing with other people?	**0.551**	0.253
	88	Do you love having your back massaged?	**0.802**	0.007
	93	Do you feel very close to your friends?	**<u>0.425</u>**	**<u>0.349</u>**
	98	Is trying new foods something you have always enjoyed?	**0.521**	0.317
	101	When are things bothering you do you like to talk to other people about it?	**0.443**	0.220

The stronger of the two factor loadings for each item are bolded. Underline indicates cross-loaded items.

Expert Item Content Review

During expert item content review, items from each factor were assessed on whether they adequately reflected their assigned dimension of Schizotypy. Coste and colleagues (1997) strongly recommend expert item content review during item refinement to preserve content validity. Content review was completed on all items by one expert in the field of Schizotypy and Schizophrenia. All items loading on the UnuExp and IntAnh factors were judged to reflect their respective dimensions. However, only a subset of items (10 in total) loading on the CogDis factor were judged to accurately depict the construct (e.g., *Are you easily distracted when you read or talk to someone?*). The remaining CogDis items appeared to describe experiences of anxiety (e.g., *Do you worry about things you should not have done or said?*) or depression (e.g., *Do you often feel that there is no purpose to life?*). To test this hypothesis, a 3-factor CFA was conducted which split the 24 CogDis items into cognitive disorganisation (CogDis10; 10 items), anxiety-related (CogDisAnx 8 items), and depression-related (CogDisDep 6 items) items. This model strongly fit the data (CFI=.995, TLI=.995, RMSEA=.025, 90%CI=.015-.033) and manifest a significantly better fit when compared to the original unidimensional structure ($\Delta\chi^2$=61.35, Δdf=3, p<.001).

To further support the validity of this architecture, a hierarchical regression was conducted where the sum of Schizophrenia Thought Disorder subscale items from the PAI-SF (PAI-SCZ-T) were regressed onto CogDis factor scores (i.e., items from each factor summed with equal weightings) derived from the three manifest factors noted above. Predictor variables were entered in two blocks: the CogDisAnx and CogDisDep factor scores in the first block and CogDis10 factor score in the second block. The omnibus model was significant, $F(2,434) = 56.48$, p<.001, and the predictors accounted for roughly 20% of the variance in PAI-SCZ-T scores (adj.R^2=.203). Although both CogDisAnx and CogDisDep factor scores were significantly associated with PAI-SCZ-T (β=.146, t= 2.730, p= .007, and β=.352, t=6.595, p<.001, respectively), CogDis10 predicted reliable variance over and above these variables, $\Delta F(1,433) = 42.01$, p< .001, ΔR^2=.07. These results suggest that CogDis10 is a separable factor that has a stronger association with a concurrent measure of cognitive disorganisation than the other two factors. Consequently, only the CogDis10 items were retained as representing the CogDis factor in further analyses.

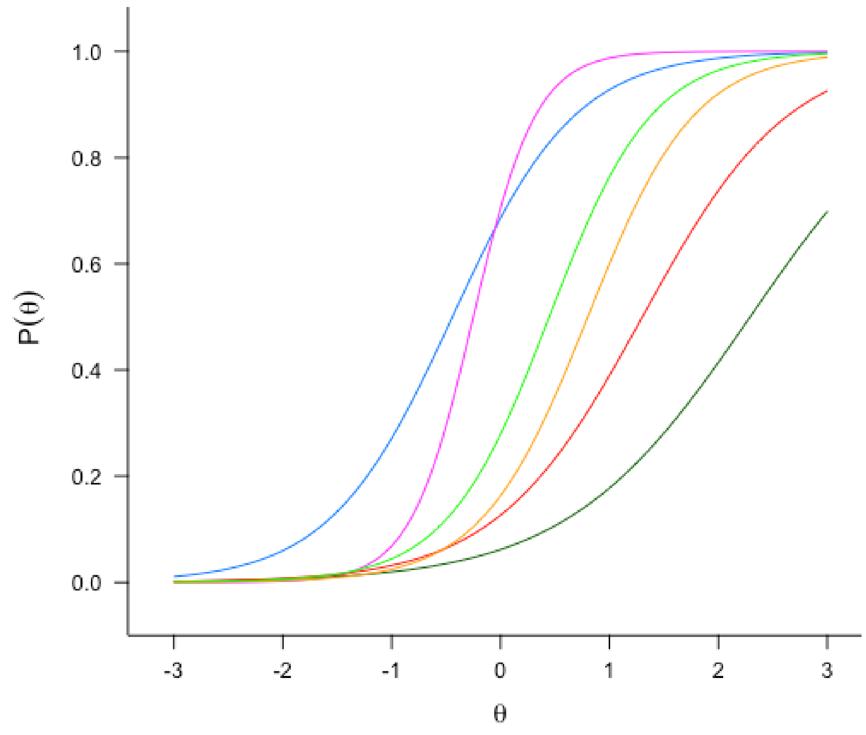
Similarly, the ImpNon factor items were judged to reflect constructs other than Schizotypy. Although Mason and colleagues (1995) argue that the ImpNon factor provides a broader view of psychosis and SSD, we posit, based on the five arguments below, that the ImpNon factor measures symptoms beyond those that are theoretically and empirically established as indicators of SSD. First, the source of several items constituting the ImpNon factor were generated in relation to other constructs. For example, 13 of the 23 ImpNon items were borrowed from the Borderline Personality scale (STB, Claridge & Broks, 1984) and the Eysenck's Personality Questionnaire (EPQ; Eysenck & Eysenck, 1975) Lie Scale, leaving only ten items from the EPQ Psychoticism scale (Eysenck & Eysenck, 1975) and Chapman Hypomanic scale (Eckblad & Chapman, 1986). The content of these items is aligned with their scales of origin and generally reflect aggressive or impulsive behaviours (e.g., *Do you often have an urge to hit someone?* or *Do you ever have the urge to break or smash things?*) and/or self-abusive behaviour or ideation (*Have you ever felt the urge to injure yourself?*). Although there are historical linkages between borderline, neurotic, and psychotic symptoms, these relationships have since been rebuked, with new models delineating borderline traits and the Schizophrenia spectrum as separate constructs (see Barnow et al., 2010 for a review). Second, previous studies assessing the factorial structure of the OLIFE and its short form (including factor analysis and exploratory structural equation modelling), have demonstrated that both three- (without ImpNon) and four-factor models adequately fit the data (Cella et al., 2013; Sierro et al., 2016). These results have been shown in normative (Fonseca-Pedrero, Ortuño-Sierra, Sierro, et al., 2015) and at-risk-for-psychosis populations (Lin et al., 2013). Third, one of the fundamental tenets of Claridge's fully dimensional model of Schizotypy, upon which the OLIFE is based, considers Schizotypy to be a personality dimension continuously distributed in the population that is necessary, but not sufficient, for the development of psychotic illness (Rawlings et al., 2008a). Therefore, measurement of Schizotypy should, in theory, be able to differentiate those at increased risk of developing a psychotic disorder over those who are not (i.e., predictive validity). However, ImpNon was measured at low levels in relatives of Schizophrenia patients and did not significantly predict increased risk of psychotic disorders in community samples (Chapman et al., 1994). Furthermore, according to the fully dimensional model, ImpNon should also demonstrate differentiate between patients with SSD and healthy controls. However, when comparing healthy controls with Schizophrenia patients, ImpNon scores could not statistically distinguish between these groups, in contrast to the excellent discriminating power exhibited by UnuExp, CogDis, and IntAnh (Cochrane et al., 2010). For these reasons, the ImpNon factor was judged to be invalid as a core aspect of Schizotypy and removed from subsequent analyses. A total of 31 items were removed in the expert item content review, leaving 54 items retained for item refinement.

Item Refinement

The magnitude of factor loadings of the remaining 54 items across three factors (30 UnuExp, 10 CogDis, 14 IntAnh) were assessed; items with loadings <.32 were removed (12 items). The remaining 42 items were then submitted to a 3-factor EFA to assess their communalities. Items with communality <.20 were removed (10 items), leaving a final pool of 32 items retained. Prior to IRT modelling, these items were entered into a 3-factor CFA which confirmed strong model fit estimates; χ^2=687.07, *df*=431, *p*<.001, *CFI*=.958, *TLI*=.955, *RMSEA*=.037, *90%CI*=.032 – .042.

Unidimensional 2-Parameter IRT conducted on the final item pool was used to evaluate item discrimination (*a*) and severity (*b*) parameters. These parameters, along with item probability functions (IPF), item information curves (IIC), and test information curves (TIC), were used to reduce redundant and poorly performing items. For confirmation, items removed based on poor factor loading and/or low communality in the previous step were also submitted to unidimensional IRT modelling within their respective factor. Items 40 (CogDis) and 66 (UnuExp) were identified as providing unique breadth along the severity axis (i.e., theta, θ) and, therefore, retained. As such, a total of 34 items were assessed for the final model: 19 UnuExp, 8 CogDis, and 7 IntAnh. The 19 items in the initial UnuExp model were systematically assessed based on their IPFs and IICs. Items possessing similar severity and low discrimination were systematically removed as they were deemed redundant. During this process, item 66 was retained despite poor severity (*a*=1.186) as it provided strong discrimination at the upper end of θ (*b*=2.292). The final UnuExp scale consisted of six items with a strong model fit (*CFI*=.999, *TLI*=.999, *RMSEA*=.009) excellent breadth across severity, and good discrimination (*a*=1.186 to 3.478 and *b*=-0.442 to 2.292). A similar approach was used for CogDis items with the initial eight item model. Item 40 was retained as it provided the strongest discrimination (*b*=0.804) despite poor severity (*a*=0.839) comparative to other items. The final CogDis scale consists of five items with strong model fit indices (*CFI*=.973, *TLI*=.946, *RMSEA*=.07) and good model parameter estimates (*a*=0.839 to 2.769 and *b*=-0.504 to 0.835). IRT analysis of the IntAnh scale followed the same procedure. Only two items were removed from the initial seven item model, both due to redundancy based on their severity and discrimination parameters relative to the other items. The final IntAnh scale consists of five items. Again, strong model fit estimates (*CFI*=.987, *TLI*=.974, *RMSEA*=.04) and parameter estimates (*a*=1.223 to 2.070 and *b*=0.002 to 0.835) were demonstrated. Following IRT, the final model consisted of 16 items; see Figure 4.2 and Figure 4.3 for final item probability functions and the total item information curve, respectively. Refer to Appendix C for extended details of item refinement through IRT.

A. Unusual Experiences



OLIFE27_U Do you ever feel sure that something is about to happen, even though there does not seem to be any reason for you thinking that?

OLIFE60_U Do your thoughts sometimes seem so real as actual events in your life?

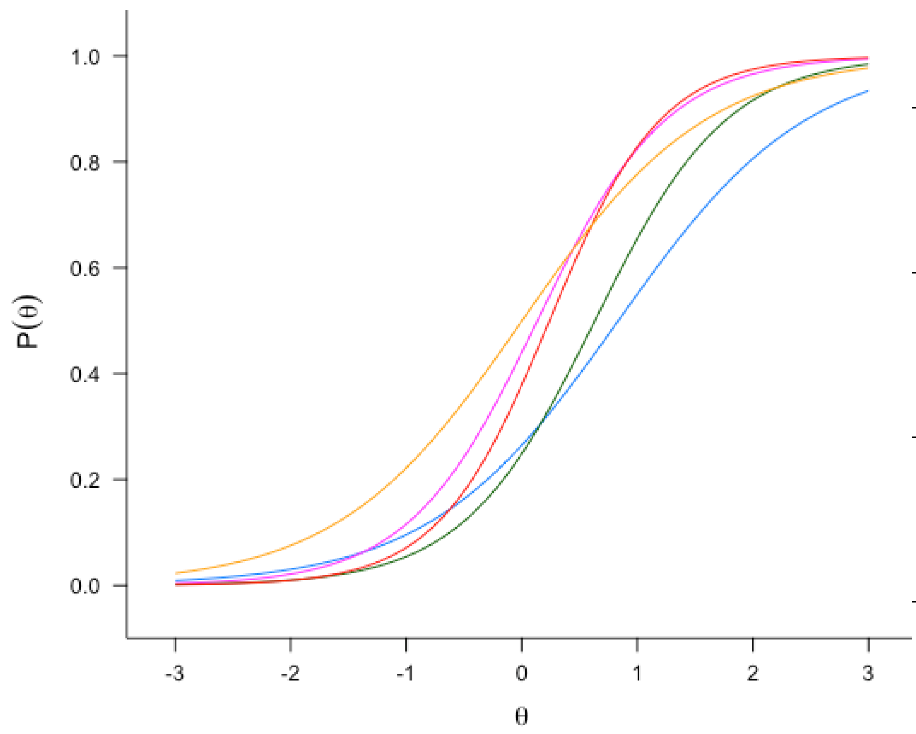
OLIFE99_U Does a passing thought ever seem so real it frightens you?

OLIFE92_U Do people in your daydreams seem so true to life that you sometimes think they are real?

OLIFE87_U Have you occasionally felt as though your body did not exist?

OLIFE66_U On occasions, have you seen a person's face in front of you when no one was in fact there?

B. Introvertive Anhedonia



OLIFE67_S Do you prefer watching television over going out with other people?

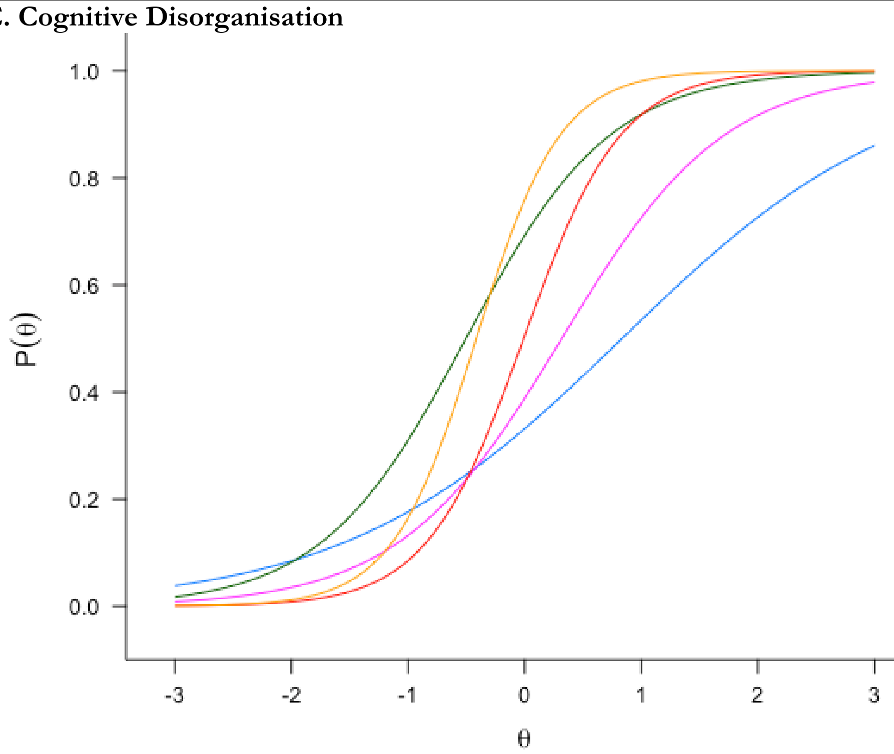
OLIFE19_S Are you much too independent to really get involved with other people?

OLIFE33_S Do you feel that making new friends isn't worth the energy it takes?

OLIFE29_S Do people who try to get to know you better usually give up after a while?

OLIFE7_S Are people usually better if they stay aloof from emotional involvement with people?

C. Cognitive Disorganisation



OLIFE76_C Do you frequently have difficulty in starting to do things?

OLIFE94_C No matter how hard you try to concentrate do unrelated thoughts creep into your mind?

OLIFE81_C Do you often have difficulties in controlling your thoughts?

OLIFE61_C Are you sometimes so nervous that you are 'blocked'?

OLIFE40_C Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense?

Figure 4.2 Item response functions for final items in each OLIFE factor. Items are ordered based on level of severity.

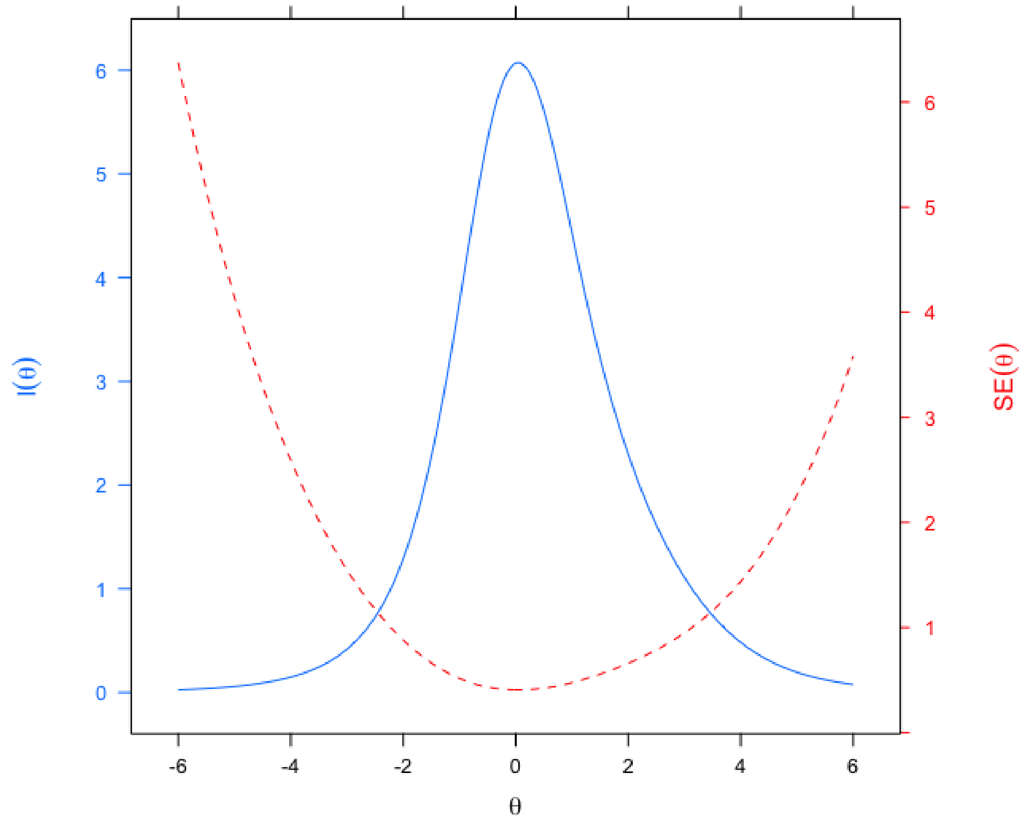


Figure 4.3 Test information curve and standard error of the final 16 OLIFE items.

Assessment of Overall Latent Structure

The overall latent structure of the final 16 items was tested by assessing three latent architectures: (1) 3-Factor with no higher-order factor, (2) hierarchical model with a higher-order factor mediated by three sub-factors, and (3) bifactor model with a general factor, onto which all items load, and three orthogonal secondary factors. Please refer to Table 4.6 for summary of model fit and Table 4.7 for model comparison statistics.

Table 4.6. Model fit statistics for three models tested during assessment of overall latent structure.

Index	**3 Factor**	**Hierarchical**	**Bifactor**
Comparative Fit Index (CFI)	0.995	0.995	0.994
Tucker-Lewis Index (TLI)	0.994	0.994	0.993
Root mean square error of approximation (RMSEA)	0.018	0.018	0.019
RMSEA 90% CI lower bound	0.000	0.000	0.000
RMSEA 90% CI upper bound	0.032	0.032	0.033
Standardised root mean square residual (SRMR)	0.044	0.044	0.044

3 Factor = 3 factor structure with no higher-order, Hierarchical = 3 factor structure with higher-order total score, Bifactor = bifactor model. Estimator = DWLS.

Table 4.7. Overall latent structure model comparison statistics.

	Baseline test				**Difference test**		
	n	χ^2	***df***	***p***	$\Delta\chi^2$	Δdf	***p***
Bifactor	437	83.631	100	0.135			
3 Factor	437	120.702	101	0.150	-1.229e-9	1	1.000
Hierarchical	437	115.728	101	0.150	-3.304e-10	0	

Model comparison showed no difference in fit between the three models. For parsimony, the 3-Factor with no higher-order factor was chosen as the final model. This 16-item, 3-factor solution was named the OLIFE16. To avoid confusion during subsequent comparisons with the parent scale, the subscales of the OLIFE16 will be denoted as UnuExp16 (Unusual Experiences), IntAnh16 (Introvertive Anhedonia), and CogDis16 (Cognitive Disorganisation). The total score, constituting the sum of subscale scores, will be denoted as Total16. Table 4.8 provides the standardised factor loadings for the OLIFE16 items.

Table 4.8. Standardised factor loadings for OLIFE16 items.

OLIFE16 Item	Original OLIFE item	Subscale	Std. Factor Loading
		Unusual Experiences (UnuExp16)	
3	27	Do you ever feel sure that something is about to happen, even though there does not seem to be any reason for you thinking that?	0.416
7	60	Do your thoughts sometimes seem so real as actual events in your life?	0.504
16	99	Does a passing thought ever seem so real it frightens you?	0.656
14	92	Do people in your daydreams seem so true to life that you sometimes think they are real?	0.543
13	87	Have you occasionally felt as though your body did not exist?	0.463
9	66	On occasions, have you seen a person's face in front of you when no one was in fact there?	0.444
		Introvertive Anhedonia (IntAnh16)	
10	67	Do you prefer watching television over going out with other people?	0.418
2	19	Are you much too independent to really get involved with other people?	0.523
5	33	Do you feel that making new friends isn't worth the energy it takes?	0.606
4	29	Do people who try to get to know you better usually give up after a while?	0.616
1	7	Are people usually better if they stay aloof from emotional involvement with people?	0.470
		Cognitive Disorganisation (CogDis16)	
11	76	Do you frequently have difficulty in starting to do things?	0.489
15	94	No matter ow hard you try to concentrate do unrelated thoughts creep into your mind?	0.566
12	81	Do you often have difficulties in controlling your thoughts?	0.633
8	61	Are you sometimes so nervous that you are 'blocked?'	0.466
6	40	Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense?	0.507

Items are organised by factor and severity along θ.

4.3.3 Theory Confirmation Phase

The second phase of this study focussed on confirming the latent structure revealed in the development phase with an independent sample (*n*=548). The summary demographics for this sample can be found in Table 4.2. A summary of clinical characteristics is provided in Table 4.9, below.

Table 4.9. Summary of clinical characteristics of Sample 2 (confirmatory sample).

Measure	***n***	**Mean**	**SD**	**Scale Range**
OLIFE				
Unusual Experiences	548	10.76	6.53	0-30
Cognitive Disorganisation	548	14.08	6.07	0-24
Introvertive Anhedonia	548	9.83	5.55	0-26
Impulsive Nonconformity	548	7.28	3.41	0-18
CAPE-15	413	5.51	5.077	0-33
Persecutory Ideation	413	3.59	2.572	0-15
Bizarre Experiences	413	1.42	2.415	0-16
Perceptual Abnormalities	413	0.49	1.138	0-8
PID-5-FBF Psychoticism	413	0.60	0.524	0-2.83
Eccentricity	413	1.03	0.842	0-4.00
Unusual Beliefs	413	0.49	0.617	0-2.75
Perceptual Dysregulation	413	0.28	0.479	0-3.00
Mini IPIP				
Extraversion	413	2.49	0.946	1.00-5.00
Agreeableness	413	3.70	0.811	1.00-5.00
Conscientiousness	413	3.34	0.818	1.25-5.00
Neuroticism	413	3.05	0.909	1.00-5.00
Openness to Experience	413	3.70	0.876	1.00-5.00
PAI Subscales				
Anxiety	548	60.83	14.99	39-105
Depression	548	61.83	15.30	38-109
Schizophrenia	548	57.21	12.59	38-99
Paranoia	548	54.67	10.64	33-101
Alcohol Abuse	548	51.29	11.35	45-106
Drug Abuse	548	51.25	10.14	42-102
Positive Impression Management	548	46.12	9.49	23-67
Negative Impression Management	548	53.90	10.55	46-85

OLIFE = Oxford Liverpool Inventory of Feelings and Experiences, CAPE-15 = Community Assessment of Psychic Experiences 15 item, PID-5-FBF Psychoticism = Personality Inventory for DSM-5 Faceted Brief Form, Mini IPIP = Mini International Personality Item Pool, PAI = Personality Assessment Inventory.

Model Fit

The new OLIFE16 model was tested using CFA and demonstrated strong model fit indices and significant improvement from the original OLIFE (χ^2=156.263, *df*=101, *p*<.001, *CFI*=.971, *TLI*=.965, *RMSEA*=.031, *90%CI*=.021 – .041. A path diagram of the OLIFE16 is also provided in Figure 4.4.

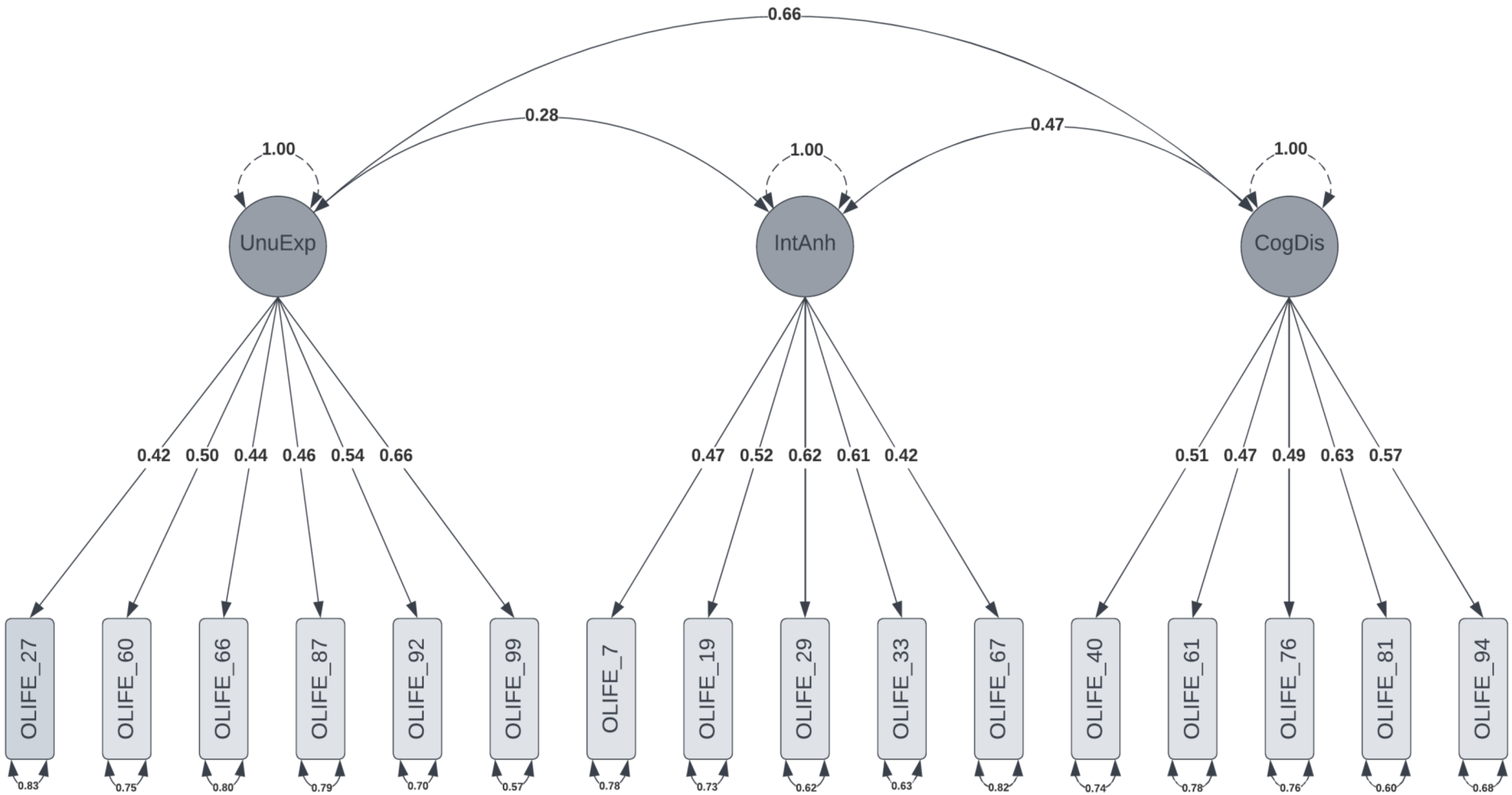


Figure 4.4 Structure of the OLIFE16. All parameter estimates are standardised. UnuExp = Unusual Experiences, IntAnh = Introvertive Anhedonia, CogDis = Cognitive Disorganisation, items are coded based on parent scale item number, e.g., OLIFE_27 = 27th item from the original OLIFE scale.

Reliability

Tests of internal consistency revealed adequate McDonald's omega for each of the factors and the total score: UnuExp16 ω = 0.677 (*95% CI* ω = 0.635-0.719), IntAnh16 ω = 0.656 (*95% CI* ω = 0.611-0.702), CogDis16 ω = 0.667 (*95% CI* ω = 0.623-0.710), Total16 ω = 0.762 (*95% CI* ω = 0.733-0.792). Although not as strong as the original OLIFE, the reduction of internal consistency is likely to be, at least in part, influenced by the significant reduction in the number of items making up the scale and its respective factors (Dunn et al., 2014).

Validity

Spearman's rho correlations were used to assess convergent and discriminant validity of the OLIFE16 with the parent scale and other clinical measures. The OLIFE16 factors demonstrated strong correlations with their respective factors in the parent scale, with Spearman's rho (ρ) correlations ranging from 0.763 to 0.890. See Table 4.10 for correlation coefficients. Spearman rho correlations between the OLIFE16 total score and other measures of psychotic experience demonstrated strong convergent validity, including with the PAI-SF Schizophrenia subscale (ρ = .727), the PID Psychoticism facet (ρ = .629), and the CAPE15 (ρ = .627). Strong correlations with PAI Anxiety and Depression were also observed (ρ = .638 and .632, respectively). These correlations are comparable to those of the parent scale; however, an improvement in discriminability between the OLIFE to OLIFE16 was observed for PAI Depression (ρ = .712). A strong correlation was also observed between the OLIFE16 and Neuroticism from the Mini IPIP (ρ = .543), which was expected, given previous research showing comorbidity between psychoticism and neuroticism (Premkumar et al., 2018). Conversely, a strong negative association was observed between OLIFE16 and Extraversion (ρ = -.268) and Conscientiousness (ρ = -.407). The correlation between OLIFE16 and Openness to Experience was non-significant (ρ = -.037). Overall, the pattern of correlation between the

OLIFE16 to other measures mirrors relationships with the parent scale and indicates strong convergent validity for the new scale.

Review of the three factors within the OLIFE16 revealed strong convergent and discriminant validity. UnuExp16 correlated strongly with all subscales of the CAPE15 and PID Psychoticism, with Spearman's rho ranging from 0.413 to 0.607. Within the CAPE15, the strongest subscale correlation was with Bizarre Experiences (ρ = .547); similarly, in the PID Psychoticism scale, the strongest correlation was observed with Unusual Beliefs (ρ = .607). Positive associations were also observed with PAI-SF Schizophrenia (ρ = .443), Neuroticism (ρ = .385), and Openness to Experience (ρ = .132). Divergence was demonstrated between UnuExp16 and Extraversion (ρ = .038), Conscientiousness (ρ = -.257), and Agreeableness (ρ = .086). These associations with the Big 5 personality traits are consistent with those observed between the parent scale and previous research (Kwapil et al., 2008; Premkumar et al., 2018). CogDis16 demonstrated strong convergence with the CAPE15 (ρ = .503) and PID Psychoticism (ρ = .515), with the most robust subscale correlations observed with Persecutory Ideation (CAPE15; ρ = .438) and Eccentricity (PID Psychoticism; ρ = .448). These subscales both possess elements that embody the disordered thinking associated with the Schizophrenia spectrum and, therefore, demonstrate convergent validity with the CogDis16 factor of the OLIFE16. CogDis16 correlations with PAI-SF subscales showed strong correlation with Schizophrenia (ρ = .611), Anxiety (ρ = .649), and Depression (ρ = .618). Previous research has shown significant overlap between disordered thinking and mood-related disorders, and this is consistent with the current results for the CogDis16 factor (Horton et al., 2014; Lewandowski et al., 2006). CogDis16 converged with Neuroticism (ρ = .536) and was not associated with Agreeableness (ρ = .013) and Openness to Experience (ρ = 0.031). Negative associations were observed between CogDis16 and Extraversion (ρ = -.154) and Conscientiousness (ρ = -.487). This pattern of results is consistent with the parent factor and previous studies of convergent/divergent validity in the OLIFE (Premkumar et al., 2018). Finally, convergent/divergent validity was also demonstrated for IntAnh16. This factor showed robust association with the PAI-SF Schizophrenia subscale (ρ = .535), along with Depression (ρ = .407) and Paranoia (ρ = .352). These are consistent with IntAnh from the original OLIFE and previous studies (Premkumar et al., 2018).

Table 4.10. Spearman's rho correlation coefficient matrix for OLIFE-16 and original OLIFE.

		OLIFE16				Original OLIFE				
Scale	**Variable**	**UnuExp16**	**CogDis16**	**IntAnh16**	**Total16**	**UnuExp**	**CogDis**	**IntAnh**	**ImpNon**	**Total**
OLIFE16	**1. UnuExp16**	—				0.837***	0.436***	0.087*	0.369***	0.628***
	2. CogDis16	0.446***	—			0.522***	0.827***	0.289***	0.418***	0.740***
	3. IntAnh16	0.180***	0.302***	—		0.252***	0.393***	0.763***	0.201***	0.584***
	4. Total16	0.724***	0.794***	0.663***	—	0.725***	0.749***	0.515***	0.441***	0.890***
CAPE15	**10. Persecutory Ideation**	0.413***	0.438***	0.316***	0.526***	0.481***	0.471***	0.262***	0.407***	0.574***
	11. Bizarre Experiences	0.547***	0.415***	0.227***	0.536***	0.604***	0.362***	0.132**	0.408***	0.527***
	12. Perceptual Abnormalities	0.479***	0.339***	0.168***	0.441***	0.476***	0.264***	0.146**	0.318***	0.423***
	13. CAPE Total	0.563***	0.503***	0.327***	0.627***	0.631***	0.488***	0.260***	0.472***	0.654***
PID Psychoticism	**14. Eccentricity**	0.413***	0.448***	0.285***	0.512***	0.482***	0.455***	0.317***	0.362***	0.580***
	15. Unusual Beliefs	0.607***	0.378***	0.152**	0.512***	0.681***	0.312***	0.109*	0.286***	0.506***
	16. Perceptual Dysregulation	0.546***	0.440***	0.208***	0.530***	0.603***	0.406***	0.156**	0.462***	0.556***
	17. Psychoticism	0.602***	0.515***	0.286***	0.629***	0.684***	0.483***	0.276***	0.437***	0.672***
Mini IPIP	**18. Extraversion**	0.038	-0.154**	-0.483***	-0.268***	0.022	-0.286***	-0.642***	0.085	-0.312***
	19. Agreeableness	0.086	0.013	-0.428***	-0.147**	0.103*	0.000	-0.503***	-0.032	-0.153**
	20. Conscientiousness	-0.257***	-0.487***	-0.173***	-0.407***	-0.256***	-0.492***	-0.140**	-0.414***	-0.432***
	21. Neuroticism	0.385***	0.536***	0.282***	0.543***	0.391***	0.701***	0.254***	0.376***	0.619***
	22. Openness to Experience	0.132**	-0.031	-0.181***	-0.037	0.171***	-0.086	-0.231***	0.084	-0.025
PAI-SF Subscale	**23. Anxiety**	0.424***	0.649***	0.328***	0.638***	0.460***	0.735***	0.329***	0.266***	0.666***
	24. Depression	0.367***	0.618***	0.407***	0.632***	0.416***	0.715***	0.455***	0.370***	0.712***
	25. Schizophrenia	0.443***	0.611***	0.535***	0.727***	0.505***	0.652***	0.558***	0.368***	0.761***
	26. Paranoia	0.268***	0.261***	0.352***	0.389***	0.263***	0.343***	0.352***	0.279***	0.432***
	27. Alcohol Abuse	0.154***	0.144***	0.093*	0.175***	0.195***	0.125**	0.005	0.350***	0.211***
	28. Drug Abuse	0.114**	0.08	0.009	0.089*	0.135**	0.073	-0.066	0.243***	0.108*

*CAPE-15=Community Assessment of Psychic Experiences 15-item, PI=Persecutory Ideation, BE=Bizarre Experiences, PA=Perceptual Abnormalities. PID-5-FBF Psychoticism=Personality Inventory for DSM-5 Faceted Brief Form Psychoticism Scale, Ecc=Eccentricity, UnuBE=Unusual Beliefs and Experiences, PeDys=Perceptual Dysregulation. Mini IPIP=Mini International Personality Item Pool, EXT=Extraversion, AGR=Agreeableness, CON=Conscientiousness, NEU=Neuroticism, OPE=Openness to Experience. * p<.05, ** p<.01, ***p<.001.*

To further investigate the convergent validity of the three OLIFE16 factors and whether they discriminate dimensions within the Schizophrenia spectrum, Spearman's rho correlations were calculated with the constituent subscales within PAI-SF Schizophrenia; perceptual disturbance (SCZ-P), thought disorder (SCZ-T), and social detachment (SCZ-S) (refer to Table 4.11 for correlations). These items are taken as an analogue measure of the three factors constituting the Schizophrenia scale identified in the initial validation of the PAI (Morey, 2014) as core dimensions of Schizophrenia. These correlations demonstrate that both CogDis16 and IntAnh16 not only show strong correlation with PAI-SF SCZ, but also discriminate between their respective PAI subscales such that CogDis16 is most strongly associated with SCZ-T (ρ = .679) and IntAnh16 is most strongly associated with SCZ-S (ρ = .623). UnuExp16 was observed to be correlated with SCZ-P (ρ = .484); however, an equally robust correlation with SCZ-T was also observed (ρ = .489).

Table 4.11. Spearman's rho correlation coefficient matrix for OLIFE16 and PAI-SF Schizophrenia scale and constituent subscales.

Variable	UnuExp16	CogDis16	IntAnh16	Total16
SCZ-P	0.484***	0.398***	0.225***	0.502***
SCZ-T	0.489***	0.679***	0.305***	0.675***
SCZ-S	0.181***	0.379***	0.623***	0.537***
PAI-SF SCZ	0.443***	0.611***	0.535***	0.727***

*SCZ=Schizophrenia, SCZ-P=Schizophrenia Perceptual Disturbances, SCZ-T=Schizophrenia Thought Disorder, SCZ-S=Schizophrenia Social Detachment, PAI-SF=Personality Assessment Inventory Short Form. * p<.05, ** p<.01, ***p<.001.*

4.4 Discussion

The current study investigated the validity and factor structure of the OLIFE in an Australian sample. Specifically, it assessed the latent structure of the OLIFE using CFA, EFA, bifactor, and hierarchical modelling, refined items using both CTT and IRT, and proposed a new, brief version of the OLIFE, the OLIFE16 (refer to Appendix C for extended descriptives for the OLIFE16 in the full sample and the final questionnaire for use). The robustness of the OLIFE16 was then tested in a confirmatory sample which replicated the latent structure (three factors) and demonstrated convergent validity with other schizotypal trait measures. The development of the OLIFE16 also targeted increased validity of the CogDis factor via the culling of items measuring anxiety and depression and the elimination of the ImpNon factor from the scale. Items were further edited using CTT and IRT approaches. Measures of internal consistency across the OLIFE16 factors were adequate, especially given the significant reduction in the number of test items. Tests of validity showed a strong relationship with the longer, parent scale, and convergent and discriminant validity with other measures.

The results of this study are consistent with the fully dimensional model of Schizotypy and, more broadly, the notion of the Schizophrenia spectrum. Most notably, the removal of the ImpNon factor from the original structure has meant that the OLIFE16's three-factor structure is now in line with contemporary conceptualisations of Schizotypy (Kwapil & Barrantes-Vidal, 2015; Raine, 1991) and Schizophrenia (Basso et al., 1998; Nelson et al., 2013). As outlined earlier, previous research in the OLIFE and OLIFE-SF have identified several concerns regarding the ImpNon factor (i.e., factorial validity, predictive validity, and discriminant validity) (Chapman et al., 1994; Claridge et al., 1983; Cochrane et al., 2010), with some researchers arguing that it should be removed (Kwapil, Gross, Silvia, et al., 2018). This study supported this view and removed this factor in the proposed brief version (OLIFE16).

Assessment of the CogDis factor revealed the presence of items measuring anxiety-related and depression-related features, which, as previously discussed, cloud the measurement of veridical cognitive disorganisation in Schizotypy (Kwapil, Gross, Silvia, et al., 2018). The resultant CogDis factor is now refined with empirically demonstrated concurrent validity with an evidence-based disordered thinking measure. It should be noted, however, that the final five-item CogDis factor is still significantly associated with depression and anxiety, though to a lesser extent than that observed in the parent scale and more in line with its association with Schizophrenia psychopathology. Although traditional cognitive disorganisation measures have focussed on cognitive-behavioural deficits, contemporary conceptualisation has shifted to also include disorganisation of affect, with research examining the relationship between Schizotypy and affective symptoms, demonstrating that disorganised thinking to be strongly associated with anxiety and depression (Kemp et al., 2018). Therefore, the association observed between the CogDis scale and PAI-SF ANX and DEP subscales suggest this scale is aligned with current conceptualisations of disordered thinking. This is also consistent with the observed associations with neuroticism, which may be considered trait-level expressions of emotional instability, depression, and anxiety (McCrae & Costa, 2010). Some researchers have suggested that neuroticism may be a moderator of Schizotypy expression and increases liability for more severe SSD (Claridge & Davis, 2002). Indeed, the strong relationship between SSD and affective symptoms overall is preserved in more severe psychopathology (e.g., high comorbidity between Schizophrenia, Anxiety and Depression; American Psychiatric Association, 2013a). As such, the pattern of correlations between the CogDis scale of the OLIFE16 with anxiety and depression psychopathology is consistent with current conceptualisations of multidimensional Schizotypy. Overall assessments of validity demonstrated that the three factors of the OLIFE16, unusual experiences, introvertive anhedonia, and cognitive disorganisation, directly map onto the positive, negative, and disorganised symptoms associated with Schizophrenia.

While the OLIFE16 has demonstrated excellent convergent and divergent validity, measures of internal consistency were less robust. As reported above, the internal consistency of the OLIFE16 is reduced compared to its parent scale. While this is a relative weakness of the new scale, this is likely to have been impacted by the reduction of items in the brief scale. Guidelines on scale shortening have shown that the use of reliability as a guide for item selection is cautioned as deletion of items to maintain high internal consistency may actually suggest item redundancy and, therefore, scale narrowness (Boyle, 1991). Such practice was specifically not used in the current study for these reasons and, given the small number of items within each factor were specifically chosen as they covered the span of the underlying dimension, sacrifices in reliability are perhaps expected.

Another area for consideration in the OLIFE16 is item wording, specifically, item 9 (item 66 in the parent scale), which belongs to the Unusual Experiences scale and asks, '*On occasions, have you seen a person's face in front of you when no one was in fact there?*'. As discussed above during IRT analysis, this item was retained for its coverage of more severe forms of the underlying dimension, which provided unique breadth to the scale beyond that of other items in the subscale. However, the specificity of the words '*a person's face*', as opposed to more general wording (e.g., things, objects), may reduce the item's discriminatory abilities. Wording revisions may need to be considered in future studies to improve the psychometric performance of this item.

To our knowledge, the OLIFE16 stands as the shortest measure of multidimensional Schizotypy while still retaining robust psychometric properties, which offers researchers an economical and accessible way to measure Schizotypy that aligns with current conceptual models. This new measure was constructed and validated with a sample ranging from 18 to 60 years and is recommended for use within this age range until further studies can demonstrate its validity outside of this age range. Note that most research utilising Schizotypy measures recruit samples of older adolescents and young adults (Gross et al., 2018). Given the original conceptualisation of the OLIFE as derived from the personality approach to Schizotypy, as opposed to the clinical approach, Schizotypy here is viewed as a trait that varies within the general population (Rawlings et al., 2008b). However, unlike other personality traits, such as conscientiousness, where most people fall in the middle ranges, levels of Schizotypy, are likely to be relatively low in the general population, with increasing scores on the OLIFE16 to be associated with increased liability for pathological SSD. Furthermore, no cut-off scores are recommended for the OLIFE16 as indicative of ‘a schizotypic individual’; instead, researchers are encouraged to use OLIFE16 scores as a continuous variable.

4.5 Conclusion

Having put forth the OLIFE16 and demonstrated initial validation with the parent scale and other measures related to Schizotypy, the next steps would be to expand the pool of validation evidence. We suggest that future research on the validity of the OLIFE16 include four main approaches. First, correlation between the OLIFE16 and its subscales with other measures of Schizotypy (e.g., the Multidimensional Schizotypy Scale; MSS; Kwapil et al., 2018) to establish concurrent validity. Second, further cross-sectional investigations on how the dimensions within the OLIFE16 are differentially associated with positive, negative, and disorganised symptoms of Schizophrenia, prodromal symptoms, personality disorders, and mood-related symptoms or disorders are also recommended. These investigations could also be extended to other areas such as cognitive impairment, genetic variation, and life experiences. Third, to establish predictive validity, longitudinal studies examining whether non-clinical samples with high OLIFE16 scores have increased liability to development of Schizophrenia-spectrum psychopathology and diagnosis are suggested. These could be compared to the results from the original OLIFE in Cochrane and colleagues (2010). And finally, given the popularity of the parent scale across many different cultures, investigations on the measurement invariance of the OLIFE16 across differing demographic groups is strongly recommended.

CHAPTER 5

Study 3: Application of the OLIFE16 to Kinematic Data from Study 1

To review the current thesis thus far, it commenced with a literature review of the neurodevelopmental nature of SSD, followed by evidence of dorsal-stream specific impairment associated with symptoms and diagnoses along this spectrum. While previous research has focussed on dorsal-stream impairments through the classical Ungerleider and Miskin (1982) 'what' versus 'where' model of ventral and dorsal visual processing streams, this thesis highlighted another well-established model of human visual processing, that of the Perception-Action Model (PAM), by Goodale and Milner (1992). PAM differs from classical models of human visual processing as it focuses on the functional outputs supported by visual processing (i.e., perception or action), as opposed to the nature of visual inputs (e.g., object identification versus spatial location). The adoption of PAM for understanding human visual processing allows for assessment of the functional implications of dorsal visual stream processing deficits in SSD, focusing on visually guided movement. As previously reviewed, only one study has used this approach by measuring the kinematics of grasping in patients with Schizophrenia and healthy controls under a visual illusion paradigm (King et al., 2008). Although this study did find differences in the grasp component of kinematics between patients and controls, there has been criticism of the use of visual illusion methodology (Bruno & Franz, 2009; Franz & Gegenfurtner, 2008). Moreover, neuroimaging data has delineated two sub-pathways within the dorsal stream that subserve qualitatively different functions during visually guided movement (Grol et al., 2007). Finally, previous research has not examined visuomotor deficits across the broader Schizophrenia spectrum, such as subclinical samples varying on Schizotypy.

Given the sparse research available investigating SSD-related dorsal stream deficits using a PAM lens, Study 1 of this research program sought to address this gap in the literature by conducting a visually guided movement study with tasks that selectively targeted ventral stream and dorsal sub-pathway functioning and recruited a community sample varying on trait-level Schizotypy. Kinematic variables associated with manual size estimation were not hypothesised to be influenced by level of Schizotypy; however, reaching and grasping movements were postulated to vary across levels of Schizotypy, with greater Schizotypy linked to increased reliance on pre-planned movement during reach and overestimation in target scaling and correction during grasp. The results of this study provided partial support for the hypotheses. As hypothesised, the ventral task was not influenced by level of Schizotypy; yet the relationship between dorsal stream tasks and level of Schizotypy were more nuanced than expected. Specifically, while overreliance on pre-planned movements, as evidenced by greater $PHS_{t\%}$, was observed with increased Schizotypy, this only occurred in the reach-to-point condition and not in reach-to-grasp. Moreover, while increased Schizotypy was associated with larger PGA (i.e., overestimation of target size, during grasp), no differences in the timing of PGA (i.e., $PGA_{t\%}$) were observed. Overall, the results provided only partial support for the hypotheses of Study 1, and the effects observed were small, ranging from η_p^2 = .048 to .059.

Given these unexpected results, we elected to closely evaluate the scale used to quantify trait-level Schizotypy (i.e., the OLIFE) on the notion that poor validity in the quantification of Schizotypy may have tempered the effects observed in Study 1. A review of the OLIFE's psychometric properties revealed several misgivings regarding its factor structure and validity. As such, Study 2 sought to conduct a comprehensive psychometric analysis of the OLIFE with the goal of refining and validating the measure in an Australian sample using classical and modern psychometric techniques. This process resulted in the OLIFE16, a 16-item brief measure of Schizotypy that possesses a three-factor structure including Unusual Experiences, Introvertive Anhedonia, and Cognitive Disorganisation and demonstrates adequate reliability and strong validity. With this new measure, there is now the opportunity to reanalyse the kinematic data from Study 1 with a refined quantification of trait-level Schizotypy.

5.1 Present Study

The purpose of the present study is to re-examine the nature of SSD-related dorsal stream and sub-pathway deficits using the PAM as an explanatory framework, as investigated in Study 1, using the OLIFE16 as a measure of trait-level Schizotypy. The hypotheses for this study are the same as those identified in Study 1 and are reiterated below for reference:

1. Level of Schizotypy will not be associated with changes in Stable Grip Aperture (SGA) during manual size estimation. This will confirm intact ventral visual stream functioning across the sample.
2. Greater Schizotypy will be associated with greater proportion of overall movement time taken to reach peak hand speed ($PHS_{t\%}$) during reach-to-point and reach-to-grasp movements. If this relationship is observed, it will demonstrate Schizotypy traits to be linked to a stronger reliance on pre-planned movement and implicate dorsomedial sub-pathway dysfunction.
3. Greater Schizotypy will also be associated with greater peak grip aperture (PGA) and proportion of overall movement time taken to reach PGA ($PGA_{t\%}$) during reach-to-grasp. This will show impairment in target scaling and correction during grasp, respectively. This pattern of performance would implicate dorsolateral dysfunction in Schizotypy.

5.2 Method

5.2.1 Participants

The sample used for this re-analysis is identical to that of Study 1. Therefore, sample characteristics will only be briefly reviewed here (please refer to Table 5.1 for a summary of demographics). For full details, please refer to Table 3.2 in the Methods section of Study 1 (Chapter 3). Table 5.2 summarises the OLIFE16 descriptives of the sample.

Table 5.1 Summary of demographic characteristics of sample.

Variable	**Mean (SD)/Proportion (*n*=94)**
Age (years)	21.1 (SD=3.98) (range 18 to 44)
Gender	67 female (26 male, 1 undisclosed)
Ethnicity	32 Caucasian; 62 Asian
Handedness	94 right-handed

Table 5.2 Summary of Oxford Liverpool Inventory of Feelings and Experiences 16 item (OLIFE16) score of sample.

OLIFE16 Subscale	**Sample (*n*=94)**
	Mean (SD)
Unusual Experiences (UnuExp16; max score 6)	1.8 (1.3)
Introvertive Anhedonia (IntAnh16; max score 5)	1.7 (1.4)
Cognitive Disorganisation (CogDis16; max score 5)	2.4 (1.2)
Total Score (max score 16)	6.0 (2.6)

5.2.2 Motion Tracking Data Processing and Extraction

All motion data files were identical to those extracted for Study 1. For reference, listed below in Table 5.3 are the definitions of the kinematic variables extracted. These are the same as those used in Study 1.

Table 5.3 Summary of kinematic variables extracted from raw motion tracking data.

Variables Measured	**Variable Description**
Movement Time (MT)	Total time from movement onset to movement offset.
Movement onset (M_{on})	The onset of reach and grasp movements was determined as exceeds 5% of the respective peak wrist velocity.
Movement offset (M_{off})	The moment when the velocity of the wrist sensor in the x axis direction falls and remains below 5% of peak velocity, i.e., the target is grasped.
Grip Aperture (GA)	The vector distance between the index finger and thumb finger sensors, measured at 10% intervals of MT from 0% (movement onset) to 100% (movement offset). GA reflects the opening and closing of the fingers that occurs when reaching to grasp an object.
Peak Grip Aperture (PGA)	The maximum three-dimensional distance between the index and thumb markers between movement onset & offset.
Time to Peak Grip Aperture (PGA_t)	Difference in time between the movement onset and when PGA is achieved.
Peak Hand Speed (PHS)	Maximum velocity reached during movement as measured by the wrist marker.
Time to Peak Hand Speed (PHS_t)	Difference in time between the movement onset and when PHS is achieved.
Stable Grip Aperture (SGA)	Averaged grip aperture across duration of each individual target size estimation.

5.3 Results

The experimental results for this study will be presented in the same order as those in Study 1. The first section will report on the control variables, which were used as validity checks. Subsequent sections will address each of the hypotheses. The data were analysed using repeated measures analysis of covariance (RM ANCOVA), with conditions (target size, movement type, and target type) as the independent variables; however, this time with OLIFE16 total score as a covariate, and kinematic variables as dependent variables. Additional RM ANCOVAs were also run with individual OLIFE16 factors as covariates. Where appropriate, the Bayesian counterpart RM ANCOVA was calculated to further verify effects. Main effects were assessed using BF_{10}. To isolate interaction effects, the $BF_{inclusion}$ (BF_{incl}) factor was used, which compares models with the effect to equivalent models without the effect, excluding higher-order interactions (JASP Team, 2022). The size of BF_{incl} can be interpreted in the same way as BF_{10}, such that, values below 1 indicate support for the null hypothesis, values between 1 and 3 suggest a weak effect, values between 3 and 10 suggest a moderate effect, and values greater than 10 suggesting a strong effect (van den Bergh et al., 2020). For continuity and ease of comparison, the frequentist and Bayesian results will be reported together. As the current analysis is specifically interested in the impact of Schizotypy, as measured by the OLIFE16, on kinematic variables, the main effects of experimental conditions will not be reported, unless they deviate from those found in Study 1.

5.3.1 Control Variables

Prior to reviewing the substantial results, analyses of control variables were completed to validate experimental manipulations and confirm that participants were responsive to these manipulations.

Target Size

Target size was varied to control for potential artefacts in results that may have arisen due to only using one target size. Across eight kinematic variables, three revealed significant main effects for target size. As expected, these arose in SGA, GA, and PGA. A total of 117 interaction effects were tested with target size and only two were significant, both of which were expected as they occurred in PGA and GA analysis.

Movement Time (MT)

Due to evidence of non-normality in the raw MT data (Shapiro-Wilk test *p* ranged from <.001 to .087), they were log transformed to improve normality (Shapiro-Wilk test *p* ranged from .016 to .733). Log(MT) was then submitted to a 2(Target Size) X 2(Movement Type) X 2(Target Type) RM ANCOVA. Importantly, no significant effects were observed for MT across levels of Schizotypy. Therefore, level of Schizotypy did not impact the time participants took to complete movements.

5.3.2 Hypothesis 1: Manual Estimation

Stable Grip Aperture (SGA)

Participants' perception of target size was assessed by analysing SGA in manual estimation trials. Due to violations of sphericity (Mauchly's test of sphericity $p<.01$), Greenhouse-Geisser corrected results are reported, where appropriate. As in Study 1, the main effect of Target Size was significant, suggesting that participants' perceived target size changed as a function of actual target size (i.e., shorter targets were estimated as shorter and longer targets estimated as longer). Level of Schizotypy (OLIFE16 total) and its latent dimensions were not associated with SGA (see Figure 5.1; for illustrative purposes, level of Schizotypy, here and in subsequent figures, is presented across two groups, high and low, created via a median split). These results support the first hypothesis, that level of Schizotypy is not associated with manual size estimation, a ventrally driven task. These results are also consistent with those observed in Study 1.

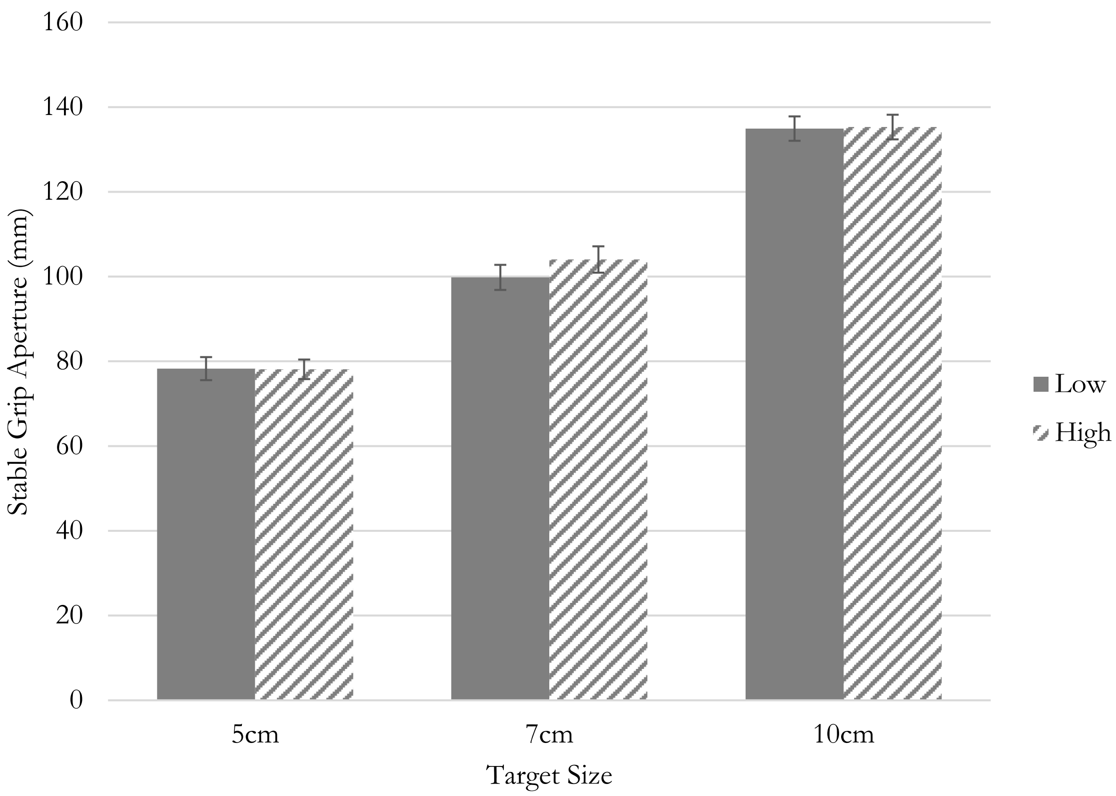


Figure 5.1 Comparison of SGA at each target size with a median split applied to level of Schizotypy (OLIFE16 Total Score). Low Schizotypy = solid bars, high Schizotypy = striped bars. Error bars represent standard error.

5.3.3 Hypothesis 2: Reliance on pre-planned movement

To test whether increased Schizotypy is associated with a stronger reliance on pre-planned movement, 2(Target Size) X 2(Movement Type) X 2(Target Type) RM ANCOVAs were computed for Peak Hand Speed (PHS) and proportion of overall MT taken to reach PHS ($PHS_{t\%}$), with OLIFE16 total score and factor scores as covariates. Observing larger $PHS_{t\%}$ with increased OLIFE16 scores would support this hypothesis.

Peak Hand Speed (PHS)

As in Study 1, Movement Type (point, grasp) revealed a significant main effect suggesting PHS was greater in reach-to-grasp compared to reach-to-point movements. No significant effects were found as a function of Schizotypy (OLIFE16 total) or its latent dimensions (see Figure 5.2).

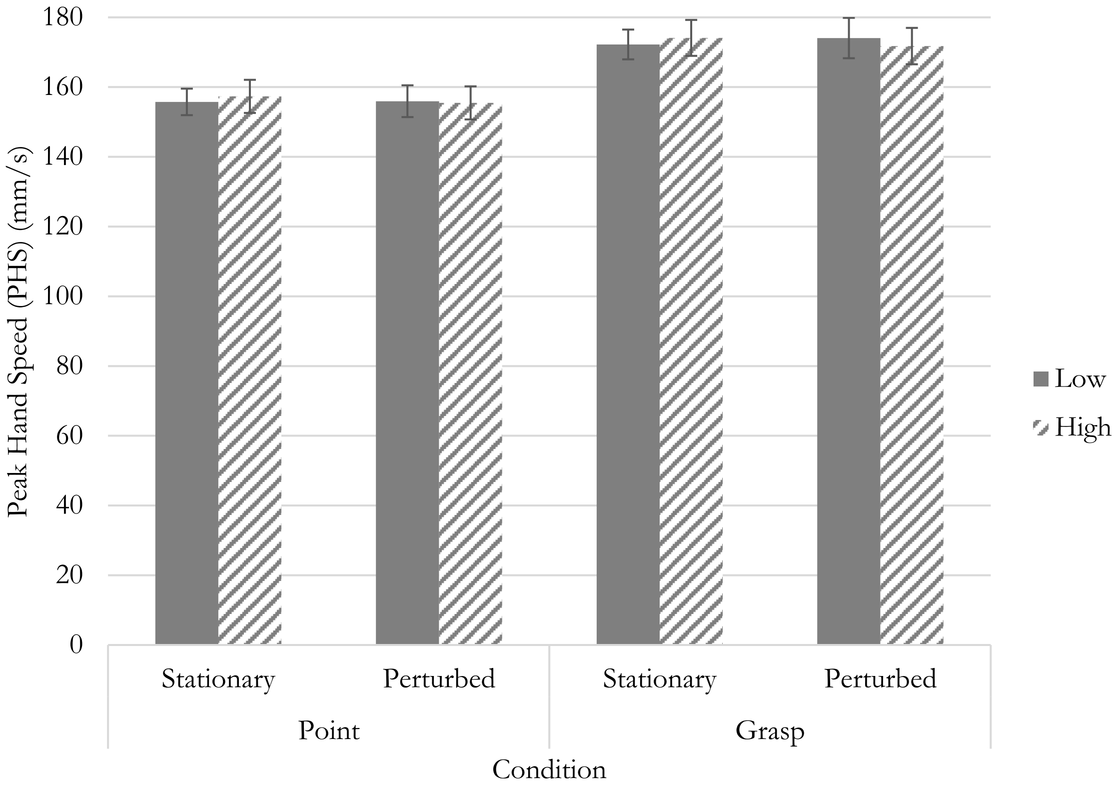


Figure 5.2 Peak hand speed for 5cm target as a function of movement type (point, grasp), target type (stationary, perturbed), and OLIFE16 total score with median split applied.

Peak Hand Speed Time (PHS_t).

To standardise PHS_t across total MT, PHS_t data were transformed to a percentage of MT and denoted by $PHS_{t\%}$. The main effect of Target Type (stationary, perturbed) was consistent with Study 1, whereby $PHS_{t\%}$ was significantly smaller (i.e., PHS occurred earlier in the movement), during perturbed compared to stationary trials. As in Study 1, very small interaction effects were observed between Target Type x Target Size (η_p^2 = .09) and Target Type x Movement Type (η_p^2 = .07). These showed that PHS occurred later (larger $PHS_{t\%}$) on stationary trials for larger targets (7cm) but earlier on perturbed trials for larger targets (7cm) compared to smaller targets (5cm) and that on perturbed trials, PHS occurred later (larger $PHS_{t\%}$) during reach-to-point compared to reach-to-grasp movements, respectively. Schizotypy was significantly associated with $PHS_{t\%}$ through the interaction between

Movement Type (point, grasp) and OLIFE16 Total, *F*(1, 78) = 4.3 *p* = .004, η_p^2 = .05; however, this was not supported in Bayesian analysis, BF_{incl} = .138. A significant interaction between Movement Type (point, grasp) and CogDis16 was also observed, *F*(1, 76) = 5.0, *p* = .029, η_p^2 = .061; however, this was not supported in Bayesian analysis, BF_{incl}=0.306. Therefore, $PHS_{t\%}$ was not observed to be impacted by level of Schizotypy (see Figure 5.3).

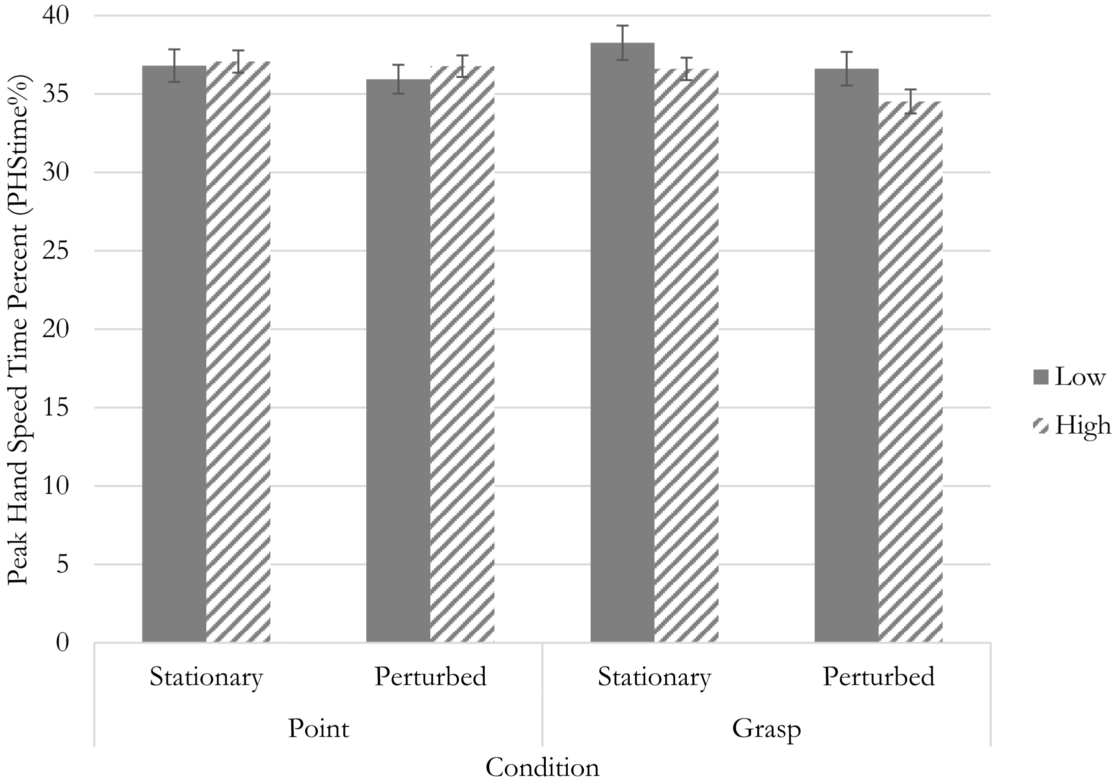


Figure 5.3 $PHS_{t\%}$ for 5cm target as a function of experimental condition and OLIFE16 total score with a median split applied. Error bars represent standard error.

Overall, the results from PHS and $PHS_{t\%}$ do not support the second hypothesis. In fact, greater Schizotypy was associated, although not significantly, with smaller $PHS_{t\%}$ indicating less time spent in pre-planned movements compared to lower Schizotypy in grasp conditions.

5.3.4 Hypothesis 3: Target size scaling and correction during grasping

To test whether increased Schizotypy is associated with impairments in target scaling and correction, 2(Target Size) X 2(Movement Type) X 2(Target Type) RM ANCOVAs were computed for Peak Grip Aperture (PGA) and proportion of overall MT taken to reach PGA ($PGA_{t\%}$), with OLIFE16 total score and factor scores as covariates. Observing larger PGA and $PGA_{t\%}$ with increased OLIFE16 scores would support this hypothesis.

Peak Grip Aperture (PGA)

Main effects of Target Size (5cm, 7cm) and Target Type (stationary, perturbed) were significant, as in Study 1, which confirms that participants correctly scaled larger grip apertures for larger targets and scaled larger apertures on perturbed compared to stationary trials. No significant interaction was observed a function of Schizotypy (OLIFE16 total score) or any of its latent dimensions (see Figure 5.4).

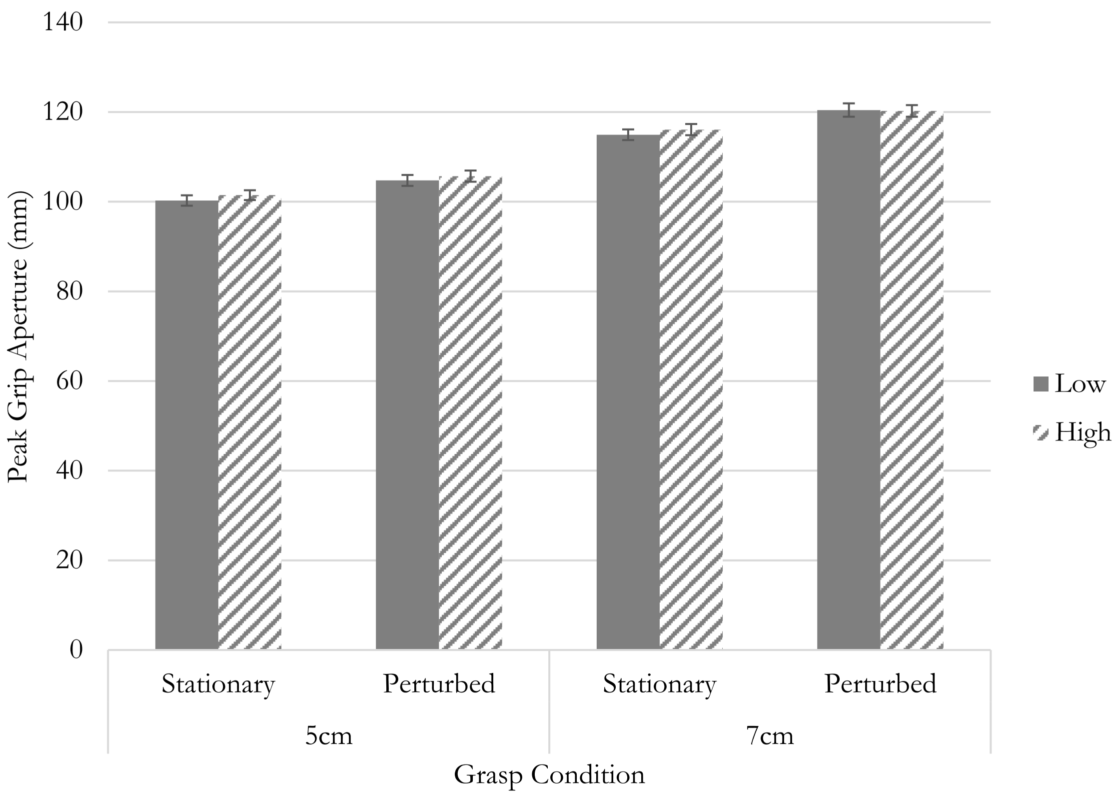


Figure 5.4 PGA as a function of grasp condition and OLIFE16 total score with a median split applied. Error bars represent standard error.

Peak Grip Aperture Time (PGA_t).

To standardise PGA_t across total MT, PGA_t data was transformed to a percentage of MT and is denoted as $PGA_{t\%}$. As in Study 1, significant main effects of Target Type (stationary, perturbed), Target Size (5cm, 7cm) were revealed. No significant effects were observed with level of Schizotypy (OLIFE16 total score) or any of its latent dimensions (see Figure 5.5). These results suggest that the percent of overall movement time taken to reach PGA is only impacted by target type, such that $PGA_{t\%}$ is delayed in perturbed compared to stationary trials and in larger (7cm) targets compared to smaller (5cm) targets.

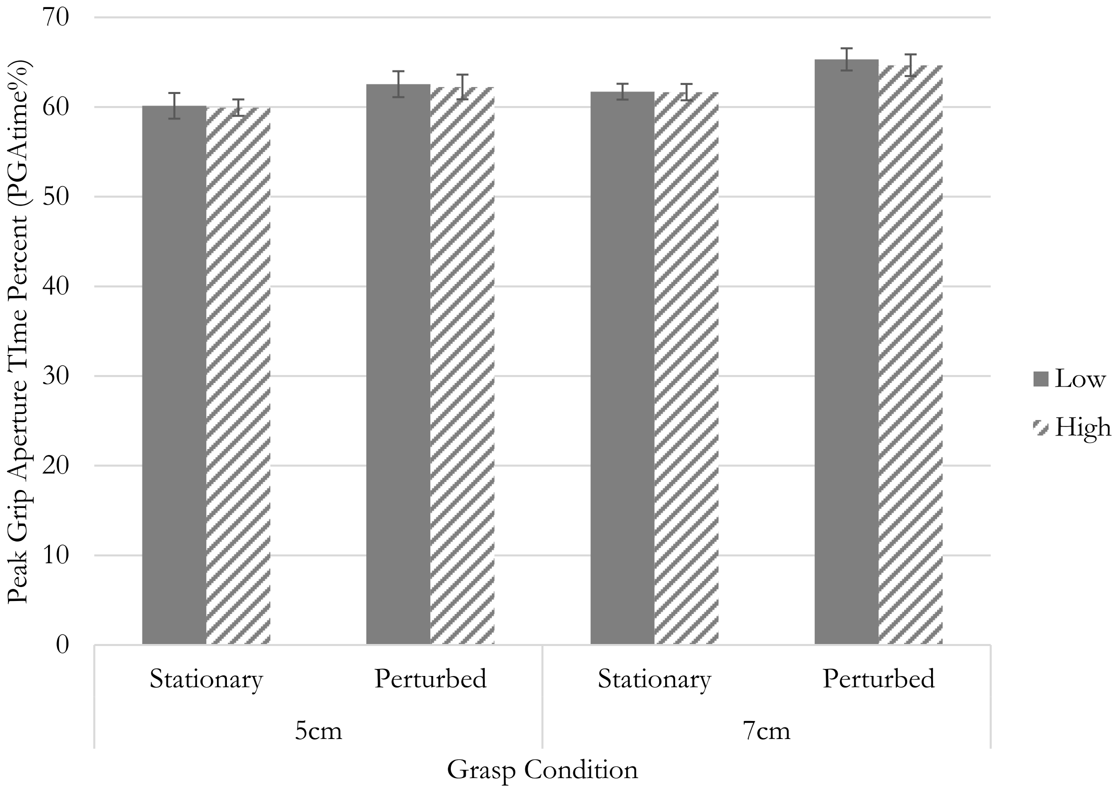


Figure 5.5 $PGA_{t\%}$ as a function of grasp condition and OLIFE16 total score with a median split applied. Error bars represent standard error.

Overall, these results do not support the third hypothesis. Increased Schizotypy was not associated with overestimation in PGA, nor was it associated with greater reliance on pre-planned movement.

Grip Aperture (GA)

Given the coarseness of using PGA, grip aperture (GA) across the entire movement was analysed in 10% increments from 0% (movement onset) to 100% (movement offset at the target). GA over 11 Intervals of Movement (0% to 100%) were submitted to an RM ANCOVA with Target Size (5cm, 7cm) and Target Type (stationary, perturbed) for grasp conditions along with OLIFE16 scores as covariates. No significant effects were observed as a function of Schizotypy or its latent dimensions. These results are consistent with those reported above for PGA and $PGA_{t\%}$, and, therefore, do not support Hypothesis 3 as increased Schizotypy was not observed to be associated with differences in target scaling and/or the time course of grip formation.

5.4 Discussion

The current study sought to re-analyse kinematic data from Study 1 (Chapter 3), which tested visually guided movement under differing task conditions to assess the integrity of ventral and dorsal streams, and the dorsolateral and dorsomedial sub-pathways, in a community sample varying on trait-level Schizotypy. It improves on the previous study by utilising a psychometrically refined measure of Schizotypy, the OLIFE16, developed in Study 2 (Chapter 4). It was hypothesised that using a refined and valid measure of Schizotypy would elucidate clearer, and potentially stronger, relationships between Schizotypy and dorsal stream functioning. Overall, differences in kinematic measures of prehension were not observed as a function of Schizotypy across dorsally driven tasks.

The results demonstrated that manual size estimation of three different target sizes varied as a function of actual target size. Since size estimation is understood to be primarily a ventral stream function, level of Schizotypy was hypothesised to not impact participant performance on this task. The results supported this hypothesis as no differences in SGA were observed as a function of Schizotypy. This effect was much clearer in this study compared to Study 1, where an effect of cognitive disorganisation, a latent dimension of Schizotypy, was observed to significantly associate with SGA. Kinematic variables recorded on dorsal stream tasks did not support the hypotheses made regarding level of Schizotypy. Level of Schizotypy was not associated with greater reliance on preplanned movement as there were no differences in $PHS_{t\%}$. On measures of grip formation, Schizotypy was also not associated with larger PGA, or the proportion of time taken to reach peak grip aperture ($PGA_{t\%}$). These results demonstrate no differences in the kinematic measures of reach-to-point and reach-to-grasp movements as a function of Schizotypy (refer to Table 5.4 for a comparison of results from Study 1 and the current study).

Table 5.4 Comparison of support for hypotheses from Study 1 and Study 3 (current study) results.

Hypothesis	**Study 1: OLIFE**	**Study 3: OLIFE16**
H1: No impact of Schizotypy on ventrally driven size estimation.	Supported	Supported
H2: Increased Schizotypy is associated with stronger reliance on pre-planned movement and less time spent in movement correction and accuracy.	Partial support	Not supported
H3: Increased Schizotypy is associated with overestimation in target scaling and a stronger reliance on pre-planned grasping with less time spent in grasp correction.	Partial support	Not supported

The current study has shown that with a refined and validated measure of Schizotypy, the patterns observed from Study 1 between subclinical SSD and dorsal-specific differences in visually guided movement are no longer observed. As such, we return to the other two possible explanations that were put forth following the results of Study 1. For reference, the three explanations offered included: (1) differences in methodology, (2) differences in sampling, and (3) validity issues in quantification of Schizotypy. Study 2 and the current study were conducted to address the issue of validity in Schizotypy measurement. This leaves methodology and sampling as avenues for further consideration. Furthermore, there is an additional limitation of this study regarding the reliability of the new Schizotypy measure, the OLIFE16. Each of these issues will be addressed in turn.

5.4.1 Methodology Related Explanations

The methodology employed in the only other study that examined dorsal-specific deficits in Schizophrenia used a visual illusion paradigm (King et al., 2008). As reviewed in the introduction, this approach has been criticised due to inconsistencies in stimuli presentation parameters. While the current studies chose to employ a different methodology to avoid the pitfalls of visual illusion paradigms, these alternative methods also come with a host of potential shortcomings. Therefore, it is important to consider the validity and reliability of the chosen methods used in Study 1. Unlike other measures in psychology, such as psychometric batteries, experimental paradigms, like the one use in Study 1, are not typically submitted to the same rigorous assessments of reliability and validity. Indeed, many of these experimental methods are unique to the individual lab from which it emanates and are used to test specific hypotheses without regard for psychometric properties. This approach also makes comparisons across studies very difficult. While there is evidence, reviewed in Study 1, that participants were adequately responding to the experimental manipulations as evidenced by consistency in the observed kinematic patterns and those from the human prehension literature, without simultaneous neuroimaging, it is unknown whether the specific tasks used in the current study were differentially activating the hypothesised neural correlates. Specifically, the ventral versus dorsal visual streams, and within the dorsal stream, the dorsomedial and dorsolateral sub-pathways. These hypotheses were guided by two major lines of research within the field of visual perception. First, Goodale and Milner's (1992) Perception-Action Model (PAM), which functionally divides the ventral and dorsal streams into vision-for-perception and vision-for-action, respectively. And second, research demonstrating functional dissociation between the dorsomedial and dorsolateral sub-pathways of the dorsal visual stream as responsible for the 'reach' and 'grasp' components of visually guided hand movements, respectively (Grol et al., 2007;

Jeannerod, 1988). The validity of the behavioural measures used in Study 1 to test these neural circuits will now be reviewed in more detail.

The functional division of the ventral and dorsal streams, as outlined in Goodale and Milner's (1992) PAM, has been well established from neuropsychological (Goodale et al., 1994a; James et al., 2003; Karnath et al., 2009), functional neuroimaging (Cavina-Pratesi et al., 2007), and behavioural studies (Ganel et al., 2008; King et al., 2008). Furthermore, the use of manual estimation and visually guided movement as behavioural measures which dissociate these visual streams has been consistently demonstrated (Culham et al., 2003; King et al., 2008; Tunik et al., 2005). The correlation of these tasks to ventral and dorsal visual streams has also been demonstrated using fMRI (Cavina-Pratesi et al., 2007). Evidence from TMS also supports the use of these methods to dissociate ventral and dorsal visual processing streams (Ellison & Cowey, 2006). Ellison and colleagues' (2006) TMS study demonstrated the ventral stream, specifically the lateral-occipital area (LO), to be casually involved in shape discrimination, and the dorsal stream, specifically the posterior parietal cortex (PPC), to be causally involved in distance discrimination. Together, these studies provide strong support for the validity of the methods used in Study 1 to adequately dissociate ventral and dorsal visual streams. Therefore, the results from the current study demonstrating no difference in size estimation (ventral task) as a function of level of Schizotypy are likely valid. However, conclusions about the dorsal sub-pathways and their associated tasks are more nuanced and require further dissection.

Another framework that has informed the current research is the further subdivision of the dorsal stream into two segregated pathways (i.e., the dorsomedial and dorsolateral sub-pathways). Research demonstrates these sub-pathways to subserve the two components of prehension; the dorsomedial stream codes for the 'reach' component while the dorsolateral stream is responsible for the 'grasp' component (Culham et al., 2003; Grol et al., 2007). As reviewed in the Introduction and Study 1, the reach component is believed to control hand transport with adjustments made primarily based on the target's extrinsic properties (e.g., location). In contrast, the grasp component is responsible for hand pre-shaping to allow for target acquisition and control with adjustments based on the intrinsic properties of the target (e.g., shape and size). Anatomically, the dorsomedial 'reach' circuit links the superior occipitoparietal complex (SPOC, also known as area V6A in macaques) and the medial intraparietal sulcus (mIPS) towards the dorsal premotor cortex (PMd) before connecting to the primary motor cortex (M1) (Grol et al., 2007). Conversely, the dorsolateral 'grasp' circuit links anterior intraparietal sulcus (aIPS, also known as AIP in macaques) within the inferior parietal lobule (IPL) and area F5 within the ventral premotor cortex (PMv). While there has been significant research, mostly from lesion studies, to support this division of function in the dorsal stream, advances in neuroimaging (e.g., multivariate pattern (MVP) analysis in fMRI) and experimental techniques such as dual-site transcranial magnetic stimulation (ds-TMS) have shown overlapping functions of these sub-pathways (Cavina-Pratesi et al., 2018; Monaco et al., 2015; Turella & Lingnau, 2014). In the dorsomedial 'reach' sub-pathway, data from neurophysiological and neuroimaging evidence indicate parietal activity to encode for both reaching and grasping movements (see Turella & Lingnau, 2014 for review). For example, neural activity in area V6A of macaques is sensitive to reach direction and grasp (Fattori et al., 2004, 2005, 2010; Turella & Lingnau, 2014). In humans, a neuroimaging study on the superior parietoccipital cortex (SPOC), the putative human homolog of area V6A, observed preparatory signals such that SPOC appeared to encode planned reach-to-touch and reach-to-grasp movements differently (Gallivan et al., 2011). The dorsal premotor cortex (PMd) has also been found to encode grasp-related components of prehension in macaques (Hao et al., 2014; Raos et al., 2004; Takahashi et al., 2017) and humans (see Turella & Lingnau, 2014 for review). More recently, a dual-site TMS study by Vesia and colleagues (2018) found that the human dorsomedial PMd to primary motor cortex (M1) circuit is responsive to handgrip formation during grasp preparation. Within the dorsolateral 'grasp' sub-pathway, areas such as aIPS and area F5 have consistently been found to encode for grasp-related aspects of prehension, especially in contexts that demand integration of visuospatial and contextual information for grasp planning and correction (Fattori et al., 2010; Verhagen et al., 2008). In particular, the aIPS has been causally linked to hand pre-shaping during reach-to-grasp (Davare et al., 2006; Rice et al., 2006; Vesia et al., 2013) and rapid online correction following target perturbation (Rice et al., 2006; Tunik et al., 2005). Taken together, these results indicate that the dorsomedial sub-pathway is more likely involved in encoding interactions in reach and grasp-related components, while the dorsolateral sub-pathway is causally involved in grasp (Turella & Lingnau, 2014). Considering this new evidence, the visually guided movement tasks used in Study 1 to differentiate dorsomedial and dorsolateral sub-pathways may have been less valid than initially proposed. It is more likely that both the reach-to-point and reach-to-grasp conditions employed the dorsomedial sub-pathway, while the reach-to-grasp also recruited the dorsolateral sub-pathway.

5.4.2 Sample Related Explanations

Another possible explanation for the unexpected results seen in this study (and Study 1) relates to differences in sampling. Specifically, King and colleagues (2008) recruited patients with diagnosis of Schizophrenia or Schizoaffective Disorder, while the current study recruited a nonclinical, community sample without a formal diagnosis and varying on trait-level of Schizotypy. When considering these differences in the context of SSD, it is important to note that the confounds often associated with clinical levels of SSD are known to impact motor control (e.g., antipsychotic medication). This raises questions as to whether the effects observed in patient populations are due to the psychopathology *per se* or the confounding effects associated with illness severity (e.g., use of medication, institutionalisation). Motor control difficulties, more generally, have been well documented in Schizophrenia (Burton et al., 2016; Manschreck et al., 2004; Vrtunski et al., 1986; Wolff & O'Driscoll, 1999). These often include symptoms such as dyskinesia, dystonia, akathisia, spontaneous Parkinsonism, and catatonic phenomena (Berna et al., 2015; Cuesta et al., 2014; Fenton, 2000; Walther & Strik, 2012). Additionally, neurological soft signs (NSS), subtle deficits in sensory integration, motor coordination, and sequencing of complex motor acts, have been a focus in Schizophrenia research as prime candidates for endophenotypes (Bachmann et al., 2014; Dazzan & Murray, 2002; Schröder et al., 1991). Simultaneously, research into antipsychotics has revealed side effects, perhaps most significantly, motor-related issues such as tardive dyskinesia (Llorca et al., 2002; Seibert & Crowell-Davis, 2019). Nevertheless, clinical investigations into antipsychotic-naïve first episode Schizophrenia patients have shown that some movement abnormalities observed in the patient population are independent of medication (Cuesta et al., 2014; Dazzan & Murray, 2002; Manschreck et al., 2004; Wolff & O'Driscoll, 1999). Furthermore, a recent systematic review by Hirjak and colleagues (2018) revealed NSS are associated with increased Schizotypy and psychosis proneness as well as unaffected first-degree relatives of Schizophrenia patients. Evidence showing that these symptoms increase in severity along the Schizophrenia spectrum has also been established (Hirjak et al., 2015; Schröder & Herold, 2022). The effects of antipsychotics on these symptoms also appears to be additive. One study, which investigated rapid hand movements in Schizophrenia, showed that patients were more impaired than their neurotypical counterparts in both amplitude and peak velocity, but not frequency of movements (Putzhammer et al., 2005). The degree of impairment also varied as a function of antipsychotic treatment such that deficits were more pronounced in those patients treated with conventional antipsychotics (e.g., haloperidol, compared to atypical antipsychotics such as risperidone and drug-naïve patients; Putzhammer et al., 2005). Indeed, even Kraepelin's (1919) initial descriptions of 'dementia praecox' identified core symptoms related to motor deficits. Taken together, this evidence suggests that motor control deficits are present across the Schizophrenia spectrum and, therefore, core to the condition. However, the assessments of motor control and NSS are almost exclusively from clinician assessment and self-report measures. Very few studies have employed more objective, fine-grained measurement methods such as motion tracking and kinematic analysis. In addition to Putzhammer and colleagues (2005), Nowak and colleagues (2006) compared five drug naïve participants with Schizophrenia, 13 medicated subjects with Schizophrenia, and 18 neurotypical controls on two manual prehension tasks. First, a vertical arm movement task that instructed participants to power grasp a cylindrical object and rapidly move it vertically between two points 30cm apart. Second, a weight catching task that instructed participants to power grasp the same cylindrical object as the first task, however, this time it was attached to a cup. Participants were instructed to keep the object and cup stationary. A small weight was then dropped into the cup, either expectedly (by the participant), or unexpectedly (by the experimenter) while the participant closed their eyes. Kinematic variables including grip force, load force, and linear acceleration were measured. Their results demonstrated that drug naive participants with Schizophrenia performed comparably to healthy controls. In contrast, medicated participants with Schizophrenia exhibited excessive grip force scaling and impaired coupling between grip and load force profiles (Nowak et al., 2006). Furthermore, these deficits were strongly correlated with the severity of both extrapyramidal side effects related to antipsychotic treatment and the negative symptoms related to the underlying pathology. These results suggest that while some motor control deficits (e.g., rapid hand movements) may be core features of the Schizophrenia spectrum, others may result from antipsychotic treatment alone (e.g., grip force scaling). These differences in underlying causes for different motor control deficits along the Schizophrenia spectrum question the interpretation of the previous study by King and colleagues (2008) suggesting differences between Schizophrenia patients and neurotypicals during visually guided grasping under visual illusion as reflective of specific dorsal stream deficits core to the condition. It is possible that the differences King and colleagues (2008) observed in peak grip aperture in the patient group may have been due to extrapyramidal side effects related to antipsychotic treatment. Although their patient population were reported to all be receiving atypical antipsychotic medication, which have been found to cause less extrapyramidal side effects than typical antipsychotics, their effects are not insignificant (Gao et al., 2008). If this is the case, it provides a possible explanation for the lack of associations between Schizotypy and kinematics of prehension in this study, i.e., the current sample are not impacted by antipsychotic treatment.

5.4.3 Reliability

For the current study, the OLIFE16 was used to test whether a refined and valid measure of Schizotypy would reveal clearer, and potentially stronger, relationships between level of Schizotypy and the kinematics of manual prehension. While refinement and purification of the parent scale to arrive at the OLIFE16 followed best psychometric practices, as outlined in Study 2, these processes likely led to sacrifices in the final scale's internal consistency. Reductions in internal consistency of the OLIFE16 and its factor scores directly affect any comparisons made between scores on the OLIFE16 and other measures, e.g., with kinematic variables. Specifically, increases in measurement error, i.e., unreliability, will immediately affect the strength of associations with the scale, such that all correlations will be diluted (Shrout & Yager, 1989). For the current study, increased measurement error in the OLIFE16 may have weakened the observed relationships between level of Schizotypy and kinematic variables of interest. While there are statistical methods to quantify the extent of attenuation, these require reliability measures of both tests in question, which, as outlined above, is unknown for the visually guided movement tasks.

5.5 Conclusion

Overall, these results have significant implications for the dorsal-specific deficits described as core to SSD. This study demonstrates, for the first time, that subclinical, trait-level Schizotypy is not strongly associated with kinematic differences in visually guided movement related to dorsal-specific processing. Conclusions about dorsomedial and dorsolateral sub-pathways are still unknown, given new evidence suggesting that these sub-pathways are not as readily dissociated by reaching and grasping as originally believed. The lack of differences as a function of Schizotypy brings into question the validity of motor-related deficits in SSD as a core feature of the condition, rather than an artefact of confounds such as antipsychotic treatment. Future research in this area is encouraged to apply other methodologies, such as visually guided movement under visual illusion, across subclinical, trait-level Schizotypy, as seen in King and colleagues (2008). This would allow for a clearer comparison of results and isolate whether the lack of results in the current study are due to sampling or methodology. Moreover, sampling across the entire Schizophrenia spectrum, including normative population varying on Schizotypy, first-episode drug-naïve patients, and Schizophrenia patients with chronic pathology is recommended. Sampling the entire Schizophrenia spectrum, rather than discrete categories, will allow for a richer analysis of how dorsal visual functioning in SSD.

CHAPTER 6

General Discussion and Conclusion

This chapter summarises the major findings from the three studies outlined in this research program, followed by a critical discussion about the contribution and implications of the results in the field. Given the thesis merges two large areas of research across SSD (i.e., dorsal visual stream deficits and psychometric assessment of Schizotypy), the forthcoming discussion is subdivided into five sections. First, a summary of the major findings from the three studies outlined in Chapters 3, 4, and 5. Second, a critical review of the dorsal-specific deficit approach to understanding SSD and the implications that Study 1 and 3 have on this area of research. Third, a broader discussion about methodological approaches that seek to correlate behavioural and self-report measures in psychology. Fourth, the utility of Schizotypy samples and measurement in Schizophrenia research at large will be addressed in the context of contemporary models. Finally, a more general discussion on psychometric measurement in psychology will be presented with a specific focus on the consequences of poorly constructed scales and recommendations for identifying psychometrically valid measures for research.

6.1 Review of Main Findings

The current research program sought to further elucidate the dorsal visual stream deficits associated with SSD through the lens of the PAM. It aimed to extend previous research by examining these deficits in subclinical, trait-level Schizotypy and applying new methods to assess sub-pathways within the dorsal stream: namely, the dorsomedial and dorsolateral circuits (Study 1). This was achieved by taking kinematic measures of the hand as participants completed a series of pointing and grasping tasks toward targets of differing sizes under stationary and perturbed conditions. It was hypothesised that reach-to-point movements and stationary targets would preferentially rely on the dorsomedial circuit while reach-to-grasp movements and perturbed targets would be driven by the dorsolateral circuit. Participants' level of Schizotypy was measured using the OLIFE, and it was hypothesised that increased Schizotypy (higher OLIFE scores) would be associated with differences in kinematic measures during visually guided movement that would implicate dorsal-specific functioning. However, the results of this study failed to support the hypothesis that Schizotypy is associated with differences in visually guided movement consistent with dorsal stream deficits. While some kinematic effects were seen, including differences in the proportion of overall movement time taken to reach Peak Hand Speed ($PHS_{t\%}$) and Peak Grip Aperture (PGA) across levels of Schizotypy, these were small. Considering these findings, validity-related issues in the measurement of Schizotypy were identified as a possible explanation (among others) for the unexpected results.

To investigate this further, a large-scale psychometric investigation was undertaken to examine the architecture and validity of the OLIFE; the measure used to quantify Schizotypy. By employing both Classical Test Theory and Item Response Theory approaches, the original OLIFE was distilled to a 16-item self-report measure with sound psychometric properties, including improved construct validity (Study 2). The OLIFE16 was then used to re-analyse kinematic data from Study 1 to examine whether the pattern of results changed with the use of a more valid measure of Schizotypy (Study 3). The results demonstrated no differences as a function of Schizotypy. Furthermore, the small effects observed in Study 1 were rendered insignificant. These outcomes suggest dorsal-specific differences in visually guided, kinematic movements are not present with increased levels of Schizotypy. Taken together, the results from this research program have significant implications for conceptualising a dorsal-specific deficit associated with SSD, the role of

Schizotypy in SSD research and, more generally, psychometric measurement in psychopathology.

6.2 Schizophrenia Spectrum Disorders and Dorsal-specific Deficits

The lack of differences in kinematic measures as a function of level of Schizotypy suggests that dorsal-specific deficits, at large, may not be present in subclinical populations of SSD. This raises the important issue of whether dorsal-specific deficits are truly core to SSD, as traces of such deficits would also be expected at lower levels of the continuum. Here, we discuss the broader considerations around observing such deficits across SSD.

SSD are associated with generalised cognitive deficits across many domains, such as memory, attention, and learning (McCutcheon et al., 2023). These studies are concerned with specific deficits, which, ideally, must use a differential deficit approach to demonstrate meaningful differences. The differential deficit approach establishes specific deficits as a greater loss in one ability as compared to one or more other, or general, abilities (Chapman & Chapman, 1973). However, the identification of specific deficits cannot rest on group performance differences alone, but also, on the discriminating power of the tasks themselves (Chapman & Chapman, 1973). First raised by Chapman and Chapman (1973), discriminating power denotes the extent to which scores on a test or task discern the "more able" from the "less able" subjects and, by extension, their respective groups. Practically, this can be achieved through matching tasks on true score variance (the product of reliability and observed variance) (Chapman & Chapman, 1975). Failure to control for differences across tasks in discriminating power can result in misleading conclusions, as the test with the greater discriminating power will consistently demonstrate a greater performance deficit between groups (Brenner et al., 2003). As such, differences observed between patient and control groups in studies demonstrating a dorsal-specific deficit in Schizophrenia may be an artifact of mismatched tasks on discriminating power, as opposed to a true difference in performance due to pathology. While some studies endeavour to match tasks using psychophysical threshold techniques, this approach caters only to the 'difficulty' aspect of discriminating power and does not match performance in terms of true score variance. To our knowledge, there has only been one study that specifically matched tasks based on true score variance in SSD using visual processing tasks. A study by Brenner and colleagues (2003) compared Schizophrenia spectrum patients with neurotypicals on discrimination and recognition tasks tapping early visual processing of form and motion. They found that when tasks were matched on difficulty only, SSD patients performed significantly worse on discrimination and recognition of motion compared to form. This demonstrated a differential deficit in motion-specific tasks. However, when tasks were matched on true score variance, the SSD group showed deficits in form discrimination and motion discrimination and recognition. These results evidence impairments in both transient and sustained channels of early visual processing channels following true score variance matching. This study demonstrates how unmatched tasks may lead to misleading conclusions about the nature of differential deficits.

Despite the significant impacts of unmatched tasks, discriminating power is rarely measured in behavioural tasks. In this vein, it is also possible that the tasks employed in Study 1 may have had such low discriminating power in the population of interest that the differences anticipated were concealed due to floor effects (i.e., the tasks were too easy and, therefore, unable to adequately differentiate as a function of level of Schizotypy). While the tasks in Study 1 were specifically chosen to preferentially recruit either the ventral, dorsomedial, or dorsolateral pathways, they are also relatively simple in the context of human perception and movement. Therefore, the potential differences in kinematic measures as a function of Schizotypy may have been too small to detect in tasks with such low discriminating power. Nevertheless, the plethora of evidence on dorsal-related deficits in SSD cannot be dismissed. Decades of research, primarily in patients with diagnosis, have demonstrated differences in performance across many tasks known to preferentially recruit the dorsal stream, for example, motion processing deficits (Chen, Levy, et al., 1999; Chen, Palafox, et al., 1999), reduced attentional spotlight modulation (Cornblatt & Keilp, 1994; Elahipanah, 2013; Elahipanah et al., 2011; Slaghuis & Thompson, 2003), attenuated attention in peripheral vision (Elahipanah et al., 2011; Slaghuis & Thompson, 2003), greater masking deficits (Butler et al., 2001; Butler & Javitt, 2005; Rassovsky et al., 2004), poor target location discrimination (Cadenhead et al., 1998; O'Donnell et al., 1996), and lower contrast sensitivity (Kéri et al., 2002). While all these studies concluded that their results reflect a dorsal-specific deficit, the evidence presented above suggests that without matching tasks on difficulty and true score variance, it is difficult to determine whether the differences observed between patients and neurotypicals are truly due to underlying pathology, an exaggeration due to unmatched tasks, or entirely due to an artefact of methodology.

6.3 Correlating Behavioural and Self-Report Measures

Another issue that arises when considering individual differences in psychological research is the relatively low associations observed between behavioural and self-report measures of the same construct observed in the literature (Dang et al., 2020). While these approaches are two of the most popular methods used in psychology, recent meta-studies in areas such as self-control (Saunders et al., 2018), empathy (Murphy & Lilienfeld, 2019), and creativity (Park et al., 2016) have demonstrated that behavioural and self-report methods purporting to measure the same construct correlate minimally (e.g., Pearson's $r = 0$ to 0.20,). Dang and colleagues (2020) highlighted this issue and proposed that the reasons underlying this problem are both methodological and conceptual in nature. Methodologically, there are fundamental differences in the meaning of reliability between behavioural and self-report measures that conflict with each other. For behavioural measures, reliability is reflected in how consistently an 'effect' is replicable. Here, within-person variability is maximised at the expense of between-person variability so that most people will respond to the experimental manipulation (Hedge et al., 2018). Conversely, reliability in self-report measures is concerned with how consistently the questionnaire can rank individuals, which seeks to maximise between-person variance (Hedge et al., 2018). Consequently, the features that support reliability in behavioural tasks are the same features that compromise reliability in self-report measures; this is known as the reliability paradox and was first observed by Hedge and colleagues (2018). Moreover, the individual reliability of two tests directly limits the maximum observable correlation between them (Metsämuuronen, 2022). Therefore, as behavioural measures typically have low reliability (for between-person variance), while self-report measures usually have high reliability (for between-person variance), the maximum possible correlation between these approaches are likely to be weakened.

Conceptually, there are also significant differences in the response processes of interest between behavioural and self-report measures. First, behavioural methods quantify responses to uncommon stimuli in specific and highly controlled conditions (e.g., reaching to grasp under specific conditions), whereas self-report measures require participants to consider their behaviours across a variety of unstructured real-life situations (e.g., '*Do you often have difficulties in controlling your thoughts?*'; Item 12 of the OLIFE16). Second, behavioural measures are performance-based (e.g., reaction time and accuracy), whereas self-report measures rely on perceptions of performance (i.e., subjective judgments about performance). Finally, behavioural measures quantify an individual's optimal performance by encouraging participants to 'do their best', while self-report measures quantify typical performance. These differences in response processes offer another explanation for the reduced correlation observed between these methods. When considered in relation to Studies 1 and 3 of this research program, the reliability paradox coupled with differences in measured response processes may have contributed to the lack of associations observed between Schizotypy and kinematic measures.

6.4 Schizotypy in Schizophrenia Research

A core guiding principle of the current research program has been viewing Schizophrenia symptomatology as lying on a continuum that spans clinically serious diagnosis to common variation in personality among non-clinical community members (i.e., Schizotypy). As previously discussed, this is referred to as the Schizophrenia spectrum and has gained increasing acceptance (Adjorlolo et al., 2021; Ahmed et al., 2013; Haslam et al., 2020). However, despite its recent popularity, the dimensional models of Schizotypy were introduced as early as 1953 by Rado. However, with the rise of the medical approach to Schizophrenia, the dimensional model at that time was largely confined to the field of personality (Nelson et al., 2013). Importantly, these two competing models differ significantly in their views on the utility and usefulness of Schizotypy in the study of SSD. From the medical perspective, Schizotypy is seen as merely a risk factor and a step in the trajectory towards Schizophrenia. Here, the point of interest is clinical psychopathology and the transition from subclinical to clinical presentations. As the conversion of those with high Schizotypy to clinical SSD is relatively low, only about 5% in a 10-year longitudinal study (Chapman et al., 1994), researchers have been more interested in other influential factors, especially genetic and environmental ones (see McCutcheon et al., 2020 for review). Therefore, the utility of understanding subclinical, Schizotypy variation through this approach is low. Conversely, the dimensional model of SSD, which views Schizotypy as an integral part of the Schizophrenia spectrum and places importance in understanding interindividual differences and processes across the spectrum. Under this approach, Schizotypy operates as an organising framework for understanding SSD as it affords rich information about the manifestation of pathology before the onset of clinical illness (Barrantes-Vidal et al., 2015; Debbané & Barrantes-Vidal, 2015). With the shift toward a neurodevelopmental and dimensional model of SSD, the use of neurotypical samples varying on level of Schizotypy as an analogue sample is warranted. The following section will review four primary utilities of such samples in the study of SSD and, more broadly, psychological research in general.

First, adoption of dimensional approaches will allow for a broader understanding of Schizophrenia that is not constricted by diagnostic labels and is in line with best practice research. Conceptualising Schizophrenia as lying on a spectrum brings together a broad range of conditions that encompasses not only clinically diagnosable disorders such as Schizophrenia and Schizotypal Personality Disorder, but also, subclinical manifestations such as prodromal and high-risk states and, more broadly, psychotic-like experiences (PLEs), Schizotypy, and psychosis (Barrantes-Vidal et al., 2015; Cohen et al., 2015; Debbané & Barrantes-Vidal, 2015). Including a broader range permits a more accurate reflection of the variation seen in the population and not just the rare, extreme occurrences in clinical disorders. Indeed, one of the primary pitfalls of traditional categorical approaches to psychopathology research is the high heterogeneity between patients diagnosed with the same condition. The Schizophrenia spectrum approach can integrate these differences meaningfully to facilitate research (Kwapil & Barrantes-Vidal, 2015; Nelson et al., 2013). This approach is consistent with the National Institute of Mental Health (NIMH) Research Domain Criteria (RDoC; Insel et al., 2010). Established in 2009, RDoC is a research framework that views mental health and psychopathology within the context of major domains of human neurobehavioural functioning, as opposed to discrete diagnostic categories. Its conception was born from recognition of several issues arising from traditional research approaches, including problems with heterogeneity, high comorbidity in patients, and lack of representation of larger spectrum functioning (Insel et al., 2010). While much research focusses on the severe end of the spectrum, studies investigating subclinical Schizotypy samples could provide crucial missing information about the heterogeneity observed at later stages.

Second, studying Schizotypy can provide insight into factors contributing to movement along the spectrum. Investigations across the Schizotypy continuum may reveal different mechanisms at different regions of the spectrum, including those that may work as catalysts for the onset of clinical disorders. Similarly, spectrum research may also unveil protective or compensatory factors in the trajectory toward severe psychopathology. For example, Schofield and Claridge (2007) demonstrated an interaction between the three factors of Schizotypy and their ability to predict evaluations of paranormal experiences. They found that Schizotypy, characterised by high unusual experiences (positive Schizotypy) and low cognitive disorganization, was associated with evaluations of paranormal experiences as pleasant. Conversely, Schizotypy, characterised by high introvertive anhedonia (negative Schizotypy) and high cognitive disorganisation, was associated with evaluations of paranormal experiences as distressing. The authors suggested that positive Schizotypy (e.g., magical thinking), may be adaptive as it enhances the ability to construct cognitive frameworks for meaning-making (Schofield & Claridge, 2007). Other researchers have come to similar conclusions such that while negative Schizotypy has been associated with poorer outcomes and increased liability for psychopathology, positive Schizotypy has been linked to enhanced openness to experience and creativity (Mohr & Claridge, 2015; Nelson & Rawlings, 2010; Schofield & Claridge, 2007; Tabak & Weisman de Mamani, 2013).

Third, the continued search for genetic and endophenotypic components of SSD is only enriched by the inclusion of Schizotypy and the broader spectrum of Schizophrenia. Finally, as previously discussed in Chapters 2 and 4, the use of subclinical samples varying on level of Schizotypy avoids the significant confounds of medication, institutionalisation, distress, and comorbidity often associated with severe forms of SSD. This is especially important, as exemplified in the current research program, should deficits often associated with Schizophrenia not be observed in subclinical populations of the spectrum, questions about the validity of these deficits as core to the condition are warranted. For the reasons outlined above, there is significant utility in the use of Schizotypy samples in SSD research.

The implication of this approach also has significant bearing on sampling more generally in Schizophrenia research. Traditionally, Schizophrenia research has adopted a group comparison method, whereby researchers specifically recruit a group of persons diagnosed with Schizophrenia and then find age and gender-matched peers to form a control group (see Kalkstein et al., 2010 for a review). Data analysis is then carried out through group comparisons on various tasks. Although this approach provides a seemingly straightforward assessment of Schizophrenia to neurotypical controls, it fundamentally ignores the dimensionality and neurodevelopmental nature of SSD. Conversely, the acceptance of the dimensional and developmental approach would consider individuals in the 'control' group as having a unique place along the Schizophrenia continuum (i.e., variation in level of Schizotypy). Furthermore, those in the 'patient' group would also likely differ in their severity, chronicity, and symptomatology along the Schizophrenia spectrum. For these reasons, it is recommended that research into SSD should sample along the continuum such that all participants are measured on level of Schizotypy, with those identified as at-risk or with diagnosis to be further characterised using additional measures, as appropriate. Furthermore, subsequent data analysis should consider 'level of Schizotypy' and variance along the Schizophrenia spectrum as a continuous variable, rather than discrete groups, as demonstrated in Studies 1 and 3 of this research program.

While the utility of Schizotypy in SSD research is readily observed, its utility outside of psychopathology is also rich with potential. Schizotypy has been of interest in a wide range of disciplines, including neuroscience (Fink et al., 2014; Wang et al., 2022), genetics (Salminen et al., 2019; Walter et al., 2016), evolution (Brüne, 2004; Cheli, 2023), personality (Fonseca-Pedrero et al., 2018; March & Springer, 2019), religion (Hanel et al., 2019), industrial and organisational psychology (Burch & Foo, 2010), anthropology (McClenon, 2012), and art (Burch et al., 2006). In particular, Schizotypy has also been recommended as an organising framework for social and affective sciences (Cohen et al., 2015). Cohen and colleagues (2015) proposed that Schizotypy has high utility outside of Schizophrenia research as the associated social and emotional differences present within Schizotypy are not only observed in SSD, but also in depression, anxiety, substance use, personality disorders, and ADHD. Therefore, Schizotypy may be an avenue for revealing underlying mechanisms at play for clinical symptoms more broadly. Furthermore, they highlight that Schizotypy has much to contribute to the theoretical constructs core to social and emotional sciences (e.g., the diathesis-stress model). Given the relatively predictable critical period for psychosis expression, Schizotypy is an excellent candidate for understanding the interaction between risk and resilience factors (Cohen et al., 2015). Taken together, this discussion emphasises the imperative role of Schizotypy not only in SSD research but also in broader areas of psychopathology and well-being. Consequently, conceptual and psychometric clarity of Schizotypy is essential for this research to be fruitful. The current research program contributed to this goal by examining both conceptual and psychometric facets of Schizotypy to provide a psychometrically robust measure that may be used in other research areas where Schizotypy is of interest.

6.5 Psychometrics: A Cautionary Tale

While the use of psychometric measures to quantify Schizotypy dates back to the mid-1900s with Paul Meehl's *Checklist of Schizotypic Signs* (Meehl, 1964), psychometrics, the area of psychology concerned with theory and techniques of measurement, began over a century earlier with work from both English (e.g., Francis Galton), and German (e.g., Johann Friedrich Herbart), scientists (Craig, 2017). Since then, psychometrics has observed prolific advancements in analytical and statistical techniques such as structural equations modelling, IRT, and most recently, machine learning (Anunciação, 2018). With increased power in statistical analyses, researchers are now able to conduct studies with increasing sophistication. However, these advancements are not infallible and, therefore, if used incorrectly, may lead to significant consequences.

One of the main issues surrounding psychometrics and measurement is that of validity, more specifically, construct validity (i.e., how well a set of indicators represent the concept it seeks to measure (Smith, 2005). Construct validity is a venerable and important idea in all of psychology as it forms the backbone of measurement. While the importance of validity and ways to measure it have been documented since the 1940s, construct validity was first coined by Paul Meehl and Lee Cronbach in their seminal 1955 paper '*Construct Validity in Psychological Tests*'. They proposed that rather than focus on criterion validity, psychology should emphasise whether tests can capture the nature of the underlying theoretical construct. They also showed construct validity to be a combination of many different types of validity that could be evaluated by constructing nomological nets, which captured theoretical concepts and their interrelations. The development of approaches to measure such theoretical constructs is then empirically tested. Ultimately, construct validity embodies how much the empirical evidence matches the theoretical networks that have been proposed.

Practically, construct validity comprises three components: substantive component, structural component, and external component (Loevinger, 1957). These components are reflected in the three stages of test construction: identifying a pool of items, analysis and selection of the internal structure of the pool of items, and correlation of test scores with other related variables. Therefore, construct validity must be considered at every stage of test construction, not merely as a final measure. As evidenced in the current research program, while the original OLIFE scale had demonstrated concurrent and discriminant validity with other measures of Schizotypy, significant issues concerning the initial item pool and internal structure were uncovered. Following a comprehensive investigation into the substantive and structural aspects of validity in the OLIFE, the new brief OLIFE16 carries a fundamentally different architecture to its predecessor, one which is more consistent with the underlying theoretical frameworks of Schizotypy. Furthermore, the application of the new measurement tool (OLIFE16) led to changes in the pattern of relationships between the behavioural (kinematic variables) and self-report (Schizotypy) measures of interest. These changes stand as an exemplar of the pitfalls of poor construct validity, i.e., that it can lead to fundamentally different, and potentially misleading, results and interpretations of data. Therefore, researchers are urged to consider all aspects of construct validity when deciding on which measures to employ in their studies, and not just the external component.

6.6 Future Research Directions

One of the main limitations of the research presented in this thesis is the lack of concurrent neuroimaging techniques alongside behavioural measures. While behavioural measures provide important insight into the functional integrity of neural networks, simultaneous measures of neural activity (e.g., fMRI) provide evidence for the validity of behavioural measures in recruiting neural networks of interest. These techniques are common in neural network mapping in normative population (Li et al., 2009); however, their application to samples along SSD are rare. Furthermore, the use of TMS over dorsal regions in low Schizotypy participants as a way of drawing causal conclusions should also be considered. TMS affords to the opportunity to create 'virtual lesions' which may be used to mimic dorsal-specific deficits observed in SSD (Silvanto & Muggleton, 2008). Comparisons in performance between 'TMS lesions' in normative populations with low Schizotypy, those with high Schizotypy, and diagnosed patients would provide rich information about dorsal-related deficits in SSD. TMS may also be used further investigate underlying networks within dorsal-related deficits, such as the dorsomedial and dorsolateral sub-pathways, with greater control comparative to behavioural studies, like the one presented in Study 1, alone.

Another area of future research from this thesis is to build the validation evidence for the OLIFE16. In particular, the OLIFE16 should be compared to other measures of Schizotypy, most notably, the Multidimensional Schizotypy Scale (MSS; Kwapil et al., 2018) and the Schizotypal Personality Scale (SPQ; Raine, 1991). In addition to other self-report measures, the OLIFE16 should also be considered along with behavioural measures known to be impaired with increased Schizotypy, for example, language (Rapp et al., 2010), processing speed (Hori et al., 2014), and verbal fluency (Cochrane et al., 2012). Evidence from both self-report and behavioural measures associated with Schizotypy would provide further validation evidence for the OLIFE16. The cross-cultural validity of the OLIFE16 should also be considered. While the original OLIFE was widely used and translated into many languages, the validity of the OLIFE16 in other cultures is unknown.

6.7 Conclusion

This thesis provides insight into SSD from both visual processing and psychometric perspectives and draws two broad conclusions. First, the presence of dorsal stream deficits in SSD, as measured by visually guided movement, is not as readily observed in subclinical populations as previously expected. While it is likely that dorsal stream deficits are important in building our understanding of SSD, their status as a *core feature* of the Schizophrenia spectrum is questioned in this thesis. Second, this thesis provides a psychometrically robust, brief measure of Schizotypy, the OLIFE16. The factor structure of the OLIFE16 is consistent with contemporary theories of Schizotypy with three latent dimensions that directly map onto the positive, negative, and disorganised dimensions of Schizophrenia. Overall, this thesis provides advancements in our understanding of the underlying processes in SSD with a specific focus on subclinical regions of the Schizophrenia spectrum.

References

Abbie, A. (2004). Cortical lamination in the Monotremata. *The Journal of Comparative Neurology*, *72*, 429–467. https://doi.org/10.1002/cne.900720302

Adjorlolo, S., Anum, A., & Adjorlolo, P. (2021). Latent structure of psychotic-like experiences in adolescents: Evidence from a multi-method taxometric study of a school-based sample in Ghana. *Psychiatry Research*, *302*, 113991. https://doi.org/10.1016/j.psychres.2021.113991

Aglioti, S., DeSouza, J. F. X., & Goodale, M. A. (1995). Size-contrast illusions deceive the eye but not the hand. *Current Biology*, *5*(6), 679–685. https://doi.org/10.1016/S0960-9822(95)00133-3

Ahmed, A. O., Green, B. A., Goodrum, N. M., Doane, N. J., Birgenheir, D., & Buckley, P. F. (2013). Does a latent class underlie schizotypal personality disorder? Implications for schizophrenia. *Journal of Abnormal Psychology*, *122*, 475–491. https://doi.org/10.1037/a0032713

Allardyce, J., Suppes, T., & van Os, J. (2007). Dimensions and the psychosis phenotype. *International Journal of Methods in Psychiatric Research*, *16*(Suppl 1), S34–S40. https://doi.org/10.1002/mpr.214

Allman, J. M., & Kaas, J. H. (1971). A representation of the visual field in the caudal third of the middle temporal gyrus of the owl monkey (Aotus trivirgatus). *Brain Research*, *31*(1), 85–105. https://doi.org/10.1016/0006-8993(71)90635-4

Alminhana, L. O., Sanseverino, M. A., Farias, M., dos Santos, O. V., Machado, W. D. L., & Claridge, G. (2020). A dimensional measure of schizotypy: Cross-cultural adaptation and validation of the Oxford-Liverpool Inventory of Feelings and Experiences short version for Brazilian Portuguese (O-LIFE-S). *Trends in Psychiatry and Psychotherapy*, *42*, 348–357. https://doi.org/10.1590/2237-6089-2019-0013

American Psychiatric Association. (1987). *Diagnostic and Statistical Manual of Mental Disorders (third ed.) revised* (3rd revised). American Psychiatric Association. https://doi.org/10.1176/appi.books.9780890420188.dsm-iii-r

American Psychiatric Association. (2013a). *Diagnostic and Statistical Manual of Mental Disorders (fifth ed.)* (5th ed.). American Psychiatric Association. https://doi.org/10.1176/appi.books.9780890425596

American Psychiatric Association. (2013b). *The Personality Inventory for DSM-5 (PID-5)—Adult*. https://www.psychiatry.org/File Library/Psychiatrists/Practice/DSM/APA_DSM5_The-Personality-Inventory-For-DSM-5-Full-Version-Adult.pdf

Andersen, R. A., & Buneo, C. A. (2002). Intentional Maps in Posterior Parietal Cortex. *Annual Review of Neuroscience*, *25*, 189–220. https://doi.org/10.1146/annurev.neuro.25.112701.142922

Anderson, J. L., Sellbom, M., & Salekin, R. T. (2018). Utility of the Personality Inventory for DSM-5–Brief Form (PID-5-BF) in the Measurement of Maladaptive Personality and Psychopathology. *Assessment*, *25*(5), 596–607. https://doi.org/10.1177/1073191116676889

Anunciação, L. (2018). An Overview of the History and Methodological Aspects of Psychometrics: History and Methodological aspects of Psychometrics. *Journal for ReAttach Therapy and Developmental Diversities*, *1*(1), Article 1. https://doi.org/10.26407/2018jrtdd.1.6

Arnold, S. E., & Trojanowski, J. Q. (1996). Recent advances in defining the neuropathology of schizophrenia. *Acta Neuropathologica*, *92*(3), 217–231. https://doi.org/10.1007/s004010050512

Bachmann, S., Degen, C., Geider, F. J., & Schröder, J. (2014). Neurological Soft Signs in the Clinical Course of Schizophrenia: Results of a Meta-Analysis. *Frontiers in Psychiatry*, *5,* Article 185. https://www.frontiersin.org/articles/10.3389/fpsyt.2014.00185

Barnow, S., Arens, E. A., Sieswerda, S., Dinu-Biringer, R., Spitzer, C., & Lang, S. (2010). Borderline Personality Disorder and Psychosis: A Review. *Current Psychiatry Reports*, *12*(3), 186–195. https://doi.org/10.1007/s11920-010-0107-9

Barrantes-Vidal, N., Gómez-de-Regil, L., Navarro, B., Vicens-Vilanova, J., Obiols, J., & Kwapil, T. (2013). Psychotic-like symptoms and positive schizotypy are associated with mixed and ambiguous handedness in an adolescent community sample. *Psychiatry Research*, *206*(2), 188–194. https://doi.org/10.1016/j.psychres.2012.12.008

Barrantes-Vidal, N., Grant, P., & Kwapil, T. (2015). The Role of Schizotypy in the Study of the Etiology of Schizophrenia Spectrum Disorders. *Schizophrenia Bulletin*, *41*(suppl 2), S408–S416. https://doi.org/10.1093/schbul/sbu191

Barrantes-Vidal, N., Ros-Morente, A., & Kwapil, T. (2009). An examination of neuroticism as a moderating factor in the association of positive and negative schizotypy with psychopathology in a nonclinical sample. *Schizophrenia Research*, *115*(2), 303–309. https://doi.org/10.1016/j.schres.2009.09.021

Basso, M. R., Nasrallah, H. A., Olson, S. C., & Bornstein, R. A. (1998). Neuropsychological correlates of negative, disorganized and psychotic symptoms in schizophrenia. *Schizophrenia Research*, *31*(2), 99–111. https://doi.org/10.1016/S0920-9964(98)00023-1

Batey, M., & Furnham, A. (2008). The relationship between measures of creativity and schizotypy. *Personality and Individual Differences*, *45*(8), 816–821. https://doi.org/10.1016/j.paid.2008.08.014

Batty, R. A., Francis, A. J. P., Innes-Brown, H., Joshua, N. R., & Rossell, S. L. (2014). Neurophysiological Correlates of Configural Face Processing in Schizotypy. *Frontiers* *in Psychiatry*, *5*, Article 101. https://www.frontiersin.org/articles/10.3389/fpsyt.2014.00101

Beauchamp, M. S., Lee, K. E., Haxby, J. V., & Martin, A. (2002). Parallel Visual Motion Processing Streams for Manipulable Objects and Human Movements. *Neuron*, *34*(1), 149–159. https://doi.org/10.1016/S0896-6273(02)00642-6

Becker, D. F., Grilo, C. M., Edell, W. S., & McGlashan, T. H. (2000). Comorbidity of Borderline Personality Disorder With Other Personality Disorders in Hospitalized Adolescents and Adults. *American Journal of Psychiatry*, *157*(12), 2011–2016. https://doi.org/10.1176/appi.ajp.157.12.2011

Bedwell, J. S., Chan, C. C., Trachik, B. J., & Rassovsky, Y. (2013). Changes in the visual-evoked P1 potential as a function of schizotypy and background color in healthy young adults. *Journal of Psychiatric Research*, *47*(4), 542–547. https://doi.org/10.1016/j.jpsychires.2012.12.012

Begliomini, C., Wall, M. B., Smith, A. T., & Castiello, U. (2007). Differential cortical activity for precision and whole-hand visually guided grasping in humans. *European Journal of Neuroscience*, *25*(4), 1245–1252. https://doi.org/10.1111/j.1460-9568.2007.05365.x

Bell, V., & Halligan, P. (2015). Schizotypy and impaired basic face recognition? Another non-confirmatory study. *Early Intervention in Psychiatry*, *9*(6), 512–515. https://doi.org/10.1111/eip.12181

Bentall, R. P., Claridge, G. S., & Slade, P. D. (1989). The multidimensional nature of schizotypal traits: A factor analytic study with normal subjects. *British Journal of Clinical Psychology*, *28*(4), 363–375. https://doi.org/10.1111/j.2044-8260.1989.tb00840.x

Berna, F., Misdrahi, D., Boyer, L., Aouizerate, B., Brunel, L., Capdevielle, D., Chereau, I., Danion, J. M., Dorey, J. M., Dubertret, C., Dubreucq, J., Faget, C., Gabayet, F., Lancon, C., Mallet, J., Rey, R., Passerieux, C., Schandrin, A., Schurhoff, F., … Zinetti-Bertschy, A. (2015). Akathisia: Prevalence and risk factors in a communitydwelling sample of patients with schizophrenia. Results from the FACE-SZ dataset. *Schizophrenia Research*, *169*(1), 255–261. https://doi.org/10.1016/j.schres.2015.10.040

Bitter, R., Mohiuddin, T., & Nawrocki, M. (2006). *LabVIEW: Advanced programming techniques*. National Instruments.

Bleuler, E. (1950). *Dementia praecox or the group of schizophrenias* (548 pages). International Universities Press.

Bosco, A., Breveglieri, R., Chinellato, E., Galletti, C., & Fattori, P. (2010). Reaching Activity in the Medial Posterior Parietal Cortex of Monkeys Is Modulated by Visual Feedback. *Journal of Neuroscience*, *30*(44), 14773–14785. https://doi.org/10.1523/JNEUROSCI.2313-10.2010

Boyle, G. J. (1991). Does item homogeneity indicate internal consistency or item redundancy in psychometric scales? *Personality and Individual Differences*, *12*(3), 291–294. https://doi.org/10.1016/0191-8869(91)90115-R

Brenner, C. A., Wilt, M., Lysaker, P., Koyfman, A., & O'Donnell, B. F. (2003). Psychometrically Matched Visual-Processing Tasks in Schizophrenia Spectrum Disorders. *Journal of Abnormal Psychology*, *112*(1), 28–37. https://doi.org/10.1037/0021-843X.112.1.28

Breveglieri, R., Borgomaneri, S., Filippini, M., Tessari, A., Galletti, C., Davare, M., & Fattori, P. (2022). Complementary contribution of the medial and lateral human parietal cortex to grasping: A repetitive TMS study. *Cerebral Cortex*, *1*, 13. https://doi.org/10.1093/cercor/bhac404

Breveglieri, R., Bosco, A., Galletti, C., Passarelli, L., & Fattori, P. (2016). Neural activity in the medial parietal area V6A while grasping with or without visual feedback. *Scientific Reports*, *6*(1), Article 1. https://doi.org/10.1038/srep28893

Breveglieri, R., De Vitis, M., Bosco, A., Galletti, C., & Fattori, P. (2018). Interplay Between Grip and Vision in the Monkey Medial Parietal Lobe. *Cerebral Cortex*, *28*(6), 2028–2042. https://doi.org/10.1093/cercor/bhx109

Broca, P. (2006). Comments Regarding the Seat of the Faculty of Spoken Language, Followed by an Observation of Aphemia (Loss of Speech) (1861). In Y. Grodzinsky & K. Amunts (Eds.), *Broca's Region*. Oxford University Press. https://doi.org/10.1093/acprof:oso/9780195177640.003.0018

Brochier, T., & Umiltà, M. A. (2007). Cortical control of grasp in non-human primates. *Current Opinion in Neurobiology*, *17*(6), 637–643. https://doi.org/10.1016/j.conb.2007.12.002

Bromet, E. J., Kotov, R., Fochtmann, L. J., Carlson, G. A., Tanenberg-Karant, M., Ruggero, C., & Chang, S. (2011). Diagnostic shifts during the decade following first admission for psychosis. *The American Journal of Psychiatry*, *168*(11), 1186–1194. https://doi.org/10.1176/appi.ajp.2011.11010048

Brüne, M. (2004). Schizophrenia—An evolutionary enigma? *Neuroscience & Biobehavioral Reviews*, *28*(1), 41–53. https://doi.org/10.1016/j.neubiorev.2003.10.002

Bruno, N., Bernardis, P., & Gentilucci, M. (2008). Visually guided pointing, the Müller-Lyer illusion, and the functional interpretation of the dorsal-ventral split: Conclusions from 33 independent studies. *Neuroscience & Biobehavioral Reviews*, *32*(3), 423–437. https://doi.org/10.1016/j.neubiorev.2007.08.006

Bruno, N., & Franz, V. H. (2009). When is grasping affected by the Müller-Lyer illusion?: A quantitative review. *Neuropsychologia*, *47*(6), 1421–1433. https://doi.org/10.1016/j.neuropsychologia.2008.10.031

Budisavljevic, S., Dell'Acqua, F., & Castiello, U. (2018). Cross-talk connections underlying dorsal and ventral stream integration during hand actions. *Cortex*, *103*, 224–239. https://doi.org/10.1016/j.cortex.2018.02.016

Burch, G. J., & Foo, G. (2010). Schizotypal and dependent personality characteristics and managerial performance. *Australian Psychologist*, *45*(4), 290–298. https://doi.org/10.1080/00050061003664803

Burch, G. S. J., Hemsley, D. R., Corr, P. J., & Gwyer, P. (2006). The relationship between incidental learning and multi-dimensional schizotypy as measured by the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE). *Personality and Individual Differences*, *40*(2), 385–394. https://doi.org/10.1016/j.paid.2005.07.010

Burch, G. S. J., Pavelis, C., Hemsley, D. R., & Corr, P. J. (2006). Schizotypy and creativity in visual artists. *British Journal of Psychology*, *97*(2), 177–190. https://doi.org/10.1348/000712605X60030

Burton, B. K., Hjorthøj, C., Jepsen, J. R., Thorup, A., Nordentoft, M., & Plessen, K. J. (2016). Research Review: Do motor deficits during development represent an endophenotype for schizophrenia? A meta-analysis. *Journal of Child Psychology and Psychiatry*, *57*(4), 446–456. https://doi.org/10.1111/jcpp.12479

Busan, P., Monti, F., Semenic, M., Pizzolato, G., & Battaglini, P. P. (2009). Parieto-occipital cortex and planning of reaching movements: A transcranial magnetic stimulation study. *Behavioural Brain Research*, *201*(1), 112–119. https://doi.org/10.1016/j.bbr.2009.01.040

Butler, P. D., & Javitt, D. C. (2005). Early-stage visual processing deficits in schizophrenia. *Current Opinion in Psychiatry*, *18*(2), 151–157. https://doi.org/ 10.1097/00001504-200503000-00008

Butler, P. D., Schechter, I., Zemon, V., Schwartz, S. G., Greenstein, V. C., Gordon, J., Schroeder, C. E., & Javitt, D. C. (2001). Dysfunction of Early-Stage Visual Processing in Schizophrenia. *American Journal of Psychiatry*, *158*(7), 1126–1133. https://doi.org/10.1176/appi.ajp.158.7.1126

Cadenhead, K. S., Serper, Y., & Braff, D. L. (1998). Transient versus sustained visual channels in the visual backward masking deficits of schizophrenia patients. *Biological Psychiatry*, *43*(2), 132–138. https://doi.org/10.1016/S0006-3223(97)00316-8

Camisa, K. M., Bockbrader, M. A., Lysaker, P., Rae, L. L., Brenner, C. A., & O'Donnell, B. F. (2005). Personality traits in schizophrenia and related personality disorders. *Psychiatry Research*, *133*(1), 23–33. https://doi.org/10.1016/j.psychres.2004.09.002

Cappe, C., Herzog, M. H., Herzig, D. A., Brand, A., & Mohr, C. (2012). Cognitive disorganisation in schizotypy is associated with deterioration in visual backward masking. *Psychiatry Research*, *200*(2), 652–659. https://doi.org/10.1016/j.psychres.2012.07.001

Capra, C., Kavanagh, D. J., Hides, L., & Scott, J. G. (2017). Current CAPE-15: A measure of recent psychotic-like experiences and associated distress. *Early Intervention in Psychiatry*, *11*(5), 411–417. https://doi.org/10.1111/eip.12245

Carlin, J. L., & Lindell, A. K. (2015). Schizotypal traits do not influence behavioral asymmetries for language processing in the non-clinical population. *Personality and Individual Differences*, *85*, 25–29. https://doi.org/10.1016/j.paid.2015.04.037

Cavina-Pratesi, C., Connolly, J. D., Monaco, S., Figley, T. D., Milner, A. D., Schenk, T., & Culham, J. C. (2018). Human neuroimaging reveals the subcomponents of grasping, reaching and pointing actions. *Cortex*, *98*, 128–148. https://doi.org/10.1016/j.cortex.2017.05.018

Cavina-Pratesi, C., Connolly, J., & Milner, A. (2013). Optic ataxia as a model to investigate the role of the posterior parietal cortex in visually guided action: Evidence from studies of patient M.H. *Frontiers in Human Neuroscience*, *7*, 336 https://www.frontiersin.org/articles/10.3389/fnhum.2013.00336

Cavina-Pratesi, C., Goodale, M. A., & Culham, J. C. (2007). FMRI Reveals a Dissociation between Grasping and Perceiving the Size of Real 3D Objects. *PLOS ONE*, *2*(5), e424. https://doi.org/10.1371/journal.pone.0000424

Cella, M., Serra, M., Lai, A., Mason, O. J., Sisti, D., Rocchi, M. B. L., Preti, A., & Petretto, D. R. (2013). Schizotypal traits in adolescents: Links to family history of psychosis and psychological distress. *European Psychiatry*, *28*(4), 247–253. https://doi.org/10.1016/j.eurpsy.2012.04.002

Chapman, L. J., & Chapman, J. P. (1973). Problems in the measurement of cognitive deficits. *Psychological Bulletin*, *79*, 380–385. https://doi.org/10.1037/h0034541

Chapman, L. J., Chapman, J. P., Kwapil, T. R., Eckblad, M., & Zinser, M. C. (1994). Putatively psychosis-prone subjects 10 years later. *Journal of Abnormal Psychology*, *103*(2), 171–183. https://doi.org/10.1037/0021-843X.103.2.171

Chapman, L. J., Chapman, J. P., & Raulin, M. L. (1976). Scales for physical and social anhedonia. *Journal of Abnormal Psychology*, *85*(4), 374–382. https://doi.org/10.1037//0021-843x.85.4.374

Chapman, L. J., Edell, W. S., & Chapman, J. P. (1980). Physical anhedonia, perceptual aberration, and psychosis proneness. *Schizophrenia Bulletin*, *6*(4), 639–653. https://doi.org/10.1093/schbul/6.4.639

Cheli, S. (2023). An evolutionary look at oddity and schizotypy: How the rise of social brain informs clinical practice. *New Ideas in Psychology*, *68*, 100993. https://doi.org/10.1016/j.newideapsych.2022.100993

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*(2), 155–161. https://doi.org/10.1001/archpsyc.56.2.155

Chen, Y., Palafox, G. P., Nakayama, K., Levy, D. L., Matthysse, S., & Holzman, P. S. (1999). Motion Perception in Schizophrenia. *Archives of General Psychiatry*, *56*(2), 149–154. https://doi.org/10.1001/archpsyc.56.2.149

Child, D. (2006). *The Essentials of Factor Analysis*. A&C Black.

Christensen, B. K., & Bilder, R. M. (2000). Dual Cytoarchitectonic Trends: An Evolutionary Model of Frontal Lobe Functioning and Its Application to Psychopathology. *The Canadian Journal of Psychiatry*, *45*(3), 247–256. https://doi.org/10.1177/070674370004500303

Ciavarro, M., Ambrosini, E., Tosoni, A., Committeri, G., Fattori, P., & Galletti, C. (2013). RTMS of Medial Parieto-occipital Cortex Interferes with Attentional Reorienting during Attention and Reaching Tasks. *Journal of Cognitive Neuroscience*, *25*(9), 1453–1462. https://doi.org/10.1162/jocn_a_00409

Claridge, G., & Beech, T. (1995). Fully and quasi-dimensional constructions of schizotypy. In A. Raine, T. Lencz, & S. A. Mednick (Eds.), *Schizotypal personality* (pp. 192–216). Cambridge University Press. https://doi.org/10.1017/CBO9780511759031.010.

Claridge, G., & Broks, P. (1984). Schizotypy and hemisphere function – I: Theoretical considerations and the measurement of schizotypy. *Personality and Individual Differences*, *5*(6), 633–648. https://doi.org/10.1016/0191-8869(84)90111-9

Claridge, G., & Davis, C. (2002). *Personality and Psychological Disorders*. Routledge. https://doi.org/10.4324/9780203766309

Claridge, G., Robinson, D. L., & Birchall, P. (1983). Characteristics of schizophrenics' and neurotics' relatives. *Personality and Individual Differences*, *4*(6), 651–664. https://doi.org/10.1016/0191-8869(83)90119-8

Cochrane, M., Petch, I., & Pickering, A. D. (2010). Do measures of schizotypal personality provide non-clinical analogues of schizophrenic symptomatology? *Psychiatry Research*, *176*(2), 150–154. https://doi.org/10.1016/j.psychres.2009.01.031

Cochrane, M., Petch, I., & Pickering, A. D. (2012). Aspects of cognitive functioning in schizotypy and schizophrenia: Evidence for a continuum model. *Psychiatry Research*, *196*(2), 230–234. https://doi.org/10.1016/j.psychres.2012.02.010

Cohen, A. S., Mohr, C., Ettinger, U., Chan, R. C. K., & Park, S. (2015). Schizotypy as An Organizing Framework for Social and Affective Sciences. *Schizophrenia Bulletin*, *41*(suppl 2), S427–S435. https://doi.org/10.1093/schbul/sbu195

Cohen, N. R., Cross, E. S., Tunik, E., Grafton, S. T., & Culham, J. C. (2009). Ventral and dorsal stream contributions to the online control of immediate and delayed grasping: A TMS approach. *Neuropsychologia*, *47*(6), 1553–1562. https://doi.org/10.1016/j.neuropsychologia.2008.12.034

Cooper, A. J., Smillie, L. D., & Corr, P. J. (2010). A confirmatory factor analysis of the Mini-IPIP five-factor model personality scale. *Personality and Individual Differences*, *48*(5), 688–691. https://doi.org/10.1016/j.paid.2010.01.004

Cornblatt, B. A., & Keilp, J. G. (1994). Impaired Attention, Genetics, and the Pathophysiology of Schizophrenia. *Schizophrenia Bulletin*, *20*(1), 31–46. https://doi.org/10.1093/schbul/20.1.31

Coste, J., Guillemin, F., Pouchot, J., & Fermanian, J. (1997). Methodological approaches to shortening composite measurement scales. *Journal of Clinical Epidemiology*, *50*(3), 247–252. https://doi.org/10.1016/S0895-4356(96)00363-0

Coste, J., Walter, E., & Venot, A. (1995). A new approach to selection and weighting of items in evaluative composite measurement scales. *Statistics in Medicine*, *14*(23), 2565–2580. https://doi.org/10.1002/sim.4780142306

Courtney, S. M., & Ungerleider, L. (1997). What fMRI has taught us about human vision. *Current Opinion in Neurobiology*, *7*(4), 554–561. https://doi.org/10.1016/S0959-4388(97)80036-0

Craig, K. (2017). The History of Psychometrics. In *Psychometric Testing* (pp. 1–14). John Wiley & Sons, Ltd. https://doi.org/10.1002/9781119183020.ch1

Cuesta, M. J., Sánchez-Torres, A. M., de Jalón, E. G., Campos, M. S., Ibáñez, B., Moreno-Izco, L., & Peralta, V. (2014). Spontaneous Parkinsonism Is Associated With Cognitive Impairment in Antipsychotic-Naive Patients With First-Episode Psychosis: A 6-Month Follow-up Study. *Schizophrenia Bulletin*, *40*(5), 1164–1173. https://doi.org/10.1093/schbul/sbt125

Cuesta, M. J., Ugarte, M. D., Goicoa, T., Eraso, S., & Peralta, V. (2007). A taxometric analysis of schizophrenia symptoms. *Psychiatry Research*, *150*(3), 245–253. https://doi.org/10.1016/j.psychres.2006.01.019

Culham, J. C., Danckert, S. L., Souza, J. F. X. D., Gati, J. S., Menon, R. S., & Goodale, M. A. (2003). Visually guided grasping produces fMRI activation in dorsal but not ventral stream brain areas. *Experimental Brain Research*, *153*(2), 180–189. https://doi.org/10.1007/s00221-003-1591-5

da Cruz, J. R., Shaqiri, A., Roinishvili, M., Favrod, O., Chkonia, E., Brand, A., Figueiredo, P., & Herzog, M. H. (2020). Neural Compensation Mechanisms of Siblings of Schizophrenia Patients as Revealed by High-Density EEG. *Schizophrenia Bulletin*, *46*(4), 1009–1018. https://doi.org/10.1093/schbul/sbz133

Dagnall, N., Denovan, A., Drinkwater, K., Parker, A., & Clough, P. (2016). Toward a Better Understanding of the Relationship between Belief in the Paranormal and Statistical Bias: The Potential Role of Schizotypy. *Frontiers in Psychology*, *7*, 1045. https://www.frontiersin.org/articles/10.3389/fpsyg.2016.01045

Danckert, J. A., Sharif, N., Haffenden, A. M., Schiff, K. C., & Goodale, M. A. (2002). A temporal analysis of grasping in the Ebbinghaus illusion: Planning versus online control. *Experimental Brain Research*, *144*(2), 275–280. https://doi.org/10.1007/s00221-002-1073-1

Dang, J., King, K. M., & Inzlicht, M. (2020). Why Are Self-Report and Behavioral Measures Weakly Correlated? *Trends in Cognitive Sciences*, *24*(4), 267–269. https://doi.org/10.1016/j.tics.2020.01.007

Davare, M., Andres, M., Cosnard, G., Thonnard, J.L., & Olivier, E. (2006). Dissociating the Role of Ventral and Dorsal Premotor Cortex in Precision Grasping. *Journal of Neuroscience*, *26*(8), 2260–2268. https://doi.org/10.1523/JNEUROSCI.3386-05.2006

Dazzan, P., & Murray, R. M. (2002). Neurological soft signs in first-episode psychosis: A systematic review. *The British Journal of Psychiatry*, *181*(S43), s50–s57. https://doi.org/10.1192/bjp.181.43.s50

Debbané, M., & Barrantes-Vidal, N. (2015). Schizotypy From a Developmental Perspective. *Schizophrenia Bulletin*, *41*(Suppl 2), S386–S395. https://doi.org/10.1093/schbul/sbu175

Del Fabro, L., Schmidt, A., Fortea, L., Delvecchio, G., D'Agostino, A., Radua, J., Borgwardt, S., & Brambilla, P. (2021). Functional brain network dysfunctions in subjects at high-risk for psychosis: A meta-analysis of resting-state functional connectivity. *Neuroscience & Biobehavioral Reviews*, *128*, 90–101. https://doi.org/10.1016/j.neubiorev.2021.06.020

Desmurget, M., Epstein, C. M., Turner, R. S., Prablanc, C., Alexander, G. E., & Grafton, S. T. (1999). Role of the posterior parietal cortex in updating reaching movements to a visual target. *Nature Neuroscience*, *2*(6), Article 6. https://doi.org/10.1038/9219

Doniger, G. M., Foxe, J. J., Murray, M. M., Higgins, B. A., & Javitt, D. C. (2002). Impaired Visual Object Recognition and Dorsal/Ventral Stream Interaction in Schizophrenia. *Archives of General Psychiatry*, *59*(11), 1011–1020. https://doi.org/10.1001/archpsyc.59.11.1011

Donnellan, M. B., Oswald, F. L., Baird, B. M., & Lucas, R. E. (2006). The Mini-IPIP Scales: Tiny-yet-effective measures of the Big Five Factors of Personality. *Psychological Assessment*, *18*(2), 192–203. https://doi.org/10.1037/1040-3590.18.2.192

Dubrowski, A., Bock, O., Carnahan, H., & Jüngling, S. (2002). The coordination of hand transport and grasp formation during single- and double-perturbed human prehension movements. *Experimental Brain Research*, *145*(3), 365–371. https://doi.org/10.1007/s00221-002-1120-y

Dunn, T. J., Baguley, T., & Brunsden, V. (2014). From alpha to omega: A practical solution to the pervasive problem of internal consistency estimation. *British Journal of Psychology*, *105*(3), 399–412. https://doi.org/10.1111/bjop.12046

Eckblad, M., & Chapman, L. J. (1983). Magical ideation as an indicator of schizotypy. *Journal of Consulting and Clinical Psychology*, *51*(2), 215–225. https://doi.org/10.1037/0022-006X.51.2.215

Eckblad, M., & Chapman, L. J. (1986). Development and validation of a scale for hypomanic personality. *Journal of Abnormal Psychology*, *95*(3), 214–222. https://doi.org/10.1037/0021-843X.95.3.214

Ehrsson, H. H., Fagergren, A., Jonsson, T., Westling, G., Johansson, R. S., & Forssberg, H. (2000). Cortical Activity in Precision- Versus Power-Grip Tasks: An fMRI Study. *Journal of Neurophysiology*, *83*(1), 528–536. https://doi.org/10.1152/jn.2000.83.1.528

Elahipanah, A. (2013). *Visual Attention among Patients with Schizophrenia: A Study of Visual Span and Selectivity in Visual Search* [Doctoral Dissertation, University of Toronto]. https://central.bac-lac.gc.ca/.item?id=TC-OTU-43553&op=pdf&app=Library&is_thesis=1&oclc_number=1032918595

Elahipanah, A., Christensen, B. K., & Reingold, E. M. (2011). Controlling the spotlight of attention: Visual span size and flexibility in schizophrenia. *Neuropsychologia*, *49*(12), 3370–3376. https://doi.org/10.1016/j.neuropsychologia.2011.08.011

Elliott, D., Helsen, W. F., & Chua, R. (2001). A century later: Woodworth's (1899) two-component model of goal-directed aiming. *Psychological Bulletin*, *127*, 342–357. https://doi.org/10.1037/0033-2909.127.3.342

Ellison, A., & Cowey, A. (2006). TMS can reveal contrasting functions of the dorsal and ventral visual processing streams. *Experimental Brain Research*, *175*(4), 618–625. https://doi.org/10.1007/s00221-006-0582-8

Erickson, K. M., & van Kan, P. L. (2005). Kinematics of reach-to-grasp movements in subjects with cerebellar deficits. *Journal of Neurologic Physical Therapy*, *29*(4), 206. https://doi.org/10.1097/01.NPT.0000282376.57194.f2

Evans, D. W., Lusk, L. G., Slane, M. M., Michael, A. M., Myers, S. M., Uljarević, M., Mason, O., Claridge, G., & Frazier, T. (2018). Dimensional assessment of schizotypal, psychotic, and other psychiatric traits in children and their parents: Development and validation of the Childhood Oxford-Liverpool Inventory of Feelings and Experiences on a representative US sample. *Journal of Child Psychology and Psychiatry*, *59*(5), 574–585. https://doi.org/10.1111/jcpp.12827

Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck personality questionnaire (junior and adult)*. Hodder and Stoughton.

Farias, M., Underwood, R., & Claridge, G. (2013). Unusual but sound minds: Mental health indicators in spiritual individuals. *British Journal of Psychology*, *104*(3), 364–381. https://doi.org/10.1111/j.2044-8295.2012.02128.x

Fattori, P., Breveglieri, R., Amoroso, K., & Galletti, C. (2004). Evidence for both reaching and grasping activity in the medial parieto-occipital cortex of the macaque. *European Journal of Neuroscience*, *20*(9), 2457–2466. https://doi.org/10.1111/j.1460-9568.2004.03697.x

Fattori, P., Kutz, D. F., Breveglieri, R., Marzocchi, N., & Galletti, C. (2005). Spatial tuning of reaching activity in the medial parieto-occipital cortex (area V6A) of macaque monkey. *European Journal of Neuroscience*, *22*(4), 956–972. https://doi.org/10.1111/j.1460-9568.2005.04288.x

Fattori, P., Raos, V., Breveglieri, R., Bosco, A., Marzocchi, N., & Galletti, C. (2010). The Dorsomedial Pathway Is Not Just for Reaching: Grasping Neurons in the Medial Parieto-Occipital Cortex of the Macaque Monkey. *Journal of Neuroscience*, *30*(1), 342–349. https://doi.org/10.1523/JNEUROSCI.3800-09.2010

Faugier-Grimaud, S., Frenois, C., & Stein, D. G. (1978). Effects of posterior parietal lesions on visually guided behavior in monkeys. *Neuropsychologia*, *16*(2), 151–168. https://doi.org/10.1016/0028-3932(78)90103-3

Fenton, W. S. (2000). Prevalence of spontaneous dyskinesia in schizophrenia. *The Journal of Clinical Psychiatry*, *61 Supplementary 4*, 10–14. https://doi.org/ 10.1192/bjp.171.3.265

Filimon, F. (2010). Human Cortical Control of Hand Movements: Parietofrontal Networks for Reaching, Grasping, and Pointing. *The Neuroscientist*, *16*(4), 388–407. https://doi.org/10.1177/1073858410375468

Fink, A., Weber, B., Koschutnig, K., Benedek, M., Reishofer, G., Ebner, F., Papousek, I., & Weiss, E. M. (2014). Creativity and schizotypy from the neuroscience perspective. *Cognitive, Affective, & Behavioral Neuroscience*, *14*(1), 378–387. https://doi.org/10.3758/s13415-013-0210-6

Fluet, M.C., Baumann, M. A., & Scherberger, H. (2010). Context-Specific Grasp Movement Representation in Macaque Ventral Premotor Cortex. *Journal of Neuroscience*, *30*(45), 15175–15184. https://doi.org/10.1523/JNEUROSCI.3343-10.2010

Fonseca-Pedrero, E., Chan, R. C. K., Debbané, M., Cicero, D., Zhang, L. C., Brenner, C., Barkus, E., Linscott, R. J., Kwapil, T., Barrantes-Vidal, N., Cohen, A., Raine, A., Compton, M. T., Tone, E. B., Suhr, J., Muñiz, J., de Albéniz, A. P., Fumero, A., Giakoumaki, S., … Ortuño-Sierra, J. (2018). Comparisons of schizotypal traits across

12 countries: Results from the International Consortium for Schizotypy Research. *Schizophrenia Research*, *199*, 128–134. https://doi.org/10.1016/j.schres.2018.03.021

Fonseca-Pedrero, E., Ortuño-Sierra, J., Mason, O. J., & Muñiz, J. (2015). The Oxford–Liverpool Inventory of Feelings and Experiences short version: Further validation. *Personality and Individual Differences*, *86*, 338–343. https://doi.org/10.1016/j.paid.2015.06.041

Fonseca-Pedrero, E., Ortuño-Sierra, J., Sierro, G., Daniel, C., Cella, M., Preti, A., Mohr, C., & Mason, O. J. (2015). The Measurement Invariance of Schizotypy in Europe. *European Psychiatry*, *30*(7), 837–844. https://doi.org/10.1016/j.eurpsy.2015.07.005

Foulds, G. A., & Bedford, A. (1975). Hierarchy of classes of personal illness. *Psychological Medicine*, *5*(2), 181–192. https://doi.org/10.1017/S0033291700056452

Franz, V. H. (2003). Manual size estimation: A neuropsychological measure of perception? *Experimental Brain Research*, *151*(4), 471–477. https://doi.org/10.1007/s00221-003-1477-6

Franz, V. H., Bülthoff, H. H., & Fahle, M. (2003). Grasp effects of the Ebbinghaus illusion: Obstacle avoidance is not the explanation. *Experimental Brain Research*, *149*(4), 470–477. https://doi.org/10.1007/s00221-002-1364-6

Franz, V. H., & Gegenfurtner, K. R. (2008). Grasping visual illusions: Consistent data and no dissociation. *Cognitive Neuropsychology*, *25*(7–8), 920–950. https://doi.org/10.1080/02643290701862449

Franz, V. H., Gegenfurtner, K. R., Bülthoff, H. H., & Fahle, M. (2000). Grasping Visual Illusions: No Evidence for a Dissociation Between Perception and Action. *Psychological Science*, *11*(1), 20–25. https://doi.org/10.1111/1467-9280.00209

Galletti, C., & Fattori, P. (2018). The dorsal visual stream revisited: Stable circuits or dynamic pathways? *Cortex*, *98*, 203–217. https://doi.org/10.1016/j.cortex.2017.01.009

Galletti, C., Kutz, D. F., Gamberini, M., Breveglieri, R., & Fattori, P. (2003). Role of the medial parieto-occipital cortex in the control of reaching and grasping movements. *Experimental Brain Research*, *153*(2), 158–170. https://doi.org/10.1007/s00221-003-1589-z

Gallivan, J. P., McLean, D. A., Valyear, K. F., Pettypiece, C. E., & Culham, J. C. (2011). Decoding Action Intentions from Preparatory Brain Activity in Human Parieto-Frontal Networks. *Journal of Neuroscience*, *31*(26), 9599–9610. https://doi.org/10.1523/JNEUROSCI.0080-11.2011

Ganel, T., Chajut, E., & Algom, D. (2008). Visual coding for action violates fundamental psychophysical principles. *Current Biology*, *18*(14), R599–R601. https://doi.org/10.1016/j.cub.2008.04.052

Gao, K., Kemp, D. E., Ganocy, S. J., Gajwani, P., Xia, G., & Calabrese, J. R. (2008). Antipsychotic-Induced Extrapyramidal Side Effects in Bipolar Disorder and Schizophrenia. *Journal of Clinical Psychopharmacology*, *28*(2), 203–209. https://doi.org/10.1097/JCP.0b013e318166c4d5

Giaccio, R. G. (2006). The dual origin hypothesis: An evolutionary brain-behavior framework for analyzing psychiatric disorders. *Neuroscience & Biobehavioral Reviews*, *30*(4), 526–550. https://doi.org/10.1016/j.neubiorev.2005.04.021

Girard, T. A., Axelrod, B. N., Patel, R., & Crawford, J. R. (2015). Wechsler Adult Intelligence Scale–IV Dyads for Estimating Global Intelligence. *Assessment*, *22*(4), 441–448. https://doi.org/10.1177/1073191114551551

Goetz, C., Coste, J., Lemetayer, F., Rat, A.C., Montel, S., Recchia, S., Debouverie, M., Pouchot, J., Spitz, E., & Guillemin, F. (2013). Item reduction based on rigorous methodological guidelines is necessary to maintain validity when shortening composite measurement scales. *Journal of Clinical Epidemiology*, *66*(7), 710–718. https://doi.org/10.1016/j.jclinepi.2012.12.015

Goldberg, G. (1985). Supplementary motor area structure and function: Review and hypotheses. *Behavioral and Brain Sciences*, *8*, 567–588. https://doi.org/10.1017/S0140525X00045167

Golden, R. R., & Meehl, P. E. (1979). Detection of the schizoid taxon with MMPI indicators. *Journal of Abnormal Psychology*, *88*(3), 217–233. https://doi.org/10.1037/0021-843X.88.3.217

Goodale, M. A., Meenan, J. P., Bülthoff, H. H., Nicolle, D. A., Murphy, K. J., & Racicot, C. I. (1994a). Separate neural pathways for the visual analysis of object shape in perception and prehension. *Current Biology*, *4*(7), 604–610. https://doi.org/10.1016/S0960-9822(00)00132-9

Goodale, M. A., Meenan, J. P., Bülthoff, H. H., Nicolle, D. A., Murphy, K. J., & Racicot, C. I. (1994b). Separate neural pathways for the visual analysis of object shape in perception and prehension. *Current Biology*, *4*(7), 604–610. https://doi.org/10.1016/S0960-9822(00)00132-9

Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. *Trends in Neurosciences*, *15*(1), 20–25. https://doi.org/10.1016/0166-2236(92)90344-8

Goodale, M. A., Milner, A. D., Jakobson, L. S., & Carey, D. P. (1991). A neurological dissociation between perceiving objects and grasping them. *Nature*, *349*(6305), Article 6305. https://doi.org/10.1038/349154a0

Goodale, M. A., Pelisson, D., & Prablanc, C. (1986). Large adjustments in visually guided reaching do not depend on vision of the hand or perception of target displacement. *Nature*, *320*(6064), Article 6064. https://doi.org/10.1038/320748a0

Goulding, A. (2004). Schizotypy models in relation to subjective health and paranormal beliefs and experiences. *Personality and Individual Differences*, *37*(1), 157–167. https://doi.org/10.1016/j.paid.2003.08.008

Granger, K. T., Prados, J., & Young, A. M. J. (2012). Disruption of overshadowing and latent inhibition in high schizotypy individuals. *Behavioural Brain Research*, *233*(1), 201–208. https://doi.org/10.1016/j.bbr.2012.05.003

Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., Smith, S. M., Dawson, D. A., Pulay, A. J., & Pickering, R. P. (2008). Prevalence, Correlates, Disability, and Comorbidity of DSM-IV Borderline Personality Disorder: Results From the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *The Journal of Clinical Psychiatry*, *69*(4), 15398.

Grant, P., Green, M. J., & Mason, O. J. (2018). Models of Schizotypy: The Importance of Conceptual Clarity. *Schizophrenia Bulletin*, *44*(Suppl 2), S556–S563. https://doi.org/10.1093/schbul/sby012

Grant, P., Kuepper, Y., Mueller, E., Wielpuetz, C., Mason, O., & Hennig, J. (2013). Dopaminergic foundations of schizotypy as measured by the German version of the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE)—A suitable endophenotype of schizophrenia. *Frontiers in Human Neuroscience*, *7*. https://www.frontiersin.org/articles/10.3389/fnhum.2013.00001

Grol, M. J., Majdandžić, J., Stephan, K. E., Verhagen, L., Dijkerman, H. C., Bekkering, H., Verstraten, F. A. J., & Toni, I. (2007). Parieto-Frontal Connectivity during Visually Guided Grasping. *Journal of Neuroscience*, *27*(44), 11877–11887. https://doi.org/10.1523/JNEUROSCI.3923-07.2007

Gross, G. M., Kwapil, T. R., Raulin, M. L., Silvia, P. J., & Barrantes-Vidal, N. (2018). The multidimensional schizotypy scale-brief: Scale development and psychometric properties. *Psychiatry Research*, *261*, 7–13. https://doi.org/10.1016/j.psychres.2017.12.033

Gunderson, J. G., & Singer, M. T. (1975). Defining borderline patients: An overview. *The American Journal of Psychiatry*, *132*, 1–10. https://doi.org/10.1176/ajp.132.1.1

Haffenden, A. M., & Goodale, M. A. (1998). The Effect of Pictorial Illusion on Prehension and Perception. *Journal of Cognitive Neuroscience*, *10*(1), 122–136. https://doi.org/10.1162/089892998563824

Hanel, P. H. P., Demmrich, S., & Wolfradt, U. (2019). Centrality of Religiosity, Schizotypy, and Human Values: The Impact of Religious Affiliation. *Religions*, *10*(5), Article 5. https://doi.org/10.3390/rel10050297

Hanssen, M., Bak, M., Bijl, R., Vollebergh, W., & Van Os, J. (2005). The incidence and outcome of subclinical psychotic experiences in the general population. *British Journal of Clinical Psychology*, *44*(2), 181–191. https://doi.org/10.1348/014466505X29611

Hao, Y., Zhang, Q., Controzzi, M., Cipriani, C., Li, Y., Li, J., Zhang, S., Wang, Y., Chen, W., Carrozza, M. C., & Zheng, X. (2014). Distinct neural patterns enable grasp types decoding in monkey dorsal premotor cortex. *Journal of Neural Engineering*, *11*(6), 066011. https://doi.org/10.1088/1741-2560/11/6/066011

Harlow, J. (1848). Passage of an iron rod through the head. *The Boston Medical and Surgical Journal (1828-1851)*, *39*(20), 0_1. https://doi.org/10.1176/jnp.11.2.281

Haslam, N., Holland, E., & Kuppens, P. (2012). Categories versus dimensions in personality and psychopathology: A quantitative review of taxometric research. *Psychological Medicine*, *42*(5), 903–920. https://doi.org/10.1017/S0033291711001966

Haslam, N., McGrath, M. J., Viechtbauer, W., & Kuppens, P. (2020). Dimensions over categories: A meta-analysis of taxometric research. *Psychological Medicine*, *50*(9), 1418–1432. https://doi.org/10.1017/S003329172000183X

Haxby, J. V., Grady, C. L., Horwitz, B., Ungerleider, L. G., Mishkin, M., Carson, R. E., Herscovitch, P., Schapiro, M. B., & Rapoport, S. I. (1991). Dissociation of object and spatial visual processing pathways in human extrastriate cortex. *Proceedings of the National Academy of Sciences*, *88*(5), 1621–1625. https://doi.org/10.1073/pnas.88.5.1621

Heath, M., Rival, C., Westwood, D. A., & Neely, K. (2005). Time Course Analysis of Closed- and Open-Loop Grasping of the Müller-Lyer Illusion. *Journal of Motor Behavior*, *37*(3), 179–185. https://doi.org/10.3200/JMBR.37.3.179-185

Hedge, C., Powell, G., & Sumner, P. (2018). The reliability paradox: Why robust cognitive tasks do not produce reliable individual differences. *Behavior Research Methods*, *50*(3), 1166–1186. https://doi.org/10.3758/s13428-017-0935-1

Heydasch, T., Haubrich, J., & Renner, K.-H. (2013). The Short Version of the Hagen Matrices Test (HMT-S): 6-Item Induction Intelligence Test. *Methods, Data, Analyses*, *7*(2), Article 2. https://doi.org/10.12758/mda.2013.011

Hirjak, D., Meyer-Lindenberg, A., Kubera, K. M., Thomann, P. A., & Wolf, R. C. (2018). Motor dysfunction as research domain in the period preceding manifest schizophrenia: A systematic review. *Neuroscience & Biobehavioral Reviews*, *87*, 87–105. https://doi.org/10.1016/j.neubiorev.2018.01.011

Hirjak, D., Thomann, P. A., Kubera, K. M., Wolf, N. D., Sambataro, F., & Wolf, R. C. (2015). Motor dysfunction within the schizophrenia-spectrum: A dimensional step towards an underappreciated domain. *Schizophrenia Research*, *169*(1), 217–233. https://doi.org/10.1016/j.schres.2015.10.022

Hoffman, R. E., & McGlashan, T. H. (2001). Neural network models of schizophrenia. *The Neuroscientist: A Review Journal Bringing Neurobiology, Neurology and Psychiatry*, *7*(5), 441–454. https://doi.org/10.1177/107385840100700513

Holmes, E. A., & Steel, C. (2004). Schizotypy: A Vulnerability Factor for Traumatic Intrusions. *The Journal of Nervous and Mental Disease*, *192*(1), 28. https://doi.org/10.1097/01.nmd.0000105997.28152.4b

Hori, H., Teraishi, T., Sasayama, D., Matsuo, J., Kinoshita, Y., Ota, M., Hattori, K., & Kunugi, H. (2014). A latent profile analysis of schizotypy, temperament and character in a nonclinical population: Association with neurocognition. *Journal of Psychiatric Research*, *48*(1), 56–64. https://doi.org/10.1016/j.jpsychires.2013.10.006

Horton, L. E., Barrantes-Vidal, N., Silvia, P. J., & Kwapil, T. R. (2014). Worries about Being Judged versus Being Harmed: Disentangling the Association of Social Anxiety and Paranoia with Schizotypy. *PLOS ONE*, *9*(6), e96269. https://doi.org/10.1371/journal.pone.0096269

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, *6*(1), 1–55. https://doi.org/10.1080/10705519909540118

Huber, S., & Huber, O. W. (2012). The Centrality of Religiosity Scale (CRS). *Religions*, *3*(3), Article 3. https://doi.org/10.3390/rel3030710

Huey, E. D., & Lieberman, J. A. (2012). From known to unknown; old to new: Can lesion studies inform psychiatry about mental illness in the 21st century? *Neuropsychiatry*, *2*(5), 369–372. https://doi.org/10.2217/npy.12.54

Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., Sanislow, C., & Wang, P. (2010). Research Domain Criteria (RDoC): Toward a New Classification Framework for Research on Mental Disorders. *American Journal of Psychiatry*, *167*(7), 748–751. https://doi.org/10.1176/appi.ajp.2010.09091379

Insel, T. R. (2010). Rethinking schizophrenia. *Nature*, *468*(7321), Article 7321. https://doi.org/10.1038/nature09552

International Schizophrenia Consortium. (2009). Common polygenic variation contributes to risk of schizophrenia that overlaps with bipolar disorder. *Nature*, *460*(7256), 748–752. https://doi.org/10.1038/nature08185

Jaaro-Peled, H., & Sawa, A. (2020). Neurodevelopmental Factors in Schizophrenia. *Psychiatric Clinics*, *43*(2), 263–274. https://doi.org/10.1016/j.psc.2020.02.010

Jakobson, L. S., Archibald, Y. M., Carey, D. P., & Goodale, M. A. (1991). A kinematic analysis of reaching and grasping movements in a patient recovering from optic ataxia. *Neuropsychologia*, *29*(8), 803–809. https://doi.org/10.1016/0028-3932(91)90073-H

Jakobson, L. S., & Goodale, M. A. (1991). Factors affecting higher-order movement planning: A kinematic analysis of human prehension. *Experimental Brain Research*, *86*(1), 199–208. https://doi.org/10.1007/BF00231054

James, T. W., Culham, J., Humphrey, G. K., Milner, A. D., & Goodale, M. A. (2003). Ventral occipital lesions impair object recognition but not object-directed grasping: An fMRI study. *Brain*, *126*(11), 2463–2475. https://doi.org/10.1093/brain/awg248

Jang, K. L., Woodward, T. S., Lang, D., Honer, W. G., & Livesley, W. J. (2005). The Genetic and Environmental Basis of the Relationship Between Schizotypy and Personality: A Twin Study. *The Journal of Nervous and Mental Disease*, *193*(3), 153. https://doi.org/10.1097/01.nmd.0000154842.26600.bd

JASP Team. (2022). *JASP* (0.16.3). https://jasp-stats.org/

Jeannerod, M. (1988). *The neural and behavioural organization of goal-directed movements* (pp. xii, 283). Clarendon Press/Oxford University Press.

Johnson, P. B., Ferraina, S., Bianchi, L., & Caminiti, R. (1996). Cortical networks for visual reaching: Physiological and anatomical organization of frontal and parietal lobe arm regions. *Cerebral Cortex*, *6*(2), 102–119. https://doi.org/10.1093/cercor/6.2.102

Jolley, S., Jones, S. H., & Hemsley, D. R. (1999). Causal processing and schizotypy. *Personality and Individual Differences*, *27*(2), 277–291. https://doi.org/10.1016/S0191-8869(98)00239-6

Kaess, M., Ceumern-Lindenstjerna, I. A. von, Parzer, P., Chanen, A., Mundt, C., Resch, F., & Brunner, R. (2013). Axis I and II Comorbidity and Psychosocial Functioning in

Female Adolescents with Borderline Personality Disorder. *Psychopathology*, *46*(1), 55–62. https://doi.org/10.1159/000338715

Kalkstein, S., Hurford, I., & Gur, R. C. (2010). Neurocognition in Schizophrenia. In N. R. Swerdlow (Ed.), *Behavioral Neurobiology of Schizophrenia and Its Treatment* (pp. 373–390). Springer. https://doi.org/10.1007/7854_2010_42

Karnath, H. O., Rüter, J., Mandler, A., & Himmelbach, M. (2009). The Anatomy of Object Recognition—Visual Form Agnosia Caused by Medial Occipitotemporal Stroke. *The Journal of Neuroscience*, *29*(18), 5854–5862. https://doi.org/10.1523/JNEUROSCI.5192-08.2009

Kemp, K., Gross, G. M., Barrantes-Vidal, N., & Kwapil, T. R. (2018). Association of positive, negative, and disorganized schizotypy dimensions with affective symptoms and experiences. *Psychiatry Research*, *270*, 1143–1149. https://doi.org/10.1016/j.psychres.2018.10.031

Kéri, S., Antal, A., Szekeres, G., Benedek, G., & Janka, Z. (2002). Spatiotemporal Visual Processing in Schizophrenia. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *14*(2), 190–196. https://doi.org/10.1176/jnp.14.2.190

King, J. P., Christensen, B. K., & Westwood, D. A. (2008). Grasping behavior in schizophrenia suggests selective impairment in the dorsal visual pathway. *Journal of Abnormal Psychology*, *117*(4), 799–811. https://doi.org/10.1037/a0013500

Knight, R. P. (1953). Borderline states. In *Drives, Affects, Behavior* (pp. 203–215). International Universities Press, Inc. https://doi.org/10.1037/10633-012

Kocsis-Bogár, K., Nemes, Z., & Perczel-Forintos, D. (2016). Factorial structure of the Hungarian version of Oxford-Liverpool Inventory of Feelings and Experiences and its applicability on the schizophrenia-schizotypy continuum. *Personality and Individual Differences*, *90*, 130–136. https://doi.org/10.1016/j.paid.2015.10.039

Kong, L., Herold, C. J., Cheung, E. F. C., Chan, R. C. K., & Schröder, J. (2020). Neurological Soft Signs and Brain Network Abnormalities in Schizophrenia. *Schizophrenia Bulletin*, *46*(3), 562–571. https://doi.org/10.1093/schbul/sbz118

Kotov, R., Jonas, K. G., Carpenter, W. T., Dretsch, M. N., Eaton, N. R., Forbes, M. K., Forbush, K. T., Hobbs, K., Reininghaus, U., Slade, T., South, S. C., Sunderland, M., Waszczuk, M. A., Widiger, T. A., Wright, A. G. C., Zald, D. H., Krueger, R. F., Watson, D., & Workgroup, H. U. (2020). Validity and Utility of Hierarchical Taxonomy of Psychopathology (HiTOP): I. Psychosis superspectrum. *World Psychiatry*, *19*(2), 151–172. https://doi.org/10.1002/wps.20730

Kotov, R., Jonas, K. G., Lian, W., Docherty, A. R., & Carpenter, W. T. (2022). Reconceptualizing schizophrenia in the Hierarchical Taxonomy Of Psychopathology (HiTOP). *Schizophrenia Research*, *242*, 73–77. https://doi.org/10.1016/j.schres.2022.01.053

Kotov, R., Krueger, R. F., & Watson, D. (2018). A paradigm shift in psychiatric classification: The Hierarchical Taxonomy Of Psychopathology (HiTOP). *World Psychiatry*, *17*(1), 24–25. https://doi.org/10.1002/wps.20478

Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., Brown, T. A., Carpenter, W. T., Caspi, A., Clark, L. A., Eaton, N. R., Forbes, M. K., Forbush, K. T., Goldberg, D., Hasin, D., Hyman, S. E., Ivanova, M. Y., Lynam, D. R., Markon, K., … Zimmerman, M. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology*, *126*, 454–477. https://doi.org/10.1037/abn0000258

Koychev, I., El-Deredy, W., Haenschel, C., & Deakin, J. F. W. (2010). Visual information processing deficits as biomarkers of vulnerability to schizophrenia: An event-related potential study in schizotypy. *Neuropsychologia*, *48*(7), 2205–2214. https://doi.org/10.1016/j.neuropsychologia.2010.04.014

Kraepelin, E. (1919). *Dementia praecox and paraphrenia*. ES Livingstone.

Króliczak, G., Cavina-Pratesi, C., Goodman, D. A., & Culham, J. C. (2007). What Does the Brain Do When You Fake It? An fMRI Study of Pantomimed and Real Grasping. *Journal of Neurophysiology*, *97*(3), 2410–2422. https://doi.org/10.1152/jn.00778.2006

Kwapil, T., & Barrantes-Vidal, N. (2015). Schizotypy: Looking Back and Moving Forward. *Schizophrenia Bulletin*, *41*(suppl_2), S366–S373. https://doi.org/10.1093/schbul/sbu186

Kwapil, T., Barrantes-Vidal, N., & Silvia, P. J. (2008). The Dimensional Structure of the Wisconsin Schizotypy Scales: Factor Identification and Construct Validity. *Schizophrenia Bulletin*, *34*(3), 444–457. https://doi.org/10.1093/schbul/sbm098

Kwapil, T., Gross, G. M., Burgin, C., Raulin, M. L., Silvia, P. J., & Barrantes-Vidal, N. (2018). Validity of the Multidimensional Schizotypy Scale: Associations with Schizotypal Traits and Normal Personality. *Personality Disorders: Theory, Research, and Treatment*, *9*(5), 458–466. https://doi.org/ 10.1037/per0000288

Kwapil, T., Gross, G. M., Silvia, P. J., Raulin, M. L., & Barrantes-Vidal, N. (2018). Development and psychometric properties of the Multidimensional Schizotypy Scale: A new measure for assessing positive, negative, and disorganized schizotypy. *Schizophrenia Research*, *193*, 209–217. https://doi.org/10.1016/j.schres.2017.07.001

Launay, G., & Slade, P. (1981). The measurement of hallucinatory predisposition in male and female prisoners. *Personality and Individual Differences*, *2*(3), 221–234. https://doi.org/10.1016/0191-8869(81)90027-1

Lenzenweger, M. F. (1994). Psychometric High-risk Paradigm, Perceptual Aberrations, and Schizotypy: An Update. *Schizophrenia Bulletin*, *20*(1), 121–135. https://doi.org/10.1093/schbul/20.1.121

Lenzenweger, M. F. (2011). *Schizotypy and Schizophrenia: The View from Experimental Psychopathology*. Guilford Press.

Lewandowski, K. E., Barrantes-Vidal, N., Nelson-Gray, R. O., Clancy, C., Kepley, H. O., & Kwapil, T. R. (2006). Anxiety and depression symptoms in psychometrically identified schizotypy. *Schizophrenia Research*, 11. https://doi.org/10.1016/j.schres.2005.11.024

Li, K., Guo, L., Nie, J., Li, G., & Liu, T. (2009). Review of methods for functional brain connectivity detection using fMRI. *Computerized Medical Imaging and Graphics*, *33*(2), 131–139. https://doi.org/10.1016/j.compmedimag.2008.10.011

Lin, A., Wigman, J. T. W., Nelson, B., Wood, S. J., Vollebergh, W. A. M., van Os, J., & Yung, A. R. (2013). Follow-up factor structure of schizotypy and its clinical associations in a help-seeking sample meeting ultra-high risk for psychosis criteria at baseline. *Comprehensive Psychiatry*, *54*(2), 173–180. https://doi.org/10.1016/j.comppsych.2012.06.011

Lin, K. C., Chen, Y., Chen, C., Wu, C., & Chang, Y. (2010). The Effects of Bilateral Arm Training on Motor Control and Functional Performance in Chronic Stroke: A Randomized Controlled Study. *Neurorehabilitation and Neural Repair*, *24*(1), 42–51. https://doi.org/10.1177/1545968309345268

Lin, K. C., Wu, C. Y., Wei, T. H., Gung, C., Lee, C. Y., & Liu, J. S. (2007). Effects of modified constraint-induced movement therapy on reach-to-grasp movements and functional performance after chronic stroke: A randomized controlled study. *Clinical Rehabilitation*, *21*(12), 1075–1086. https://doi.org/10.1177/0269215507079843

Linney, Y. M., Murray, R. M., Peters, E. R., MacDonald, A. M., Rijsdijk, F., & Sham, P. C. (2003). A quantitative genetic analysis of schizotypal personality traits. *Psychological Medicine*, *33*(5), 803–816. https://doi.org/10.1017/S0033291703007906

Linscott, R. J., & van Os, J. (2010). Systematic Reviews of Categorical Versus Continuum Models in Psychosis: Evidence for Discontinuous Subpopulations Underlying a Psychometric Continuum. Implications for DSM-V, DSM-VI, and DSM-VII. *Annual*

Review of Clinical Psychology, *6*(1), 391–419. https://doi.org/10.1146/annurev.clinpsy.032408.153506

Llorca, P. M., Chereau, I., Bayle, F. J., & Lancon, C. (2002). Tardive dyskinesias and antipsychotics: A review. *European Psychiatry*, *17*(3), 129–138. https://doi.org/10.1016/S0924-9338(02)00647-8

Loas, G., Pham-Scottez, A., Cailhol, L., Perez-Diaz, F., Corcos, M., & Speranza, M. (2013). Axis II Comorbidity of Borderline Personality Disorder in Adolescents. *Psychopathology*, *46*(3), 172–175. https://doi.org/10.1159/000339530

Manschreck, T. C., Maher, B. A., & Candela, S. F. (2004). Earlier Age of First Diagnosis in Schizophrenia Is Related to Impaired Motor Control. *Schizophrenia Bulletin*, *30*(2), 351–360. https://doi.org/10.1093/oxfordjournals.schbul.a007084

March, E., & Springer, J. (2019). Belief in conspiracy theories: The predictive role of schizotypy, Machiavellianism, and primary psychopathy. *PLOS ONE*, *14*(12), e0225964. https://doi.org/10.1371/journal.pone.0225964

Mason, O., & Claridge, G. (2006). The Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE): Further description and extended norms. *Schizophrenia Research*, *82*(2–3), 203–211. https://doi.org/10.1016/j.schres.2005.12.845

Mason, O., Claridge, G., & Jackson, M. (1995). New scales for the assessment of schizotypy. *Personality and Individual Differences*, *18*(1), 7–13. https://doi.org/10.1016/0191-8869(94)00132-C

Mason, O. (2015). The Assessment of Schizotypy and Its Clinical Relevance. *Schizophrenia Bulletin*, *41*(Suppl 2), S374–S385. https://doi.org/10.1093/schbul/sbu194

Mason, O., Booth, H., & Olivers, C. (2004). Proneness to psychosis and selection of objects of visual attention: Individual differences in visual marking. *Personality and Individual Differences*, *36*(8), 1771–1779. https://doi.org/10.1016/j.paid.2003.07.015

Mason, O., Linney, Y., & Claridge, G. (2005). Short scales for measuring schizotypy. *Schizophrenia Research*, *78*(2), 293–296. https://doi.org/10.1016/j.schres.2005.06.020

McClenon, J. (2012). A community survey of psychological symptoms: Evaluating evolutionary theories regarding shamanism and schizophrenia. *Mental Health, Religion & Culture*, *15*(8), 799–816. https://doi.org/10.1080/13674676.2011.637913

McCrae, R., & Costa, P. (2010). *NEO inventories for the NEO Personality Inventory-3 (NEO-PI-3), NEO Five-Factor Inventory-3 (NEO-FFI-3), NEO Personality Inventory-Revised (NEO PI-R): Professional manual*. PAR Inc.

McCutcheon, R. A., Keefe, R. S. E., & McGuire, P. K. (2023). Cognitive impairment in schizophrenia: Aetiology, pathophysiology, and treatment. *Molecular Psychiatry*, 1–17. https://doi.org/10.1038/s41380-023-01949-9

McCutcheon, R. A., Reis Marques, T., & Howes, O. D. (2020). Schizophrenia—An Overview. *JAMA Psychiatry*, *77*(2), 201–210. https://doi.org/10.1001/jamapsychiatry.2019.3360

McIntosh, A. R., Grady, C. L., Ungerleider, L. G., Haxby, J. V., Rapoport, S. I., & Horwitz, B. (1994). Network analysis of cortical visual pathways mapped with PET. *Journal of Neuroscience*, *14*(2), 655–666. https://doi.org/10.1523/JNEUROSCI.14-02-00655.1994

Meade, A. W., & Craig, S. B. (2012). Identifying careless responses in survey data. *Psychological Methods*, *17*(3), 437–455. https://doi.org/10.1037/a0028085

Meehl, P. E. (1962). Schizotaxia, Schizotypy, Schizophrenia. In *Schizophrenia: Seven Approaches*. Routledge.

Meehl, P. E. (1973). *Manual for use with Checklist of Schizotypic Signs*. University of Minnesota. http://conservancy.umn.edu/handle/11299/151571

Meehl, P. E. (1989). Schizotaxia revisited. *Archives of General Psychiatry*, *46*(10), 935–944. https://doi.org/10.1001/archpsyc.1989.01810100077015

Meehl, P. E. (1990). Toward an Integrated Theory of Schizotaxia, Schizotypy, and Schizophrenia. *Journal of Personality Disorders*, *4*(1), 1–99. https://doi.org/10.1521/pedi.1990.4.1.1

Meehl, P. E., & Cronbach, L. J. (1955). Construct Validity in Psychological Tests. *Psychological Bulletin*, *52*, 281–302. http://dx.doi.org/10.1037/h0040957

Metsämuuronen, J. (2022). Attenuation-Corrected Estimators of Reliability. *Applied Psychological Measurement*, *46*(8), 720–737. https://doi.org/10.1177/01466216221108131

Milner, A. D., Dijkerman, H. C., Pisella, L., McIntosh, R. D., Tilikete, C., Vighetto, A., & Rossetti, Y. (2001). Grasping the past: Delay can improve visuomotor performance. *Current Biology*, *11*(23), 1896–1901. https://doi.org/10.1016/S0960-9822(01)00591-7

Mishkin, M., Ungerleider, L. G., & Macko, K. A. (1983). Object vision and spatial vision: Two cortical pathways. *Trends in Neurosciences*, *6*, 414–417. https://doi.org/10.1016/0166-2236(83)90190-X

Mohr, C., & Claridge, G. (2015). Schizotypy—Do Not Worry, It Is Not All Worrisome. *Schizophrenia Bulletin*, *41*(suppl_2), S436–S443. https://doi.org/10.1093/schbul/sbu185

Monaco, S., Sedda, A., Cavina-Pratesi, C., & Culham, J. C. (2015). Neural correlates of object size and object location during grasping actions. *European Journal of Neuroscience*, *41*(4), 454–465. https://doi.org/10.1111/ejn.12786

Moran, P. M., Al-Uzri, M. M., Watson, J., & Reveley, M. A. (2003). Reduced Kamin blocking in non paranoid schizophrenia: Associations with schizotypy. *Journal of Psychiatric Research*, *37*(2), 155–163. https://doi.org/10.1016/S0022-3956(02)00099-7

Morey, L. C. (2014). *Personality Assessment Inventory™*. Odessa, FL: Psychological Assessment Resources.

Murphy, B. A., & Lilienfeld, S. O. (2019). Are self-report cognitive empathy ratings valid proxies for cognitive empathy ability? Negligible meta-analytic relations with behavioral task performance. *Psychological Assessment*, *31*, 1062–1072. https://doi.org/10.1037/pas0000732

Muthén, B., du Toit, S., & Spisic, D. (1997). Robust inference using weighted least squares and quadratic estimating equations in latent variable modeling with categorical and continuous outcomes. *Psychometrika*. https://cir.nii.ac.jp/crid/1572261550775850752

Neill, E. (2014). Methodological Considerations in the Recruitment and Analysis of Schizotypy Samples. *Frontiers in Psychiatry*, *5*. https://www.frontiersin.org/articles/10.3389/fpsyt.2014.00156

Nelson, B., & Rawlings, D. (2010). Relating Schizotypy and Personality to the Phenomenology of Creativity. *Schizophrenia Bulletin*, *36*(2), 388–399. https://doi.org/10.1093/schbul/sbn098

Nelson, M. T., Seal, M. L., Pantelis, C., & Phillips, L. J. (2013). Evidence of a dimensional relationship between schizotypy and schizophrenia: A systematic review. *Neuroscience & Biobehavioral Reviews*, *37*(3), 317–327. https://doi.org/10.1016/j.neubiorev.2013.01.004

Nielsen, T. C., & Petersen, K. E. (1976). Electrodermal correlates of extraversion, trait anxiety and schizophrenia. *Scandinavian Journal of Psychology*, *17*(1), 73–80. https://doi.org/10.1111/j.1467-9450.1976.tb00214.x

Nowak, D. A., Connemann, B. J., Alan, M., & Spitzer, M. (2006). Sensorimotor dysfunction of grasping in schizophrenia: A side effect of antipsychotic treatment? *Journal of Neurology, Neurosurgery & Psychiatry*, *77*(5), 650–657. https://doi.org/10.1136/jnnp.2005.084749

Nunn, J., & Peters, E. (2001). Schizotypy and patterns of lateral asymmetry on hemisphere-specific language tasks. *Psychiatry Research*, *103*(2), 179–192. https://doi.org/10.1016/S0165-1781(01)00273-6

O'Donnell, B. F., Swearer, J. M., Smith, L. T., Nestor, P. G., Shenton, M. E., & McCarley, R. W. (1996). Selective deficits in visual perception and recognition in schizophrenia. *The American Journal of Psychiatry*, *153*(5), 687–692. https://doi.org/10.1176/ajp.153.5.687

Oezgen, M., & Grant, P. (2018). Odd and disorganized–Comparing the factor structure of the three major schizotypy inventories. *Psychiatry Research*, *267*, 289–295. https://doi.org/10.1016/j.psychres.2018.06.009

Olivier, E., Davare, M., Andres, M., & Fadiga, L. (2007). Precision grasping in humans: From motor control to cognition. *Current Opinion in Neurobiology*, *17*(6), 644–648. https://doi.org/10.1016/j.conb.2008.01.008

Orjiakor, C. T., Watt, A., Iorfa, S. K., Onu, D. U., & Okonkwo, A. I. (2019). Associations between schizotypal traits and antisocial behaviours in a sub-Saharan sample. *Archives of Psychiatric Nursing*, *33*(6), 138–143. https://doi.org/10.1016/j.apnu.2019.10.001

Pandya, D. N., Seltzer, B., & Barbas, H. (1986). Input-output organization of the primate cerebral cortex. In *Comparative Primate Biology* (pp. 39–80). A.R. Liss. https://scholar.google.com/scholar_lookup?title=Input-output+organization+of+the+primate+cerebral+cortex&author=Pandya%2C+D.N.&publication_year=1986

Pandya, D. N., & Yeterian, E. H. (1985). Architecture and Connections of Cortical Association Areas. In A. Peters & E. G. Jones (Eds.), *Association and Auditory Cortices* (pp. 3–61). Springer US. https://doi.org/10.1007/978-1-4757-9619-3_1

Pandya, D. N., & Barnes, C. L. (1987). Architecture and Connections of the Frontal Lobe. In *The Frontal Lobes Revisited*. Psychology Press.

Park, N. K., Chun, M. Y., & Lee, J. (2016). Revisiting Individual Creativity Assessment: Triangulation in Subjective and Objective Assessment Methods. *Creativity Research Journal*, *28*(1), 1–10. https://doi.org/10.1080/10400419.2016.1125259

Philipsen, A. (2006). Differential diagnosis and comorbidity of attention-deficit/hyperactivity disorder (ADHD) and borderline personality disorder (BPD) in adults. *European Archives of Psychiatry and Clinical Neuroscience*, *256*(1), i42–i46. https://doi.org/10.1007/s00406-006-1006-2

Pickering, A. D. (2004). Chapter 23 - The Neuropsychology of Impulsive Antisocial Sensation Seeking Personality Traits: From Dopamine to Hippocampal Function? In R. M. Stelmack (Ed.), *On the Psychobiology of Personality* (pp. 453–476). Elsevier. https://doi.org/10.1016/B978-008044209-9/50024-5

Prather, S. C., Votaw, J. R., & Sathian, K. (2004). Task-specific recruitment of dorsal and ventral visual areas during tactile perception. *Neuropsychologia*, *42*(8), 1079–1087. https://doi.org/10.1016/j.neuropsychologia.2003.12.013

Premkumar, P., Onwumere, J., Betts, L., Kibowski, F., & Kuipers, E. (2018). Schizotypal traits and their relation to rejection sensitivity in the general population: Their mediation by quality of life, agreeableness and neuroticism. *Psychiatry Research*, *267*, 201–209. https://doi.org/10.1016/j.psychres.2018.06.002

Price, J. L., & Drevets, W. C. (2012). Neural circuits underlying the pathophysiology of mood disorders. *Trends in Cognitive Sciences*, *16*(1), 61–71. https://doi.org/10.1016/j.tics.2011.12.011

Putzhammer, A., Perfahl, M., Pfeiff, L., Ibach, B., Johann, M., Zitzelsberger, U., & Hajak, G. (2005). Performance of diadochokinetic movements in schizophrenic patients. *Schizophrenia Research*, *79*(2), 271–280. https://doi.org/10.1016/j.schres.2005.05.020

R Core Team. (2022). *R: A language and environment for statistical computing* (v4.2.1). R Foundation for Statistical Computing. http://www.R-project.org/

Rado, S. (1953). Dynamics and classification of disordered behavior. *American Journal of Psychiatry*, *110*(6), 406–416. https://doi.org/10.1176/ajp.110.6.406

Raine, A. (1991). The SPQ: A Scale for the Assessment of Schizotypal Personality Based on DSM-III-R Criteria. *Schizophrenia Bulletin*, *17*(4), 555–564. https://doi.org/10.1093/schbul/17.4.555

Raine, A., Reynolds, C., Lencz, T., Scerbo, A., Triphon, N., & Kim, D. (1994). Cognitive-perceptual, interpersonal, and disorganized features of schizotypal personality. *Schizophrenia Bulletin*, *20*(1), 191–201. https://doi.org/10.1093/schbul/20.1.191

Raos, V., Umiltá, M. A., Gallese, V., & Fogassi, L. (2004). Functional Properties of Grasping-Related Neurons in the Dorsal Premotor Area F2 of the Macaque Monkey. *Journal of Neurophysiology*, *92*(4), 1990–2002. https://doi.org/10.1152/jn.00154.2004

Rapp, A. M., Mutschler, D. E., Wild, B., Erb, M., Lengsfeld, I., Saur, R., & Grodd, W. (2010). Neural correlates of irony comprehension: The role of schizotypal personality traits. *Brain and Language*, *113*(1), 1–12. https://doi.org/10.1016/j.bandl.2009.11.007

Rassovsky, Y., Green, M. F., Nuechterlein, K. H., Breitmeyer, B., & Mintz, J. (2004). Paracontrast and metacontrast in schizophrenia: Clarifying the mechanism for visual masking deficits. *Schizophrenia Research*, *71*(2), 485–492. https://doi.org/10.1016/j.schres.2004.02.018

Rawlings, D., Williams, B., Haslam, N., & Claridge, G. (2008a). Is schizotypy taxonic? Response to Beauchaine, Lenzenweger, and Waller. *Personality and Individual Differences*, *44*(8), 1663–1672. https://doi.org/10.1016/j.paid.2008.01.021

Rawlings, D., Williams, B., Haslam, N., & Claridge, G. (2008b). Taxometric analysis supports a dimensional latent structure for schizotypy. *Personality and Individual Differences*, *44*(8), 1640–1651. https://doi.org/10.1016/j.paid.2007.06.005

Regier, D. A., Narrow, W. E., Clarke, D. E., Kraemer, H. C., Kuramoto, S. J., Kuhl, E. A., & Kupfer, D. J. (2013). DSM-5 field trials in the United States and Canada, Part II:

Test-retest reliability of selected categorical diagnoses. *The American Journal of Psychiatry*, *170*(1), 59–70. https://doi.org/10.1176/appi.ajp.2012.12070999

Rice, N. J., Tunik, E., & Grafton, S. T. (2006). The Anterior Intraparietal Sulcus Mediates Grasp Execution, Independent of Requirement to Update: New Insights from Transcranial Magnetic Stimulation. *Journal of Neuroscience*, *26*(31), 8176–8182. https://doi.org/10.1523/JNEUROSCI.1641-06.2006

Rizzolatti, G., Fadiga, L., Matelli, M., Bettinardi, V., Paulesu, E., Perani, D., & Fazio, F. (1996). Localization of grasp representations in humans by PET: 1. Observation versus execution. *Experimental Brain Research*, *111*(2), 246–252. https://doi.org/10.1007/BF00227301

Rossetti, Y., Revol, P., McIntosh, R., Pisella, L., Rode, G., Danckert, J., Tilikete, C., Dijkerman, H. C., Boisson, D., Vighetto, A., Michel, F., & Milner, A. D. (2005). Visually guided reaching: Bilateral posterior parietal lesions cause a switch from fast visuomotor to slow cognitive control. *Neuropsychologia*, *43*(2), 162–177. https://doi.org/10.1016/j.neuropsychologia.2004.11.004

Rossi, A., & Daneluzzo, E. (2002). Schizotypal dimensions in normals and schizophrenic patients: A comparison with other clinical samples. *Schizophrenia Research*, *54*(1), 67–75. https://doi.org/10.1016/S0920-9964(01)00353-X

RStudio Team. (2020). *RStudio: Integrated Development for R*. RStudio, PBC.

Rund, B. R. (2018). The research evidence for schizophrenia as a neurodevelopmental disorder. *Scandinavian Journal of Psychology*, *59*(1), 49–58. https://doi.org/10.1111/sjop.12414

Ruscio, J., Ruscio, A. M., & Meron, M. (2007). Applying the Bootstrap to Taxometric Analysis: Generating Empirical Sampling Distributions to Help Interpret Results. *Multivariate Behavioral Research*, *42*(2), 349–386. https://doi.org/10.1080/00273170701360795

Salminen, I., Read, S., Hurd, P., & Crespi, B. (2019). Genetic variation of UBE3A is associated with schizotypy in a population of typical individuals. *Psychiatry Research*, *275*, 94–99. https://doi.org/10.1016/j.psychres.2019.03.019

Sanides, F. (1969). Comparative Architectonics of the Neocortex of Mammals and Their Evolutionary Interpretation. *Annals of the New York Academy of Sciences*, *167*(1), 404–423. https://doi.org/10.1111/j.1749-6632.1969.tb20459.x

Saunders, B., Milyavskaya, M., Etz, A., Randles, D., & Inzlicht, M. (2018). Reported Self-control is not Meaningfully Associated with Inhibition-related Executive Function: A Bayesian Analysis. *Collabra: Psychology*, *4*(1), 39. https://doi.org/10.1525/collabra.134

Schofield, K., & Claridge, G. (2007). Paranormal experiences and mental health: Schizotypy as an underlying factor. *Personality and Individual Differences*, *43*(7), 1908–1916. https://doi.org/10.1016/j.paid.2007.06.014

Schröder, J., & Herold, C. J. (2022). Neurological Soft Signs in Schizophrenia Spectrum Disorders. In A. L. Teixeira, E. Furr Stimming, W. G. Ondo, A. Teixeira, E. Furr Stimming, & W. G. Ondo (Eds.), *Movement Disorders in Psychiatry* (pp. 169-C10.P105). Oxford University Press. https://doi.org/10.1093/med/9780197574317.003.0010

Schröder, J., Niethammer, R., Geider, F. J., Reitz, C., Binkert, M., Jauss, M., & Sauer, H. (1991). Neurological soft signs in schizophrenia. *Schizophrenia Research*, *6*(1), 25–30. https://doi.org/10.1016/0920-9964(91)90017-L

Scott, J., Martin, G., Bor, W., Sawyer, M., Clark, J., & McGrath, J. (2009). The prevalence and correlates of hallucinations in Australian adolescents: Results from a national survey. *Schizophrenia Research*, *107*(2), 179–185. https://doi.org/10.1016/j.schres.2008.11.002

Sehatpour, P., Dias, E. C., Butler, P. D., Revheim, N., Guilfoyle, D. N., Foxe, J. J., & Javitt, D. C. (2010). Impaired Visual Object Processing Across an Occipital-Frontal-Hippocampal Brain Network in Schizophrenia: An Integrated Neuroimaging Study.

Archives of General Psychiatry, *67*(8), 772–782. https://doi.org/10.1001/archgenpsychiatry.2010.85

Seibert, L., & Crowell-Davis, S. (2019). Antipsychotics. In *Veterinary Psychopharmacology* (pp. 201–215). John Wiley & Sons, Ltd. https://doi.org/10.1002/9781119226253.ch14

Sellen, J. L., Oaksford, M., & Gray, N. S. (2005). Schizotypy and Conditional Reasoning. *Schizophrenia Bulletin*, *31*(1), 105–116. https://doi.org/10.1093/schbul/sbi012

Shaw, J., Claridge, G., & Clark, K. (2001). Schizotypy and the shift from dextrality: A study of handedness in a large non-clinical sample. *Schizophrenia Research*, *50*(3), 181–189. https://doi.org/10.1016/S0920-9964(00)00167-5

Shrout, P. E., & Yager, T. J. (1989). Reliability and validity of screening scales: Effect of reducing scale length. *Journal of Clinical Epidemiology*, *42*(1), 69–78. https://doi.org/10.1016/0895-4356(89)90027-9

Sierro, G., Rossier, J., Mason, O. J., & Mohr, C. (2016). French validation of the O-LIFE short questionnaire. *European Journal of Psychological Assessment*, *32*(3), 195–203. https://doi.org/10.1027/1015-5759/a000249

Silvanto, J., & Muggleton, N. G. (2008). New light through old windows: Moving beyond the “virtual lesion” approach to transcranial magnetic stimulation. *NeuroImage*, *39*(2), 549–552. https://doi.org/10.1016/j.neuroimage.2007.09.008

Sinclair, S. J., Siefert, C. J., Shorey, H. S., Antonius, D., Shiva, A., Kehl-Fie, K., & Blais, M. A. (2009). A psychometric evaluation of the Personality Assessment Inventory – short form clinical scales in an inpatient psychiatric sample. *Psychiatry Research*, *170*(2), 262–266. https://doi.org/10.1016/j.psychres.2008.11.001

Slaghuis, W. L., & Thompson, A. K. (2003). The effect of peripheral visual motion on focal contrast sensitivity in positive- and negative-symptom schizophrenia. *Neuropsychologia*, *41*(8), 968–980. https://doi.org/10.1016/S0028-3932(02)00321-4

Smeets, J. B. J., & Brenner, E. (2006). 10 years of illusions. *Journal of Experimental Psychology: Human Perception and Performance*, *32*, 1501–1504. https://doi.org/10.1037/0096-1523.32.6.1501

Smith, G. T. (2005). On Construct Validity: Issues of Method and Measurement. *Psychological Assessment*, *17*(4), 396–408. https://doi.org/10.1037/1040-3590.17.4.396

Smith, L., Riley, S., & Peters, E. R. (2009). Schizotypy, delusional ideation and well-being in an American new religious movement population. *Clinical Psychology & Psychotherapy*, *16*(6), 479–484. https://doi.org/10.1002/cpp.645

Snellen, H. (1873). *Probebuchstaben zur Bestimmung der Sehschärfe.* (P.W. van de Weijer, Utrecht). ISBN-13: 978-1149649404.

Stanciu, M., & Papasteri, C. C. (2017). Validation of the Romanian Oxford-Liverpool Inventory of Feelings and Experiences (OLIFE): Description, Psychometric Properties and Factor Structure. *Journal of Evidence-Based Psychotherapies*, *17*(1), 67–88. https://doi.org/10.24193/jebp.2017.1.5

Startup, M. (1999). Schizotypy, dissociative experiences and childhood abuse: Relationships among self-report measures. *British Journal of Clinical Psychology*, *38*(4), 333–344. https://doi.org/10.1348/014466599162908

Steel, C., Hemsley, D. R., & Pickering, A. D. (2002). Distractor cueing effects on choice reaction time and their relationship with schizotypal personality. *British Journal of Clinical Psychology*, *41*(2), 143–156. https://doi.org/10.1348/014466502163930

Stevens, S. S. (1946). On the theory of scales of measurement. *Science*, *103*, 677–680. https://doi.org/10.1126/science.103.2684.677

Tabachnick, B. G., Fidell, L. S., & Ullman, J. B. (2019). *Using Multivariate Statistics* (Seventh edition). Pearson.

Tabak, N. T., & Weisman de Mamani, A. G. (2013). Latent profile analysis of healthy schizotypy within the extended psychosis phenotype. *Psychiatry Research*, *210*(3), 1008–1013. https://doi.org/10.1016/j.psychres.2013.08.006

Takahashi, K., Best, M. D., Huh, N., Brown, K. A., Tobaa, A. A., & Hatsopoulos, N. G. (2017). Encoding of Both Reaching and Grasping Kinematics in Dorsal and Ventral Premotor Cortices. *Journal of Neuroscience*, *37*(7), 1733–1746. https://doi.org/10.1523/JNEUROSCI.1537-16.2016

Tellegen, A., & Briggs, P. F. (1967). Old wine in new skins: Grouping Wechsler subtests into new scales. *Journal of Consulting Psychology*, *31*(5), 499–506. https://doi.org/10.1037/h0024963

Thomas, E. H., Rossell, S. L., Tan, E. J., Neill, E., Van Rheenen, T. E., Carruthers, S. P., Sumner, P. J., Louise, S., Bozaoglu, K., & Gurvich, C. (2019). Do schizotypy dimensions reflect the symptoms of schizophrenia? *Australian & New Zealand Journal of Psychiatry*, *53*(3), 236–247. https://doi.org/10.1177/0004867418769746

Tomko, R. L., Trull, T. J., Wood, P. K., & Sher, K. J. (2014). Characteristics of Borderline Personality Disorder in a Community Sample: Comorbidity, Treatment Utilization, and General Functioning. *Journal of Personality Disorders*, *28*(5), 734–750. https://doi.org/10.1521/pedi_2012_26_093

Trull, T. J., Freeman, L. K., Vebares, T. J., Choate, A. M., Helle, A. C., & Wycoff, A. M. (2018). Borderline personality disorder and substance use disorders: An updated review. *Borderline Personality Disorder and Emotion Dysregulation*, *5*(1), 15. https://doi.org/10.1186/s40479-018-0093-9

Tsakanikos, E., & Reed, P. (2003). Visuo-spatial processing and dimensions of schizotypy: Figure-ground segregation as a function of psychotic-like features. *Personality and Individual Differences*, *35*(3), 703–712. https://doi.org/10.1016/S0191-8869(02)00263-5

Tsakanikos, E., Sverdrup-Thygenson, L., & Reed, P. (2003). Latent inhibition and psychosis-proneness: Visual search as a function of pre-exposure to the target and schizotypy level. *Personality and Individual Differences*, *34*(4), 575–589. https://doi.org/10.1016/S0191-8869(02)00029-6

Tunik, E., Frey, S. H., & Grafton, S. T. (2005). Virtual lesions of the anterior intraparietal area disrupt goal-dependent on-line adjustments of grasp. *Nature Neuroscience*, *8*(4), Article 4. https://doi.org/10.1038/nn1430

Turella, L., & Lingnau, A. (2014). Neural correlates of grasping. *Frontiers in Human Neuroscience*, *8*, 686. https://www.frontiersin.org/articles/10.3389/fnhum.2014.00686

Ungerleider, L., & Haxby, J. V. (1994). 'What' and 'where' in the human brain. *Current Opinion in Neurobiology*, *4*(2), 157–165. https://doi.org/10.1016/0959-4388(94)90066-3

Ungerleider, L., & Mishkin, M. (1982). Two cortical visual systems. In *Analysis of Visual Behavior* (pp. 549–586). MIT Press. https://cir.nii.ac.jp/crid/1570854174261014272

Vaillancourt, D. E., Thulborn, K. R., & Corcos, D. M. (2003). Neural Basis for the Processes That Underlie Visually Guided and Internally Guided Force Control in Humans. *Journal of Neurophysiology*, *90*(5), 3330–3340. https://doi.org/10.1152/jn.00394.2003

van den Bergh, D., van Doorn, J., Marsman, M., Draws, T., van Kesteren, E. J., Derks, K., Dablander, F., Gronau, Q. F., Kucharský, Š., Gupta, A. R. K. N., Sarafoglou, A., Voelkel, J. G., Stefan, A., Ly, A., Hinne, M., Matzke, D., & Wagenmakers, E.-J. (2020). A Tutorial on Conducting and Interpreting a Bayesian ANOVA in JASP. *L'Année Psychologique*, *120*(1), 73–96. https://doi.org/10.3917/anpsy1.201.0073

Van Essen, D. C., & Gallant, J. L. (1994). Neural mechanisms of form and motion processing in the primate visual system. *Neuron*, *13*(1), 1–10. https://doi.org/10.1016/0896-6273(94)90455-3

Van Rossum, G., & Drake, F. L. J. (1995). *Python reference manual*. Centrum voor Wiskunde en Informatica.

Verdoux, H., Maurice-Tison, S., Gay, B., Van Os, J., Salamon, R., & Bourgeois, M. L. (1998). A survey of delusional ideation in primary-care patients. *Psychological Medicine*, *28*(1), 127–134. https://doi.org/10.1017/S0033291797005667

Verhagen, L., Dijkerman, H. C., Grol, M. J., & Toni, I. (2008). Perceptuo-Motor Interactions during Prehension Movements. *Journal of Neuroscience*, *28*(18), 4726–4735. https://doi.org/10.1523/JNEUROSCI.0057-08.2008

Vesia, M., Barnett-Cowan, M., Elahi, B., Jegatheeswaran, G., Isayama, R., Neva, J. L., Davare, M., Staines, W. R., Culham, J. C., & Chen, R. (2017). Human dorsomedial parieto-motor circuit specifies grasp during the planning of goal-directed hand actions. *Cortex*, *92*, 175–186. https://doi.org/10.1016/j.cortex.2017.04.007

Vesia, M., Bolton, D. A., Mochizuki, G., & Staines, W. R. (2013). Human parietal and primary motor cortical interactions are selectively modulated during the transport and grip formation of goal-directed hand actions. *Neuropsychologia*, *51*(3), 410–417. https://doi.org/10.1016/j.neuropsychologia.2012.11.022

Vesia, M., Culham, J. C., Jegatheeswaran, G., Isayama, R., Le, A., Davare, M., & Chen, R. (2018). Functional interaction between human dorsal premotor cortex and the ipsilateral primary motor cortex for grasp plans: A dual-site TMS study. *NeuroReport*, *29*(16), 1355–1359. https://doi.org/10.1097/WNR.0000000000001117

Vollema, M. G., & van den Bosch, R. J. (1995). The Multidimensionality of Schizotypy. *Schizophrenia Bulletin*, *21*(1), 19–31. https://doi.org/10.1093/schbul/21.1.19

Vrtunski, B. P., Simpson, D. M., Weiss, K. M., & Davis, G. C. (1986). Abnormalities of fine motor control in schizophrenia. *Psychiatry Research*, *18*(3), 275–284. https://doi.org/10.1016/0165-1781(86)90114-9

Walker, E., Kestler, L., Bollini, A., & Hochman, K. (2004). Schizophrenia: Etiology and Course. *Annual Review of Psychology*, *55*, 401–430. https://doi.org/10.1146/annurev.psych.55.090902.141950

Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua*. Sage Publications, Inc.

Walter, E. E., Fernandez, F., Snelling, M., & Barkus, E. (2016). Genetic Consideration of Schizotypal Traits: A Review. *Frontiers in Psychology*, *7*, 1769. https://www.frontiersin.org/articles/10.3389/fpsyg.2016.01769

Walther, S., & Strik, W. (2012). Motor Symptoms and Schizophrenia. *Neuropsychobiology*, *66*(2), 77–92. https://doi.org/10.1159/000339456

Wang, C. Y., Hwang, W. J., Fang, J. J., Sheu, C. F., Leong, I. F., & Ma, H. I. (2011). Comparison of Virtual Reality Versus Physical Reality on Movement Characteristics of Persons with Parkinson's Disease: Effects of Moving Targets. *Archives of Physical Medicine and Rehabilitation*, *92*(8), 1238–1245. https://doi.org/10.1016/j.apmr.2011.03.014

Wang, J., Zhou, T., Qiu, M., Du, A., Cai, K., Wang, Z., Zhou, C., Meng, M., Zhuo, Y., Fan, S., & Chen, L. (1999). Relationship between ventral stream for object vision and dorsal stream for spatial vision: An fMRI+ERP study. *Human Brain Mapping*, *8*(4), 170–181. https://doi.org/10.1002/(SICI)1097-0193(1999)8:4<170::AID-HBM2>3.0.CO;2-W

Wang, S. M., Kuo, L. C., Ouyang, W. C., Hsu, H. M., & Ma, H. I. (2018). Effects of object size and distance on reaching kinematics in patients with schizophrenia. *Hong Kong Journal of Occupational Therapy*, *31*(1), 22–29. https://doi.org/10.1177/1569186118759610

Wang, Y., Cai, X., Zhang, R., Zhang, Y., Zhou, H., Wang, Y., Wang, Y., Huang, J., Wang, Y., Cheung, E. F. C., & Chan, R. C. K. (2022). Altered brain structural and functional connectivity in schizotypy. *Psychological Medicine*, *52*(5), 834–843. https://doi.org/10.1017/S0033291720002445

Watts, A. L., Poore, H. E., Lilienfeld, S. O., & Waldman, I. D. (2019). Clarifying the associations between Big Five personality domains and higher-order psychopathology dimensions in youth. *Journal of Research in Personality*, *82*, 103844. https://doi.org/10.1016/j.jrp.2019.07.002

Wechsler, D. (2008). *Wechsler Adult Intelligence Scale—Fourth Edition*. Pearson Publishing. https://doi.org/10.1037/t15169-000

Weidner, R., & Fink, G. R. (2007). The Neural Mechanisms Underlying the Müller-Lyer Illusion and Its Interaction with Visuospatial Judgments. *Cerebral Cortex*, *17*(4), 878–884. https://doi.org/10.1093/cercor/bhk042

Wernicke, C. (1970). The Aphasic Symptom-Complex: A Psychological Study on an Anatomical Basis*. *Archives of Neurology*, *22*(3), 280–282. https://doi.org/10.1001/archneur.1970.00480210090013

Westwood, D., & Goodale, M. (2003). Perceptual illusion and the real-time control of action. *Spatial Vision*, *16*(3), 243–254. https://doi.org/10.1163/156856803322467518

Wing, J. K., & Agrawal, N. (2003). Concepts and Classification of Schizophrenia. In *Schizophrenia* (pp. 1–14). John Wiley & Sons, Ltd. https://doi.org/10.1002/9780470987353.ch1

Wolff, A. L., & O'Driscoll, G. A. (1999). Motor deficits and schizophrenia: The evidence from neuroleptic-naïve patients and populations at risk. *Journal of Psychiatry and Neuroscience*, *24*(4), 304–314.

Wu, C., Wong, M., Lin, K., & Chen, H. (2001). Effects of Task Goal and Personal Preference on Seated Reaching Kinematics After Stroke. *Stroke*, *32*(1), 70–76. https://doi.org/10.1161/01.STR.32.1.70

Wuthrich, V. M., & Bates, T. C. (2006). Confirmatory Factor Analysis of the Three-Factor Structure of the Schizotypal Personality Questionnaire and Chapman Schizotypy Scales. *Journal of Personality Assessment*, *87*(3), 292–304. https://doi.org/10.1207/s15327752jpa8703_10

Yaghoubi, H., & Mohammadzadeh, A. (2012). Validation of the Oxford- Liverpool Inventory of Feelings and Experiences (O-LIFE) Questionnaire. *Zahedan Journal of Research in Medical Sciences*, *14*(9), 24-29. https://brieflands.com/articles/zjrms-93220.html#abstract

Yong, A. G., & Pearce, S. (2013). A Beginner's Guide to Factor Analysis: Focusing on Exploratory Factor Analysis. *Tutorials in Quantitative Methods for Psychology*, *9*(2), 79–94. https://doi.org/10.20982/tqmp.09.2.p079

Zandersen, M., & Parnas, J. (2019a). Identity Disturbance, Feelings of Emptiness, and the Boundaries of the Schizophrenia Spectrum. *Schizophrenia Bulletin*, *45*(1), 106–113. https://doi.org/10.1093/schbul/sbx183

Zandersen, M., & Parnas, J. (2019b). Borderline personality disorder or a disorder within the schizophrenia spectrum? A psychopathological study. *World Psychiatry*, *18*(1), 109–110. https://doi.org/10.1002/wps.20598

Zandersen, M., & Parnas, J. (2020). Exploring schizophrenia spectrum psychopathology in borderline personality disorder. *European Archives of Psychiatry and Clinical Neuroscience*, *270*(8), 969–978. https://doi.org/10.1007/s00406-019-01039-4

Zeki, S., Watson, J. D., Lueck, C. J., Friston, K. J., Kennard, C., & Frackowiak, R. S. (1991). A direct demonstration of functional specialization in human visual cortex. *Journal of Neuroscience*, *11*(3), 641–649. https://doi.org/10.1523/JNEUROSCI.11-03-00641.1991

APPENDICES

APPENDIX A

Meta-contrast Masking and Schizophrenia Spectrum Disorders [Manuscript]

During the initial stages of this research program, meta-contrast masking was identified as a possible paradigm for investigation of dorsal stream deficits in Schizophrenia Spectrum Disorders (SSD). Upon further review of the literature using meta-contrast masking as a paradigm for elucidating early visual processing, i.e., parvocellular and magnocellular pathways, which feed into ventral and dorsal cortical streams, respectively, it became clear that there was an inconsistency between the data from these studies and subsequent interpretation of underlying mechanisms of action. Specifically, meta-contrast masking deficits in SSD are largely attributed to an underperforming magnocellular system (M-system), however, the predominant theory explaining the mechanisms of meta-contrast masking emphasis overperformance on the M-system. The following commentary provides a review of the meta-contrast masking phenomena, its application to understanding dorsal deficits in SSD, and highlights the consistency between theory and data along with possible alternative explanations which considers recent neurophysiological and neuroanatomical literature. From this review, the current research program turned to an alternative method of understanding dorsal stream deficits in SSD, namely, visually guided movement.

This commentary is written as a manuscript for submission to peer-reviewed journals.

Meta-contrast Masking and Schizophrenia Spectrum Disorders

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Running head: Meta-contrast masking and schizophrenia

Abstract

Early visual processing deficits have been consistently demonstrated in Schizophrenia Spectrum Disorders. Much of this research has relied on the use of visual masking, especially meta-contrast masking. While the results of these masking studies have consistently shown poor perceptual performance (i.e., greater masking) in those further along on the Schizophrenia spectrum compared to control groups, clear explanations of the underlying mechanisms at play are controversial. Meta-contrast masking deficits in Schizophrenia patients are largely attributed to an underperforming magnocellular system (M-system), however, the predominant theory explaining the mechanisms of meta-contrast masking emphasis overperformance on the M-system. We discuss this inconsistency between theory and data along with possible alternative explanations which considers recent neurophysiological and neuroanatomical literature.

Keywords: Schizophrenia, Visual Masking, Metacontrast Masking, Feedforward Feedback Model

Over the past several decades, the visual information-processing deficits associated with Schizophrenia (SCZ) have been the target of considerable research. These studies have employed a range of different psychophysical and electrophysiological methods. In this regard, luminance pedestals (Delord et al., 2006), orientation tuning (Ishikawa et al., 2006), spatial frequency discrimination (Kéri et al., 2002; O'Donnell et al., 2002; W. A. Slaghuis, 1998), electroencephalography (EEG) and visually evoked potentials (VEPs) (Butler et al., 2005; Lalor et al., 2012), and perceptual closure tasks (Amiaz et al., 2016) have all been employed to show that visual perceptual deficits in SCZ are disproportionately reflective of neural abnormalities in the dorsal stream; specifically, the magnocellular (M) channel (Butler et al., 2005, 2008; Martínez et al., 2008). Furthermore, these deficits have been found to be stable across time (Chkonia et al., 2010; J. Lee et al., 2008) and relatively independent of medication (Butler et al., 2003) and cognitive deficits (Kéri et al., 2001; Koelkebeck et al., 2005). As such, it has been suggested that these visual perceptual deficits, especially those linked to M channel/dorsal stream impairment, are central to the disorder (Butler & Javitt, 2005).

A commonly used tool to investigate the human visual system, and by extension, SCZ-related visual deficits, is visual masking, in particular, visual backward masking (see Green et al., 2011). In visual backward masking tasks, the visibility of a stimulus (i.e., the target) is reduced or eliminated when another visual stimulus (i.e., the mask) temporally follows the target within a specific timeframe (Breitmeyer, 1984). This interaction is reliably observed in both neurotypical (Breitmeyer & Öğmen, 2006; Bruchmann et al., 2010; Ogmen et al., 2003) and SCZ patient populations (Cadenhead et al., 1998; M. Herzog et al., 2013; Schechter et al., 2003). The most prominent theoretical explanation offered for understanding the mechanisms underlying this phenomenon has appealed to the dual visual processing streams framework (Green et al., 1994; Herzog & Brand, 2015). This framework, drawing on knowledge of the properties of cells that process information in the early stages of vision, proposes that visual backward masking arises from magno- (M) and parvo- (P) cell interactions. More specifically, visual backward masking occurs because M cell activity suppresses, or inhibits, P cell activity (Breitmeyer & Ganz, 1976). The details of this interaction; known as the Dual Channel Model (DCM; Breitmeyer & Ganz, 1976) will be reviewed in more detail later. For now, however, it is important to note that according to this model, greater M cell activity should be associated with greater masking and therefore reduced perception, while weaker M cell activity should be associated with weaker masking and therefore increased perception.

While studies of visual backward masking performance in neurotypicals reliably show reductions in accuracy and increases in reaction time under masked compared to unmasked conditions, these deficits are consistently more pronounced in SCZ (Cadenhead et al., 1998; Green et al., 1994; Herzog et al., 2013; Schechter et al., 2003) These results suggest that patients with SCZ experience greater masking; that is, the disruptive impact of the mask on perception of the target is greater for patients than controls. This deficit is typically interpreted as consistent with the hypothesised *underactive* M channel in SCZ (Butler et al., 2003; McClure, 2001; Rassovsky et al., 2004), however, the DCM of masking would predict the opposite pattern of results for a group with an underactive M channel; that is, an underactive M channel would lead to weaker masking, and therefore, increased perception (Breitmeyer & Ganz, 1976).

Here, we consider this inconsistency and propose an alternative explanation which accommodates both previous and contemporary research findings and offers clear directions for future research on this topic. In doing so, we will first examine the dual visual processing streams explanation of SCZ-related masking impairment. This will be followed by a discussion outlining the limitations of this model and introduce an alternative explanation, along with its implications and future directions for research.

The Visual System

Both the visual-perceptual deficits associated with SCZ, in general, and the impairments in visual masking, more specifically, have been framed within the dual visual processing streams framework (Butler & Javitt, 2005). Therefore, to contextualize the forthcoming discussion of visual masking impairments among persons with SCZ, the major features of the dual visual processing streams model are first reviewed. Subcortically, the presence of dual visual processing streams arises at the level of the retinal ganglion cells and is characterised by the differential distribution of two cell types: M and P cells (Kim et al., 2021; Livingstone & Hubel, 1988). The M cells (also called parasol cells at the retinal level) project from the retinal ganglion layer to the lateral geniculate nucleus of the thalamus, to V1, and then preferentially to dorsal cortical areas, e.g. parietal cortex (Kim et al., 2021; Livingstone & Hubel, 1988). In contrast, the P channel cells (also called midget cells at the retinal level) project along similar subcortical pathways to preferentially innervate the ventral cortical areas, e.g. inferior temporal cortex (Livingstone & Hubel, 1988). At the population level, M and P cells also possess differing physiological properties and functions, (Kim et al., 2021; Livingstone & Hubel, 1988) which allow them to process complementary, and sometimes overlapping, aspects of visual scenes (Edwards et al., 2021). When combined, these form a stable percept of the environment. An important differentiating characteristic of population level responses of P versus M cells is their relative sensitivity to spatial versus temporal information (Derrington & Lennie, 1984; Livingstone & Hubel, 1988; Shapley, 1990). Here, spatial information denotes elements within a visual scene that reflect changes in luminance across space, while temporal information refers to elements that reflect changes in luminance across time. While there is considerable overlap in their response properties, population M cell responses posses greater sensitivity to higher temporal frequencies with fast transmission speeds and transient responses that provide temporal information such as location and movement (Edwards et al., 2021; Levitt et al., 2001; Livingstone & Hubel, 1988). Conversely, population P cell responses posses relatively superior spatial acuity with relatively slow transmission speeds and sustained responses. These cells primarily convey spatial information, such as the fine details of a visual scene (Livingstone & Hubel, 1988). Due to their differing response profiles, M and P channels are often referred to as transient and sustained channels, respectively (Breitmeyer, 1984). Another dimension along which M and P cells differ is their receptive field size (Breitmeyer, 1984). A cell's receptive field is a region of space upon which the presence of stimuli will alter cell firing (Breitmeyer, 1984). P cells have relatively smaller receptive fields compared to M cells, which allows for their advantage in processing high spatial frequencies (Breitmeyer, 1984).

Although these channels possess differential, but overlapping, cell properties and function, visual perception occurs through the coordinated use of both systems (Breitmeyer, 1984). For any given visual task, the relative recruitment of M and P channels is governed by internal, top-down (e.g. attention) and external, bottom-up (e.g. stimulus properties) inputs (Breitmeyer, 1984). Therefore, different visual tasks utilise different levels of input from M and P channels, which leads to differential interactions between these channels for visual perception to occur. For these reasons, assessment of visual system dysfunction must consider interactions between M and P channels. Moreover, given M and P cells largely projected into dorsal and ventral cortical visual processing streams, respectively, assessment of early visual system interactions may provide insight into the integrity of late-stage processing.

Schizophrenia and the Visual System

As previously discussed, visual information-processing deficits are recognised as core features of SCZ. These deficits have been characterised by research investigating visual perception in patients using a range of different psychophysical methods (see Ishikawa et al., 2006; Kéri et al., 1998; Lalor et al., 2012). Common to most of these studies, however, is the use of stimuli that differentially stimulate M and P channels. These studies have consistently shown that patients with SCZ perform worse than healthy controls in tasks that rely more extensively on M channel, as opposed to P channel, processing. For example, patients with SCZ are worse at discriminating low versus high spatial frequency (Kéri et al., 2002; O'Donnell et al., 2002). Since M cells are preferentially stimulated by low spatial frequency, these results suggest an M channel deficit. Other studies have also come to similar conclusions using different methodologies such as luminance pedestal (Delord et al., 2006), Vernier threshold (Kéri et al., 2004), and orientation tuning (Ishikawa et al., 2006) tasks. Further evidence from electrophysiology, specifically visual evoked potentials (VEPs), show that patients with SCZ have decrements in the amplitude of the occipital P1 component (Butler et al., 2007; Haenschel et al., 2007; Lalor et al., 2012). The P1 component is a positive deflection of an EEG wave and occurs around 100ms post-stimulus. Although there is still contention regarding the source of this component, it is consistently associated with reduced activity in the dorsal steam (Foxe et al., 2005). As such, M channel deficits have been implicated as a core characteristic of SCZ to the point where some researchers have suggested it to be a possible endophenotype to the disorder (Kéri et al., 2004).

Schizophrenia and Higher Cortical Regions

Schizophrenia-related M channel deficits have also been found beyond V1 and include higher cortical regions governing visual motion processing, visuomotor action, visual attention, and visual working memory (Chen, 2011; Fuller et al., 2006, 2009; King et al., 2008). In humans, cells with sensitivity to motion are first found in V1, however, due to their small receptive fields, each of these neurons only captures motion in a small part of the entire visual space (Edwards et al., 2021). As such, complex pooling and comparison of motion signals across space is required and occurs in higher cortical regions; primarily the medial temporal (MT) cortical area, also known as V5 (visual area 5) (Born & Bradley, 2005). At V5/ MT, features of motion including speed and direction are processed. Patients with SCZ show impairments in (1) speed discrimination and (2) decoding motion patterns across the visual field. Results from studies using varying visual stimuli (e.g. grating, single dots, and random dot patterns) have consistently demonstrated poorer performance in patients with SCZ compared to controls (Chen, Palafox, et al., 1999; Chen, Nakayama, et al., 1999; Clementz et al., 2007; Hong et al., 2009; Kim et al., 2006). An effective stimulus used to understand V5/MT motion pooling stage is the global motion stimulus developed by Newsome and Pare (1988). Global motion stimulus consists of a random-dot kinematogram where a portion of the dots move in the same direction (the signal dots), while the others in random directions (the noise dots). Moreover, the ratio between signal to noise is varied to produce varying levels of difficulty. Importantly, the signal dots are randomly assigned on initiation of each frame. Therefore, successful global motion decoding during low signal/high noise conditions requires the integration of local motion detector outputs across space and time (Edwards & Badcock, 1994). Patients with SCZ perform with reduced accuracy and require stronger signals to detect global motion relative to healthy controls (Chen et al., 2003; Slaghuis et al., 2007). Bennett and colleagues (2016) further demonstrated dorsal specific visual motion deficits by showing patients with SCZ were selectively impaired in global motion processing, a specific M channel/dorsal stream function; however, performed equivalent to controls on global form processing, a specific P channel/ventral stream function. In the realm of visuomotor action, King and colleagues (2008) showed that patients with SCZ perform worse than controls on dorsally driven visuomotor tasks compared to ventral visual tasks using size-contrast visual illusions. Taken together, these results show visual information-processing impairments in SCZ to arise at the subcortical (M and P channel) level and persist into higher cortical regions. Therefore, using methodological tools which are easily manipulated to not only assess the integrity of the M channel, but also the interaction of M and P channels are required. One of the most prominent methodological tools used to examine M and P channel integrity in patient populations is visual masking.

Visual Masking

As previously noted, visual masking tasks reduce or eliminate the visibility of a target stimulus via the presentation of a second stimulus (i.e., the mask) within a prescribed temporal window, either before (forward masking) or after (backward masking) the target (Breitmeyer & Öğmen, 2006). Backward masking, in particular, has captured significant scientific interest, owing perhaps to the counterintuitive nature of the phenomenon; that a stimulus *following* a target impedes perception of the target itself. In meta-contrast masking, a variant of backward masking, the target and mask do not occupy the same spatial locations, even if they are superimposed, see Figure 1. The fact that a spatially disparate mask attenuates target processing suggests that the visual system may not operate in a simple linear, feedforward fashion; if it were, then incoming stimuli would be processed such that proceeding stimuli, especially those that are spatially disparate, would not impact processing of preceding stimuli. However, occurrence of the meta-contrast masking phenomenon implies that differing cell inputs (both spatially and temporally) interact to achieve resultant perception (Breitmeyer & Öğmen, 2000). Several studies have examined the parameters governing meta-contrast masking and suggest that it occurs due to M cell inhibition of P cell activity (Albrecht et al., 2010; Breitmeyer & Öğmen, 2000; Ishikawa et al., 2006). Interestingly, patients with SCZ consistently perform worse on meta-contrast masking tasks compared to their healthy counterparts (Lee, 1985; Merritt et al., 1986; Rassovsky et al., 2004, 2005). These studies have reliably found patient performance to be characterised by reduced target discrimination/identification accuracy and higher thresholds - i.e., longer temporal gaps between mask and target, to achieve comparable performance to healthy controls. Given these results, and the importance of masking paradigms as an avenue toward greater understanding of the visual system and visual awareness, a brief review of meta-contrast masking is now provided.

Meta-contrast masking is a well-studied and distinctive form of backward masking. Meta-contrast masking employs temporal event sequences identical to backward masking i.e., the target stimulus precedes the masking stimulus. However, the spatial relationship between target and mask differs. In meta-contrast masking, the mask spatially surrounds the location of the preceding target, whereas in traditional backward masking, the mask spatially overlaps with the location of the preceding target. Refer to Figure 1 for illustrated examples.

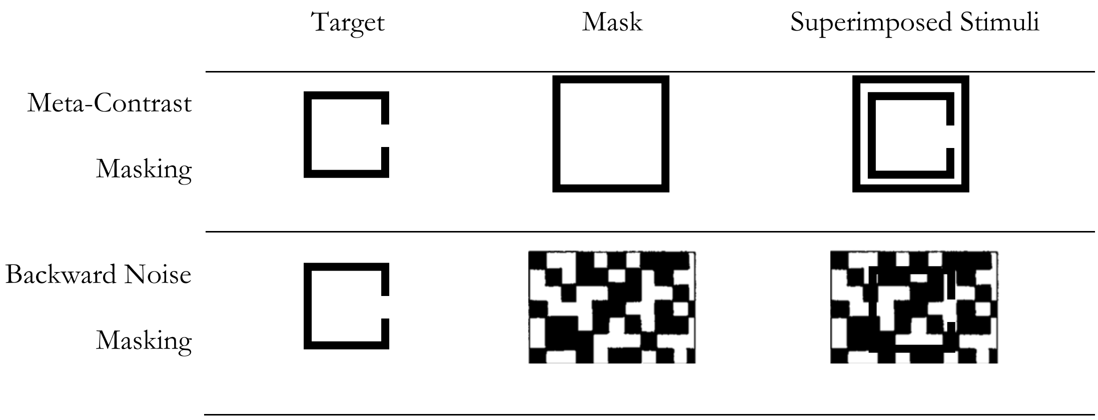


Figure A.1 Examples of target and mask in meta-contrast masking and backward noise masking. Notably, in meta-contrast masking, the target and mask are spatially exclusive even when superimposed.

Temporal variation between the onsets of the target and mask (i.e., Stimulus Onset Asynchrony; SOA), is typically manipulated during visual masking experiments (see Figure 2). A distinctive pattern of performance is observed as a function of SOA, which is referred to as a 'masking function'. Different visual masking paradigms produce different masking functions, such that the shape of a masking function indicates the type of visual mask employed.

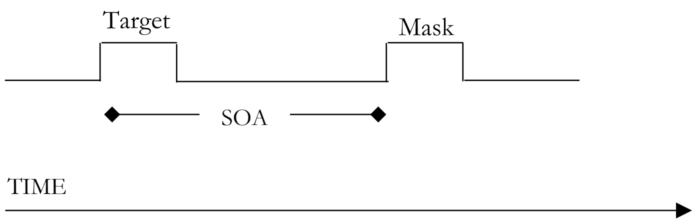


Figure A.2. Temporal sequence of events for backward and metacontrast masking.

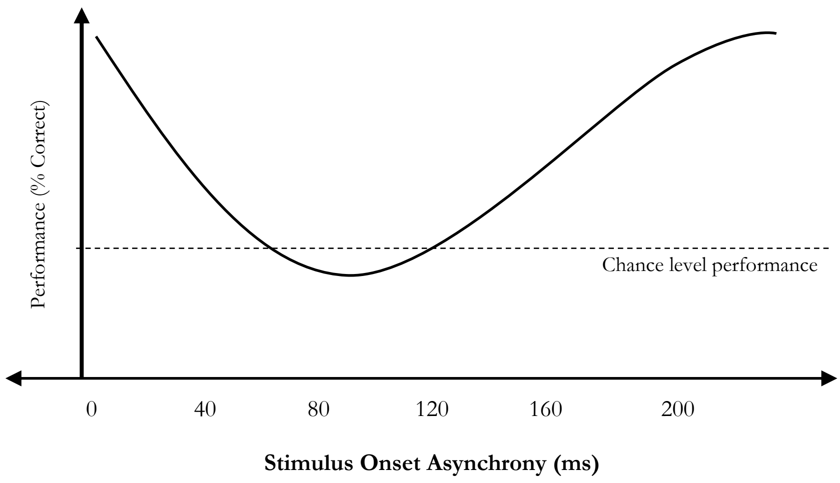


Figure A.3. Schematic of typical meta-contrast masking function.

Meta-contrast masking typically manifests a non-monotonic, specifically U-shaped, masking function (refer to Figure 3). This contrasts with studies investigating traditional backwards masking, where monotonic functions are the norm (Breitmeyer & Öğmen, 2006; Schiller & Smith, 1965). Furthermore, the trough of the non-monotonic function is reliably observed within the SOA window of 80-120ms (Breitmeyer & Öğmen, 2006), with only minor horizontal and vertical axis translations with target/mask manipulations. Upon further examination of Figure 3, a lag is observed following the mask onset and before target perception if fully attenuated. This implies a temporal delay of the processes underlying the masking phenomenon. Moreover, this delay is gradual, rather than instantaneous and/or sharply declining at a particular SOA. This gradual change is also observable on recovery; that is, at SOAs greater than 120ms, target perception is steadily reinstituted. Meta-contrast masking has also been observed both monoptically (where the target and mask are presented to the same eye) and dichoptically (where the target and mask are presented to different eyes simultaneously (see Kolers & Rosner, 1960; McFadden & Gummerman, 1973; Schiller & Smith, 1965). This suggests the mechanisms through which meta-contrast masking occurs is at a level where input from both eyes has converged (i.e., within V1 or downstream from this brain region).

Breitmeyer & Ganz's Sustained-Transient Dual-Channel Model (DCM)

The presence of the non-monotonic, U-shaped function as a manifestation of meta-contrast masking has sparked interest in the mechanisms giving rise to the form of this function. Several explanations have been proposed; arguably, however, the most dominant explanation has been the Dual Channel Model (DCM) by Breitmeyer and Ganz (1976). The temporal dynamics of the masking function led Breitmeyer and Ganz (1976) to propose that meta-contrast masking resulted from the inhibition of one visual channel input (P channel) by another (M channel). At the core of their logic is the differential temporal properties of the two channels. Recall, that M and P channels differ in their relative speed of transmission and persistence, with the M channel being faster (hence why P channel is often called *sustained* and M channel *transient*). The pattern of M channel and P channel activity elicited by a stimulus is shown in Figure 4. M channel responses can inhibit P channel responses. Accordingly, when a mask is presented after a target, the faster M channel activity elicited by the mask can 'catch up' and inhibit P channel activity from the target. Since P channel activity represents information about form (e.g., what the stimulus is), then perception of the target is most impaired when the M channel activity from the mask maximally overlaps with the P channel activity representing the target. This point of maximal overlap is when the mask appears about 80-120ms after the target. This is explained in more detail below.

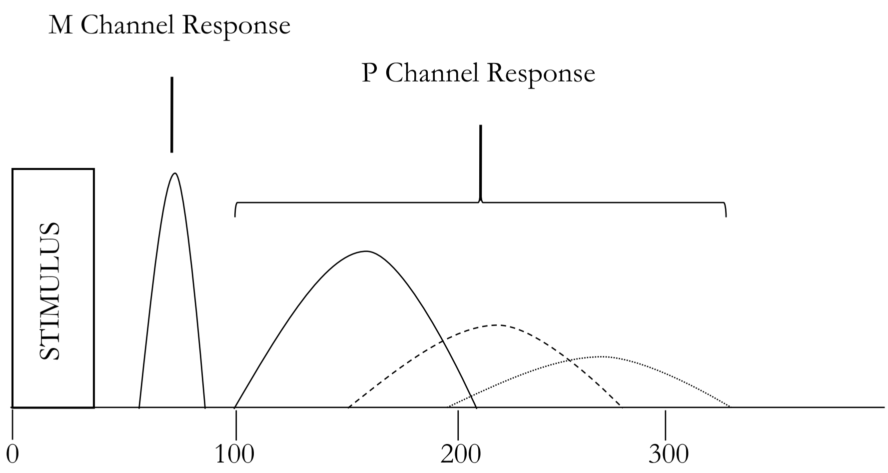


Figure A.4. Adapted from Breitmeyer & Ganz (1976). Temporal sequencing of visual cell activity in the human visual system at the level of the primary visual cortex. There is an initial spike in activity from the transient/M channel.

The overlap in the temporal offset of the mask and the onset of the target is posited to create interference between the visual channels. Specifically, the M channel response from mask onset inhibits the P channel response to the target. Figure 5 provides a schematic of the proposed interference.

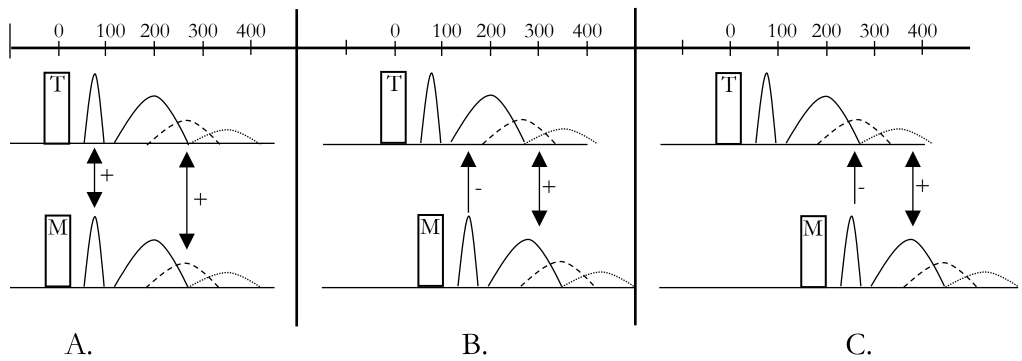


Figure A.5. Adapted from Breitmeyer & Ganz (1976). Schematic representation of the mechanisms of meta-contrast masking as proposed by the DCM, where the x axis is time in milliseconds. A. Simultaneous target and mask onset. B. Medium target and mask offset within masking window, SOA≈100ms. C. Large target and mask offset outside of masking window, SOAs ≥ 150ms.

The DCM proposes that at short SOAs (<80ms), M cell activity does not interfere with P cell activity, since there is minimal temporal overlap between inputs reaching V1. However, as SOA increases, there is a shift in the M cell activity from the mask, which moves closer and closer to that of the P cell activity from the preceding target (as in Figure 5B. Within the SOA window of 80-120ms, the M cell activity from the mask occurs simultaneously with P cell activity from the target. This causes inhibition of P cell activity by the M cells. Inhibition of the P channel results in loss of spatial information, which is required for successful perception of the target. Further increases in SOA leads to M cell activity from the mask to occur past the initial phases of target sustained activity; thereby leading to reduced masking. Breitmeyer and Ganz's (1976) DCM provides a straight-forward explanation of the U-shaped masking function that exemplifies the temporal characteristics of the mask's impact on target perception. In doing so, it suggests that meta-contrast masking is a result of interactions between early-stage visual processing inputs, i.e. M and P cell activity, which are directed up stream to higher cortical areas.

Schizophrenia and Visual Masking Research

Research using meta-contrast masking to assess visual information processing deficits in people with SCZ consistently reveals worse performance among patients relative to healthy controls (see Butler et al., 2003; Cadenhead et al., 1998; Green et al., 2003; Merritt et al., 1986; Rassovsky et al., 2004; Saccuzzo et al., 1996). These disparities in performance are characterised by patients (1) having a reduction in target discrimination/identification accuracy (Butler et al., 2003; Rassovsky et al., 2004), and (2) requiring longer SOAs to achieve comparable performance to controls (Butler et al., 2003; Rassovsky et al., 2004). These studies often attribute results to deficits in magnocellular (M) channel functioning in the patient group (Butler et al., 2003; McClure, 2001; Rassovsky et al., 2004). This is consistent with previous research using other methodologies to suggest M channel impairments, e.g., spatial frequency discrimination tasks (Kéri et al., 2002; O'Donnell et al., 2002), global form versus global motion tasks (Bennett et al., 2016), and EEG (Lalor et al., 2012). Conclusions from visual masking studies, and inferences of specific M channel deficits, are usually predicated on traditional theories of masking such as Breitmeyer and Ganz's (1976) DCM.

However, despite the face validity of these explanations, there is an inconsistency between the findings from data reflecting SCZ performance and the theoretical explanation of the phenomenon offered by Breitmeyer and Ganz's (1976) DCM. As previously presented, the DCM model proposes that meta-contrast masking, and the distinctive U-shaped masking function that it produces, results from M/transient channel suppression of P/sustained channel activity. Therefore, increased M channel activity (hyper-activity) would result in *greater* masking, while reduced M channel activity (hypo-activity) would lead to *reduced* masking. Applying this logic to Breitmeyer and Ganz's (1976) DCM, the observed results of increased masking among SCZ patients contradicts the known M channel impairment in this patient group. In fact, increased masking suggests enhanced M channel function. In light of this inconsistency, alternative frameworks for understanding visual masking and visual masking in SCZ are required.

With recent developments in technology allowing for more detailed examination of brain circuitry, the notion of feedforward and feedback connections in cortical areas has become an increasingly popular framework for understanding the visual system (Bullier, 2001; Tapia & Beck, 2014). In this vein, the following discussion will propose an alternative framework which utilises the feedforward/feedback framework to reconcile the SCZ-related meta-contrast masking deficits and the neurophysiology putatively underlying these deficits.

Alternative Explanation for SCZ Meta-Contrast Masking Performance

Anatomically, feedforward and feedback pathways are organised in a hierarchical and dynamic manner (Bullier, 2001; Lamme & Roelfsema, 2000). Electrophysiological studies have found that the initial 'feedforward sweep' from the retina to the primary visual cortex (V1) occurs approximately 55-70 ms post stimulus onset (Boehler et al., 2008; Di Russo et al., 2003;

Foxe et al., 2005; Vanni et al., 2001). From there, the feedforward connections continue onto temporal, parietal, and frontal regions via the dorsal and ventral streams (Bullier, 2001). Importantly, feedback connections, from hierarchically higher cortical regions to lower cortical regions, simultaneously propagate information laterally and backward (Bullier, 2001; Felleman & Van Essen, 1991; Tapia & Beck, 2014). Successful visual perception is, therefore, achieved by the integration of feedforward and feedback signals (Adámek et al., 2022). Neurophysiological and neuroanatomical evidence of such feedforward and feedback pathways have been substantiated via EEG, TMS, and single cell recording experiments (Bridgeman, 1980; Fahrenfort et al., 2007; Tapia & Beck, 2014). For example, in a seminal study by Pascual-Leone and Walsh (2001), perception of motion was only significantly compromised when application of TMS to early visual cortex (V1) occurred between 5 and 45ms *after* application to motion processing areas (V5/MT). These results were interpreted as TMS interference of fast feedback connections from V5/MT to V1. Similarly, shape visibility was compromised when TMS was applied to the intraparietal sulcus (IPS) at an SOA of 90ms (Koivisto et al., 2014) and 150ms post stimulus onset on lateral occipital cortex (LOC; Koivisto et al., 2011).

It is generally understood that feedback signals amplify and focus the activity of neurons in lower visual areas; however, the specific function of feedback processes and the mechanics upon which they operate are still under debate (Hupé et al., 1998). Martinez-Conde and Macknik (2007) suggests that feedback acts as attentional modulation of the feedforward sweep, while others propose that feedback modulates activity in early sensory areas because of expectations and/or minimisation of prediction error. The most accepted hypothesis proposes that feedback signals serve to guide input of details to initial, coarse visual representations generated by feedforward sweeps (Breitmeyer, 2007; Fahrenfort et al., 2007, 2008). This process is similar to that of the 'frame-and-fill' model of visual perception, which proposes that fast neural inputs reach higher order visual processing areas quickly and prime or 'frame' the slower neural inputs which arrive later and carry the 'fill' signal, i.e., richer information such as dense form and colour information (Chen et al., 2007). Application of the 'frame-and-fill' theory to masking is not a novel concept. For example, one of the theories put forward to explain object substitution masking (OSM; a phenomenon whereby a sparse, temporally trailing four dot mask interferes with target perception, despite differing contour and spatial location of target and mask) is that of neural re-entrant processing, or neural feedback (Di Lollo et al., 2000; Goodhew et al., 2012).

In the case of meta-contrast masking, a similar explanation has been proffered. Using the feedforward-feedback model, meta-contrast masking is hypothesized to occur through the interaction of feedforward and feedback signals. This dual process can be likened to the interactions between M and P channels, as presented the DCM, but the focus is displaced from early, pre-cortical visual pathways to late-level, cortical circuits. During masking, the feedback signals from the target are interrupted by the feedforward signals from the mask. This model is reminiscent of Öğmen's (1993) Retino-Cortical Dynamics (RECOD) model, which proposes that the real-time dynamics of visual perception occurs in three distinct phases; (1) a 'feed-forward dominant' phase where afferent signals from early visual system travel to later cortical areas, (2) a 'feedback dominant' phase where re-entrant signals contribute to visual processing, and (3) a 'reset' phase which allows smooth transitions between phase 1 and 2 (Purushothaman et al., 1998). Under this framework, meta-contrast masking, occurs due to disruption of the 'feedback dominant' phase for target perception by the 'feed-forward dominant' phase for mask perception. Using this new model, the consistent performance deficit in people with SCZ on meta-contrast masking tasks could be explained by defective feedback signals from either active or passive suppression of feedback from feedforward signals, rather than necessarily reflecting differential performance of the M and P cells.

Experimental Evidence for the Feedforward-Feedback Explanation of Meta-Contrast Masking Performance

Studies examining the neural effects of meta-contrast masking yield results consistent with the notion that masking occurs due to interference of feedback signals. These studies have used a range of different methods including single cell recording (see Martinez-Conde & Macknik, 2007) and figure-ground segmentation experiments (see (Lamme et al., 2002) in non-human primates. EEG recordings of humans produce findings consistent with those of single cell recordings. In a backward masking study by Fahrenfort and colleagues (2007), three distinct stages of visual processing, with regards to target detection, were identified. First, during the initial 110ms post-stimulus onset period, activity was recorded from the occipito-temporal region, which was interpreted (i.e., the initial feedforward sweep). Second, starting at 110ms post-stimulus onset, occipital activity was recorded, which was inferred to be reactivation of occipital areas due to feedback processing. Third, and finally, around 200-300ms post stimulus onset, another wave of activity from occipito-temporal regions. This was taken to reflect additional feedback processing. The presence of these three distinct stages is especially important when masking is applied. Fahrenfort and colleagues (2007) found that when masking was successful (i.e., when participants could not correctly detect the target), only the second and third stages of activity were no longer observed, while the first stage remained intact. This suggests that during backward masking, it is primarily feedback signals that are suppressed. Collectively, these studies support the feedforward-feedback account of meta-contrast masking.

The feedforward-feedback model provides an alternative and parsimonious explanation for, not only, the phenomenon of normative meta-contrast masking, but also the pattern of results found in patients with SCZ on these tasks. Moreover, the theory is consistent with recent advances in neurophysiological and neuroanatomical studies of the visual system. The use of a feedforward-feedback model for understanding visual masking, especially, meta-contrast masking, has significant implications both for neurotypical visual perception and also clinical presentations demonstrating visual perception deficits at its core (e.g., SCZ). For neurotypical visual perception, it suggests that the visual system can no longer be conceptualised as an arrangement of cells which receive input from the environment and processes it in a unidirectional, feedforward manner through the hierarchy of cortical regions. Rather, there are substantial forward and backward interactions between early and late cortical regions which, in culmination with top-down and bottom-up influences, allow for visual processing and subsequent responses to occur.

Given the implications for neurotypical visual processing, research into disorders such as SCZ, which have visual processing deficits at its core, should consider not only the impact of early visual processing streams (i.e., M and P channels) on later cortical processing areas, but also the influence of feedforward and feedback processing, especially within the dorsal stream. To date, there are no known methods available to directly manipulate feedforward and/or feedback pathways involved in visual processing to assess causality. However, from an ecological perspective, vision is a tool which allows us to interact with the environment (Goodale & Milner, 1992). Hence, investigation of the impact of dorsal stream dysfunction, possibly due to impaired feedforward/feedback networks, on visually guided, volitional, goal-directed action may further elucidate visual processing deficits in SCZ.

Other Explanations

Although the feedforward-feedback model provides a logical and sound framework upon which both meta-contrast masking studies and those with its application to SCZ can be interpreted, other models have also been offered which attempt to resolve the inconsistency in the literature. Once such model is Herzog and colleagues' (2013) Neuromodulation Model, which proposes that the phenomenon of masking is not due to visual deficits such as M channel impairment or feedback suppression, but rather an instantiation of a general deficit in neuromodulation. Here, neuromodulation refers to the physiological process whereby a given neuron uses one or more neurotransmitters (e.g., acetylcholine), to regulate diverse populations of neurons (e.g., the cholinergic system). This model posits that, when fleeting task-relevant elements (e.g., targets) occur, the brain must amplify input information in order to consciously detect and/or identify the target; this process of amplification is hypothesised to be controlled by neuromodulators or attention (M. Herzog et al., 2013; M. H. Herzog & Brand, 2015). Support for this approach comes from findings that attention can increase neuronal responses in visual areas of both human (Gandhi et al., 1999) and non-human primates (Treue & Maunsell, 1996), especially, when stimuli are weak. Furthermore, EEG recordings during masking of SCZ patients have found diminished neuronal responses to targets even prior mask onset, suggesting diminished enhancement of neural activity from the target (Plomp et al., 2013). However, it should be noted that no baseline measure of neural activity without masking was measured; therefore, a fair comparison of no masking, control masking, and patient masking cannot be made. Without such a comparison, it is difficult to assess the validity of the neuromodulation model as it would be expected that an explanation for deficits in masking for patients with SCZ would be an amplification of the phenomenon of masking in controls. If diminished neuromodulation is indeed the underlying cause of meta-contrast masking, then reductions in neuronal activity of control subjects would also be observed.

Summary, Future Directions & Recommendations

Visual masking, in particular, meta-contrast masking, is an experimental method widely used to understanding neural processing among people with SCZ. Consistently, studies have found patients with SCZ to experience greater masking deficits in the form of reductions in target discrimination/identification accuracy and the need for longer SOAs to achieve comparable performance to controls. Despite relatively robust results, explanations of the mechanisms through which masking occurs are questionable, with significant inconsistencies between theoretical frameworks used to understand masking and the conclusions drawn from studies using masking to explore SCZ. The dominant framework used to explain meta-contrast masking in the past few decades has been the Breitmeyer and Ganz's (1976) DCM, which suggests meta-contrast masking to occur due to interruptions between systems in early visual processing, i.e., M-channel on P-channel suppression. However, this model predicts that greater masking results from enhanced M-channel activity, whereas the greater masking observed empirically in SCZ is often attributed to *reduced* M-channel activity, a phenomenon demonstrated via other measures in SCZ. This contradiction highlights the need for alternative theoretical frameworks for understanding the pattern of masking observed in SCZ.

Recent findings from visual neurophysiology and neuroanatomy suggest a higher cortical feedforward-feedback framework to provide are more convincing explanation able to accommodate for research results. This model proposes that meta-contrast masking arises due to suppression of feedback signals from target stimuli because of the feedforward signals produced by the onset of a mask. Using a 'frame-and-fill' approach, feedback signals are believed to provide spatial sharpening, required for perception, of input from the initial feedforward sweep. Although some research has endeavoured to elucidate feedforward and feedback circuitry, significant advances in technology and methodology is needed before this model is able to provide any specificity in how meta-contrast masking occurs in the brain. Nevertheless, the implication of feedforward and feedback signals in meta-contrast masking suggest that successful visual processing goes beyond a simple input/output model and that such impairments in SCZ more likely due to widespread network or connection issues rather than acute structural damage. In this vein, adopting a neurodevelopmental approach to understanding visual information processing deficits in SCZ is recommended. Given technological limitations for investigating feedforward and feedback pathway integrity, reorienting toward the functional and ecological impacts of visual information processing deficits in SCZ using a neurodevelopmental approach is needed.

References

Adámek, P., Langová, V., & Horáček, J. (2022). Early-stage visual perception impairment in schizophrenia, bottom-up and back again. *Schizophrenia*, *8*(1), 1–12. https://doi.org/10.1038/s41537-022-00237-9

Albrecht, T., Klapötke, S., & Mattler, U. (2010). Individual differences in metacontrast masking are enhanced by perceptual learning. *Consciousness and Cognition*, *19*(2), 656–666. https://doi.org/10.1016/j.concog.2009.12.002

Amiaz, R., Vainiger, D., Gershon, A. A., Weiser, M., Lavidor, M., & Javitt, D. C. (2016). Applying Transcranial Magnetic Stimulation (TMS) Over the Dorsal Visual Pathway Induces Schizophrenia-like Disruption of Perceptual Closure. *Brain Topography*, *29*(4), 552–560. https://doi.org/10.1007/s10548-016-0487-1

Bennett, D., Dluzniak, A., Cropper, S. J., Partos, T., Sundram, S., & Carter, O. (2016). Selective impairment of global motion integration, but not global form detection, in schizophrenia and bipolar affective disorder. *Schizophrenia Research: Cognition*, *3*, 11–14. https://doi.org/10.1016/j.scog.2015.11.003

Boehler, C. N., Schoenfeld, M. A., Heinze, H.-J., & Hopf, J.-M. (2008). Rapid recurrent processing gates awareness in primary visual cortex. *Proceedings of the National Academy of Sciences*, *105*(25), 8742–8747. https://doi.org/10.1073/pnas.0801999105

Born, R. T., & Bradley, D. C. (2005). Structure and function of visual area MT. *Annual Review of Neuroscience*, *28*(1), 157–189. https://doi.org/10.1146/annurev.neuro.26.041002.131052

Breitmeyer, B. G. (1984). *Vision: Visual Masking. An Integrative Approach*. Oxford University Press. https://www.science.org/doi/10.1126/science.228.4701.864.b

Breitmeyer, B. G. (2007). Visual masking: Past accomplishments, present status, future developments. *Advances in Cognitive Psychology*, *3*(1–2), 9–20. https://doi.org/10.2478/v10053-008-0010-7

Breitmeyer, B. G., & Ganz, L. (1976). Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression, and information processing. *Psychological Review*, *83*(1), 1–36. https://doi.org/10.1037/0033-295X.83.1.1

Breitmeyer, B. G., & Öğmen, H. (2000). Recent models and findings in visual backward masking: A comparison, review, and update. *Perception & Psychophysics*, *62*(8), 1572–1595. https://doi.org/10.3758/BF03212157

Breitmeyer, B., & Öğmen, H. (2006). *Visual Masking: Time Slices Through Conscious and Unconscious Vision*. OUP Oxford.

Bridgeman, B. (1980). Temporal response characteristics of cells in monkey striate cortex measured with metacontrast masking and brightness discrimination. *Brain Research*, *196*(2), 347–364. https://doi.org/10.1016/0006-8993(80)90400-X

Bruchmann, M., Breitmeyer, B. G., & Pantev, C. (2010). Metacontrast masking within and between visual channels: Effects of orientation and spatial frequency contrasts. *Journal of Vision*, *10*(6), 12. https://doi.org/10.1167/10.6.12

Bullier, J. (2001). Integrated model of visual processing. *Brain Research Reviews*, *36*(2), 96–107. https://doi.org/10.1016/S0165-0173(01)00085-6

Butler, P. D., DeSanti, L. A., Maddox, J., Harkavy-Friedman, J. M., Amador, X. F., Goetz, R. R., Javitt, D. C., & Gorman, J. M. (2003). Visual backward-masking deficits in schizophrenia: Relationship to visual pathway function and symptomatology. *Schizophrenia Research*, *59*(2), 199–209. https://doi.org/10.1016/S0920-9964(01)00341-3

Butler, P. D., & Javitt, D. C. (2005). Early-stage visual processing deficits in schizophrenia. *Current Opinion in Psychiatry*, *18*(2), 151–157.

Butler, P. D., Martinez, A., Foxe, J. J., Kim, D., Zemon, V., Silipo, G., Mahoney, J., Shpaner, M., Jalbrzikowski, M., & Javitt, D. C. (2007). Subcortical visual dysfunction in schizophrenia drives secondary cortical impairments. *Brain*, *130*(2), 417–430. https://doi.org/10.1093/brain/awl233

Butler, P. D., Silverstein, S. M., & Dakin, S. C. (2008). Visual Perception and Its Impairment in Schizophrenia. *Biological Psychiatry*, *64*(1), 40–47. https://doi.org/10.1016/j.biopsych.2008.03.023

Butler, P. D., Zemon, V., Schechter, I., Saperstein, A. M., Hoptman, M. J., Lim, K. O., Revheim, N., Silipo, G., & Javitt, D. C. (2005). Early-Stage Visual Processing and Cortical Amplification Deficits in Schizophrenia. *Archives of General Psychiatry*, *62*(5), 495–504. https://doi.org/10.1001/archpsyc.62.5.495

Cadenhead, K. S., Serper, Y., & Braff, D. L. (1998). Transient versus sustained visual channels in the visual backward masking deficits of schizophrenia patients. *Biological Psychiatry*, *43*(2), 132–138. https://doi.org/10.1016/S0006-3223(97)00316-8

Chen, C.-M., Lakatos, P., Shah, A. S., Mehta, A. D., Givre, S. J., Javitt, D. C., & Schroeder, C. E. (2007). Functional Anatomy and Interaction of Fast and Slow Visual Pathways in Macaque Monkeys. *Cerebral Cortex*, *17*(7), 1561–1569. https://doi.org/10.1093/cercor/bhl067

Chen, Y. (2011). Abnormal Visual Motion Processing in Schizophrenia: A Review of Research Progress. *Schizophrenia Bulletin*, *37*(4), 709–715. https://doi.org/10.1093/schbul/sbr020

Chen, Y., Nakayama, K., Levy, D. L., Matthysse, S., & Holzman, P. S. (1999). Psychophysical isolation of a motion-processing deficit in schizophrenics and their relatives and its association with impaired smooth pursuit. *Proceedings of the National Academy of Sciences*, *96*(8), 4724–4729. https://doi.org/10.1073/pnas.96.8.4724

Chen, Y., Nakayama, K., Levy, D., Matthysse, S., & Holzman, P. (2003). Processing of global, but not local, motion direction is deficient in schizophrenia. *Schizophrenia Research*, *61*(2), 215–227. https://doi.org/10.1016/S0920-9964(02)00222-0

Chen, Y., Palafox, G. P., Nakayama, K., Levy, D. L., Matthysse, S., & Holzman, P. S. (1999). Motion Perception in Schizophrenia. *Archives of General Psychiatry*, *56*(2), 149–154. https://doi.org/10.1001/archpsyc.56.2.149

Chkonia, E., Roinishvili, M., Makhatadze, N., Tsverava, L., Stroux, A., Neumann, K., Herzog, M. H., & Brand, A. (2010). The Shine-Through Masking Paradigm Is a Potential Endophenotype of Schizophrenia. *PLOS ONE*, *5*(12), e14268. https://doi.org/10.1371/journal.pone.0014268

Clementz, B. A., McDowell, J. E., & Dobkins, K. R. (2007). Compromised speed discrimination among schizophrenia patients when viewing smooth pursuit targets. *Schizophrenia Research*, *95*(1), 61–64. https://doi.org/10.1016/j.schres.2007.05.043

Delord, S., Ducato, M. G., Pins, D., Devinck, F., Thomas, P., Boucart, M., & Knoblauch, K. (2006). Psychophysical assessment of magno- and parvocellular function in schizophrenia. *Visual Neuroscience*, *23*(3–4), 645–650. https://doi.org/10.1017/S0952523806233017

Derrington, A. M., & Lennie, P. (1984). Spatial and temporal contrast sensitivities of neurones in lateral geniculate nucleus of macaque. *The Journal of Physiology*, *357*(1), 219–240. https://doi.org/10.1113/jphysiol.1984.sp015498

Di Lollo, V., Enns, J. T., & Rensink, R. A. (2000). Competition for consciousness among visual events: The psychophysics of reentrant visual processes. *Journal of Experimental Psychology: General*, *129*(4), 481–507. https://doi.org/10.1037/0096-3445.129.4.481

Di Russo, F., Martínez, A., & Hillyard, S. A. (2003). Source Analysis of Event-related Cortical Activity during Visuo-spatial Attention. *Cerebral Cortex*, *13*(5), 486–499. https://doi.org/10.1093/cercor/13.5.486

Edwards, M., & Badcock, D. R. (1994). Global motion perception: Interaction of the ON and OFF pathways. *Vision Research*, *34*(21), 2849–2858. https://doi.org/10.1016/0042-6989(94)90054-X

Edwards, M., Goodhew, S. C., & Badcock, D. R. (2021). Using perceptual tasks to selectively measure magnocellular and parvocellular performance: Rationale and a user's guide.

Psychonomic Bulletin & Review, *28*(4), 1029–1050. https://doi.org/10.3758/s13423-020-01874-w

Fahrenfort, J. J., Scholte, H. S., & Lamme, V. A. F. (2007). Masking Disrupts Reentrant Processing in Human Visual Cortex. *Journal of Cognitive Neuroscience*, *19*(9), 1488–1497. https://doi.org/10.1162/jocn.2007.19.9.1488

Fahrenfort, J. J., Scholte, H. S., & Lamme, V. A. F. (2008). The spatiotemporal profile of cortical processing leading up to visual perception. *Journal of Vision*, *8*(1), 12. https://doi.org/10.1167/8.1.12

Felleman, D. J., & Van Essen, D. C. (1991). Distributed hierarchical processing in the primate cerebral cortex. *Cerebral Cortex (New York, N.Y.*, *1*(1), 1–47. https://doi.org/10.1093/cercor/1.1.1-a

Foxe, J. J., Murray, M. M., & Javitt, D. C. (2005). Filling-in in Schizophrenia: A High-density Electrical Mapping and Source-analysis Investigation of Illusory Contour Processing. *Cerebral Cortex*, *15*(12), 1914–1927. https://doi.org/10.1093/cercor/bhi069

Fuller, R. L., Luck, S. J., Braun, E. L., Robinson, B. M., McMahon, R. P., & Gold, J. M. (2006). Impaired control of visual attention in schizophrenia. *Journal of Abnormal Psychology*, *115*(2), 266–275. https://doi.org/10.1037/0021-843X.115.2.266

Fuller, R. L., Luck, S. J., Braun, E. L., Robinson, B. M., McMahon, R. P., & Gold, J. M. (2009). Impaired visual working memory consolidation in schizophrenia. *Neuropsychology*, *23*(1), 71–80. https://doi.org/10.1037/a0013854

Gandhi, S. P., Heeger, D. J., & Boynton, G. M. (1999). Spatial attention affects brain activity in human primary visual cortex. *Proceedings of the National Academy of Sciences*, *96*(6), 3314–3319. https://doi.org/10.1073/pnas.96.6.3314

Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. *Trends in Neurosciences*, *15*(1), 20–25. https://doi.org/10.1016/0166-2236(92)90344-8

Goodhew, S. C., Dux, P. E., Lipp, O. V., & Visser, T. A. W. (2012). Understanding recovery from object substitution masking. *Cognition*, *122*(3), 405–415. https://doi.org/10.1016/j.cognition.2011.11.010

Green, M. F., Nuechterlein, K. H., Breitmeyer, B., Tsuang, J., & Mintz, J. (2003). Forward and backward visual masking in schizophrenia: Influence of age. *Psychological Medicine*, *33*(5), 887–895. https://doi.org/10.1017/S003329170200716X

Green, M. F., Nuechterlein, K. H., & Mintz, J. (1994). Backward Masking in Schizophrenia and Mania: I. Specifying a Mechanism. *Archives of General Psychiatry*, *51*(12), 939–944. https://doi.org/10.1001/archpsyc.1994.03950120011003

Haenschel, C., Bittner, R. A., Haertling, F., Rotarska-Jagiela, A., Maurer, K., Singer, W., & Linden, D. E. J. (2007). Contribution of Impaired Early-Stage Visual Processing to Working Memory Dysfunction in Adolescents With Schizophrenia: A Study With Event-Related Potentials and Functional Magnetic Resonance Imaging. *Archives of General Psychiatry*, *64*(11), 1229–1240. https://doi.org/10.1001/archpsyc.64.11.1229

Herzog, M. H., & Brand, A. (2015). Visual masking & schizophrenia. *Schizophrenia Research: Cognition*, *2*(2), 64–71. https://doi.org/10.1016/j.scog.2015.04.001

Herzog, M., Roinishvili, M., Chkonia, E., & Brand, A. (2013). Schizophrenia and visual backward masking: A general deficit of target enhancement. *Frontiers in Psychology*, *4*. https://www.frontiersin.org/articles/10.3389/fpsyg.2013.00254

Hong, L. E., Turano, K. A., O'Neill, H. B., Hao, L., Wonodi, I., McMahon, R. P., & Thaker, G. K. (2009). Is Motion Perception Deficit in Schizophrenia a Consequence of Eye-Tracking Abnormality? *Biological Psychiatry*, *65*(12), 1079–1085. https://doi.org/10.1016/j.biopsych.2008.10.021

Hupé, J. M., James, A. C., Payne, B. R., Lomber, S. G., Girard, P., & Bullier, J. (1998). Cortical feedback improves discrimination between figure and background by V1, V2 and V3 neurons. *Nature*, *394*(6695), 784–787. https://doi.org/10.1038/29537

Ishikawa, A., Shimegi, S., & Sato, H. (2006). Metacontrast masking suggests interaction between visual pathways with different spatial and temporal properties. *Vision Research*, *46*(13), 2130–2138. https://doi.org/10.1016/j.visres.2005.12.013

Kéri, S., Antal, A., Szekeres, G., Benedek, G., & Janka, Z. (2002). Spatiotemporal Visual Processing in Schizophrenia. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *14*(2), 190–196. https://doi.org/10.1176/jnp.14.2.190

Kéri, S., Antal, A., Szekeres, G., Benedek, G., Janka, Z., & Szent-Györgyi, A. (1998). Transient visual channel functions in schizophrenia. *International Journal of Psychophysiology*, *1–2*(30), 170. https://doi.org/10.1016/S0167-8760(98)90442-1

Kéri, S., Kelemen, O., Benedek, G., & Janka, Z. (2004). Vernier Threshold in Patients With Schizophrenia and in Their Unaffected Siblings. *Neuropsychology*, *18*(3), 537–542. https://doi.org/10.1037/0894-4105.18.3.537

Kéri, S., Szendi, I., Kelemen, O., Benedek, G., & Janka, Z. (2001). Remitted schizophrenia-spectrum patients with spared working memory show information processing abnormalities. *European Archives of Psychiatry and Clinical Neuroscience*, *251*(2), 60–65. https://doi.org/10.1007/s004060170054

Kim, D., Wylie, G., Pasternak, R., Butler, P. D., & Javitt, D. C. (2006). Magnocellular contributions to impaired motion processing in schizophrenia. *Schizophrenia Research*, *82*(1), 1–8. https://doi.org/10.1016/j.schres.2005.10.008

Kim, U. S., Mahroo, O. A., Mollon, J. D., & Yu-Wai-Man, P. (2021). Retinal Ganglion Cells—Diversity of Cell Types and Clinical Relevance. *Frontiers in Neurology*, *12*. https://www.frontiersin.org/articles/10.3389/fneur.2021.661938

King, J. P., Christensen, B. K., & Westwood, D. A. (2008). Grasping behavior in schizophrenia suggests selective impairment in the dorsal visual pathway. *Journal of Abnormal Psychology*, *117*(4), 799–811. https://doi.org/10.1037/a0013500

Koelkebeck, K., Ohrmann, P., Hetzel, G., Arolt, V., & Suslow, T. (2005). Visual backward masking: Deficits in locating targets are specific to schizophrenia and not related to intellectual decline. *Schizophrenia Research*, *78*(2), 261–268. https://doi.org/10.1016/j.schres.2005.01.017

Koivisto, M., Lähteenmäki, M., Kaasinen, V., Parkkola, R., & Railo, H. (2014). Overlapping activity periods in early visual cortex and posterior intraparietal area in conscious visual shape perception: A TMS study. *NeuroImage*, *84*, 765–774. https://doi.org/10.1016/j.neuroimage.2013.09.051

Koivisto, M., Railo, H., Revonsuo, A., Vanni, S., & Salminen-Vaparanta, N. (2011). Recurrent Processing in V1/V2 Contributes to Categorization of Natural Scenes. *Journal of Neuroscience*, *31*(7), 2488–2492. https://doi.org/10.1523/JNEUROSCI.3074-10.2011

Kolers, P. A., & Rosner, B. S. (1960). On Visual Masking (Metacontrast): Dichoptic Observation. *The American Journal of Psychology*, *73*(1), 2–21. https://doi.org/10.2307/1419113

Lalor, E. C., De Sanctis, P., Krakowski, M. I., & Foxe, J. J. (2012). Visual sensory processing deficits in schizophrenia: Is there anything to the magnocellular account? *Schizophrenia Research*, *139*(1), 246–252. https://doi.org/10.1016/j.schres.2012.05.022

Lamme, V. A. F., & Roelfsema, P. R. (2000). The distinct modes of vision offered by feedforward and recurrent processing. *Trends in Neurosciences*, *23*(11), 571–579. https://doi.org/10.1016/S0166-2236(00)01657-X

Lamme, V. A. F., Zipser, K., & Spekreijse, H. (2002). Masking Interrupts Figure-Ground Signals in V1. *Journal of Cognitive Neuroscience*, *14*(7), 1044–1053. https://doi.org/10.1162/089892902320474490

Lee, J., Nuechterlein, K. H., Subotnik, K. L., Sugar, C. A., Ventura, J., Gretchen-Doorly, D., Kelly, K., & Green, M. F. (2008). Stability of visual masking performance in recent-onset schizophrenia: An 18-month longitudinal study. *Schizophrenia Research*, *103*(1), 266–274. https://doi.org/10.1016/j.schres.2008.03.005

Lee, R. (1985). *A comparison of forward and backward masking in schizophrenics and controls* [Doctoral Dissertation]. Bowling Green State University.

Levitt, J. B., Schumer, R. A., Sherman, S. M., Spear, P. D., & Movshon, J. A. (2001). Visual Response Properties of Neurons in the LGN of Normally Reared and Visually Deprived Macaque Monkeys. *Journal of Neurophysiology*, *85*(5), 2111–2129. https://doi.org/10.1152/jn.2001.85.5.2111

Livingstone, M., & Hubel, D. (1988). Segregation of form, color, movement, and depth: Anatomy, physiology, and perception. *Science (New York, N.Y.)*, *240*(4853), 740–749. https://doi.org/10.1126/science.3283936

Martínez, A., Hillyard, S. A., Dias, E. C., Hagler, D. J., Butler, P. D., Guilfoyle, D. N., Jalbrzikowski, M., Silipo, G., & Javitt, D. C. (2008). Magnocellular Pathway Impairment in Schizophrenia: Evidence from Functional Magnetic Resonance Imaging. *Journal of Neuroscience*, *28*(30), 7492–7500. https://doi.org/10.1523/JNEUROSCI.1852-08.2008

Martinez-Conde, S., & Macknik, S. L. (2007). Mind tricks. *Nature*, *448*(7152), 414–414. https://doi.org/10.1038/448414a

McClure, R. K. (2001). The visual backward masking deficit in Schizophrenia. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *25*(2), 301–311. https://doi.org/10.1016/S0278-5846(00)00166-4

McFadden, D., & Gummerman, K. (1973). Monoptic and dichoptic metacontrast across the vertical meridian. *Vision Research*, *13*(1), 185–196. https://doi.org/10.1016/0042-6989(73)90173-9

Merritt, R. D., Balogh, D. W., & Leventhal, D. B. (1986). Use of a metacontrast and a paracontrast procedure to assess the visual information processing of hypothetically schizotypic college students. *Journal of Abnormal Psychology*, *95*(1), 74–80. https://doi.org/10.1037/0021-843X.95.1.74

Newsome, W. T., & Pare, E. B. (1988). A selective impairment of motion perception following lesions of the middle temporal visual area (MT). *Journal of Neuroscience*, *8*(6), 2201–2211. https://doi.org/10.1523/JNEUROSCI.08-06-02201.1988

O'Donnell, B. F., Potts, G. F., Nestor, P. G., Stylianopoulos, K. C., Shenton, M. E., & McCarley, R. W. (2002). Spatial frequency discrimination in schizophrenia. *Journal of Abnormal Psychology*, *111*(4), 620–625. https://doi.org/10.1037/0021-843X.111.4.620

Öğmen, H. (1993). A neural theory of retino-cortical dynamics. *Neural Networks*, *6*(2), 245–273. https://doi.org/10.1016/0893-6080(93)90020-W

Ogmen, H., Breitmeyer, B. G., & Melvin, R. (2003). The what and where in visual masking. *Vision Research*, *43*(12), 1337–1350. https://doi.org/10.1016/S0042-6989(03)00138-X

Pascual-Leone, A., & Walsh, V. (2001). Fast Backprojections from the Motion to the Primary Visual Area Necessary for Visual Awareness. *Science*, *292*(5516), 510–512. https://doi.org/10.1126/science.1057099

Plomp, G., Roinishvili, M., Chkonia, E., Kapanadze, G., Kereselidze, M., Brand, A., & Herzog, M. H. (2013). Electrophysiological Evidence for Ventral Stream Deficits in Schizophrenia Patients. *Schizophrenia Bulletin*, *39*(3), 547–554. https://doi.org/10.1093/schbul/sbr175

Purushothaman, G., Öğmen, H., Chen, S., & Bedell, H. E. (1998). Motion deblurring in a neural network model of retino-cortical dynamics1This study was presented in part at the 1995 annual meeting of ARVO and appeared in abstract form in Investigative Ophthalmology and Visual Science 36, S52.1. *Vision Research*, *38*(12), 1827–1842. https://doi.org/10.1016/S0042-6989(97)00350-7

Rassovsky, Y., Green, M. F., Nuechterlein, K. H., Breitmeyer, B., & Mintz, J. (2004). Paracontrast and metacontrast in schizophrenia: Clarifying the mechanism for visual masking deficits. *Schizophrenia Research*, *71*(2), 485–492. https://doi.org/10.1016/j.schres.2004.02.018

Rassovsky, Y., Green, M. F., Nuechterlein, K. H., Breitmeyer, B., & Mintz, J. (2005). Modulation of Attention During Visual Masking in Schizophrenia. *American Journal of Psychiatry*, *162*(8), 1533–1535. https://doi.org/10.1176/appi.ajp.162.8.1533

Saccuzzo, D. S., Cadenhead, K. S., & Braff, D. L. (1996). Backward versus forward visual masking deficits in schizophrenic patients: Centrally, not peripherally, mediated? *The American Journal of Psychiatry*, *153*(12), 1564–1570. https://doi.org/10.1176/ajp.153.12.1564

Schechter, I., Butler, P. D., Silipo, G., Zemon, V., & Javitt, D. C. (2003). Magnocellular and parvocellular contributions to backward masking dysfunction in schizophrenia. *Schizophrenia Research*, *64*(2), 91–101. https://doi.org/10.1016/S0920-9964(03)00008-2

Schiller, P. H., & Smith, M. C. (1965). A comparison of forward and backward masking. *Psychonomic Science*, *3*(1), 77–78. https://doi.org/10.3758/BF03343028

Shapley, R. (1990). Visual sensitivity and parallel retinocortical channels. *Annual Review of Psychology*, *41*, 635–658. https://doi.org/10.1146/annurev.ps.41.020190.003223

Slaghuis, W. A. (1998). Contrast sensitivity for stationary and drifting spatial frequency gratings in positive- and negative-symptom schizophrenia. *Journal of Abnormal Psychology*, *107*(1), 49–62. https://doi.org/10.1037/0021-843X.107.1.49

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*(3), 415–426. https://doi.org/10.1007/s00221-007-1003-3

Tapia, E., & Beck, D. M. (2014). Probing feedforward and feedback contributions to awareness with visual masking and transcranial magnetic stimulation. *Frontiers in Psychology*, *5*. https://www.frontiersin.org/articles/10.3389/fpsyg.2014.01173

Treue, S., & Maunsell, J. H. R. (1996). Attentional modulation of visual motion processing in cortical areas MT and MST. *Nature*, *382*(6591), 539–541. https://doi.org/10.1038/382539a0

Vanni, S., Tanskanen, T., Seppä, M., Uutela, K., & Hari, R. (2001). Coinciding early activation of the human primary visual cortex and anteromedial cuneus. *Proceedings of the National Academy of Sciences*, *98*(5), 2776–2780. https://doi.org/10.1073/pnas.041600898

APPENDIX B

Study 1 Supplementary Materials (Chapter 3)

B.1 Study 1 Participant Questionnaire

Australian National University

Participant Information Sheet

Researcher:
The current study is being undertaken by Elizabeth Shen (Clinical Psychology PhD candidate) and Associate Professor Bruce Christensen from the Research School of Psychology at the Australian National University (ANU) College of Health and Medicine.

Project Title: Elucidating the integrity of dorsal stream functioning via visually guided movement

General Outline of the Project:

- **Description and Methodology:** This project explores the mechanisms underlying visual perception during visually guided movement, i.e. any movement involving continuous visual feedback. You will be asked to complete several questionnaires and three experimental tasks, in which your dominant arm will be connected to a motion tracker and you will be instructed to perform a series of movements toward a specified target. Your reaction time and finger position will be recorded. The investigator will give you detailed instructions about the task. There are no known harmful consequences of participating in this task.
- **Participants:** Data from approximately 90 participants will be collected from the general university population, and participation will take approximately 90 minutes in total.
- **Use of Data and Feedback:** The data from this study will contribute to the researcher's PhD thesis, and associated research publications. Results of the study will be made available to participations upon completion of research.
- **Project Funding:** This project is funded by the Research School of Psychology at the ANU.

Participant Involvement:

- **Voluntary Participation & Withdrawal:** Participation in the project is voluntary and you may, without negative consequences, decline to take part, withdraw from the research, and/or refuse to answer questions without providing an explanation at any time until the work is prepared for publication. If you do withdraw, your data will be destroyed.
- **What does participation in the research entail?** If you decide to participate, you will meet individually in a private room with the researcher. You will be asked to complete several short questionnaires and then three tasks in which your dominant arm will be connected to a motion tracker and you will be instructed to make a series of movements toward a target.
- **Transfer of Questionnaire Data from Previous Studies (if applicable):** If you have already participated in the study "The Impact of Personality, Hand Position and Perceptual Expectations on Visual Perception" (Protocol 2016/491), you can elect to have your questionnaire data transferred to this study. Transfer of data will be confidential and de-identified. This choice is entirely optional and will not impact on your participation and reimbursement in either study.
- **Location and Duration:** The research will take place in a private room in the Research School of Psychology at the ANU campus. You will only be required to attend one session. The study will take approximately 90 minutes to complete.
- **Remuneration:** Participants in this research will be offered either 20 dollars ($20) in cash or psychology students can elect to receive 90 minutes course credit toward research participation requirements. This will be provided upon attending the study session. If you choose to withdraw you will still be provided with $20 or 90 minutes of course credit.
- **Risks:** There are no known risks, hazards, or side effects arising from participation in this study. However, the nature of some of the questions asked may cause participants mild distress, along with asking about sensitive personal information. If this occurs, you are free to cease participation immediately without explanation or penalty. If you become severely distressed as a direct result of participating in the experiment, a debrief and follow-up with a clinical psychologist appointment will be offered, along with contacts for ANU Counselling Centre, and Lifeline.
- **Benefits:** This research will increase our understanding of visual perception processes during visually guided movement.

Australian National University

Exclusion Criteria:
Eligibility for participation in the current study is restricted to adult participants, aged between 18 and 55-years-old, with normal or corrected-to-normal visual acuity and normal or corrected-to-normal movement ability in their dominant arm.

Confidentiality:
All data will be completely confidential, and will be protected in so far as the law allows. Your name will not appear on any research documentation except the consent form and your data will be recorded in a de-identified form. Data collected from this study will be aggregated with other participants' data when reported in a thesis or in publications. If you decide to withdraw from the study, your data will be destroyed.

Privacy Notice:
In collecting your personal information within this research, the ANU must comply with the Privacy Act 1988. The ANU Privacy Policy is available at https://policies.anu.edu.au/ppl/document/ANUP_010007 and it contains information about how a person can:

- Access or seek correction to their personal information;
- Complain about a breach of an Australian Privacy Principle by ANU, and how ANU will handle the complaint.

Data Storage:

- **Where:** All data management procedures will be in compliance with the *Privacy Act 1988* (Cth) and the ANU Code of Research Conduct. Research information will be stored in a secure filing cabinet in a secure office (in the Psychology Building) at the ANU Research School of Psychology and electronic information will be stored on password protected computer. Only the investigators (Elizabeth Shen and Associate Professor Bruce Christensen) will have access to this information.
- **How long:** All data and associated consent forms will be kept for five years following use for thesis or publications arising from the research.
- **Handling of Data following the required storage period:** After five years all electronic and paper information will be deleted/destroyed.

Queries and Concerns:

- **Contact Details for More Information:** If you have any questions are concerns about the study, please contact Elizabeth Shen (elizabeth.shen@anu.edu.au) or Associate Professor Bruce Christensen (bruce.christensen@anu.edu.au).
- **Contact Details if in Distress:** If you experience any distress as a result of your participation in this study you may wish to contact of the following services:

ANU Counselling Service (for ANU students)
Phone: 02 6125 2442, Website: http://counselling.anu.edu.au
ANU Psychology Clinic (for ANU students and members of the public)
Phone: 02 6125 8498, Website: psychology.clinic@anu.edu.au
Lifeline Phone: 13 11 14

Ethics Committee Clearance:
The ethical aspects of this research have been approved by the ANU Human Research Ethics Committee (Protocol 2017/152). If you have any concerns or complaints about how this research has been conducted, please contact:
Ethics Manager
The ANU Human Research Ethics Committee
The Australian National University
Telephone: +61 2 6125 3427
Email: Human.Ethics.Officer@anu.edu.au

Australian National University

WRITTEN CONSENT for Participants

Visual perception during visually guided movement

Investigators: Elizabeth Shen (PhD Candidate) and A/Prof. Bruce Christensen

I ________________________________ (print name) consent to take part in the "*Visual perception during visually guided movement*" study. I have read and understood the Information Sheet given to me about the research project, and I have had any questions and concerns about the project

(listed here:

____

____)

addressed to my satisfaction. I understand the nature and purpose of this research.

I agree to participate in the project. (tick one box only) YES ☐ NO ☐

I agree to have my questionnaire data from previous studies (protocol 2016/491: The Impact of Personality, Hand Position and Perceptual Expectations on Visual Perception) to be used in the current study.

YES ☐ NO ☐

Signature: …………………………………………….

Date: …………………………………………….

Would you like to receive correspondence about the outcomes of this study? (tick one box only)

YES ☐ NO ☐

If yes, please provide your email address:

____

The Australian National University | Canberra ACT 2601 Australia | CRICOS Provider No. 00120C

Q1.1 Please enter your participant code. Contact the experimenter if you are unsure of your participant code.

____

Q1.2 Please enter the month of your birth followed by the year of your birth (MMYYYY).

For example, if you were born in March 1994, enter 031994

____

Q1.3 What is your age (in years)?

▼ 18 (18) ... 60 (60)

Q1.4 What gender do you identify as?

- ◯ Male (1)
- ◯ Female (0)
- ◯ Trans* (2)
- ◯ Non-binary (3)
- ◯ Prefer not to say (4)

Q1.5 Are you left- or right-handed?

- ◯ Right (0)
- ◯ Left (1)

Q1.6 What is your ethnicity?

▼ Caucasian (0), Asian (1), African (2), Aboriginal and/or Torres Strait Islander (3), Other (4)

Display This Question:

If Q1.6 = Other

Q1.7 Please provide more information about your ethnicity:

____

Q1.8 What is your highest level of education you have currently achieved?

▼ Year 10 Certificate or equivalent (0) ... Doctoral Degree or equivalent (5)

Q1.9 What is your current occupation?

- ○ Full time student (0)
- ○ Part time student (1)
- ○ Other (2)

Display This Question:

If Q1.9 = Full time student

Or Q1.9 = Part time student

Q1.10 What year of study are you currently in?

▼ 1st Year (1) ... 5th Year or beyond (5)

Display This Question:

If Q1.9 = Part time student

Or Q1.9 = Other

Q1.11 Please specify your occupation / occupation in addition to student.

____

Q1.12 Is English your first language?

- ○ Yes (1)
- ○ No (0)

Display This Question:

If Q1.12 = No

Q1.13 At what age (in years) did you start learning English?

____

Q1.14 Have you ever been diagnosed with a mental illness or psychiatric disorder?

- Yes (1)
- No (0)

Display This Question:

If Q1.14 = Yes

Q1.15 Please provide more information about your mental illness or psychiatric disorder diagnosis(es).

____

Q1.16 Do you have a first-degree relative (i.e. mother, father, sister, or brother) with a diagnosed mental illness or psychiatric disorder?

- Yes (1)
- No (0)

Display This Question:

If Q1.16 = Yes

Q1.17 Please provide more information about your first-degree relatives with a diagnosed mental illness or psychiatric disorder.

____

Q1.18 Have you ever been diagnosed with a neurological disorder (e.g. traumatic brain injury) or had a neurological episode (e.g. a concussion, loss of consciousness for longer than 2 minutes)?

- Yes (1)
- No (0)

Display This Question:

If Q1.18 = Yes

Q1.19 Please provide more information about your current and/or previous neurological disorder(s) or neurological episode(s)

____

Q1.20 Have you ever had a medical illness that has affected your cognition or perception?

- ◯ Yes (1)
- ◯ No (0)

Display This Question:

If Q1.20 = Yes

Q1.21 Please provide more information about your medical illness(es) that have affected your cognition or perception.

____

Q1.22 Do you have a history of a learning disorder or learning difficulties?

- ◯ Yes (1)
- ◯ No (0)

Display This Question:

If Q1.22 = Yes

Q1.23 Please provide more information about your current and/or previous learning disorder(s) or learning difficulties.

____

Q1.24 Do you have a history of Attention Deficit-Hyperactivity Disorder (ADHD) or school-related behavioural problems?

- ◯ Yes (1)
- ◯ No (0)

Display This Question:

If Q1.24 = Yes

Q1.25 Please provide more information about your history of ADHD and/or school-related behavioural problems.

____

Q1.26 Do you have colourblindness?

- ○ Yes (1)
- ○ No (0)

Q1.27 Do you have 20/20 vision (normal or corrected-to-normal glasses or contact lens)?

- ○ Yes, normal vision (1)
- ○ Yes, corrected-to-normal vision (2)
- ○ No (0)

Q2.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
My friends are available if I need them. (1)	○	○	○	○
I have some inner struggles that cause problems for me. (2)	○	○	○	○
My health condition has restricted my activities. (3)	○	○	○	○
I am so tense in certain situations that I have great difficulty getting by. (4)	○	○	○	○
I have to do some things a certain way or I get nervous. (5)	○	○	○	○
Much of the time I'm sad for no real reason. (6)	○	○	○	○
Often I think and talk so quickly that other people cannot follow my train of thought. (7)	○	○	○	○
Most of the people I know can be trusted. (8)	○	○	○	○
Sometimes I cannot remember who I am. (9)	○	○	○	○
I have some ideas that others think are strange. (10)	○	○	○	○
I was usually well-behaved at school. (11)	○	○	○	○
I've seen a lot of doctors over the years. (12)	○	○	○	○
I'm a very sociable person. (13)	○	○	○	○
My mood can shift quite suddenly. (14)	○	○	○	○
Sometimes I feel guilty about how much I drink. (15)	○	○	○	○

Q3.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I'm a "take charge" type of person. (16)	○	○	○	○
My attitude about myself changes a lot. (17)	○	○	○	○
People would be surprised if I yelled at someone. (18)	○	○	○	○
My relationships have been stormy. (19)	○	○	○	○
At times I wish I were dead. (20)	○	○	○	○
People are afraid of my temper. (21)	○	○	○	○
Sometimes I use drugs to feel better. (22)	○	○	○	○
I've tried just about every type of drug. (23)	○	○	○	○
Sometimes I let little things bother me too much. (24)	○	○	○	○
I often have trouble concentrating because I'm nervous. (25)	○	○	○	○
I often fear I might slip up and say something wrong. (26)	○	○	○	○
I feel that I've let everyone down. (27)	○	○	○	○
I have many brilliant ideas. (28)	○	○	○	○
Certain people go out of their way to bother me. (29)	○	○	○	○
I just don't seem to relate to people very well (30)	○	○	○	○

Q4.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I've borrowed money knowing I wouldn't pay it back. (31)	○	○	○	○
Much of the time I don't feel well. (32)	○	○	○	○
I often feel jittery. (33)	○	○	○	○
I keep reliving something horrible that happened to me. (34)	○	○	○	○
I hardly have any energy. (35)	○	○	○	○
I can be very demanding when I want things done quickly. (36)	○	○	○	○
People usually treat me pretty fairly. (37)	○	○	○	○
My thinking has become confused. (38)	○	○	○	○
I get a kick out of doing dangerous things. (39)	○	○	○	○
My favourite poet is Raymond Kertezc. (40)	○	○	○	○
I like being around my family. (41)	○	○	○	○
I need to make some important changes in my life. (42)	○	○	○	○
I've had illnesses that my doctors could not explain. (43)	○	○	○	○
I can't do some things well because of nervousness. (44)	○	○	○	○
I have impulses that I fight to keep under control. (45)	○	○	○	○

Q5.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I've forgotten what it's like to feel happy. (46)	○	○	○	○
I take on so many commitments that I can't keep up. (47)	○	○	○	○
I have to be alert to the possibility that people will be unfaithful. (48)	○	○	○	○
I have visions in which I see myself forced to commit crimes. (49)	○	○	○	○
Other people sometimes put thoughts into my head. (50)	○	○	○	○
I've deliberately damaged someone's property. (51)	○	○	○	○
My health problems are very complicated. (52)	○	○	○	○
It's easy for me to make new friends. (53)	○	○	○	○
My moods get quite intense. (54)	○	○	○	○
I have trouble controlling my use of alcohol. (55)	○	○	○	○
I'm a natural leader. (56)	○	○	○	○
Sometimes I feel terribly empty inside. (57)	○	○	○	○
I tell people off when they deserve it. (58)	○	○	○	○
I want to let certain people know how much they've hurt me. (59)	○	○	○	○
I've thought about ways to kill myself. (60)	○	○	○	○

Q6.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
Sometimes my temper explodes and I completely lose control. (61)	○	○	○	○
People have told me that I have a drug problem. (62)	○	○	○	○
I never use drugs to help me cope with the world. (63)	○	○	○	○
Sometimes I'll avoid someone I really don't like. (64)	○	○	○	○
It's often hard for me to enjoy myself because I am worrying about things. (65)	○	○	○	○
I have exaggerated fears. (66)	○	○	○	○
Sometimes I think I'm worthless. (67)	○	○	○	○
I have some very special talents that few others have. (68)	○	○	○	○
Some people do things to make me look bad. (69)	○	○	○	○
I don't have much to say to anyone. (70)	○	○	○	○
I'll take advantage of others if they leave themselves open to it. (71)	○	○	○	○
I suffer from a lot of pain. (72)	○	○	○	○
I worry so much that at times I feel like I am going to faint. (73)	○	○	○	○
Thoughts about my past often bother me while I'm thinking about something else. (74)	○	○	○	○
I have no trouble falling asleep. (75)	○	○	○	○

Q7.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I get quite irritated if people try to keep me from accomplishing my goals. (76)	○	○	○	○
I seem to have as much luck in life as others do. (77)	○	○	○	○
My thoughts get scrambled sometimes. (78)	○	○	○	○
I do a lot of wild things just for the thrill of it. (79)	○	○	○	○
Sometimes I get ads in the mail that I don't really want. (80)	○	○	○	○
If I'm having problems, I have people I can talk to. (81)	○	○	○	○
I need to change some things about myself, even if it hurts. (82)	○	○	○	○
I've had numbness in parts of my body that I can't explain. (83)	○	○	○	○
Sometimes I am afraid for no reason. (84)	○	○	○	○
It bothers me when things are out of place. (85)	○	○	○	○
Everything seems like a big effort. (86)	○	○	○	○
Recently I've had much more energy than usual. (87)	○	○	○	○
Most people have good intentions. (88)	○	○	○	○
Since the day I was born, I was destined to be unhappy. (89)	○	○	○	○
Sometimes it seems that my thoughts are broadcast so that others can hear them. (90)	○	○	○	○

Q8.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I've done some things that weren't exactly legal. (91)	○	○	○	○
It's a struggle for me to get things done with the medical problem I have. (92)	○	○	○	○
I like to meet new people. (93)	○	○	○	○
My mood is very steady. (94)	○	○	○	○
There have been times when I've had to cut down on my drinking. (95)	○	○	○	○
I would be good at a job where I tell others what to do. (96)	○	○	○	○
I worry a lot about other people leaving me. (97)	○	○	○	○
When I get mad at other drivers on the road, I let them know. (98)	○	○	○	○
People once close to me have let me down. (99)	○	○	○	○
I've made plans about how to kill myself. (100)	○	○	○	○
Sometimes I'm very violent. (101)	○	○	○	○
My drug use has caused me financial strain. (102)	○	○	○	○
I've never had problems at work because of drugs. (103)	○	○	○	○
I sometimes complain too much. (104)	○	○	○	○
I'm often worried and nervous that I can barely stand it. (105)	○	○	○	○

Q9.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I get very nervous when I have to do something in front of others. (106)	○	○	○	○
I don't feel like trying anymore. (107)	○	○	○	○
My plans will make me famous someday. (108)	○	○	○	○
People around me a faithful to me. (109)	○	○	○	○
I'm a loner. (110)	○	○	○	○
I'll do most things if the price is right. (111)	○	○	○	○
I am in good health. (112)	○	○	○	○
Sometimes I feel dizzy when I've been under a lot of pressure. (113)	○	○	○	○
I've been troubled by memories of a bad experience for a long time. (114)	○	○	○	○
I rarely have trouble sleeping. (115)	○	○	○	○
Sometimes I get upset because others don't understand my plans. (116)	○	○	○	○
I've given a lot, but I haven't gotten much in return. (117)	○	○	○	○
Sometimes I have trouble keeping different thoughts separate. (118)	○	○	○	○
My behaviour is pretty wild at times. (119)	○	○	○	○
My favourite sports event on television is the high jump. (120)	○	○	○	○

Q10.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I spend most of my time alone. (121)	○	○	○	○
I need some help to deal with important problems. (122)	○	○	○	○
I've had episodes of double vision or blurred vision. (123)	○	○	○	○
I'm not the kind of person who panics easily. (124)	○	○	○	○
I can relax even if my home is a mess. (125)	○	○	○	○
Nothing seems to give me much pleasure. (126)	○	○	○	○
At times my thoughts move very quickly. (127)	○	○	○	○
I usually assume people are telling the truth. (128)	○	○	○	○
I think I have three to four completely different personalities inside me. (129)	○	○	○	○
Others can read my thoughts. (130)	○	○	○	○
I used to lie a lot to get out of tight situations. (131)	○	○	○	○
My medical problems always seem to be hard to treat. (132)	○	○	○	○
I am a warm person. (133)	○	○	○	○
I have little control over my anger. (134)	○	○	○	○
My drinking seems to cause problems in my relationships with others. (135)	○	○	○	○

Q11.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I have trouble standing up for myself. (136)	○	○	○	○
I often wonder what I should do with my life. (137)	○	○	○	○
I'm not afraid to yell at someone to get my point across. (138)	○	○	○	○
I rarely feel very lonely. (139)	○	○	○	○
I've recently been thinking about suicide. (140)	○	○	○	○
Sometimes I smash things when I'm upset. (141)	○	○	○	○
I never use illegal drugs. (142)	○	○	○	○
I sometimes do things so impulsively that I get into trouble. (143)	○	○	○	○
Sometimes I'm too impatient. (144)	○	○	○	○
My friends say I worry too much. (145)	○	○	○	○
I'm not easily frightened. (146)	○	○	○	○
I can't seem to concentrate very well. (147)	○	○	○	○
I have accomplished some remarkable things. (148)	○	○	○	○
Some people try to keep me from getting ahead. (149)	○	○	○	○
I don't feel close to anyone. (150)	○	○	○	○

Q12.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I can talk my way out of just about anything. (151)	○	○	○	○
I seldom have complaints about how I feel physically. (152)	○	○	○	○
I can often feel my heart pounding. (153)	○	○	○	○
I can't seem to get over something from my past. (154)	○	○	○	○
I've been moving more slowly that usual. (155)	○	○	○	○
I have great plans and it irritates me that people try to interfere. (156)	○	○	○	○
People don't appreciate what I've done for them. (157)	○	○	○	○
Sometimes it feels as if somebody is blocking my thoughts. (158)	○	○	○	○
If I get tired of a place, I just pick up and leave. (159)	○	○	○	○
Most people would rather win than lose. (160)	○	○	○	○

Q13.1 Please check the answer for each item as it applies to you.

	Never (0)	Sometimes (1)	Often (2)	Almost Always (3)
My thoughts seem as real as actual events in my life. (1)	○	○	○	○
No matter how much I try to concentrate on my work, unrelated thoughts always creep into my mind. (2)	○	○	○	○
I have had the experience of hearing a person's voice and then found that there was no one there. (3)	○	○	○	○
The sounds I hear in my daydreams are usually clear and distinct. (4)	○	○	○	○
The people in my daydreams seem so true to life that I think they are real. (5)	○	○	○	○
In my daydreams I can hear the sound of a tune almost as clearly as if I were actually listening to it. (6)	○	○	○	○
I hear a voice speaking my thoughts aloud. (7)	○	○	○	○
I have been troubled by hearing voices in my head. (8)	○	○	○	○
I have seen a person's face in front of me when no one was there. (9)	○	○	○	○
When I look at things they appear strange to me. (10)	○	○	○	○
I see shadows and shapes when there is nothing there. (11)	○	○	○	○
When I look at things, they look unreal to me. (12)	○	○	○	○
When I look at myself in the mirror I look different. (13)	○	○	○	○

Q14.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Do you find the bright lights of a city exciting to look at? (1)	○	○
Does your sense of smell sometimes become unusually strong? (2)	○	○
Have you sometimes sensed an evil presence around you, even though you could not see it? (3)	○	○
Have you had very little fun from physical activities like walking, swimming, or sports? (4)	○	○
Are you usually in an average sort of mood, not too high and not too low? (5)	○	○
Do you often feel that there is no purpose to life? (6)	○	○
Are people usually better if they stay aloof from emotional involvements with people? (7)	○	○
Do you think you could learn to read other's minds if you wanted to? (8)	○	○
Do you often overindulge in alcohol or food? (9)	○	○
Are there very few things that you have ever really enjoyed doing? (10)	○	○

Q15.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Can some people make you aware of them just by thinking about you? (11)	○	○
Do you often feel lonely? (12)	○	○
Do you worry about awful things that might happen? (13)	○	○
Can just being with friends make you feel really good? (14)	○	○
Do you ever have a sense of vague danger or sudden dread for reasons that you do not understand? (15)	○	○
Do you stop to think things over before doing anything? (16)	○	○
Do you find it difficult to keep interested in the same thing for a long time? (17)	○	○
Do you ever have the urge to break or smash things? (18)	○	○
Are you much too independent to really get involved with other people? (19)	○	○
Do ideas and insights sometimes come to you so fast that you cannot express them all? (20)	○	○

Q16.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Are you easily distracted when you read or talk to someone? (21)	○	○
Would being in debt worry you? (22)	○	○
Do you often feel “fed up”? (23)	○	○
Do you believe in telepathy? (24)	○	○
Do people who drive carefully annoy you? (25)	○	○
Can you usually let yourself go and enjoy yourself at a lively party? (26)	○	○
Do you ever feel sure that something is about to happen, even though there does not seem to be any reason for you thinking that? (27)	○	○
Is it hard for you to make decisions? (28)	○	○
Do people who try to get to know you better usually give up after a while? (29)	○	○
Do you often feel like doing the opposite of what other people suggest, even though you know they are right? (30)	○	○

Q17.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Does your voice ever seem distant or faraway? (31)	○	○
Are you easily confused if too much happens at the same time? (32)	○	○
Do you feel that making new friends isn't worth the energy it takes? (33)	○	○
Do you often feel the impulse to spend money which you know you can't afford? (34)	○	○
Do you worry too long after an embarrassing experience? (35)	○	○
Do you ever suddenly feel distracted by distant sounds that you are not normally aware of? (36)	○	○
Do you often have an urge to hit someone? (37)	○	○
Do you often have days when indoor lights seem so bright that they bother your eyes? (38)	○	○
Do you like going out a lot? (39)	○	○
Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense? (40)	○	○

Q18.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Would it make you nervous to play the clown in front of other people? (41)	○	○
Do you have many friends? (42)	○	○
Have you ever felt that you have special, almost magical powers? (43)	○	○
Are you a person whose mood goes up and down easily? (44)	○	○
Are you rather lively? (45)	○	○
Have you ever blamed someone for doing something you know was really your fault? (46)	○	○
Have you sometimes had the feeling of gaining or losing energy when certain people look at you or touch you? (47)	○	○
Would you call yourself a nervous person? (48)	○	○
Do you like mixing with people? (49)	○	○
Have you felt as though your head or limbs were somehow not your own? (50)	○	○

Q19.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Do you sometimes talk about things you know nothing about? (51)	○	○
Do you often experience an overwhelming sense of emptiness? (52)	○	○
Do you at times have an urge to do something harmful or shocking? (53)	○	○
Do you think having close friends is not as important as some people say? (54)	○	○
Are the sounds that you hear in your daydreams really clear and distinct? (55)	○	○
Are you easily hurt when people find fault with you or the work you do? (56)	○	○
Have you wondered whether the spirits of the dead can influence the living? (57)	○	○
Do you often change between intense liking and disliking of the same person? (58)	○	○
Do you enjoy many different kinds of play and recreation? (59)	○	○
Do your thoughts sometimes seem as real as actual events in your life? (60)	○	○

Q20.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Are you sometimes so nervous that you are "blocked"? (61)	○	○
Do you feel so good at controlling others that it sometimes scares you? (62)	○	○
Do you think people spend too much time safeguarding their future with savings and insurance? (63)	○	○
Does it often feel good to massage your muscles when they are tired or sore? (64)	○	○
Do you dread going into a room by yourself where other people have already gathered and are talking? (65)	○	○
On occasions, have you seen a person's face in front of you when no one was in fact there? (66)	○	○
Do you prefer watching television over going out with other people? (67)	○	○
Have you ever cheated at a game? (68)	○	○
On seeing a soft thick carpet have you sometimes had the impulse to take off your shoes and walk barefoot on it? (69)	○	○
When in the dark do you often see shapes and forms even though there's nothing there? (70)	○	○

Q21.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Do you easily lose your courage when criticised or failing in something? (71)	○	○
Has dancing or the idea of it always seemed dull to you? (72)	○	○
Have you ever felt the urge to injure yourself? (73)	○	○
Are your thoughts sometimes so strong that you can almost hear them? (74)	○	○
Do you sometimes feel that your accidents are caused by mysterious forces? (75)	○	○
Do you frequently have difficulty in starting to do things? (76)	○	○
Is it fun to sing with other people? (77)	○	○
When you catch a train do you often arrive at the last minute? (78)	○	○
When you look in the mirror does your face sometimes seem quite different from usual? (79)	○	○
Do you usually have very little desire to buy new kinds of food? (80)	○	○

Q22.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Do you often have difficulties in controlling your thoughts? (81)	○	○
When in a group of people do you usually prefer to let someone else be the centre of attention? (82)	○	○
Have you ever thought you heard people talking only to discover that it was in fact some nondescript noise? (83)	○	○
Have you often felt uncomfortable when friends touch you? (84)	○	○
Do you worry about things you should not have done or said? (85)	○	○
Would you take drugs which may have strange or dangerous effects? (86)	○	○
Have you occasionally felt as though your body did not exist? (87)	○	○
Do you love having your back massaged? (88)	○	○
Does it happen that nearly every thought immediately and automatically suggests an enormous number of ideas? (89)	○	○
Do you often hesitate when you are going to say something in a group of people whom you more or less know? (90)	○	○

Q23.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Do you consider yourself to be pretty much an average kind of person? (91)	○	○
Do people in your daydreams seem so true to life that you sometimes think they are real? (92)	○	○
Do you feel very close to your friends? (93)	○	○
No matter how hard you try to concentrate do unrelated thoughts creep into your mind? (94)	○	○
Have you ever taken advantage of someone? (95)	○	○
Is your hearing sometimes so sensitive that ordinary sounds become uncomfortable? (96)	○	○
When in a crowded room, do you often have difficulty in following a conversation? (97)	○	○
Is trying new foods something you have always enjoyed? (98)	○	○
Does a passing thought ever seem so real it frightens you? (99)	○	○
Would you like other people to be afraid of you? (100)	○	○

Q24.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
When things are bothering you do you like to talk to other people about it? (101)	○	○
Have you felt that you might cause something to happen just by thinking too much about it? (102)	○	○
Are you easily distracted from work by daydreams? (103)	○	○
Is it true that your relationships with other people never get very intense? (104)	○	○

Thank-you for completed these questionnaires. Please let the experimenter know you have finished.

B.2 Data Screening and Cleaning

B.2.1 Missing data

Table B.1 Summary of participants with missing data by experimental condition.

ID	Pointing				Grasping			
	5cm		7cm		5cm		7cm	
	Stationary	**Perturbed**	**Stationary**	**Perturbed**	**Stationary**	**Perturbed**	**Stationary**	**Perturbed**
1188	X	X						
1507						X		
1554		X	X			X		
1556	X		X	X			X	X
1560		X	X	X				
1565					X		X	X
1569				X			X	X
1576	X	X	X	X				
1579	X	X	X	X			X	X
1589		X						
1590			X	X				
1591			X	X				
1592			X	X	X	X	X	X
1593			X	X		X		
Total	**4**	**6**	**9**	**9**	**2**	**4**	**5**	**5**

Note: X indicates condition where data are missing. For conditions with data, these participants were included in the respective analyses.

B.2.2 Extended Descriptives of Final Sample

Table B.2 Descriptives for OLIFE.

	OLIFE_Total	Usu_Exp	Cog_Dis	Int_Anh	Imp_Non
Valid	94	94	94	94	94
Missing	0	0	0	0	0
Mean	41.021	10.457	14.340	8.798	7.426
Std. Deviation	12.669	6.016	4.972	4.957	3.064
Skewness	0.511	0.584	-0.319	0.340	0.875
Std. Error of Skewness	0.249	0.249	0.249	0.249	0.249
Shapiro-Wilk	0.971	0.970	0.978	0.974	0.946
P-value of Shapiro-Wilk	0.034	0.032	0.116	0.058	< .001
Minimum	11.000	0.000	1.000	0.000	2.000
Maximum	82.000	29.000	24.000	22.000	19.000
50th percentile	40.000	10.000	14.000	8.000	7.000
90th percentile	54.700	19.000	20.000	15.000	12.000

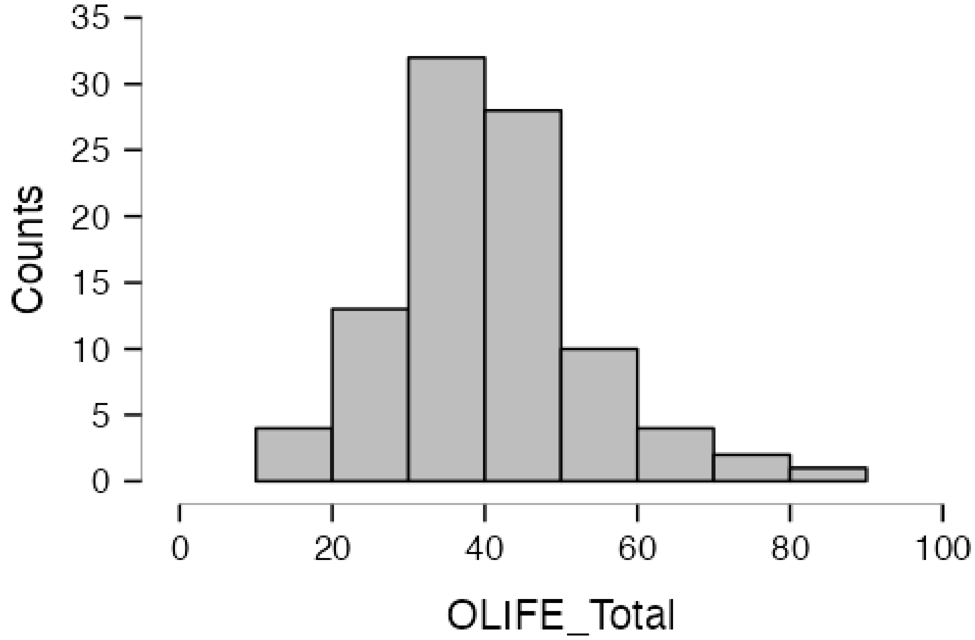


Figure B.1 OLIFE total score distribution.

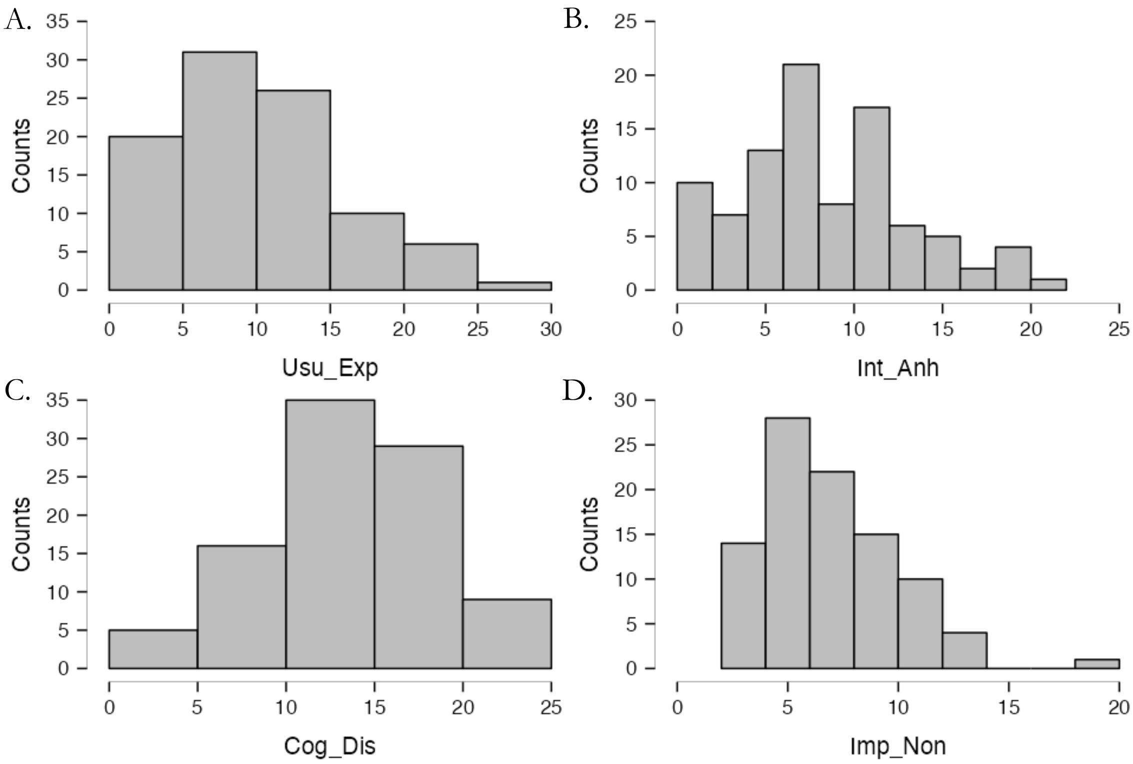


Figure B.2 OLIFE subscale distributions. A. Unusual Experiences, B. Introvertive Anhedonia, C. Cognitive Disorganisation, D. Impulsive Nonconformity.

B.3 Data Analysis Supplementary Outputs

B.3.1 Movement Time

Table B.3 Movement time (MT) descriptives and log transformed MT Shapiro-Wilk values.

		Point				Grasp			
		5cm		7cm		5cm		7cm	
		Stationary	Perturbed	Stationary	Perturbed	Stationary	Perturbed	Stationary	Perturbed
Raw Values	Valid	90	88	85	85	92	90	89	89
	Missing	4	6	9	9	2	4	5	5
	Mean	4.022	4.104	3.983	4.092	4.239	4.458	4.241	4.597
	Std. Deviation	0.841	0.853	0.912	0.822	0.877	0.904	0.898	0.998
	Minimum	2.50	2.55	2.42	2.72	2.69	2.94	2.61	2.97
	Maximum	6.35	6.56	6.73	6.28	6.81	6.86	6.52	7.62
	Shapiro-Wilk (SW)	0.968	0.953	0.945	0.934	0.969	0.965	0.975	0.952
	P-value SW	0.027	0.003	0.001	< .001	0.026	0.015	0.087	0.002
Log Transformed	Shapiro-Wilk (SW)	0.988	0.983	0.978	0.963	0.988	0.981	0.99	0.978
	P-value SW	0.599	0.306	0.151	0.016	0.551	0.202	0.733	0.131

Note: Log transformations corrected MT data distributions to normal in all conditions except reach-to-point under perturbed conditions with the 7cm target.

B.3.2 Stable Grip Aperture

OLIFE Total

Table B.4 Within subjects effects for Stable Grip Aperture with OLIFE total score.

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
Target Size	None	153660.369[a]	2.000[a]	76830.184[a]	842.168[a]	< .001[a]	0.596	0.902
	Greenhouse-Geisser	153660.369	1.762	87193.121	842.168	< .001	0.596	0.902
	Huynh-Feldt	153660.369	1.794	85648.469	842.168	< .001	0.596	0.902
Target Size ∗ OLIFE_Total	None	388.152[a]	2.000[a]	194.076[a]	2.127[a]	0.122[a]	0.002	0.023
	Greenhouse-Geisser	388.152	1.762	220.253	2.127	0.129	0.002	0.023
	Huynh-Feldt	388.152	1.794	216.351	2.127	0.128	0.002	0.023
Residuals	None	16786.146	184.000	91.229				
	Greenhouse-Geisser	16786.146	162.132	103.534				
	Huynh-Feldt	16786.146	165.056	101.700				

Note. Type II Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

Table B.5 Between subjects effects for Stable Grip Aperture with OLIFE total score..

Cases	Sum of Squares	df	Mean Square	F	p
OLIFE_Total	552.701	1	552.701	0.587	0.446
Residuals	86618.958	92	941.510		

Note. Type II Sum of Squares

Table B.6 Test of Sphericity for Stable Grip Aperture.

	Mauchly's W	Approx. X^2	df	p-value	Greenhouse-Geisser ε	Huynh-Feldt ε	Lower Bound ε
Target Size	0.865	13.185	2	0.001	0.881	0.897	0.500

Table B.7 Post Hoc Comparisons – Target Size

		Mean Difference	SE	t	p_{bonf}	p_{holm}
5cm	7cm	-23.915	1.393	-17.165	< .001	< .001
	10cm	-56.936	1.393	-40.867	< .001	< .001
7cm	10cm	-33.021	1.393	-23.702	< .001	< .001

Note. P-value adjusted for comparing a family of 3

OLIFE subscales

Table B.8 Within subjects effects for Stable Grip Aperture with OLIFE subscale scores.

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
Target Size	None	153660.369[a]	2.000[a]	76830.184[a]	867.923[a]	< .001[a]	0.595	0.907
	Greenhouse-Geisser	153660.369	1.785	86065.441	867.923	< .001	0.595	0.907
	Huynh-Feldt	153660.369	1.819	84452.982	867.923	< .001	0.595	0.907
Target Size ∗ Usu_Exp	None	300.760[a]	2.000[a]	150.380[a]	1.699[a]	0.186[a]	0.001	0.019
	Greenhouse-Geisser	300.760	1.785	168.456	1.699	0.189	0.001	0.019
	Huynh-Feldt	300.760	1.819	165.300	1.699	0.189	0.001	0.019
Target Size ∗ Cog_Dis	None	642.569[a]	2.000[a]	321.284[a]	3.629[a]	0.029[a]	0.002	0.039
	Greenhouse-Geisser	642.569	1.785	359.904	3.629	0.034	0.002	0.039
	Huynh-Feldt	642.569	1.819	353.161	3.629	0.033	0.002	0.039
Target Size ∗ Int_Anh	None	287.745[a]	2.000[a]	143.873[a]	1.625[a]	0.200[a]	0.001	0.018
	Greenhouse-Geisser	287.745	1.785	161.167	1.625	0.203	0.001	0.018
	Huynh-Feldt	287.745	1.819	158.147	1.625	0.202	0.001	0.018
Target Size ∗ Imp_Non	None	54.141[a]	2.000[a]	27.071[a]	0.306[a]	0.737[a]	2.098e-4	0.003
	Greenhouse-Geisser	54.141	1.785	30.325	0.306	0.712	2.098e-4	0.003
	Huynh-Feldt	54.141	1.819	29.756	0.306	0.716	2.098e-4	0.003
Residuals	None	15756.897	178.000	88.522				
	Greenhouse-Geisser	15756.897	158.900	99.163				
	Huynh-Feldt	15756.897	161.934	97.305				

Note. Type II Sum of Squares. [a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Table B.9 Between subjects effects for Stable Grip Aperture with OLIFE subscale scores.

Cases	**Sum of Squares**	**df**	**Mean Square**	**F**	**p**	**η^2**	**η^2_p**
Usu_Exp	135.722	1	135.722	0.143	0.706	5.26E-04	0.002
Cog_Dis	1409.986	1	1409.986	1.49	0.225	0.005	0.016
Int_Anh	212.864	1	212.864	0.225	0.636	8.25E-04	0.003
Imp_Non	1343.771	1	1343.771	1.42	0.237	0.005	0.016
Residuals	84234.803	89	946.458				

Note. Type II Sum of Squares

Table B.10 Test of Sphericity for Stable Grip Aperture with OLIFE subscale scores.

	Mauchly's W	**Approx. X^2**	**df**	**p-value**	**Greenhouse-Geisser ε**	**Huynh-Feldt ε**	**Lower Bound ε**
Target Size	0.880	11.270	2	0.004	0.893	0.910	0.500

B.3.3 Grip Aperture

Table B.11 Within subjects effects for Grip Aperture with OLIFE total score.

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
Target Size	None	105439.917	1	105439.917	214.657	< .001	0.035	0.719
Target Size ✻ OLIFE_Total	None	776.639	1	776.639	1.581	0.212	2.59E-04	0.018
Residuals	None	41261.039	84	491.203				
Target Type	None	16155.244	1	16155.244	56.833	< .001	0.005	0.404
Target Type ✻ OLIFE_Total	None	197.289	1	197.289	0.694	0.407	6.58E-05	0.008
Residuals	None	23877.857	84	284.26				
Interval of Movement	None	2.19E+06	10	218744.243	752.473	< .001	0.73	0.9
	Greenhouse-Geisser	2.19E+06	2.343	933453.875	752.473	< .001	0.73	0.9
Interval of Movement ✻ OLIFE_Total	None	5842.627	10	584.263	2.01	0.03	0.002	0.023
	Greenhouse-Geisser	5842.627	2.343	2493.242	2.01	0.129	0.002	0.023
Residuals	None	244188.491	840	290.701				
	Greenhouse-Geisser	244188.491	196.844	1240.515				
Target Size ✻ Target Type	None	263.225	1	263.225	1.187	0.279	8.78E-05	0.014
Target Size ✻ Target Type ✻ OLIFE_Total	None	4.207	1	4.207	0.019	0.891	1.40E-06	2.26E-04
Residuals	None	18625.407	84	221.731				
Target Size ✻ Interval of Movement	None	44630.929	10	4463.093	78.554	< .001	0.015	0.483
	Greenhouse-Geisser	44630.929	2.852	15648.276	78.554	< .001	0.015	0.483
Target Size ✻ Interval of Movement ✻ OLIFE_Total	None	400.494	10	40.049	0.705	0.72	1.34E-04	0.008
	Greenhouse-Geisser	400.494	2.852	140.419	0.705	0.543	1.34E-04	0.008
Residuals	None	47725.087	840	56.816				
	Greenhouse-Geisser	47725.087	239.579	199.204				
Target Type ✻ Interval of Movement	None	6730.838	10	673.084	16.007	< .001	0.002	0.16
	Greenhouse-Geisser	6730.838	3.361	2002.777	16.007	< .001	0.002	0.16
Target Type ✻ Interval of Movement ✻ OLIFE_Total	None	406.611	10	40.661	0.967	0.471	1.36E-04	0.011
	Greenhouse-Geisser	406.611	3.361	120.988	0.967	0.416	1.36E-04	0.011
Residuals	None	35320.691	840	42.048				
	Greenhouse-Geisser	35320.691	282.303	125.116				
Target Size ✻ Target Type ✻ Interval of Movement	None	811.081	10	81.108	2.472	0.006	2.71E-04	0.029
	Greenhouse-Geisser	811.081	2.794	290.313	2.472	0.067	2.71E-04	0.029
Target Size ✻ Target Type ✻ Interval of Movement ✻ OLIFE_Total	None	512.987	10	51.299	1.563	0.113	1.71E-04	0.018
	Greenhouse-Geisser	512.987	2.794	183.615	1.563	0.202	1.71E-04	0.018
Residuals	None	27564.617	840	32.815				
	Greenhouse-Geisser	27564.617	234.681	117.456				

Note. Sphericity corrections not available for factors with 2 levels. Type II Sum of Squares. [a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Table B.12 Between subjects effects for Grip Aperture across the movement with OLIFE total score.

Cases	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
OLIFE_Total	10414.4	1	10414.4	4.894	0.03	0.003	0.055
Residuals	178747	84	2127.95				

Note. Type II Sum of Squares

Table B.13 Test of Sphericity for Grip Aperture with OLIFE subscale scores

	Mauchly's W	Approx. X^2	df	p-value	Greenhouse-Geisser ε	Huynh-Feldt ε	Lower Bound ε
Interval of Movement	5.28E-11	1904.253	54	< .001	0.234	0.241	0.1
Target Size ✻ Interval of Movement	6.39E-08	1333.005	54	< .001	0.285	0.296	0.1
Target Type ✻ Interval of Movement	1.70E-06	1068.942	54	< .001	0.336	0.352	0.1
Target Size ✻ Target Type ✻ Interval of Movement	8.69E-07	1122.946	54	< .001	0.279	0.29	0.1

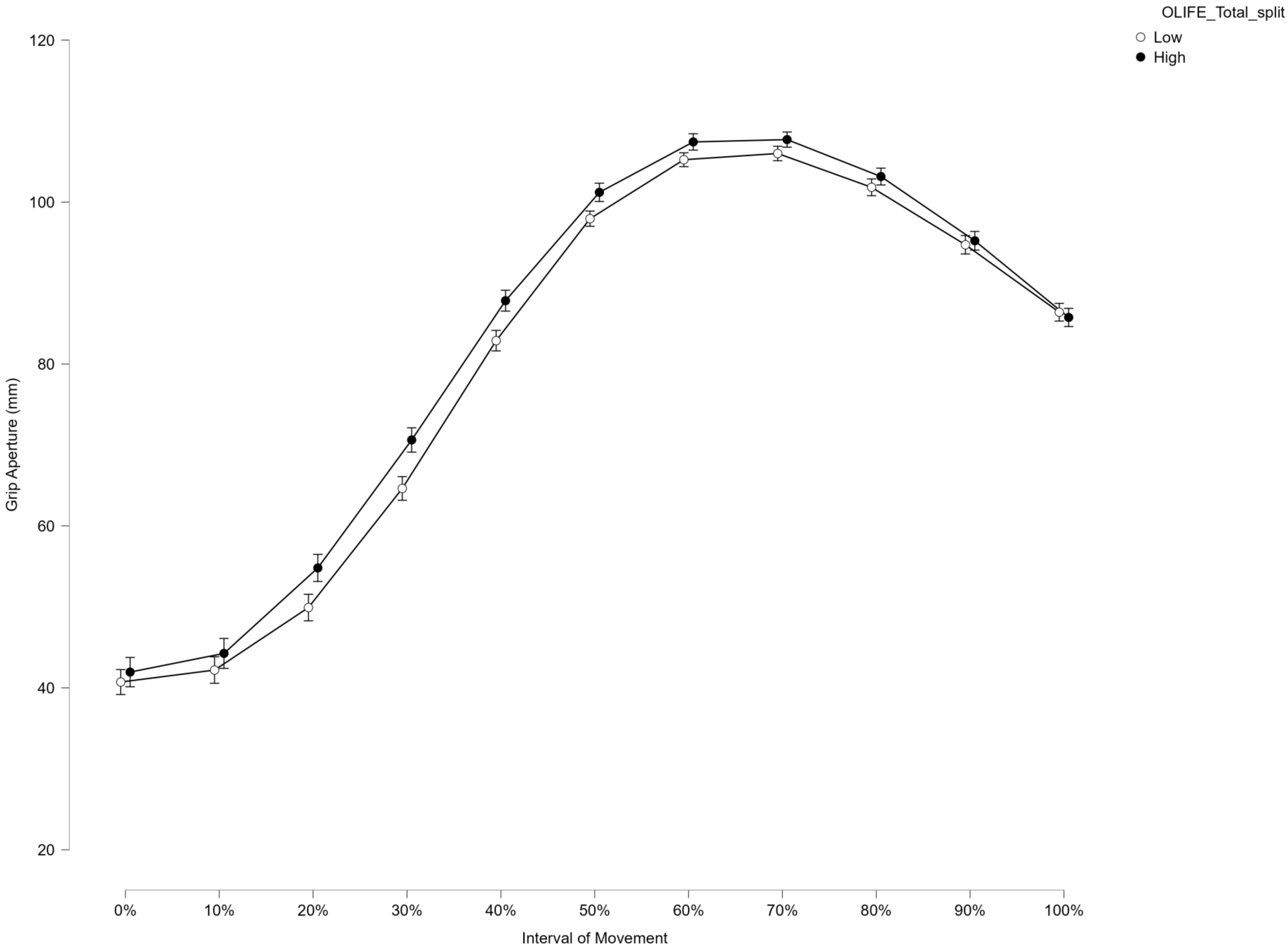


Figure B.3 Grip aperture as a function of interval of movement (0-100%) and OLIFE total score with a median split applied. Error bars represent standard error.

APPENDIX C

Study 2 Supplementary Materials (Chapter 4)

C.1 Study 2 Participant Questionnaire

Welcome to the Personality and Unique Experiences Survey!

This project explores your unique experiences and personality traits. You will be asked to complete several questionnaires and your responses will be saved.
Data from 1000 participants will be collected from the general population, and participation will take approximately 30 to 40 minutes. The data from this study will contribute to the researcher's Ph.D. thesis and related research publications. Results of the study will be made available to participants upon completion of the research. This project is funded by the Research School of Psychology at the ANU.

Voluntary Participation & Withdrawal: Participation in the project is voluntary and you may, without negative consequences, decline to take part, withdraw from the research, and/or refuse to answer questions without providing an explanation at any time until the work is prepared for publication. If you do withdraw, your data will be destroyed.

- **What does participation in the research entail?** If you decide to participate, you will be asked to complete a series of questionnaires online.
- **Location and Duration:** The research will take place online. The study will take approximately 30 to 40 minutes to complete.

Remuneration: Participants in this research will be offered £3.75 (approx. $7AUD) for participation. This will be provided upon completion of the survey by following the link back to Prolific.

Risks: There are no known risks, hazards, or side effects arising from participation in this study. However, the nature of some of the questions asked may cause some participants mild distress, along with asking about sensitive personal information. If this occurs, you are free to cease participation immediately without explanation or penalty and request your data to be withdrawn. If you become severely distressed as a direct result of participating in the experiment, a debrief and follow-up with a clinical psychologist appointment will be offered along with contacts for ongoing support.

Benefits: This research will increase our understanding of personality and unusual experiences within the community.

Confidentiality: All data will be completely confidential and will be protected in so far as the law allows. Your name will not appear on any research documentation except the consent form and your data will be recorded in a de-identified form. Data collected from this study will be aggregated with other participants' data when reported in a thesis or in publications. If you decide to withdraw from the study, your data will be destroyed.

Privacy Notice: In collecting your personal information within this research, the ANU must comply with the Privacy Act 1988. The ANU Privacy Policy is available at https://policies.anu.edu.au/ppl/document/ANUP_010007 and it contains information about how a person can:

- Access or seek correction to their personal information;
- Complain about a breach of an Australian Privacy Principle by ANU, and how ANU will handle the complaint.

Data Storage:

- **Where:** All data management procedures will be in compliance with the *Privacy Act 1988* (Cth) and the ANU Code of Research Conduct. Research information will be stored in a secure filing cabinet in a secure office (in the Psychology Building) at the ANU Research School of Psychology and electronic information will be stored on password protected computer. Only the investigators (Elizabeth Shen and Associate Professor Bruce Christensen) will have access to this information.
- **How long:** All data and associated consent forms will be kept for five years following use for thesis or publications arising from the research.
- **Handling of Data following the required storage period:** After five years all electronic and paper information will be deleted/destroyed.

Queries and Concerns:

- **Contact Details for More Information:** If you have any questions are concerns about the study, please contact Elizabeth Shen (elizabeth.shen@anu.edu.au) or Associate Professor Bruce Christensen (bruce.christensen@anu.edu.au).
- **Contact Details if in Distress:** If you experience any distress as a result of your participation in this study you may wish to contact Lifeline on 13 11 14.

Ethics Committee Clearance: The ethical aspects of this research have been approved by the ANU Human Research Ethics Committee (Protocol 2017/152). If you have any concerns or complaints about how this research has been conducted, please contact:

Ethics Manager
The ANU Human Research Ethics Committee
The Australian National University
Telephone: +61 2 6125 3427
Email: Human.Ethics.Officer@anu.edu.au

Consent
Declaration of Consent

I have read and understood the information provided above and agree to the terms outlined. I consent to participating in this study, titled 'Personality and Unique Experiences Survey'.

○ Yes, I consent to participating in this study. (1)

○ No, I do NOT consent to participating in this study. (2)

Display This Question:

If Previous question = No, I do NOT consent to participating in this study

Q3.1 As you do not wish to participate in this study, please return your submission on Prolific by selecting the 'Stop without completing' button.

What is your current age (in years)?

▼ Under 18 () ... Over 60 ()

In what country do you currently reside?

▼ Australia (1) ... Other (4)

Do you have normal or corrected-to-normal vision? (i.e. if you need glasses, you are wearing them or contact lenses)

◯ Yes (1)

◯ No (2)

◯ Rather not say (0)

Display This Question:

If age = "Under 18" or "Over 60" OR

If Country of residence ≠ "Australia" OR

If Vision = "No".

Q5.1 You are ineligible for this study, as you have provided information which is inconsistent with your Prolific pre-screening responses. Please return your submission on Prolific by selecting the 'Stop without completing' button.

What is your Prolific ID?

____

What is your residential postcode? (Please enter 4 digits only).

This information is collected to assess the spread of participants across Australia.

____

What device are you using to complete this survey?

○ Mobile phone (e.g. Iphone 12, Samsung S21 etc.) (1)

○ Tablet (e.g. Ipad, Surface Pro without attached keyboard) (2)

○ Laptop (e.g. Macbook, HP Probook) (3)

○ Desktop (e.g. iMac, Dell PC) (4)

What gender do you identify as?

○ Male (1)

○ Female (0)

○ Trans* (2)

○ Non-binary (3)

○ Prefer not to say (4)

Which ethnic group do you most identify with?

▼ Caucasian or White (0) ... Other (6)

Display This Question:

If Ethnicity = Other

Please provide more information about your ethnicity:

____

Which religion do you identify with the most?

- No religion (0)
- Buddhism (1)
- Christianity (2)
- Hinduism (3)
- Islam (4)
- Judaism (5)
- Decline to answer (6)
- Other (7)

Display This Question:

If Religion = Other

Please provide more information about your religion:

____

____

____

____

____

CRS1 How often do you think about religious issues?

- Very Often (4)
- Often (3)
- Occasionally (2)
- Rarely (1)
- Never (0)

CRS2 To what extent do you believe that God, deities, or something divine exists?

- Very much so (4)
- Quite a bit (3)
- Moderately (2)
- Not very much (1)
- Not at all (0)

CRS3 How often do you take part in religious services?

- Once a week or more (4)
- One to three times a month (3)
- A few times a year (2)
- One to two times a year (1)
- Never (0)

CRS4 How often do you pray or meditate?

- Once a day or more (4)
- More than once a week (3)
- Once to four times a month (2)
- Once to a few times a year (1)
- Never (0)

CRS5 How often do you experience situations in which you have the feeling that God or something divine allows for an intervention in your life?

- Very often (4)
- Often (3)
- Occasionally (2)
- Rarely (1)
- Never (0)

Please indicate if you have ever suffered from or witnessed any of these events. Select all that apply.

- Natural disaster (e.g. flood, bushfire, earthquake) (1)
- Fire or explosion (2)
- Transportation accident (e.g. car accident, plane crash) (3)
- Serious accident at work, home, or during recreational activity (4)
- Physical assault (with or without a weapon) (6)
- Sexual assault or unwanted sexual experiences (with or without a weapon) (7)
- Combat or exposure to a war-zone (8)
- Capitivity (e.g. kidnapped, abducted, held hostage, prisoner of war) (9)
- LIfe threatening or serious illness (including chronic illnesses) (10)
- Sudden violent and/or accidental death (e.g. death by homocide or unexpected medical reason) (11)
- Serious injury, harm, or death you caused to someone else (12)
- Serious relationship breakdown (e.g. divorce, family estrangement) (13)
- Severe human suffering (14)

In your **lifetime**, how many significant adverse life events, like those described in the previous question, have you experienced?

If you are unsure, just take your best guess.

○ None to 5 events (0)

○ 6 to 10 events (1)

○ 11 to 15 events (2)

○ More than 15 events (3)

In the **last 12 months**, how many significant adverse life events, like those described in the previous question, have you experienced?

If you are unsure, just take your best guess.

○ None to 5 events (0)

○ 6 to 10 events (1)

○ 11 to 15 events (2)

○ More than 15 events (3)

What is your highest level of education you have currently achieved?

▼ Less than Year 10 Certificant or equivalent (6) ... Doctoral Degree or equivalent (5)

What is your current occupation? (e.g. nurse, student, carpenter)

____

On average, how many hours do you work per week? Include hours from all occupations, including study as a student.

- <10 hours (0)
- 11 - 20 hours (1)
- 21 - 30 hours (2)
- 31 - 40 hours (3)
- 40+ hours (4)

On average, how many hours of sleep do you get per night?

- Less than 4 hours (0)
- 4 to 6 hours (1)
- 6 to 8 hours (2)
- 8 to 10 hours (3)
- More than 10 hours (4)

On average, how would you rate your sleep quality overall?

- Very good (0)
- Fairly good (1)
- Fairly bad (2)
- Very bad (3)

Is English your first language?

- Yes (1)
- No (0)

Display This Question:

If Language_eng = No

At what age (in years) did you start learning English?

____

Which option is closest in meaning to '**bright**'?

- Pale (0)
- Exciting (1)
- Shining (2)
- Correct (3)

Which option is closest in meaning to '**afraid**'?

- Scared (0)
- Intrepid (1)
- Fake (2)
- Reluctant (3)

Which option is closest in meaning to '**impulse**'?

- Reality (0)
- Heart rate (1)
- Critical (2)
- Desire (3)

Have you ever been diagnosed with a mental illness or psychiatric disorder?

- ◯ Yes (1)
- ◯ No (0)

Display This Question:

If Dem_MentalDx = Yes

Please provide more information about your mental illness or psychiatric disorder diagnosis(es).

____

Do you have a first-degree relative (i.e. mother, father, sister, or brother) with a diagnosed mental illness or psychiatric disorder?

- ◯ Yes (1)
- ◯ No (0)

Display This Question:

If Dem_MentalDxRel = Yes

Please provide more information about your first-degree relatives with a diagnosed mental illness or psychiatric disorder.

____

Have you ever been diagnosed with a neurological disorder (e.g. traumatic brain injury) or had a neurological episode (e.g. a concussion, loss of consciousness for longer than 2 minutes)?

- ◯ Yes (1)
- ◯ No (0)

Display This Question:

If Dem_NeuDx = Yes

Please provide more information about your current and/or previous neurological disorder(s) or neurological episode(s)

____

Have you ever had a medical illness that has affected your thinking skills or perception (your ability to hear, see, or taste)?

- Yes (1)
- No (0)

Display This Question:

If Dem_PercDx = Yes

Please provide more information about your medical illness(es) that have affected your cognition or perception.

____

Do you have a history of a learning disorder or learning difficulties?

- Yes (1)
- No (0)

Display This Question:

If Dem_LearnDx = Yes

Please provide more information about your current and/or previous learning disorder(s) or learning difficulties.

____

Do you have a history of Attention Deficit-Hyperactivity Disorder (ADHD) or school-related behavioural problems?

- Yes (1)
- No (0)

Display This Question:

If Dem_ADHDDx = Yes

Please provide more information about your history of ADHD and/or school-related behavioural problems.

____

Q7.1 You are onto the next questionnaire!
Please read each question and decide if it is an accurate statement about you. Even if it's unclear, take your best guess. Give your own opinion of yourself. There are no right or wrong answers. Be sure to answer every question.

	Yes (1)	No (0)
Do you find the bright lights of a city exciting to look at? (OLIFE1R_S)	○	○
Does your sense of smell sometimes become unusually strong? (OLIFE2_U)	○	○
Have you sometimes sensed an evil presence around you, even though you could not see it? (OLIFE3_U)	○	○
Have you had very little fun from physical activities like walking, swimming, or sports? (OLIFE4_S)	○	○
Are you usually in an average sort of mood, not too high and not too low? (OLIFE5R_I)	○	○
Do you often feel that there is no purpose to life? (OLIFE6_C)	○	○
Are people usually better if they stay aloof from emotional involvements with people? (OLIFE7_S)	○	○
Do you think you could learn to read other’s minds if you wanted to? (OLIFE8_U)	○	○
Do you often overindulge in alcohol or food? (OLIFE9_I)	○	○
Are there very few things that you have ever really enjoyed doing? (OLIFE10_S)	○	○

Q8.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Can some people make you aware of them just by thinking about you? (OLIFE11_U)	○	○
Do you often feel lonely? (OLIFE12_C)	○	○
Do you worry about awful things that might happen? (OLIFE13_C)	○	○
Can just being with friends make you feel really good? (OLIFE14R_S)	○	○
Do you ever have a sense of vague danger or sudden dread for reasons that you do not understand? (OLIFE15_U)	○	○
Do you stop to think things over before doing anything? (OLIFE16R_I)	○	○
Do you find it difficult to keep interested in the same thing for a long time? (OLIFE17_C)	○	○
Do you ever have the urge to break or smash things? (OLIFE18_I)	○	○
Are you much too independent to really get involved with other people? (OLIFE19_S)	○	○
Do ideas and insights sometimes come to you so fast that you cannot express them all? (OLIFE20_U)	○	○

Q9.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Are you easily distracted when you read or talk to someone? (OLIFE21_C)	○	○
Would being in debt worry you? (OLIFE22R_I)	○	○
Do you often feel “fed up”? (OLIFE23_C)	○	○
Do you believe in telepathy? (OLIFE24_U)	○	○
Do people who drive carefully annoy you? (OLIFE25_I)	○	○
Can you usually let yourself go and enjoy yourself at a lively party? (OLIFE26R_S)	○	○
Do you ever feel sure that something is about to happen, even though there does not seem to be any reason for you thinking that? (OLIFE27_U)	○	○
Is it hard for you to make decisions? (OLIFE28_C)	○	○
Do people who try to get to know you better usually give up after a while? (OLIFE29_S)	○	○
Do you often feel like doing the opposite of what other people suggest, even though you know they are right? (OLIFE30_I)	○	○

Q10.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Does your voice ever seem distant or faraway? (OLIFE31_U)	◯	◯
Are you easily confused if too much happens at the same time? (OLIFE32_C)	◯	◯
Do you feel that making new friends isn't worth the energy it takes? (OLIFE33_S)	◯	◯
Do you often feel the impulse to spend money which you know you can't afford? (OLIFE34_I)	◯	◯
Do you worry too long after an embarrassing experience? (OLIFE35_C)	◯	◯
Do you ever suddenly feel distracted by distant sounds that you are not normally aware of? (OLIFE36_U)	◯	◯
Do you often have an urge to hit someone? (OLIFE37_I)	◯	◯
Do you often have days when indoor lights seem so bright that they bother your eyes? (OLIFE38_U)	◯	◯
Do you like going out a lot? (OLIFE39R_S)	◯	◯
Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense? (OLIFE40_C)	◯	◯

Q11.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Would it make you nervous to play the clown in front of other people? (OLIFE41R_I)	○	○
Do you have many friends? (OLIFE42R_S)	○	○
Have you ever felt that you have special, almost magical powers? (OLIFE43_U)	○	○
Are you a person whose mood goes up and down easily? (OLIFE44_C)	○	○
Are you rather lively? (OLIFE45R_S)	○	○
Have you ever blamed someone for doing something you know was really your fault? (OLIFE46_I)	○	○
Have you sometimes had the feeling of gaining or losing energy when certain people look at you or touch you? (OLIFE47_U)	○	○
Would you call yourself a nervous person? (OLIFE48_C)	○	○
Do you like mixing with people? (OLIFE49R_S)	○	○
Please select 'No' for this question. (VAL1)	○	○

Q12.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Have you felt as though your head or limbs were somehow not your own? (OLIFE50_U)	○	○
Do you sometimes talk about things you know nothing about? (OLIFE51_I)	○	○
Do you often experience an overwhelming sense of emptiness? (OLIFE52_C)	○	○
Do you at times have an urge to do something harmful or shocking? (OLIFE53_I)	○	○
Do you think having close friends is not as important as some people say? (OLIFE54_S)	○	○
Are the sounds that you hear in your daydreams really clear and distinct? (OLIFE55_U)	○	○
Are you easily hurt when people find fault with you or the work you do? (OLIFE56_C)	○	○
Have you wondered whether the spirits of the dead can influence the living? (OLIFE57_U)	○	○
Do you often change between intense liking and disliking of the same person? (OLIFE58_I)	○	○
Do you enjoy many different kinds of play and recreation? (OLIFE59R_S)	○	○

Q13.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Do your thoughts sometimes seem as real as actual events in your life? (OLIFE60_U)	○	○
Are you sometimes so nervous that you are “blocked”? (OLIFE61_C)	○	○
Do you feel so good at controlling others that it sometimes scares you? (OLIFE62_U)	○	○
Do you think people spend too much time safeguarding their future with savings and insurance? (OLIFE63_I)	○	○
Does it often feel good to massage your muscles when they are tired or sore? (OLIFE64R_S)	○	○
Do you dread going into a room by yourself where other people have already gathered and are talking? (OLIFE65_C)	○	○
Do you often feel there is purpose to life? (INC1)	○	○
On occasions, have you seen a person’s face in front of you when no one was in fact there? (OLIFE66_U)	○	○
Do you prefer watching television over going out with other people? (OLIFE67_S)	○	○
Have you ever cheated at a game? (OLIFE68_I)	○	○

Q14.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
On seeing a soft thick carpet have you sometimes had the impulse to take off your shoes and walk barefoot on it? (OLIFE69R_S)	○	○
When in the dark do you often see shapes and forms even though there's nothing there? (OLIFE70_U)	○	○
Is it easy for you to make decisions? (INC2)	○	○
Do you easily lose your courage when criticised or failing in something? (OLIFE71_C)	○	○
Has dancing or the idea of it always seemed dull to you? (OLIFE72_S)	○	○
Have you ever felt the urge to injure yourself? (OLIFE73_I)	○	○
Are your thoughts sometimes so strong that you can almost hear them? (OLIFE74_U)	○	○
Do you sometimes feel that your accidents are caused by mysterious forces? (OLIFE75_U)	○	○
Do you frequently have difficulty in starting to do things? (OLIFE76_C)	○	○
Do you have few friends? (INC3)	○	○

Q15.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Is it fun to sing with other people? (OLIFE77R_S)	○	○
When you catch a train do you often arrive at the last minute? (OLIFE78_I)	○	○
When you look in the mirror does your face sometimes seem quite different from usual? (OLIFE79_U)	○	○
Do you usually have very little desire to buy new kinds of food? (OLIFE80_S)	○	○
Do you often have difficulties in controlling your thoughts? (OLIFE81_C)	○	○
When in a group of people do you usually prefer to let someone else be the centre of attention? (OLIFE82R_I)	○	○
Have you ever thought you heard people talking only to discover that it was in fact some nondescript noise? (OLIFE83_U)	○	○
Have you often felt uncomfortable when friends touch you? (OLIFE84_S)	○	○
Do you worry about things you should not have done or said? (OLIFE85_C)	○	○
Do you think having close friends is as important as some people say? (INC4)	○	○

Q16.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Would you take drugs which may have strange or dangerous effects? (OLIFE86_I)	○	○
Have you occasionally felt as though your body did not exist? (OLIFE87_U)	○	○
Do you love having your back massaged? (OLIFE88R_S)	○	○
Does it happen that nearly every thought immediately and automatically suggests an enormous number of ideas? (OLIFE89_U)	○	○
Do you often hesitate when you are going to say something in a group of people whom you more or less know? (OLIFE90_C)	○	○
Do you consider yourself to be pretty much an average kind of person? (OLIFE91R_I)	○	○
Do people in your daydreams seem so true to life that you sometimes think they are real? (OLIFE92_U)	○	○
Do you feel very close to your friends? (OLIFE93R_S)	○	○
No matter how hard you try to concentrate do unrelated thoughts creep into your mind? (OLIFE94_C)	○	○
Have you ever taken advantage of someone? (OLIFE95_I)	○	○

Q17.1 Read each question and decide if it is an accurate statement about you. Give your own opinion of yourself. Be sure to answer every question.

	Yes (1)	No (0)
Is your hearing sometimes so sensitive that ordinary sounds become uncomfortable? (OLIFE96_U)	○	○
When in a crowded room, do you often have difficulty in following a conversation? (OLIFE97_C)	○	○
Is trying new foods something you have always enjoyed? (OLIFE98R_S)	○	○
Does a passing thought ever seem so real it frightens you? (OLIFE99_U)	○	○
Would you like other people to be afraid of you? (OLIFE100_I)	○	○
When things are bothering you do you like to talk to other people about it? (OLIFE101R_S)	○	○
Have you felt that you might cause something to happen just by thinking too much about it? (OLIFE102_U)	○	○
Are you easily distracted from work by daydreams? (OLIFE103_C)	○	○
Is it true that your relationships with other people never get very intense? (OLIFE104_S)	○	○

Q18.1 This task is about solving puzzles.
Each task shows an incomplete jigsaw puzzle. The patterns you will see follow rules which may apply to a row, a column or to a diagonal. They may apply to the figure as a whole or to parts of it only. They may involve addition, subtraction, the alignment of figures or single components.

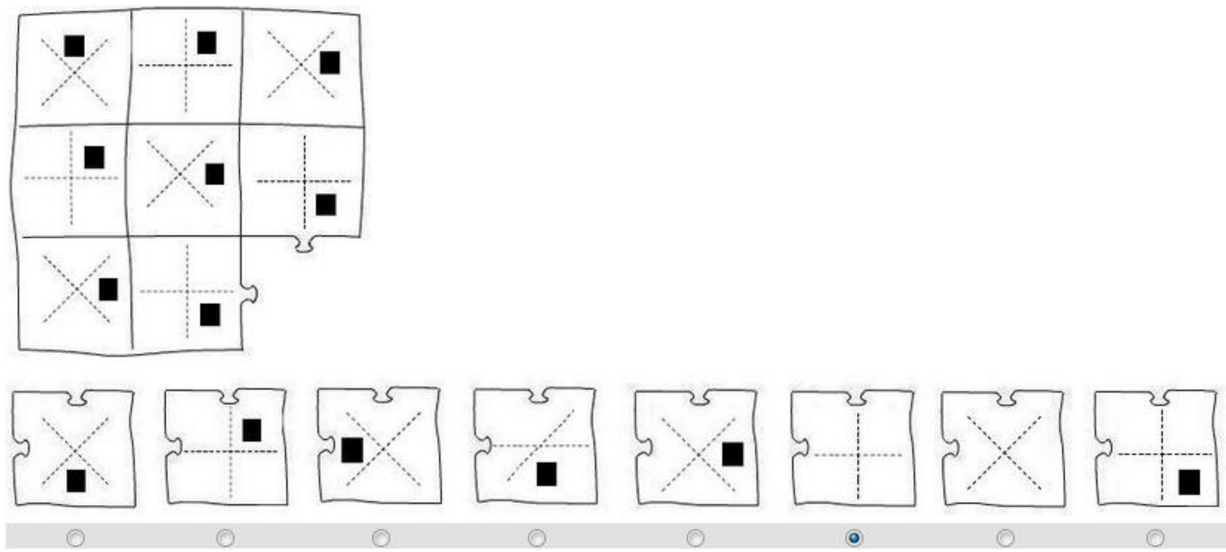
Only one of the eight pieces given is the correct one required to complete the design. It is your task to select the piece which completes the jigsaw puzzle. Each task needs to be completed within 2:00 minutes.

Let's start with a practice. If you are using a **mobile phone or tablet**, please **turn your screen to landscape** for these questions.

[Participants were shown two untimed sample items and provided corrective feedback upon giving their answers.]

[EXAMPLE OF HMT-S ITEM]

Which piece is the one required to complete the design? (Select the correct piece and click the arrow button to continue)



[Following the two sample items, the participants were presented with six test items.]

Q21.1 Now you will be presented with the test items.

Mark the piece which is, in your opinion, the correct one and click on the arrow button, which is shown after a short moment. Each task is timed at 2:00 minutes. The next jigsaw puzzle will be shown automatically after the 2:00 minute period has elapsed.

If you are using a **mobile phone or tablet**, please **turn your screen to landscape** for these questions.

Good luck!

[The specific HMT-S items are not presented here. Please contact the author of this research program for specific items, if needed.]

Q28.1
You are onto the next questionnaire!

Please read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. There are no right or wrong answers. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I am so tense in certain situations that I have great difficulty getting by. (PAI4_ANX-A)	○	○	○	○
Much of the time I'm sad for no real reason. (PAI6_DEP-A)	○	○	○	○
Most of the people I know can be trusted. (PAI8_PAR-H_R)	○	○	○	○
Sometimes I cannot remember who I am. (PAI9_NIM)	○	○	○	○
I have some ideas that others think are strange. (PAI10_SCZ-P)	○	○	○	○
Sometimes I feel guilty about how much I drink. (PAI15_ALC)	○	○	○	○
Sometimes I use drugs to feel better. (PAI22_DRG)	○	○	○	○
I've tried just about every type of drug. (PAI23_DRG)	○	○	○	○
Sometimes I let little things bother me too much. (PAI24_PIM_R)	○	○	○	○
I often have trouble concentrating because I'm nervous. (PAI25_ANX-C)	○	○	○	○

Q29.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I feel that I've let everyone down. (PAI27_DEP-C)	○	○	○	○
Certain people go out of their way to bother me. (PAI29_PAR-P)	○	○	○	○
I just don't seem to relate to people very well. (PAI30_SCZ-S)	○	○	○	○
I often feel jittery. (PAI33_ANX-P)	○	○	○	○
Please select 'Slighty True' for this question. (VAL2)	○	○	○	○
I hardly have any energy. (PAI35_DEP-P)	○	○	○	○
People usually treat me pretty fairly. (PAI37_PAR-R_R)	○	○	○	○
My thinking has become confused. (PAI38_SCZ-T)	○	○	○	○
I can't do some things well because of nervousness. (PAI44_ANX-A)	○	○	○	○
I've forgotten what it's like to feel happy. (PAI46_DEP-A)	○	○	○	○

Q30.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I have to be alert to the possibility that people will be unfaithful. (PAI48_PAR-H)	○	○	○	○
I have visions in which I see myself forced to commit crimes. (PAI49_NIM)	○	○	○	○
Other people sometimes put thoughts into my head. (PAI50_SCZ-P)	○	○	○	○
I have trouble controlling my use of alcohol. (PAI55_ALC)	○	○	○	○
People have told me that I have a drug problem. (PAI62_DRG)	○	○	○	○
I never use drugs to help me cope with the world. (PAI63_DRG_R)	○	○	○	○
Sometimes I'll avoid someone I really don't like. (PAI64_PIM_R)	○	○	○	○
It's often hard for me to enjoy myself because I am worrying about things. (PAI65_ANX-C)	○	○	○	○
Sometimes I think I'm worthless. (PAI67_DEP-C)	○	○	○	○
Some people do things to make me look bad. (PAI69_PAR-P)	○	○	○	○

Q31.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I don't have much to say to anyone. (PAI70_SCZ-S)	○	○	○	○
I worry so much that at times I feel like I am going to faint. (PAI73_ANX-P)	○	○	○	○
I have no trouble falling asleep. (PAI75_DEP-P_R)	○	○	○	○
I seem to have as much luck in life as others do. (PAI77_PAR-R_R)	○	○	○	○
My thoughts get scrambled sometimes. (PAI78_SCZ-T)	○	○	○	○
Sometimes I am afraid for no reason. (PAI84_ANX-A)	○	○	○	○
Everything seems like a big effort. (PAI86_DEP-A)	○	○	○	○
Most people have good intentions. (PAI88_PAR-H_R)	○	○	○	○
Since the day I was born, I was destined to be unhappy. (PAI89_NIM)	○	○	○	○
Sometimes it seems that my thoughts are broadcast so that others can hear them. (PAI90_SCZ-P)	○	○	○	○

Q32.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
There have been times when I've had to cut down on my drinking. (PAI95_ALC)	○	○	○	○
My drug use has caused me financial strain. (PAI102_DRG)	○	○	○	○
I've never had problems at work because of drugs. (PAI103_DRG_R)	○	○	○	○
I sometimes complain too much. (PAI104_PIM_R)	○	○	○	○
I'm often worried and nervous that I can barely stand it. (PAI105_ANX-C)	○	○	○	○
I don't feel like trying anymore. (PAI107_DEP-C)	○	○	○	○
People around me are faithful to me. (PAI109_PAR-P_R)	○	○	○	○
I'm a loner. (PAI110_SCZ-S)	○	○	○	○
Sometimes I feel dizzy when I've been under a lot of pressure. (PAI113_ANX-P)	○	○	○	○
I rarely have trouble sleeping. (PAI115_DEP-P_R)	○	○	○	○

Q33.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
I've given a lot, but I haven't gotten much in return. (PAI117_PAR-R)	○	○	○	○
Sometimes I have trouble keeping different thoughts separate. (PAI118_SCZ-T)	○	○	○	○
I'm not the kind of person who panics easily. (PAI124_ANX-A_R)	○	○	○	○
Nothing seems to give me much pleasure. (PAI126_DEP-A)	○	○	○	○
I usually assume people are telling the truth. (PAI128_PAR-H_R)	○	○	○	○
I think I have three to four completely different personalities inside me. (PAI129_NIM)	○	○	○	○
Others can read my thoughts. (PAI130_SCZ-P)	○	○	○	○
My drinking seems to cause problems in my relationships with others. (PAI135_ALC)	○	○	○	○
I never use illegal drugs. (PAI142_DRG_R)	○	○	○	○
Sometimes I'm too impatient. (PAI144_PIM_R)	○	○	○	○

Q34.1 Read each statement and decide if it is an accurate statement about you. Mark your answer by filling in one of the circles on the answer sheet.

Give your own opinion of yourself. Be sure to answer every statement.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
My friends say I worry too much. (PAI145_ANX-C)	○	○	○	○
Please select 'False' for this question (VAL3)	○	○	○	○
I can't seem to concentrate very well. (PAI147_DEP-C)	○	○	○	○
Some people try to keep me from getting ahead. (PAI149_PAR-P)	○	○	○	○
I don't feel close to anyone. (PAI150_SCZ-S)	○	○	○	○
I can often feel my heart pounding. (PAI153_ANX-P)	○	○	○	○
I've been moving more slowly than usual. (PAI155_DEP-P)	○	○	○	○
People don't appreciate what I've done for them. (PAI157_PAR-R)	○	○	○	○
Sometimes it feels as if somebody is blocking my thoughts. (PAI158_SCZ-T)	○	○	○	○

Q35.1
Great job so far! You're onto the next part of this survey.
For each item, please indicate how often you have felt that way.

	Never (0)	Sometimes (1)	Often (2)	Nearly Always (3)
Have you felt as if people seem to drop hints about you or say things with a double meaning? (CAPE1)	○	○	○	○
Have you felt as if some people are not what they seem to be? (CAPE2)	○	○	○	○
Have you felt that you are being persecuted in anyway? (CAPE3)	○	○	○	○
Have you felt as if there is a conspiracy against you? (CAPE4)	○	○	○	○
Have you felt as if electrical devices such as computers can influence the way you think? (CAPE5)	○	○	○	○
Have you felt that people look at you oddly because of your appearance? (CAPE6)	○	○	○	○
Have you felt as if the thoughts in your head are being taken away from you? (CAPE7)	○	○	○	○
Have you felt as if the thoughts in your head are not your own? (CAPE8)	○	○	○	○

Q35.2
For each item, please indicate how often you have felt that way.

	Never (0)	Sometimes (1)	Often (2)	Nearly Always (3)
Have your thoughts been so vivid that you were worried other people would hear them? (CAPE9)	○	○	○	○
Have you heard your thoughts being echoes back to you? (CAPE10)	○	○	○	○
Have you felt as if you are under the control of some force or power other than yourself? (CAPE11)	○	○	○	○
Have you heard voices when you are alone? (CAPE12)	○	○	○	○
Have you heard voices talking to each other when you are alone? (CAPE13)	○	○	○	○
Have you felt as if a double has taken the place of a family member, friend, or acquaintance? (CAPE14)	○	○	○	○
Have you seen objects, people, or animals that other people can't see? (CAPE15)	○	○	○	○

Q36.1
You're onto the next questionnaire!

This is a list of things different people might say about themselves. We are interested in how you would describe yourself. There are no right or wrong answers.

Read each statement carefully, selecting the response that best describes you.

	Often False (1)	Somewhat False (2)	Somewhat True (3)	Often True (4)
People have told me that I think about things in a really strange way. (PDI1_Ecc_1)	○	○	○	○
Others seem to think I'm quite odd or unusual. (PDI1_Ecc_2)	○	○	○	○
I think about things in odd ways that don't make sense to most people. (PDI1_Ecc_3)	○	○	○	○
I often have thoughts that make sense to me but that other people say are strange. (PDI1_Ecc_4)	○	○	○	○

Q36.2 We are interested in how you would describe yourself. There are no right or wrong answers.

Read each statement carefully, selecting the response that best describes you.

	Often False (1)	Somewhat False (2)	Somewhat True (3)	Often True (4)
I often have unusual experiences, such as sensing the presence of someone who isn't actually there. (PDI2_UnuBE_1)	○	○	○	○
I have seen things that weren't really there. (PDI2_UnuBE_2)	○	○	○	○
Sometimes I can influence other people just by sending my thoughts to them. (PDI2_UnuBE_3)	○	○	○	○
I've had some really weird experiences that are very difficult to explain. (PDI2_UnuBE_4)	○	○	○	○

Q36.3 We are interested in how you would describe yourself. There are no right or wrong answers.

Read each statement carefully, selecting the response that best describes you.

	Often False (1)	Somewhat False (2)	Somewhat True (3)	Often True (4)
It's weird, but sometimes ordinary objects seem to be a different shape than usual. (PDI3_PeDy_1)	○	○	○	○
Sometimes I feel "controlled" by thoughts that belong to someone else. (PDI3_PeDy_2)	○	○	○	○
Sometimes I think someone else is removing thoughts from my head. (PDI3_PeDy_3)	○	○	○	○
Things around me often feel unreal, or more real than usual. (PDI3_PeDy_4)	○	○	○	○

Q37.1
Great job so far! This is the last questionnaire.

There are no right or wrong answers. How much do you agree with each statement about you as you generally are now?

	Strongly Disagree (1)	Somewhat Disagree (2)	Neither Agree nor Disagree (3)	Somewhat Agree (4)	Strongly Agree (5)
I am the life of the party. (EX1)	○	○	○	○	○
I sympathise with others' feelings. (AG1)	○	○	○	○	○
I get chores done right away. (CO1)	○	○	○	○	○
I have frequent mood swings. (NE1)	○	○	○	○	○
I have a vivid imagination. (OP1)	○	○	○	○	○
I don't talk a lot. (EX2R)	○	○	○	○	○
I am not interested in other people's problems. (AG2R)	○	○	○	○	○

Q37.2
Remember, there are no right or wrong answers.
How much do you agree with each statement about you as you generally are now?

	Strongly Disagree (1)	Somewhat Disagree (2)	Neither Agree nor Disagree (3)	Somewhat Agree (4)	Strongly Agree (5)
I often forget to put things back in their proper place. (CO2R)	○	○	○	○	○
I am relaxed most of the time. (NE2R)	○	○	○	○	○
I am not interested in abstract ideas. (OP2R)	○	○	○	○	○
I talk to a lot of different people at parties. (EX3)	○	○	○	○	○
I feel others' emotions. (AG3)	○	○	○	○	○
I like order. (CO3)	○	○	○	○	○
I get upset easily. (NE3)	○	○	○	○	○

Q37.3
Remember, there are no right or wrong answers.
How much do you agree with each statement about you as you generally are now?

	Strongly Disagree (1)	Somewhat Disagree (2)	Neither Agree nor Disagree (3)	Somewhat Agree (4)	Strongly Agree (5)
I have difficulty understanding abstract ideas. (OP3R)	○	○	○	○	○
Please select 'Strongly Agree' for this question. (VAL4)	○	○	○	○	○
I keep in the background. (EX4R)	○	○	○	○	○
I am not really interested in others. (AG4R)	○	○	○	○	○
I make a mess of things. (CO4R)	○	○	○	○	○
I seldom feel blue. (NE4R)	○	○	○	○	○
I do not have a good imagination. (OP4R)	○	○	○	○	○

Thanks for your time and effort! We have one more question for you about the quality of your response.

Please know that regardless of your answer to this question, you will be compensated based on the rate listed on Prolific for this study and in line with Prolific's guidelines for acceptable responses.

In your honest opinion, should we use your data?

○ Yes (1)

○ No (0)

C.2 Methods Flowchart

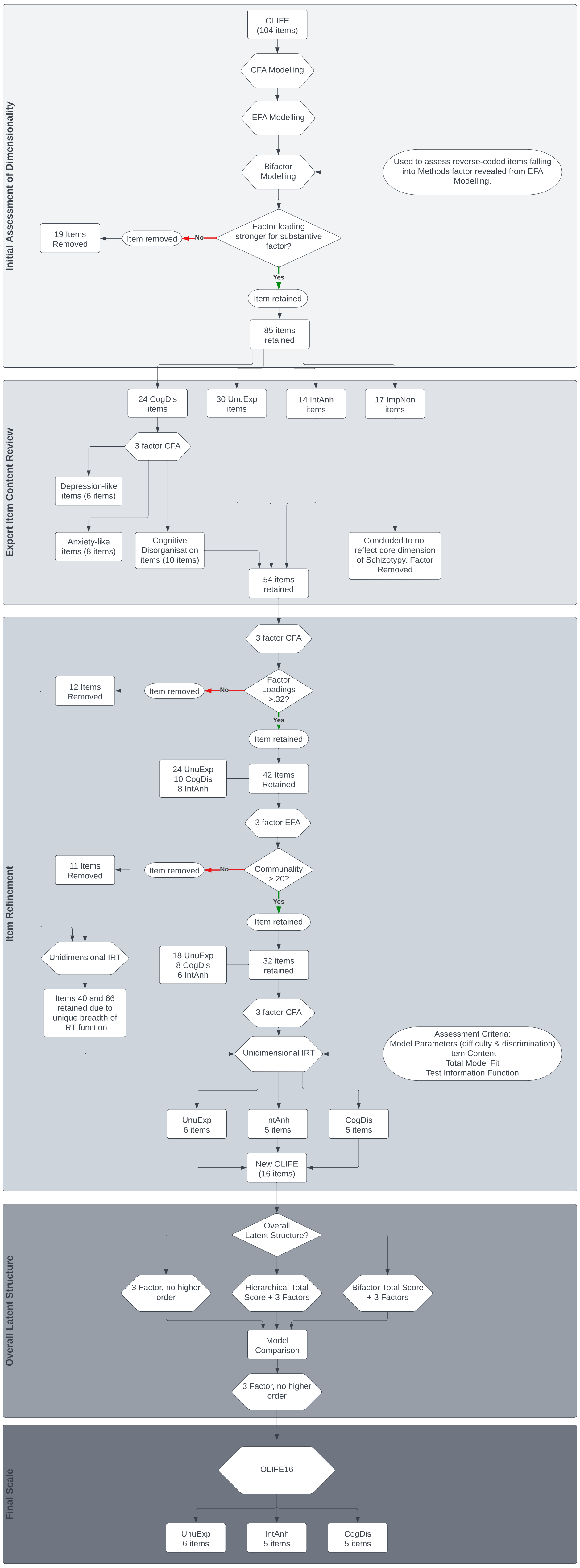


Figure C.1 Flowchart of methodology process of Phase 1 of Study 2.

C.3 Item Refinement via IRT Additional Outputs

Following item refinement based on factor loadings, through confirmatory factor analysis, and communalities, through exploratory factor analysis, the resulting 34 items were entered into 2-parameter unidimensional IRT analysis. The following section details the results of this analysis. All output is from computed using R (v4.2.1; R Core Team, 2022) through RStudio (v4.1.2; RStudio Team, 2020).

C.3.1 Unusual Experiences

Table C. 1 Proportion for each level of response – Unusual Experiences.

Item	0	1	logit
OLIFE3_U	0.5963	0.4037	-0.3902
OLIFE15_U	0.4358	0.5642	0.2583
OLIFE27_U	0.3739	0.6261	0.5157
OLIFE36_U	0.5826	0.4174	-0.3333
OLIFE47_U	0.5849	0.4151	-0.3428
OLIFE55_U	0.6995	0.3005	-0.8451
OLIFE57_U	0.5023	0.4977	-0.0092
OLIFE60_U	0.4106	0.5894	0.3617
OLIFE66_U	0.9014	0.0986	-2.2126
OLIFE70_U	0.5275	0.4725	-0.1102
OLIFE74_U	0.6032	0.3968	-0.4189
OLIFE75_U	0.7775	0.2225	-1.2513
OLIFE79_U	0.6491	0.3509	-0.615
OLIFE87_U	0.8028	0.1972	-1.4036
OLIFE89_U	0.5872	0.4128	-0.3522
OLIFE92_U	0.7294	0.2706	-0.9914
OLIFE99_U	0.6353	0.3647	-0.5551
OLIFE102_U	0.5665	0.4335	-0.2676

Table C.2 Unusual Experience items total model fit – round 1 of 4.

	M2	df	p	RMSEA	RMSEA_5	RMSEA_95	SRMSR	TLI	CFI
stats	375.208	152.000	0.000	0.058	0.051	0.065	0.054	0.959	0.964

Table C.3 Unusual Experience items model parameters – round 1 of 4.

	a	b
OLIFE3_U	**1.290**	**0.411**
OLIFE15_U	**1.495**	**-0.232**
OLIFE27_U	1.814	-0.430
OLIFE36_U	1.784	0.304
OLIFE47_U	**1.416**	**0.346**
OLIFE50_U	1.582	1.311
OLIFE55_U	1.627	0.769
OLIFE57_U	**1.202**	**0.020**
OLIFE60_U	2.204	-0.272
OLIFE66_U	1.276	2.174
OLIFE70_U	1.607	0.115
OLIFE74_U	2.019	0.361
OLIFE75_U	1.541	1.148
OLIFE79_U	1.517	0.586
OLIFE87_U	1.738	1.205
OLIFE89_U	**1.369**	**0.361**
OLIFE92_U	2.187	0.794
OLIFE99_U	2.172	0.460
OLIFE102_U	1.548	0.262

Bolded items removed.

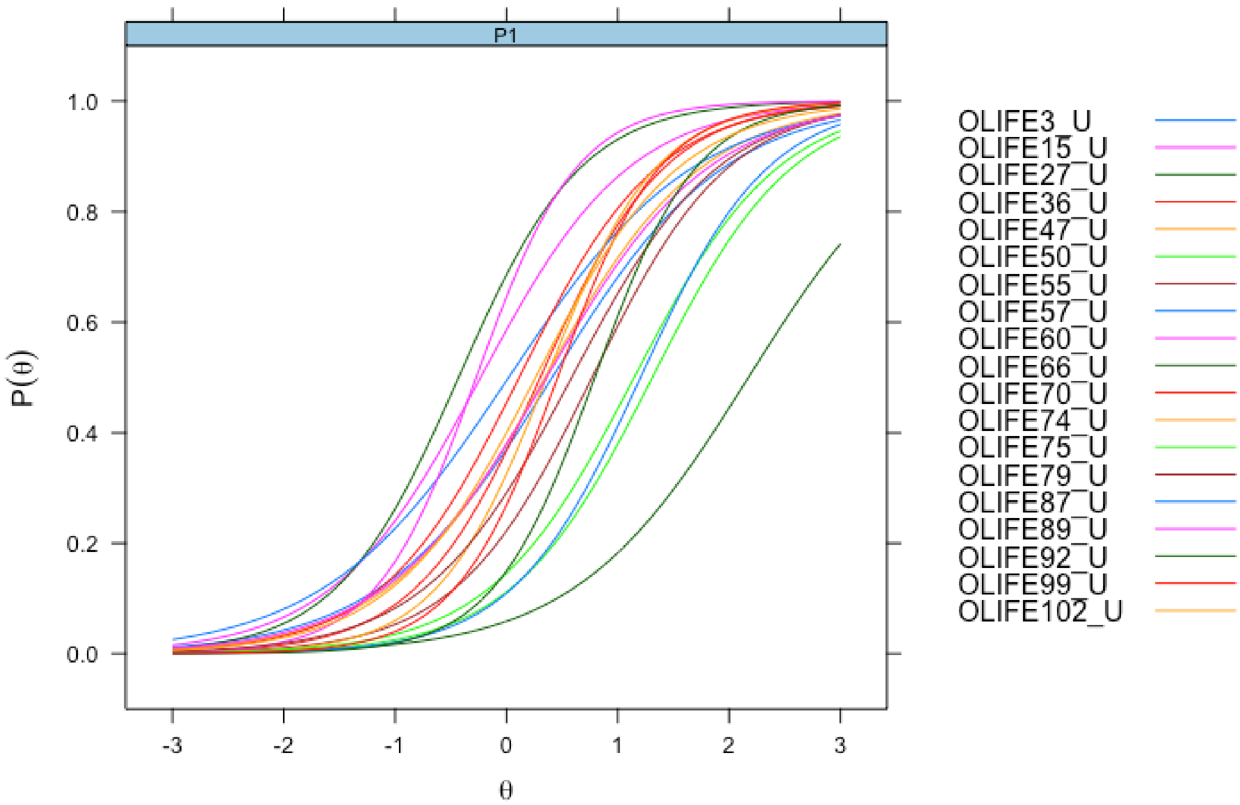


Figure C.2 Item probability functions for initial IRT analysis of Unusual Experiences items.

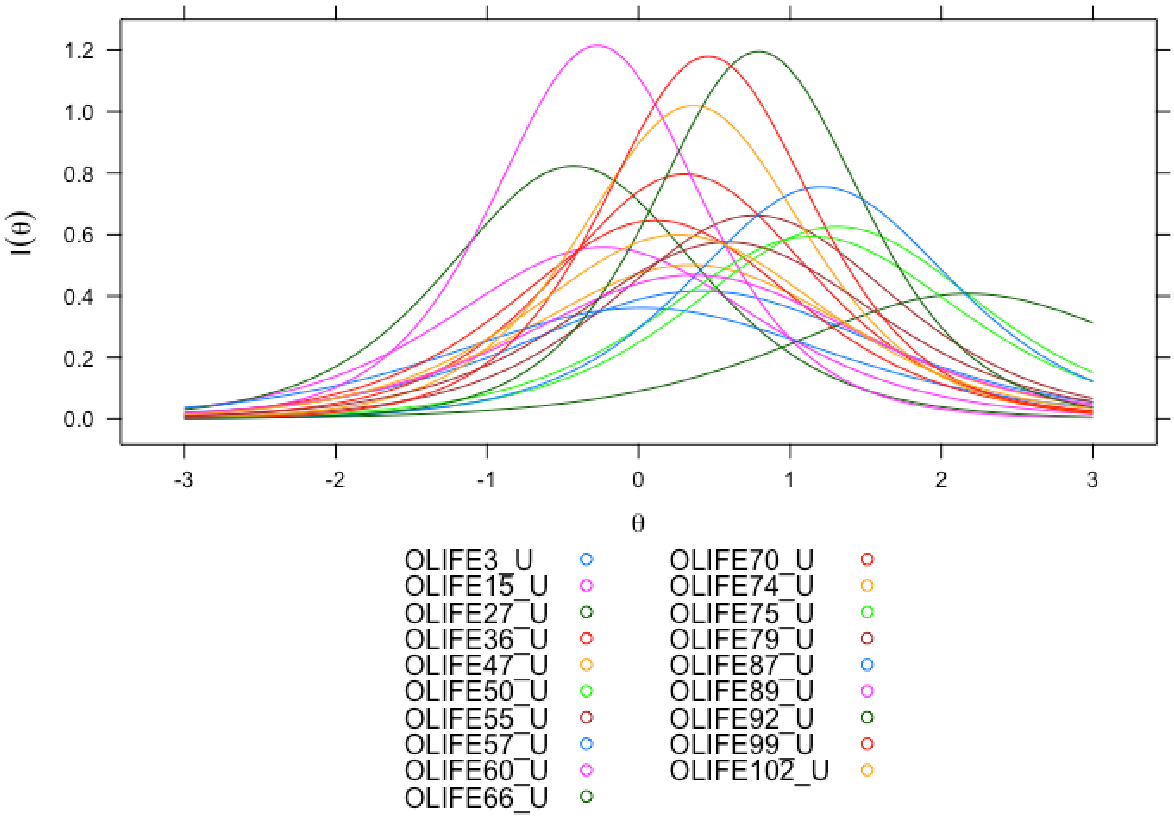


Figure C.3 Item information curves for initial Unusual Experiences items.

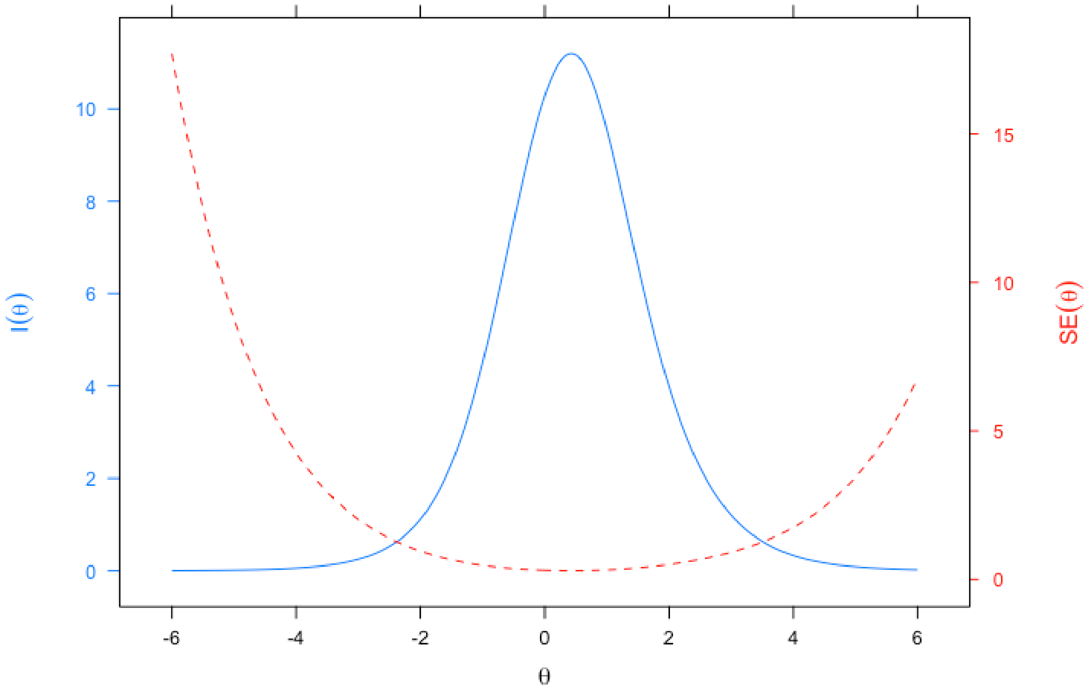


Figure C.4 Test information and standard error curves for Unusual Experience items.

Table C.4 Unusual Experience items total model fit – round 2 of 4.

	M2	df	p	RMSEA	RMSEA_5	RMSEA_95	SRMSR	TLI	CFI
stats	214.5949	90	$3.53e^{-12}$	0.056414	0.046694	0.066080	0.053211	0.96432	0.96942

Table C.5 Unusual Experience items model parameters – round 2 of 4.

	a	b
OLIFE27_U	1.598	-0.463
OLIFE36_U	1.914	0.290
OLIFE50_U	**1.587**	**1.309**
OLIFE55_U	1.676	0.755
OLIFE60_U	2.410	-0.270
OLIFE66_U	1.216	2.248
OLIFE70_U	**1.586**	**0.109**
OLIFE74_U	2.028	0.354
OLIFE75_U	**1.372**	**1.221**
OLIFE79_U	1.502	0.585
OLIFE87_U	1.680	1.224
OLIFE89_U	**1.427**	**0.349**
OLIFE92_U	2.253	0.782
OLIFE99_U	2.292	0.445
OLIFE102_U	**1.401**	**0.270**

Bolded item removed.

Table C.6 Unusual Experience items total model fit – round 3 of 4.

	M2	df	p	RMSEA	RMSEA_5	RMSEA_95	SRMSR	TLI	CFI
stats	74.7466	35	0.000105	0.051036	0.034910	0.066972	0.046028	0.97413	0.97988

Table C.7 Unusual Experience items model parameters – round 3 of 4.

	a	b
OLIFE27_U	1.461	-0.485
OLIFE36_U	**1.796**	**0.294**
OLIFE55_U	**1.812**	**0.728**
OLIFE60_U	2.82	-0.263
OLIFE66_U	1.199	2.274
OLIFE74_U	**1.954**	**0.355**
OLIFE79_U	**1.402**	**0.606**
OLIFE87_U	1.462	1.317
OLIFE92_U	2.454	0.76
OLIFE99_U	2.23	0.446

Bolded items removed.

Table C.8 Unusual Experience items total model fit – final model (round 4 of 4).

	M2	df	p	RMSEA	RMSEA_5	RMSEA_95	SRMSR	TLI	CFI
stats	9.3513	9	0.4055	0.009462	0	0.055160	0.026916	0.99901	0.99941

Table C.9 Unusual Experience items model parameters – final model (round 4 of 4).

	a	b	Item
OLIFE27_U	1.769	-0.442	Do you ever feel sure that something is about to happen, even though there does not seem to be any reason for you thinking that?
OLIFE60_U	3.478	-0.250	Do your thoughts sometimes seem as real as actual events in your life?
OLIFE66_U	1.186	2.292	On occasions, have you seen a person's face in front of you when no one was in fact there?
OLIFE87_U	1.483	1.302	Have you occasionally felt as though your body did not exist?
OLIFE92_U	2.040	0.801	Do people in your daydreams seem so true to life that you sometimes think they are real?
OLIFE99_U	2.121	0.447	Does a passing thought ever seem so real it frightens you?

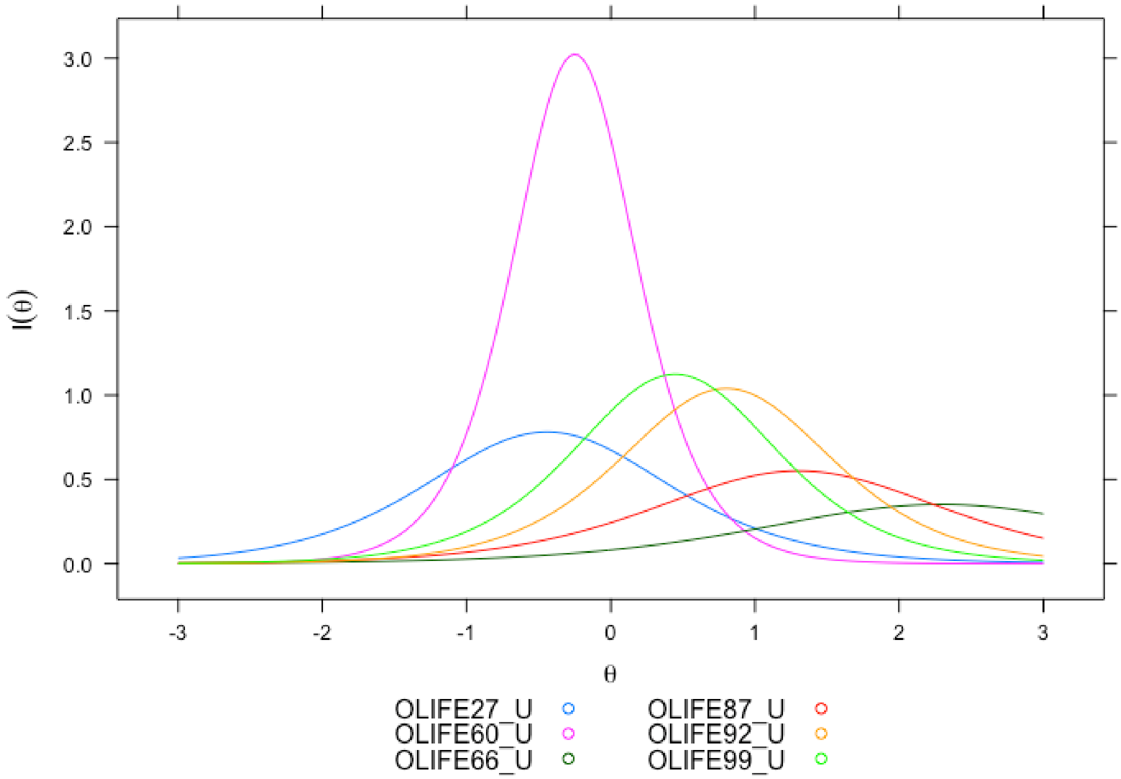


Figure C.5 Item information curves for final Unusual Experience items.

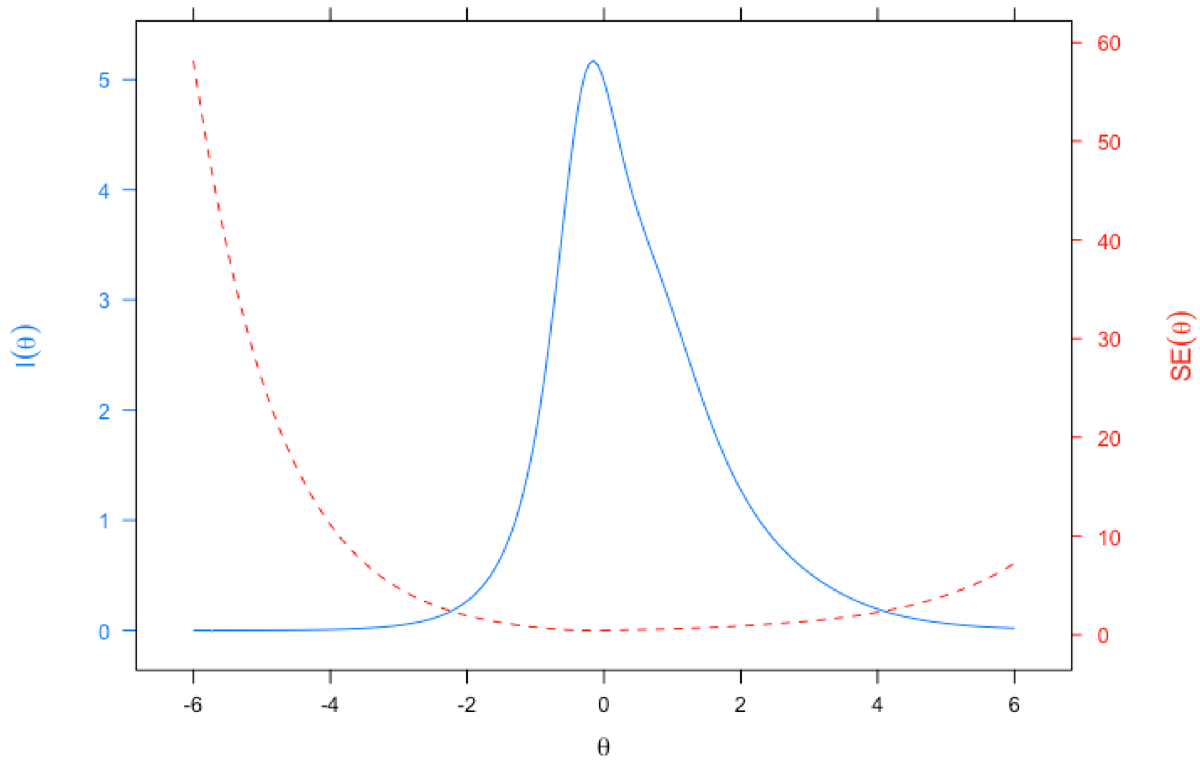


Figure C.6 Test information and standard error for final Unusual Experiences subscale.

C.3.2 Introvertive Anhedonia

Table C.10 Proportions for each level of response – Introvertive Anhedonia.

Item	0	1	logit
OLIFE7_S	0.6888	0.3112	-0.7945
OLIFE10_S	0.6201	0.3799	-0.4901
OLIFE19_S	0.54	0.46	-0.1605
OLIFE29_S	0.6751	0.3249	-0.7311
OLIFE33_S	0.5744	0.4256	-0.2997
OLIFE67_S	0.5011	0.4989	-0.0046

Table C.11 Introvertive Anhedonia items total model fit – round 1 of 2.

	M2	df	p	RMSEA	RMSEA_5	RMSEA_95	SRMSR	TLI	CFI
stats	15.010	14	0.377444	0.01286589	0	0.04891741	0.030899	0.997979	0.9986527

Table C.12 Introvertive Anhedonia items model parameters – round 1 of 2.

	a	b
OLIFE7_S	1.267	0.82
OLIFE10_S	**1.397**	**0.48**
OLIFE19_S	1.814	0.138
OLIFE29_S	1.733	0.639
OLIFE33_S	1.961	0.25
OLIFE67_S	1.267	0.005
OLIFE84_S	**1.22**	**0.489**

Bolded items removed.

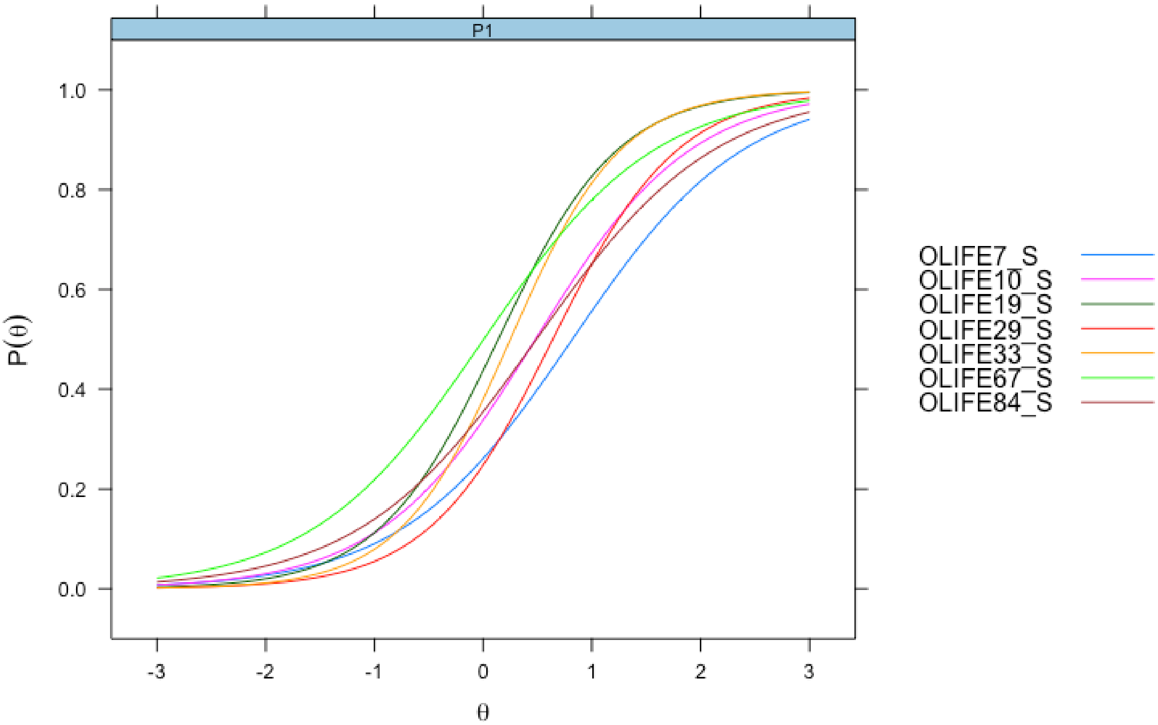


Figure C.7 Item probability functions for initial IRT analysis of Introvertive Anhedonia items.

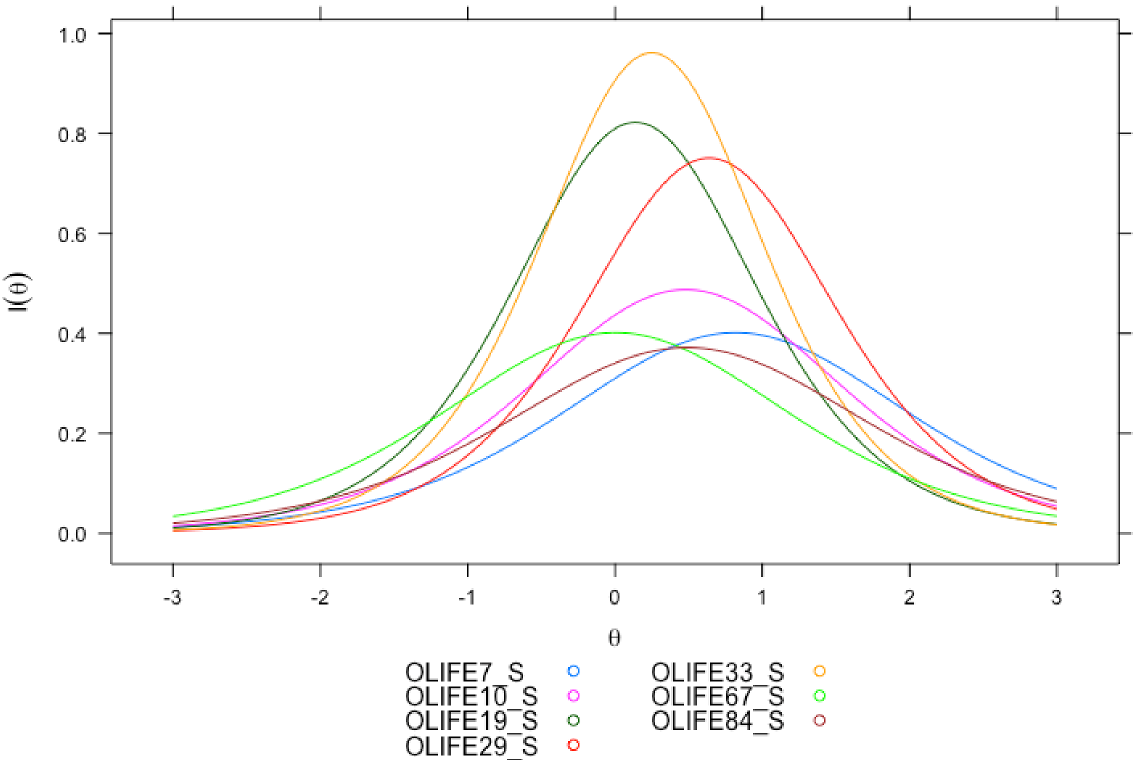


Figure C.8 Item information curves for initial Introvertive Anhedonia items.

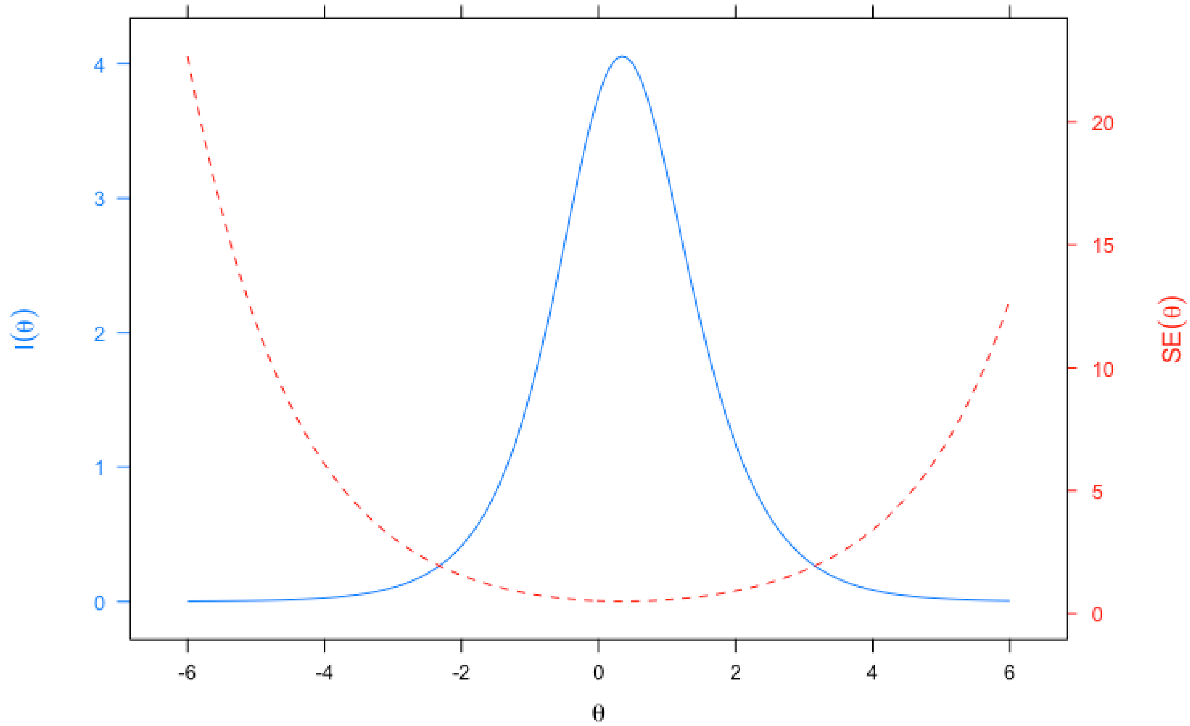


Figure C.9 Test information and standard error for initial Introvertive Anhedonia subscale.

Table C.13 Introvertive Anhedonia items total model fit – final items (round 2 of 2).

	M2	df	p	RMSEA	RMSEA_5	RMSEA_95	SRMSR	TLI	CFI
stats	10.18637	5	0.07012345	0.04877571	0	0.09179061	0.03569361	0.9741064	0.9870532

Table C.14 Introvertive Anhedonia items model parameters – final items (round 2 of 2).

	a	b	Items
OLIFE7_S	1.223	0.835	Are people usually better if they stay aloof from emotional involvements with people?
OLIFE19_S	1.791	0.135	Are you much too independent to really get involved with other people?
OLIFE29_S	1.749	0.632	Do people who try to get to know you better usually give up after a while?
OLIFE33_S	2.07	0.239	Do you feel that making new friends isn't worth the energy it takes?
OLIFE67_S	1.251	0.002	Do you prefer watching television over going out with other people?

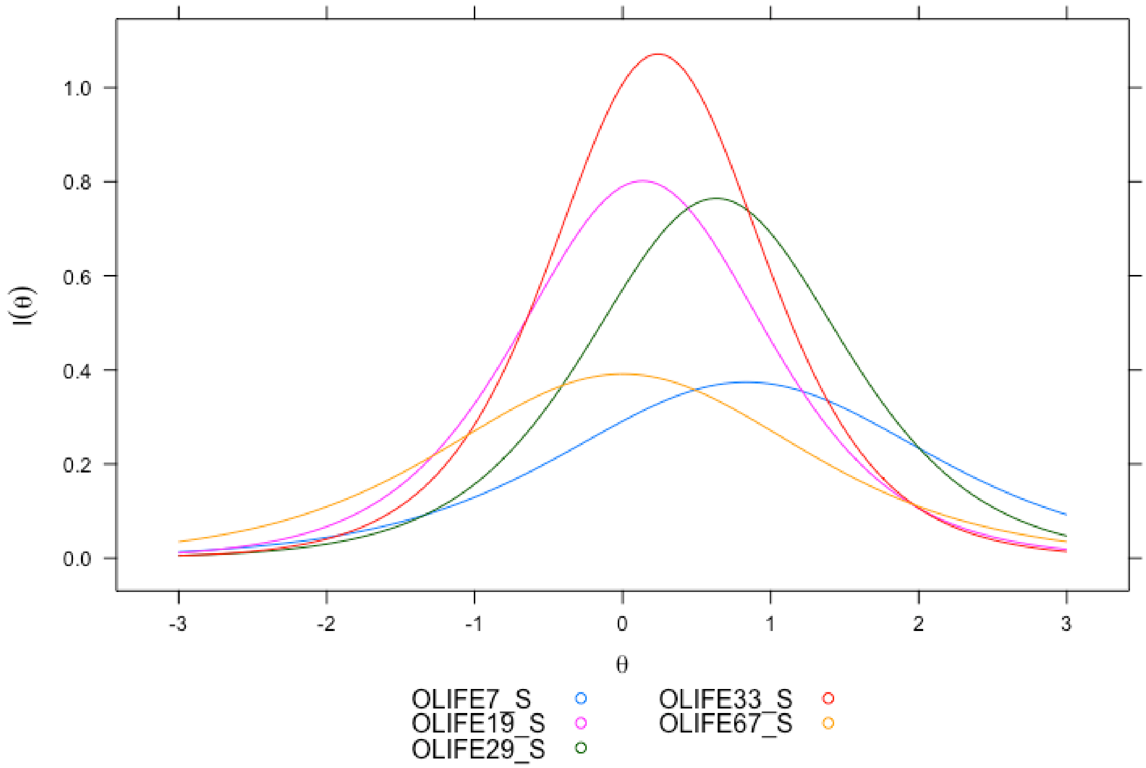


Figure C.10 Item information curves for final Introvertive Anhedonia items.

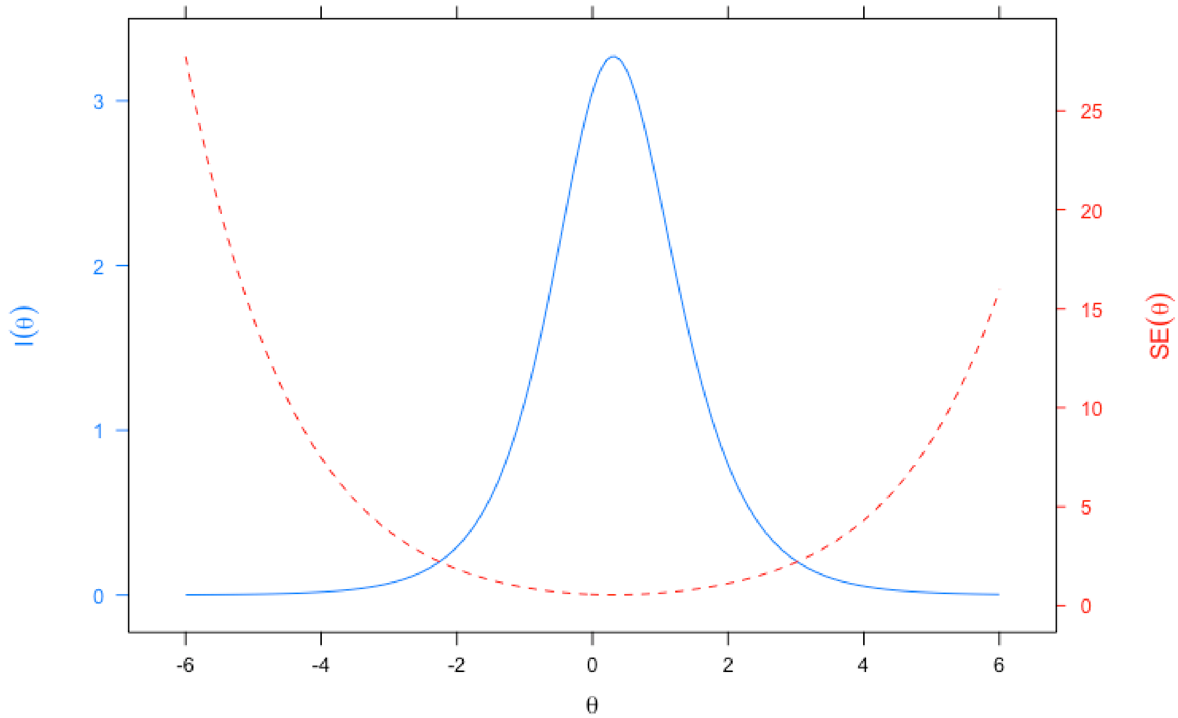


Figure C.11 Test information and standard error for final Introvertive Anhedonia subscale.

C.3.3 Cognitive Disorganisation

Table C.15 Proportion of responses at each level – Cognitive Disorganisation.

Item	**0**	**1**	**logit**
OLIFE21_C	0.4188	0.5812	0.3278
OLIFE28_C	0.4325	0.5675	0.2717
OLIFE32_C	0.4485	0.5515	0.2067
OLIFE40_C	0.6476	0.3524	-0.6085
OLIFE61_C	0.5812	0.4188	-0.3278
OLIFE76_C	0.3638	0.6362	0.5587
OLIFE81_C	0.4966	0.5034	0.0137
OLIFE94_C	0.3616	0.6384	0.5686
OLIFE103_C	0.5057	0.4943	-0.0229

Table C.16 Cognitive Disorganisation items total model fit – round 1 of 3.

	M2	**df**	**p**	**RMSEA**	**RMSEA_5**	**RMSEA_95**	**SRMSR**	**TLI**	**CFI**
stats	41.628	20	0.00309	0.049803	0.028057	0.071054	0.042724	0.97377	0.98123

Table C.17 Cognitive Disorganisation items model parameters – round 1 of 3.

	a	**b**
OLIFE21_C	1.493	-0.304
OLIFE28_C	**1.341**	**-0.267**
OLIFE40_C	0.883	0.804
OLIFE61_C	1.475	0.316
OLIFE76_C	1.982	-0.453
OLIFE81_C	2.019	-0.004
OLIFE94_C	2.374	-0.429
OLIFE103_C	**1.757**	**0.026**

Bolded items removed.

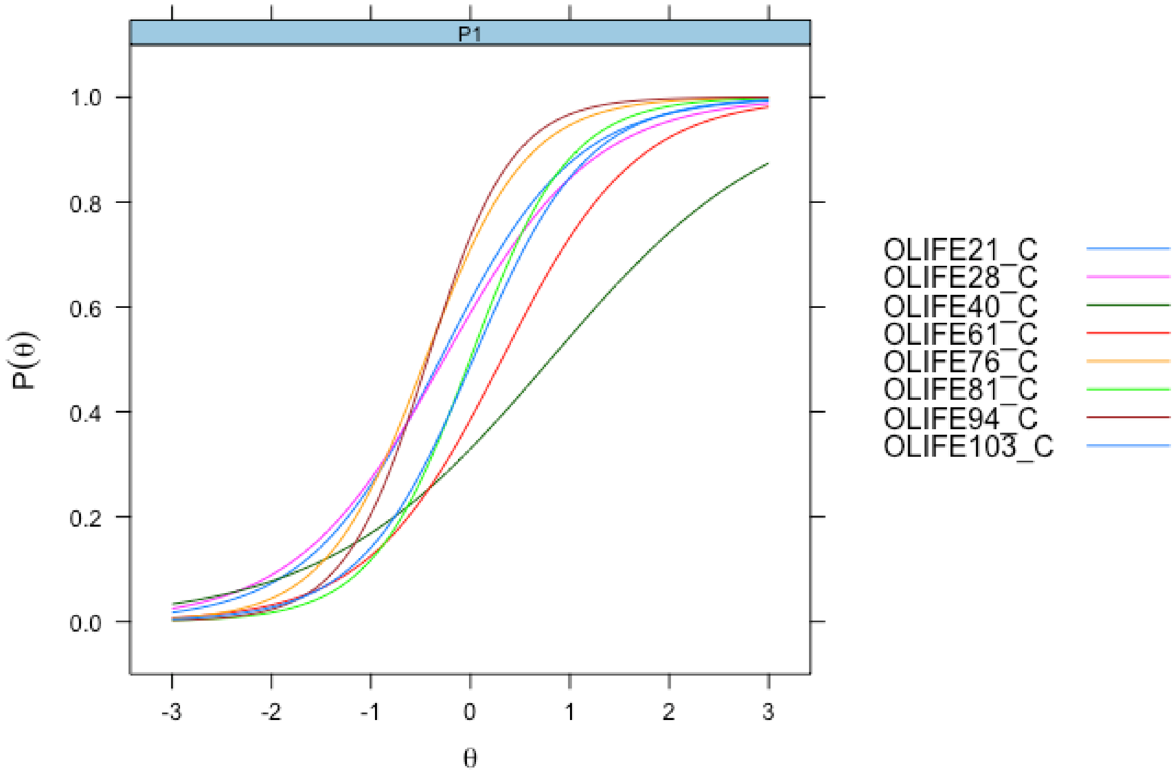


Figure C.12 Item probability functions for initial IRT analysis of Cognitive Disorganisation items.

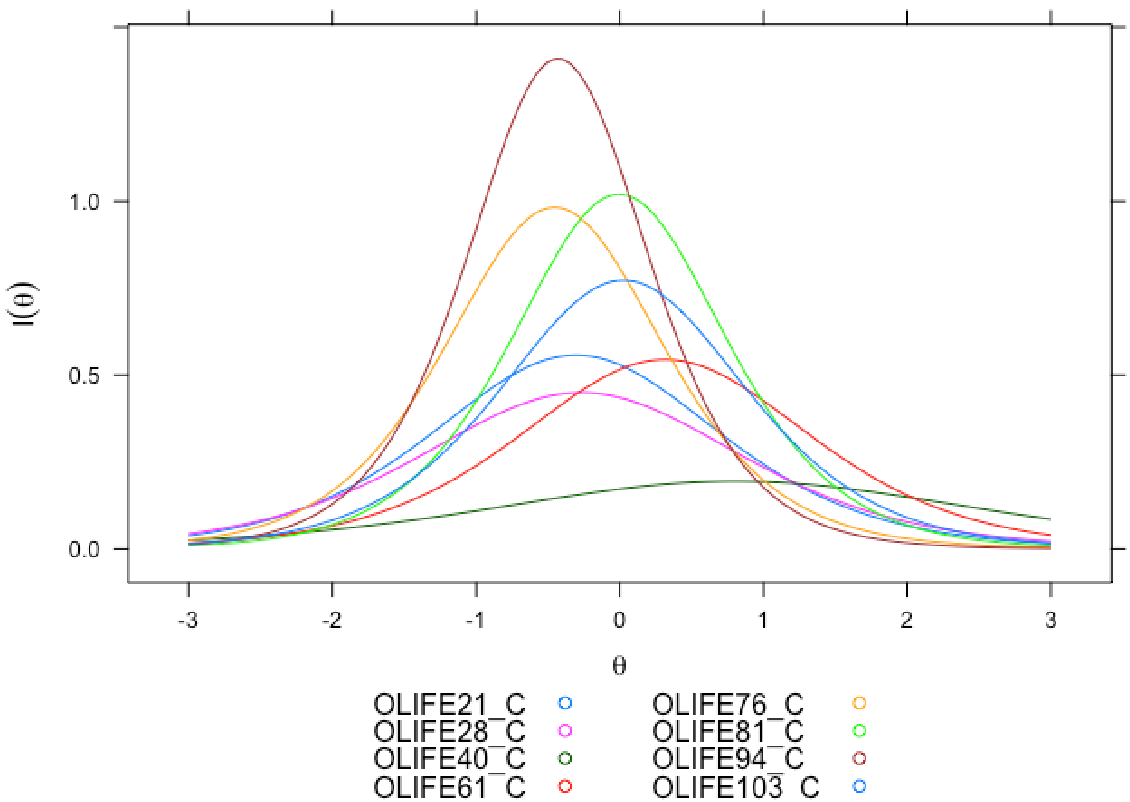


Figure C.13 Item information curves for initial Cognitive Disorganisation items.

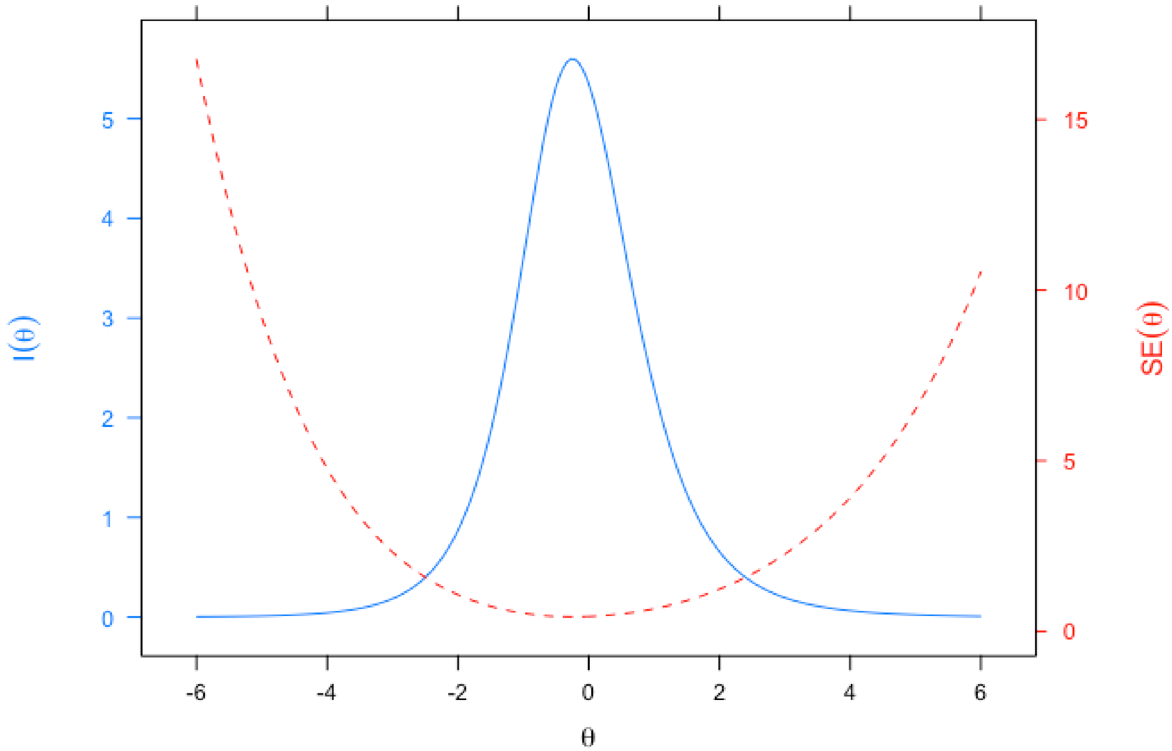


Figure C.14 Test information and standard error for initial Cognitive Disorganisation subscale.

Table C.18 Cognitive Disorganisation items total model fit – round 2 of 3.

	M2	df	p	RMSEA	RMSEA_5	RMSEA_95	SRMSR	TLI	CFI
stats	20.104	9	0.017282	0.053195	0.021241	0.084566	0.040933	0.97028	0.98217

Table C.19 Cognitive Disorganisation items model parameters – round 2 of 3.

	a	b
OLIFE21_C	**1.439**	**-0.314**
OLIFE40_C	0.879	0.806
OLIFE61_C	1.452	0.315
OLIFE76_C	1.712	-0.489
OLIFE81_C	2.238	-0.009
OLIFE94_C	2.610	-0.423

Bolded items removed.

Table C.20 Cognitive disorganisation items total model fit – final items (round 3 of 3).

	M2	df	p	RMSEA	RMSEA_5	RMSEA_95	SRMSR	TLI	CFI
stats	16.592	5	0.005342	0.0729217	0.03595814	0.1130557	0.04640205	0.945843	0.9729215

Table C.21 Cognitive Disorganisation items model parameters – final items (round 3 of 3).

	a	b	Item
OLIFE40_C	0.839	0.835	Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense?
OLIFE61_C	1.426	0.318	Are you sometimes so nervous that you are "blocked"?
OLIFE76_C	1.610	-0.504	Do you frequently have difficulty in starting to do things?
OLIFE81_C	2.391	-0.009	Do you often have difficulties in controlling your thoughts?
OLIFE94_C	2.769	-0.415	No matter how hard you try to concentrate do unrelated thoughts creep into your mind?

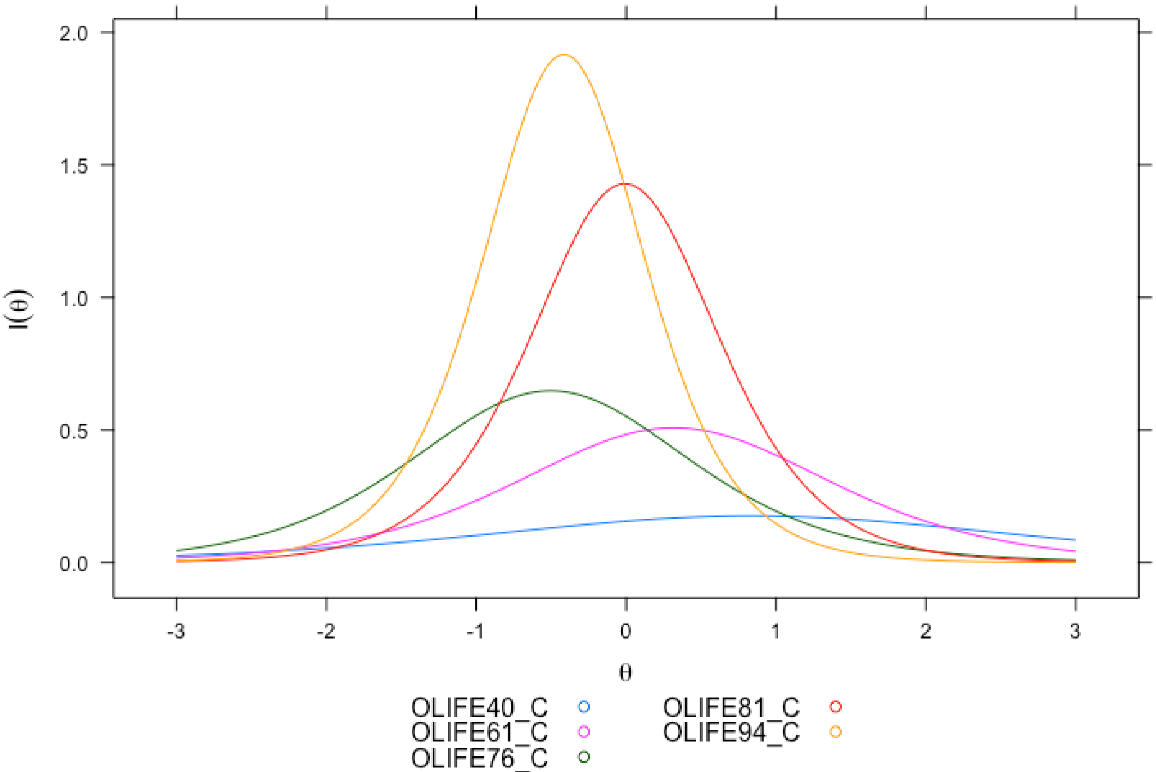


Figure C.15 Item information curves for final Cognitive Disorganisation items.

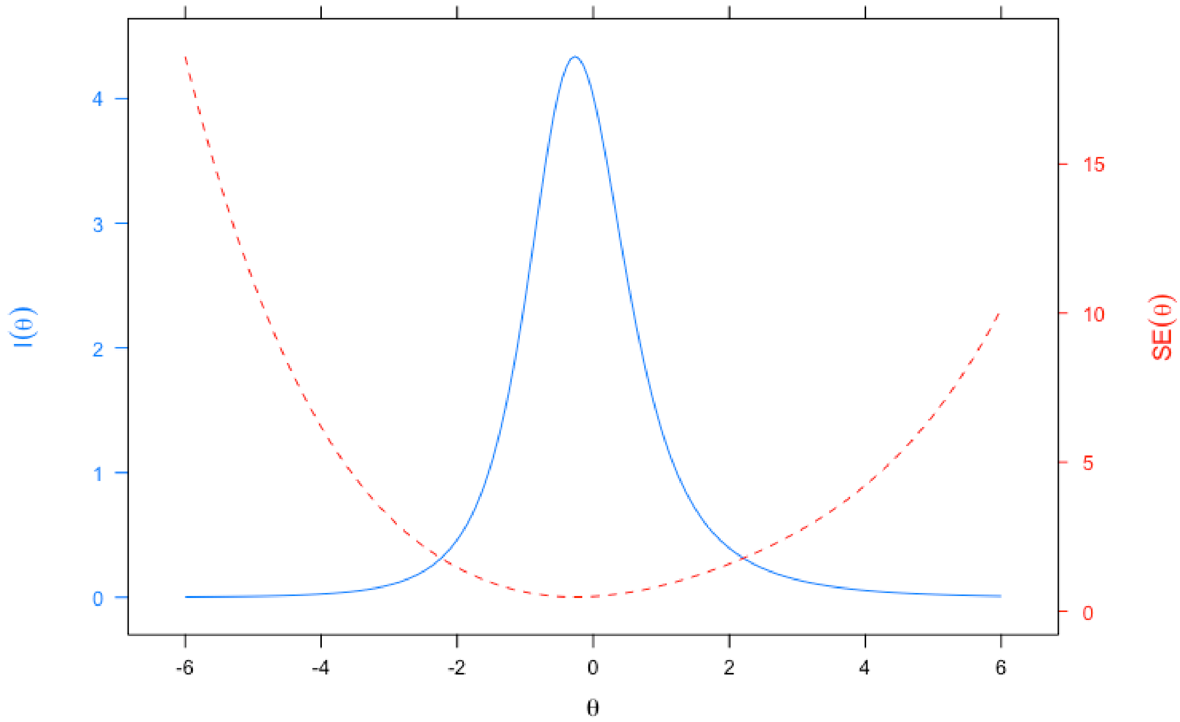


Figure C.16 Test information and standard error for final Cognitive Disorganisation subscale.

C.4 OLIFE16 Descriptives from Study 2 (Sample 1)

Descriptives

Table C.22 OLIFE16 total and subscale score descriptives from Sample 1 (n=437).

	UnuExp16	IntAnh16	CogDis16	OLIFE16_Total
Valid	437	437	437	437
Missing	0	0	0	0
Mean	2.142	2.021	2.549	7.824
Std. Deviation	1.697	1.612	1.620	4.552
IQR	2.000	2.000	3.000	7.000
Skewness	0.394	0.379	-0.115	0.287
Std. Error of Skewness	0.117	0.117	0.117	0.117
Kurtosis	-0.821	-1.060	-1.124	-0.655
Std. Error of Kurtosis	0.233	0.233	0.233	0.233
Minimum	0.000	0.000	0.000	0.000
Maximum	6.000	5.000	5.000	20.000
50th percentile	2.000	2.000	3.000	8.000
90th percentile	5.000	4.000	5.000	14.000

Table C.23 OLIFE16 total and subscale score descriptives from Sample 2 (n=437).

	UnuExp16	IntAnh16	CogDis16	OLIFE16_Total
Valid	548	548	548	548
Missing	0	0	0	0
Mean	2.104	2.015	2.670	6.788
Std. Deviation	1.615	1.569	1.583	3.508
IQR	2.000	2.000	3.000	5.000
Skewness	0.518	0.347	-0.181	0.253
Std. Error of Skewness	0.104	0.104	0.104	0.104
Kurtosis	-0.483	-0.970	-1.069	-0.544
Std. Error of Kurtosis	0.208	0.208	0.208	0.208
Minimum	0.000	0.000	0.000	0.000
Maximum	6.000	5.000	5.000	16.000
50th percentile	2.000	2.000	3.000	6.500
90th percentile	4.000	4.000	5.000	12.000

Distributions

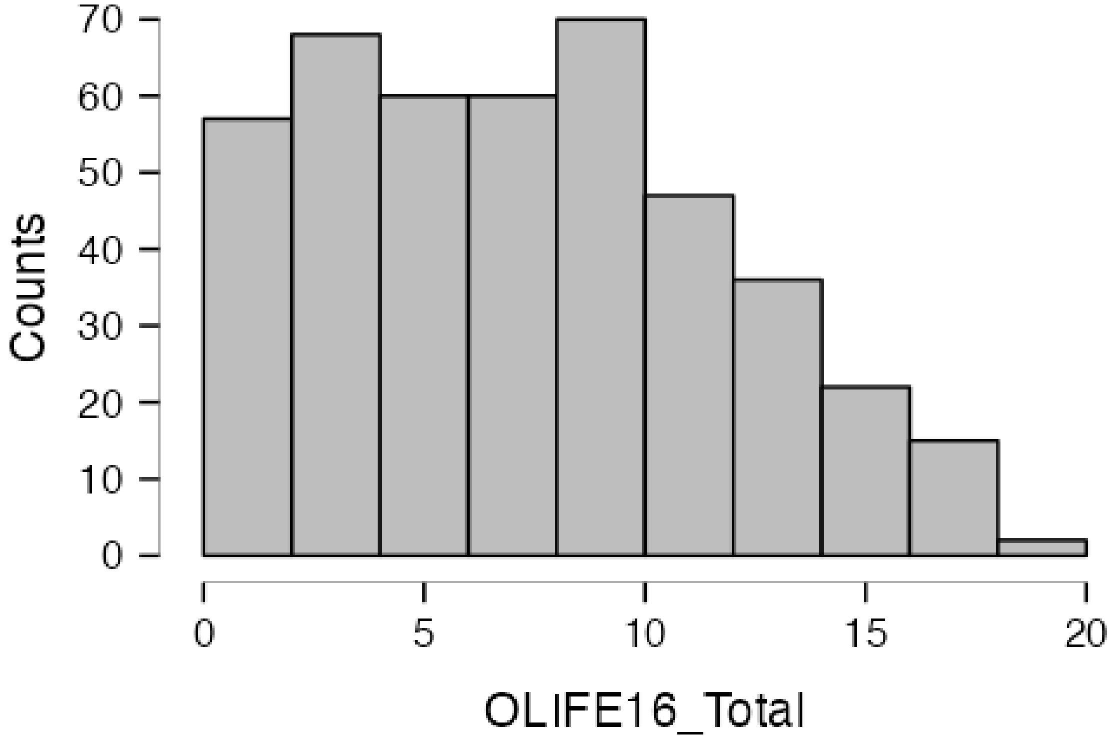


Figure C.17 OLIFE16 Total score distribution

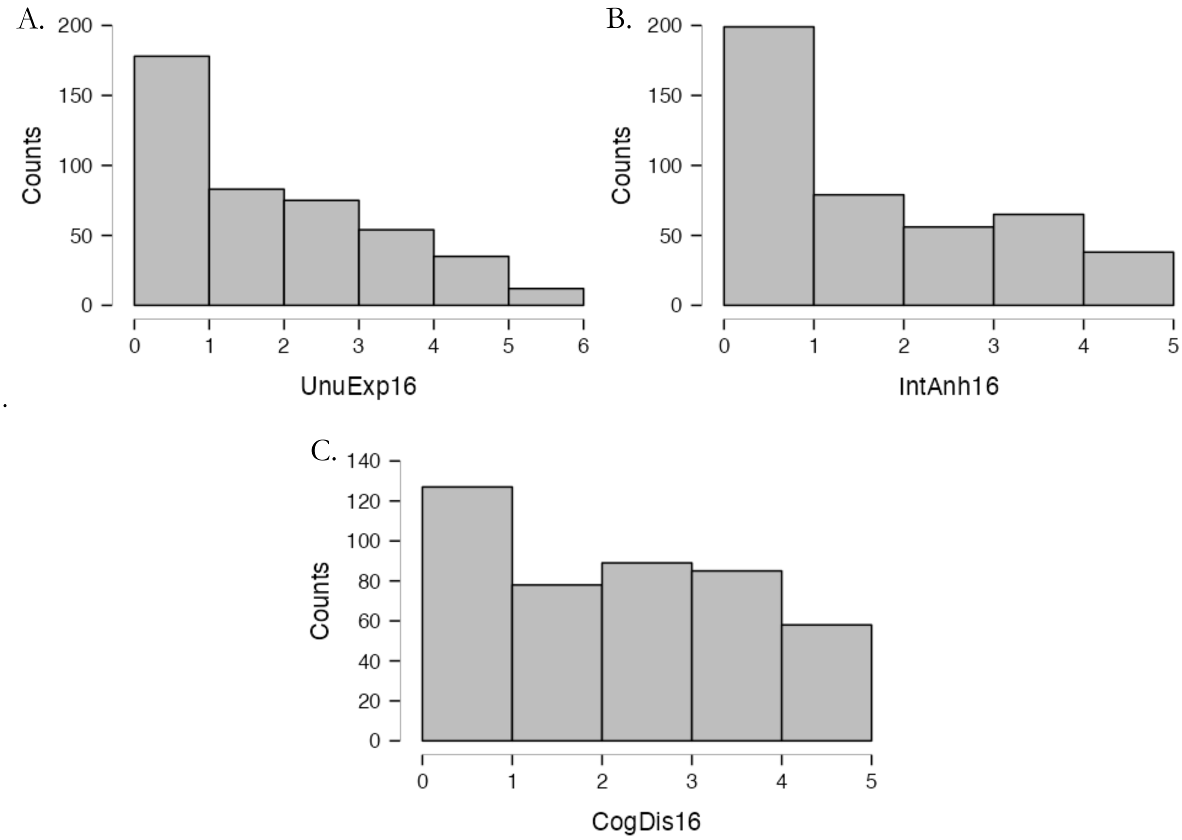


Figure C.18 OLIFE16 subscale distributions. A. Unusual Experiences, B. Introvertive Anhedonia, C. Cognitive Disorganisation.

C.5 OLIFE16 Questionnaire

OLIFE16 Questionnaire

Elizabeth Shen, Dr. Conal Monaghan, and Professor Bruce K. Christensen

Please read each question and select the response that best reflects you. There are no right or wrong answers. Please answer every question.

			UE	IA	CD
Are people usually better off if they stay aloof from emotional involvements with people?	True	False			
Are you much too independent to really get involved with other people?	True	False			
Do you ever feel sure that something is about to happen, even though there does not seem to be any reason for you thinking that?	True	False			
Do people who try to get to know you better usually give up after a while?	True	False			
Do you feel that making new friends isn't worth the energy it takes?	True	False			
Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense?	True	False			
Do your thoughts sometimes seem as real as actual events in your life?	True	False			
Are you sometimes so nervous that you are 'blocked?'	True	False			
On occasions, have you seen a person's face in front of you when no one was in fact there?	True	False			
Do you prefer watching television to going out with other people?	True	False			
Do you frequently have difficulty in starting to do things?	True	False			
Do you often have difficulties in controlling your thoughts?	True	False			
Have you occasionally felt as though your body did not exist?	True	False			
Do the people in your daydreams seem so true to life that you sometimes think they are real?	True	False			
No matter how hard you try to concentrate do unrelated thoughts creep into your mind?	True	False			
Does a passing thought ever seem so real it frightens you?	True	False			
		Totals			