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Contributing factors to cognitive deficits in psychometric schizotypy: implications for schizophrenia

PhD Thesis

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Contents

Dedication

Writing this PhD thesis was the hardest, but most rewarding process I have ever undertook. Like many others when I started my PhD, I felt that three letters after my name would make me feel secure. However, this did nothing to change how I felt. Instead, it was the friends, colleagues, and a love for research that I never knew I was missing that made me feel fulfilled. For people that may find this on a dusty bookshelf in many years' time or a dystopian Metaverse library, if I have learnt anything, it is to follow the ideas that you love and stick to the people who help you chase them.

I wish to dedicate this thesis to those who made it possible. To my mother, whose unconditional love, pride, and acceptance made me feel I could do anything, but even if I couldn't, I was not a failure (even if she still does not know what I do). To all my university friends who have supported me across the years: Sue-Lynn Mah, Ben Purvis, Jennifer Salvage, Chloe Wider and Liam Cahill. To the LGBT network who I have made lifelong friends with. To Paula Moran, who gave me the freedom to lead my own research and has been not only been an amazing supervisor but also a supportive friend. To Mark Haselgrove, whose supervision of my undergraduate project gave me the confidence to pursue research. To Claudia Danielmeier who has helped me to bring this work together. To the Economic and Social Science Research Council (ESRC) for funding this research and for their continued support. Finally, thank you to all the participants, administrative support staff, and to caffeine.

Preface

The overarching theme that connects this research is the cognitive function in schizotypy: be it neurocognition, social cognition, or metacognition. More specifically, how faulty cognition may explain the poor functional outcomes seen in psychosis patients. This research was proposed to make psychological research "more realistic", by understanding the influences that may mean cognitive tasks may measure more than just cognition, and how this may limit the predictivity of cognitive tasks of real-world ability. This was approached in two ways. Firstly, by exploring how potential confounding behavioural variables seen in people with schizophrenia, such as increased negative affect, poorer motivation and confounds of clinical status may affect cognition. Secondly, by introducing metacognition as an explanatory factor for why cognitive ability may not translate to real-world functioning. The underlying aim of this thesis is to understand how these factors affect cognitive test performance, actual cognitive ability, and subsequent associations to functioning. Ultimately, it is hoped these insights may be useful to improve daily functioning in psychosis.

Thank you for taking the time to read this body of work.

Chapter summaries

Chapter 1

Literature Review

Schizophrenia is a mental health disorder in which symptoms include hallucinations and delusions, flattened affect and reduced emotional expressiveness, and disorganised thoughts and speech. Patients commonly present reduced functional ability such as poorer quality of life, social integration, and vocational success. Much of the research into explanations for poorer functioning has pointed to cognition, specifically neurocognition (non-social cognition) and social cognition. However, together these processes explain only around 10% of the variance in functioning, with interventions based on these approaches having relatively moderate success. Recently, it has been proposed other factors may both affect cognitive task performance and the translation of ability to functioning. This Chapter reviews the current understanding of some of these factors, the influence of metacognition ('thinking about thinking'), negative affect (emotions), amotivation and confounds of clinical status. Currently, our understanding of these factors relates to schizophrenia, especially metacognition, is incomplete.

Chapter 2

The influence of metacognition, negative affect, and motivation of neurocognitive task performance in non-clinical psychometric schizotypy.

The first experimental Chapter of this thesis aimed to assess the influence of metacognition, negative affect, amotivation and confounds of clinical status on neurocognition (non-social cognition). To do so, two neurocognitive tasks were chosen: Probabilistic Reversal Learning (PRL) and attentional set-shifting. While PRL is a broad measure of neurocognition that includes visual learning, working memory and attention, set-shifting is a specific assessment of effectively shifting attention from one visual dimension (e.g., colour) to another (e.g., shape). The influence of metacognition, negative affect, and amotivation were investigated by assessing these traits psychometrically and controlling for their influence when predicting performance. People varying in psychometric schizotypy ('psychosis-proneness') were assessed to mitigate the effects of clinical confounds. The results found that positive schizotypy ('hallucinations and delusions') was associated with poorer performance on the PRL task, specifically to punishing stimuli. Similarly, set-shifting performance was impaired. In contrast, disorganised schizotypy predicted improved performance on both tasks, with the reasons behind this being unclear. None of the potential mediators explained this relationship, but several methodological limitations of both the tasks and psychometric

assessments meant this finding was not conclusive. Consequently, three additional experiments were designed to investigate this further in subsequent chapters. The first follow-up experiment adapted both of these neurocognitive tasks to also assess behavioural measures of metacognition. The second experiment extended this adaptation to social cognition to understand whether deficits were domain-specific or generalisable. Finally, the third experiment also adapted the PRL task into an immersive Virtual Reality task; assessing the influence of motivation.

Chapter 3

Metacognitive adaptations of neurocognitive tasks in psychometric schizotypy

The experimental design of the tasks in chapter 2 meant that it was unclear whether poorer performance was caused by cognition or metacognition. Moreover, self-reported metacognition scores were suggested to be too subjective, and so behavioural measures were needed. This chapter adapted these tasks by expanding the response dimensions to include accuracy judgements (cognition) and both confidence and acting on knowledge (both behavioural metacognition). For the metacognitive PRL task (M-PRL), positive schizotypy was again associated with poorer learning of punishing stimuli, supporting deficits in learning. Additionally, these participants also acted on this faulty knowledge and were overconfident when they believed they were incorrect, also supporting deficits in metacognition. However, there were no performance differences in the metacognitive set-shifting task and neither negative affect nor amotivation explained performance differences. These highlighted that while self-reported metacognition may not influence cognitive performance, behavioural assessments may be more powerful tools to assess it.

Chapter 4

High Schizotypy Predicts Emotion Recognition Deficits, but perhaps not poor real-world functioning.

This chapter expanded the current research of chapter 3 into the social cognitive domain of emotion recognition. Participants were presented with video clips of actors representing one of 14 different emotions through facial expression, body language, and nonsense syllables that participants were tasked to recognise. Negative schizotypy predicted poorer emotion recognition of negative emotions which was mediated by poorer metacognitive processes ('*I must control my thoughts at all times*'). As these effects are specific to negative emotions, this may be explained by these faces producing negative internal states that participants feel they must control – leading to greater distractibility and avoidance. However, the behavioural metacognitive assessments found that those high in negative schizotypy did not act on this faulty knowledge; illustrating impaired social cognition that is not transferred to poor performance through intact metacognition. Consistent with chapter 2,

disorganised schizotypy traits predicted improved performance to negative emotions, although the reasons for this are also unclear. There was also a trend-level association between positive schizotypy and negative emotion recognition, which is potentially consistent with poorer performance to punishing neurocognitive stimuli presented in chapter 2 and chapter 3. No measure predicted recognition of positive emotions and neither negative affect nor motivation mediated performance differences. While the effects of positive and disorganised schizotypy appear to span neurocognition and social cognition, negative schizotypy appears specific to social cognition.

Chapter 5

Can immersive Virtual Reality adaptations of cognitive tasks mitigate deficits in psychometric schizotypy? A 2D vs. Virtual Reality comparison

Chapter 2 found that motivation did not mediate performance deficits in schizotypy. However, the psychometric scale used was unvalidated, task-specific, and returned low internal consistency scores; meaning the influence of motivation was unclear. The current chapter instead manipulated motivation by adapting the PRL task of Chapter 2 into an immersive Virtual Reality assessment (VR-PRL). This adaptation was designed following open-ended participant feedback from Chapter 2, including greater task instruction clarity and contextualisation of task aims. This new sample did not differ in levels of schizotypy, negative affect, or overall task performance, but both pre-task and post-task motivation were much higher in the current study. All associations between schizotypy and performance, including the association between positive schizotypy and poorer learning of punishing cues, were mitigated. It was unclear if motivation was the underlying cause of change as other factors, such as increased comprehension and realism, may have impacted this relationship. Ultimately, it was suggested the VR adaptation may have reduced task-related anxiety, which subsequently meant punishing stimuli were no longer considered threatening.

Chapter 6

General discussion

The final Chapter discussed trends throughout these studies and implications for future research. Firstly, as both neurocognitive and social cognitive deficits were replicated in sub-clinical schizotypy, this provided indirect evidence that deficits in patients are not solely due to confounds of patient status (e.g., medication side effects). Across all tasks, these associations were specifically due to poorer performance towards negative stimuli only, which was suggested to be due to these stimuli evoking negative internal states, thereafter causing distraction or inattention. In contrast, disorganised schizotypy consistently predicted improved performance to negative stimuli, and while the reasons for this association could not be determined, it highlights that schizotypy should not be

considered pathological. A potential explanation for these findings came from the lack of association to performance in the VR-task, which may have reduced task-related anxiety. Specifically, participants may not have perceived the stimuli as negative to the same extent, as the perceived 'cost' of incorrect performance was reduced by greater enjoyment. This may also be consistent with the findings of the M-PRL, which specifically pointed to deficits in *perceived* negative stimuli. Moreover, the gamification of cognitive tasks may have benefits beyond enjoyment and willingness to return, including more accurate assessments of applied cognitive ability. Although, more controlled manipulations of the VR-PRL are needed before these claims can be supported (e.g., manipulate *only* motivation). This contrasts with trait negative affect, which did not influence the association between schizotypy and performance in any analysis; suggesting schizotypy and negative affect may influence cognition through separate causal pathways. The results on metacognition presented interesting findings. Across all Chapters, schizotypy was associated with increased self-reflection, thought monitoring, and thought control thinking styles. However, the surveys are designed for clinical samples and define greater scrutiny of thoughts as maladaptive (e.g., rumination). Critically, without cognitive ability assessments, self-report ratings may be misleading, perhaps contributing to current literature inconsistencies. Comparing subjective and behavioural metacognition led to greater insights in the current thesis. Specifically, excessive Need to Control Thoughts (metacognition) partially explained why negative schizotypy was related to poorer social cognition, which is consistent with the above suggestions of avoiding negative internal states. This maladaptive metacognitive belief contrasted with the adaptive metacognitive control technique in the same individuals, wherein those high in negative schizotypy did not act on poorer cognition. Metacognition in the psychosis-spectrum is currently not well understood, but combining psychometric and behavioural measures may help disentangle contrasting findings.

Chapter 1

Literature Review

Schizophrenia

What is schizophrenia?

Schizophrenia is a chronic and potentially debilitating mental health disorder that will affect 0.5% of the general population at some point in their lifetime[1]. The positive symptoms of schizophrenia are behaviours that are not commonly present in those without schizophrenia, while negative symptoms involve behaviours diminished in patients[2]. More recent conceptualisations also distinguish positive symptoms further into "reality distortion" and disorganisation. Reality distortion symptoms are exemplified through hallucinations and delusions and are considered the hallmark of psychosis. Hallucinations are false sensory experiences, with auditory hallucinations (i.e., hearing voices) and visual hallucinations (seeing people, spirits, or fictitious events) being the most common[3]. However, hallucinations can stem from any of the senses such, as tactile hallucinations (e.g., feeling insects crawling over one's skin), olfactory hallucinations (pungent or repulsive smells), and gustatory hallucinations (foul tastes). Commonly, these experiences cause significant distress. This distress is also seen in delusions: erroneous beliefs that are held with a high degree of certainty and are incredibly resistant to change. People may feel they are being controlled by outside forces (delusions of control), that others mean them harm (persecutory delusions), or that they have god-like powers (delusions of grandeur). The other component of positive symptoms, disorganised symptoms, refers to thoughts and speech that are logically divergent or bizarre. Patients can be quick to shift topics, continually repeat old ideas, and speak constantly and rapidly without purpose. Social behaviour can also be inappropriate, such as displaying an inappropriate facial emotion for the current social context (i.e., 'grimacing'). Movement is also sometimes bizarre, from rapid and unusual movements to not moving at all for prolonged periods (catatonia). Disorganised symptoms are sometimes further divided into disorganisation (bizarre behaviour, mannerisms, posture, conceptual disorganisation) and cognitive disorganisation (difficulties in abstract thinking, poor attention, and inappropriate affect). Finally, negative symptoms are suggested to fall into five categories[4,5]: *Avolition*, a diminished engagement in social, occupational, and intellectual interests; *Anhedonia*, a reduction in the intensity or range of positive emotions felt; *Asociality*, withdrawal or avoidance of social situations perhaps resulting in a lack of meaningful social connections; *Restricted/Blunted Affect*, reduced emotional expressiveness such as body language gestures, voice intonation, and facial expressions; and finally, *Alogia*, reduced spontaneous speech and verbalisations. These categories can also be grouped into expressive (restricted affect and alogia) and experiential symptoms (avolition and apathy, and asociality)[6]. The separability of these symptoms has been evidenced through different underlying neurological structures. Specifically, negative symptoms predict activity in the ventrolateral Prefrontal Cortex and ventral striatum, reality distortion is

associated with medial prefrontal cortex, amygdala, and hippocampus/parahippocampal activity, and disorganised symptoms predict the activity of the dorsolateral prefrontal cortex[7]. Examples of divergent behaviour in schizophrenia are often extreme to illustrate their content. However, every person with schizophrenia is different concerning the frequency and intensity of these symptoms and others. Indeed, not all symptoms are necessary for diagnosis according to both the DSM-V[2] and ICD-10[8].

Living with schizophrenia

The typical trajectory of schizophrenia commonly begins with the First Episode of Psychosis (FEP) materialising in early adulthood. Even in these prodromal stages, schizophrenia can be privately, socially, and occupationally crippling[9]. Patients not only have to contend with the symptoms of the disorder, but also the stigma associated with it[10]. After initial diagnosis, approximately 23% - 29% of FEP patients go on to develop co-morbid mood disorders[11] and 26.8% will attempt suicide[12], which is further exacerbated by this co-morbidity[13]. As a whole, schizophrenia patients show severe impairments in daily functioning[14,15] including lower rates of employment[16], smaller social networks[17], and higher levels of loneliness[18]. These impairments can be seen across illness chronicity with 50% of those with an At-Risk Mental State (ARMS) for psychosis and 67% of FEP patients being considered "socially disabled"[19]. Patients also present poorer physical health including an elevated risk of diabetes and cardiovascular disorders, which contribute to a reduced life expectancy of approximately 20 years[20]. These physiological and psychological factors also contribute to the reduced Quality of Life in patients[21–23]. Poorer functioning in daily life is associated with higher levels of negative[24,25] and disorganised symptoms[26], which may create further deterioration[27]. Even with interventions, approximately 82% of schizophrenia patients will relapse after 5 years. Although, recovery at a 2-year follow-up predicts staying in remission for up to 15 years and 40% of people do achieve long-term recovery[28].

Aside from the personal costs, in England, the total societal cost in terms of healthcare and lost productivity of schizophrenia was estimated to be £6.7 billion in 2004 and £11.8 billion in 2012[29,30], which is economically similar[i] to cancer services (£6.7 billion) and the effects of tobacco (£13.8 billion)[31]. In the UK specifically, supporting a single patient with schizophrenia costs £46,880 a year[32]. Clearly, schizophrenia is an extremely important disorder to understand and support for both societal and economic reasons.

[i] This is not meant to compare the pain or immeasurable distress of different health issues.

Interventions for schizophrenia

The 1950s saw the introduction of first-generation (or 'typical') antipsychotic medication that targeted positive symptoms. In the 1990s, these were succeeded by second-generation antipsychotics ('atypical') which additionally targeted negative symptoms and unwanted side effects. Current estimates suggest antipsychotic medication reduces relapse rates from 26% - 78% to around 0% - 12%[33,34]. However, there is less convincing evidence they reduce negative symptoms to a clinically meaningful extent. Specifically, although both second-generation anti-psychotics (Cohen's *d* = -.576) and anti-depressant medication (*d* = -.349) reduce negative symptoms, it is estimated that a group difference of *d* = -0.97 is necessary to be clinically relevant[35]. In real terms, this equates to around a 10.1% improvement in symptoms over control groups[35]. Moreover, one-third of patients show no significant response to medication[36,37], its effectiveness decreases with chronicity[38], and antipsychotics have little impact on functional outcomes such as Quality of Life and employment[33].

The side effects of (primarily first-generation[39]) antipsychotic medication also highlight the need for novel interventions, being associated with tardive dyskinesia (involuntary and repetitive movements), difficulty concentrating, parkinsonian symptoms (slowness of movement), restlessness, and weight gain. These factors are particularly important to consider because the medication is a potentially lifelong commitment[33], as side effects predict both non-adherence and relapse[40], and difficulty concentrating may exacerbate disorganised symptoms. That being said, drop-out rates are found to be higher for placebo groups (54%) relative to medication (30%)[33], suggesting patients feel the gains of treatment outweigh the perceived costs. Indeed, these advancements in tackling positive symptoms began to shift the clinical conversation from treatment to clinical recovery. However, as it is primarily the untreated negative symptoms that predict daily functioning in patients - attention began to shift to alternative interventions.

One such intervention is Cognitive Behavioural Therapy (CBT) which has been widely applied to psychosis. A recent review[41] of 20 meta-analyses comparing CBT against different comparator groups found that CBT was effective at reducing positive and negative symptoms at small to medium effect sizes (e.g., *d* = 0.40). However, CBT did not appear to be more effective than other psychotherapies in terms of symptom reduction, relapses, social functioning, or improvements in Quality of Life. Moreover, these effects are not appreciably greater than those of antipsychotics[42] and combining CBT with pharmacological interventions does not result in additive benefits[35]. Moreover, interventions such as Art Therapy[43] and aerobic exercise[44] have also been found to reduce symptoms to a similar extent.

The relatively limited effectiveness of pharmacological and psychotherapy interventions was a major catalyst for renewed effort into cognitive research in schizophrenia. The remainder of this Chapter will explain neurocognitive and social cognitive abilities in schizophrenia, outline the theoretical associations to symptoms and functioning and, critically, the limits of their explanatory power will be defined. At the end of this Chapter, it will be explained how this thesis aims to address some of the current limitations of cognitive research in psychosis.

Can neurocognition explain and improve functioning in schizophrenia?

Neurocognition

In schizophrenia, cognition broadly refers to the acquisition, storage, manipulation, and implementation of information[45] and can be broken down into neurocognition (non-social cognition) and social cognition. Early cognitive research in schizophrenia focused on neurocognition[14]: the cognitive processes attributed to specific brain regions. The specific neurocognitive domains that show relatively consistent impairment in schizophrenia were outlined in the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS)™ Cognitive Battery (MCCB), which aimed to standardize cognitive testing in schizophrenia[46]. The MATRICS domains include processing speed, attention/vigilance, Working Memory, Verbal Learning, Visual Learning, and Reasoning/Problem-solving (see **Figure 1**). The MATRICS also includes emotion processing as a measure of social cognition, which will be discussed later.

Neurocognitive deficits in schizophrenia

Neurocognitive deficits are seen by some as a core feature of schizophrenia[47] and a putative endophenotype[48]. They are, however, considered separable from the symptoms of schizophrenia and are not necessary for a diagnosis. In relation to neurotypical and psychiatric controls, a recent meta-review has summarised that although deficits in episodic memory and executive function appear across most psychiatric disorders, schizophrenia presents greater impairments relative to both neurotypical controls and psychiatric controls in all MATRICS domains[49]. These neurocognitive deficits have been found to pre-date the symptoms of schizophrenia[50–52], as well as being reported in Clinical High-Risk children and adolescents[52], with early-onset psychosis presenting the most severe deficits[53]. One systematic review[54] also outlined that deficits specifically in both Working Memory (Hedge's g = -0.29) and visual learning (g = -0.40) were predictive of transition to psychosis.

There is currently inconclusive evidence as to whether neurocognitive deficits in schizophrenia increase over time. Earlier research suggested that the Duration of Untreated Psychosis (DUP) was 'neurotoxic' for the brain, considering schizophrenia a neurodegenerative disorder. This perspective dates back to the original conceptualisation of schizophrenia as dementia praecox ("early dementia") and is supported by reviews reporting neurological changes (e.g., increases in ventricular volume)[55] and losses in grey matter over time[56]. However, two recent meta-analyses (k = 27 – 43) both concluded that DUP was largely unrelated to neurocognitive deficits[57,58], although these studies

were considered low quality[58]. In fact, DUP was associated with improved cognitive flexibility (*r* = 0.10 to 0.31) and schizophrenia patients presented similar age-related improvements in cognition to the general population (k = 25)[50]. Moreover, the cycles of relapse and remission in patients are not consistent with a progressive disorder. It has been suggested that a clinical selection bias of those with severe schizophrenia (who are also most likely to also have co-morbid disorders) may skew the clinical representation of schizophrenia as a severe, degenerative, and treatment-resistant disorder (the "clinical illusion")[59].

However, there is strong and consistent evidence of a neurocognitive decline specifically between the ages of 12 and 18 that thereafter predicts transition to psychosis[60]. Thus, neurological deterioration appears to be significant at these initial stages of schizophrenia where the deterioration peaks[61]. There is also evidence that perhaps only a subset of patients go on to further deteriorate, but due to secondary factors such as substance abuse, social and financial impoverishment and medication side effects, rather than schizophrenia itself[34].

To what extent do neurocognitive factors explain symptoms?

Neurocognition is widely considered to be associated with negative symptoms both cross-sectionally and longitudinally in schizophrenia[50], however, there is limited recent systematic evidence available. One review reported that poorer neurocognitive performance on each MATRICS domain was associated with increased negative symptoms (e.g., speed of processing, *r* = .26; working memory, *r* = -.21; verbal learning and memory, *r* = -.21), but not combined positive symptoms (*r* = .00). A second review split positive symptoms into reality distortion and disorganisation; revealing that disorganised symptoms (*r* = -.23) but not reality distortion symptoms (*r* = -.04) were significantly associated with total neurocognitive ability. In terms of MATRICS domains, reality distortion was associated most strongly with attention/vigilance (*r* = -.12), whereas disorganisation was associated equally across all domains (*r* = -.20 to -.26)[26]. Finally, a third review reported that patients with chronic negative symptoms ('deficit schizophrenia') present more severe neurocognitive deficits than those without chronic negative symptoms (*d* = 0.24 - 0.60, k = 29)[62]. Overall, negative and disorganised symptoms are the primary correlates of neurocognition with negligible impact from reality disotortion[26,63].

These findings conflict with early associative leaning literature, which suggested patients in the acute stages of illness (i.e., when positive symptoms are highest) are less able to filter and ignore irrelevant stimuli (e.g., attention/vigilance)[64–67]. This is also consistent with the aberrant salience hypothesis of schizophrenia: that patients attribute unwarranted importance and pay inappropriate

attention to irrelevant stimuli[68,69]. Consequently, aberrant salience of the environment may cause a 'sensory overload' that is exacerbated by ineffective neurocognitive filtering, increased integration of irrelevant information and an overemphasis on details[70]. This has been suggested to produce spurious associations between unrelated events[69] establishing delusional thoughts, hallucinations, and a general disconnection from reality. More recently, it has also been suggested that positive symptoms may be partially due to intrusive thoughts caused by Working Memory deficits (e.g., intrusive verbal representations into Working Memory may produce auditory hallucinations), supported through atypical functioning of language processing brain structures[71] and poorer inhibitory control relative to both healthy controls and psychiatric controls[49]. Disorganised traits have received much less attention, partially due to their pairing with reality distortion. Some potential reasons for associations may include deficits in inhibiting bizarre behaviour, slow processing speed, and reduced executive functions reducing the sharpness of cognition. However, there is a conceptual overlap between disorganised symptoms and cognition which makes understanding these potential relationships difficult. For example, current symptom assessment tools include poor attention and abstract thinking (e.g., the PANSS) and there is an overlap between poorer verbal fluency (neurocognition) and disorganised speech (disorganisation).

Surprisingly, while neurocognitive deficits are consistently associated with negative symptoms there is much less theoretical explanation for this association. The most prevalent suggestion is that it is specifically amotivation within negative symptoms that produce poor performance, meaning symptoms explain cognition, rather than cognition explaining symptoms. It has also been suggested this relationship may be reciprocal, through poor Working Memory restricting access to the meanings and value of previous and anticipated events[72]. Specifically, patients are less able to represent expected rewards (i.e., health benefits of exercise) or punishments (negative consequences of inappropriate social behaviour), which influence goal-oriented behaviour. These suggestions are also consistent with findings that patients may undervalue reward, not modify their behaviour with perceived value[73], and that neurocognitive task practice may improve symptoms through increased reward sensitivity[74].

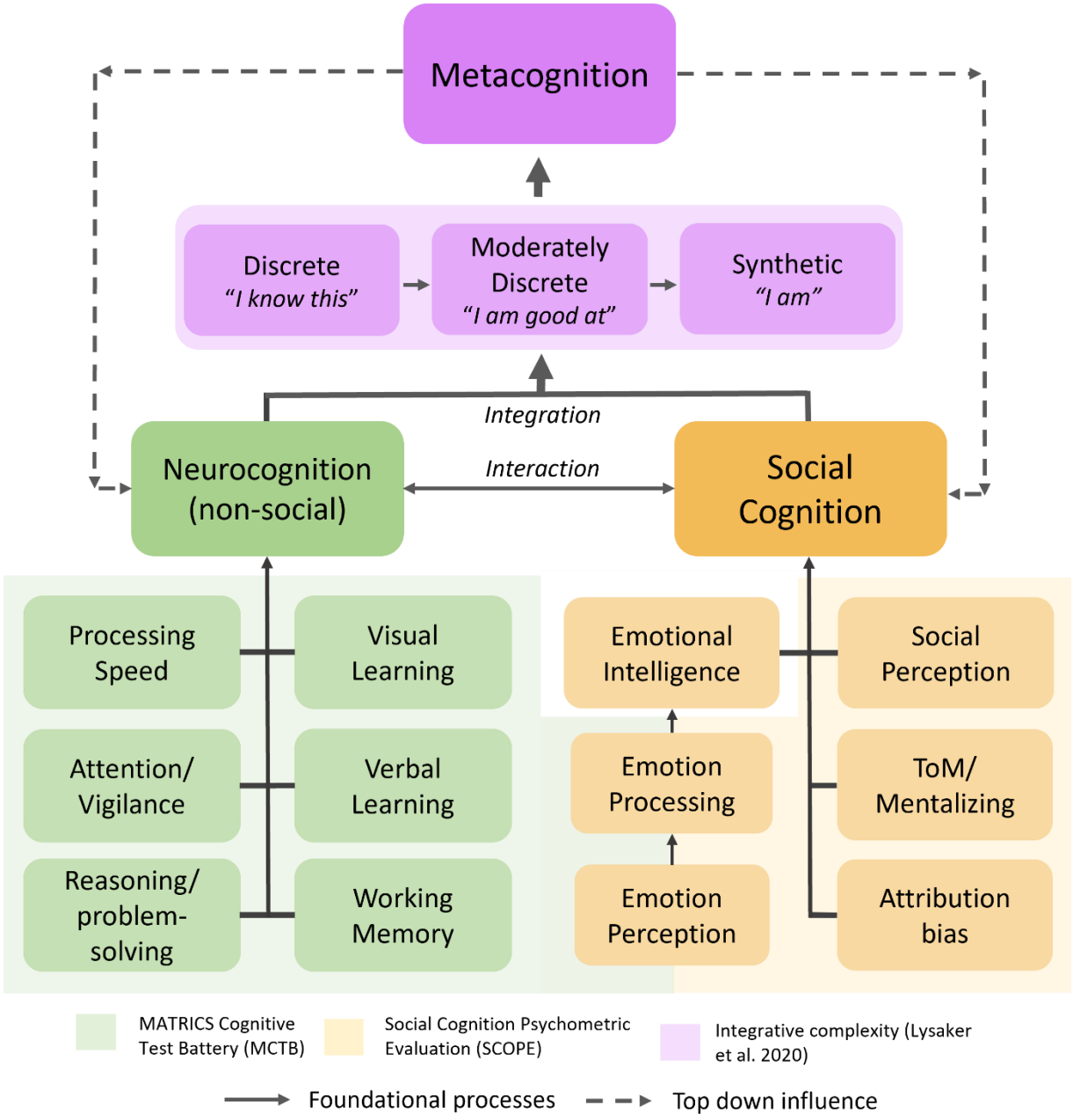

Figure 1. Hierarchical representation of the interplay between different cognitive domains. Both neurocognition and social cognition are comprised of six to eight subdomains in schizophrenia research. Information from neurocognitive and social cognitive processes interact with one another (i.e., social knowledge of face-relevant areas directs attention to the eyes) and integrate to form metacognitive knowledge (e.g., "I correctly identified my friend"). This knowledge is integrated with other knowledge to form more interactively complex metacognitive insights (e.g., "I enjoy cooking more than others").

How does neurocognition relate to functioning?

Functioning in schizophrenia is commonly grouped into four categories: social behaviour in the milieu, social skills, social problem-solving, and community functioning[75]. While all four domains assess the application of cognitive abilities, both social problem-solving and social skills are measures of functional capacity (raw ability to perform a task) and both community functioning and social behaviour in the milieu are measures of actual functioning in the real world (functional outcomes). Functional capacity may be assessed by planning activities, handling money, or arranging transportation in role-play scenarios[76], whereas functional outcomes may include independent living and employment. A recent meta-analysis has succinctly summarised the associations between both neurocognition and social cognition (discussed next) with functioning (**Figure 2.**)[75]. This review of 166 studies of 399 effect sizes (N = 12868) found that combined neurocognitive ability predicted all four functional domains at small effect sizes (r = 0.22, range: 0.14 - 0.26, R^2 = 4.84%) which was consistent across disease chronicity. This overall association was stronger than the analysis of each MATRICS domain individually; suggesting the link between neurocognition and functioning may be general and not domain-specific. Across studies, neurocognition was most closely related to vocational activity, while negative symptoms were more predictive of social outcomes[77].

The finding that general neurocognition relates to functioning is supported by the complexity of everyday tasks that rarely require a single domain (unlike cognitive tasks). For example, executive function, working memory, and vigilance have been found to predict full-time employment status[78], poor visuospatial ability may mean people misplace household items, poor vigilance may lead to difficulty in listening to instructions, and slow processing speed may mean target-driven employment environments become difficult to keep up with[47]. Another example is the transportation assessment of the UCSD Performance-based Skills Assessment (UPSA), wherein participants must read transportation timetables and plan their route[76]. While relatively simple, this task requires good verbal learning and comprehension, the abstraction of the meaning of bus times, understanding the spatial relationship between transport locations, working memory to manipulate the plan in consciousness, and verbal fluency to ask for guidance. From these findings, it may be assumed that improvements in neurocognition may create wide reaching benefits. Indeed, this is the basis of both the Cognitive Remediation and cognition-enhancing medications, as outlined below.

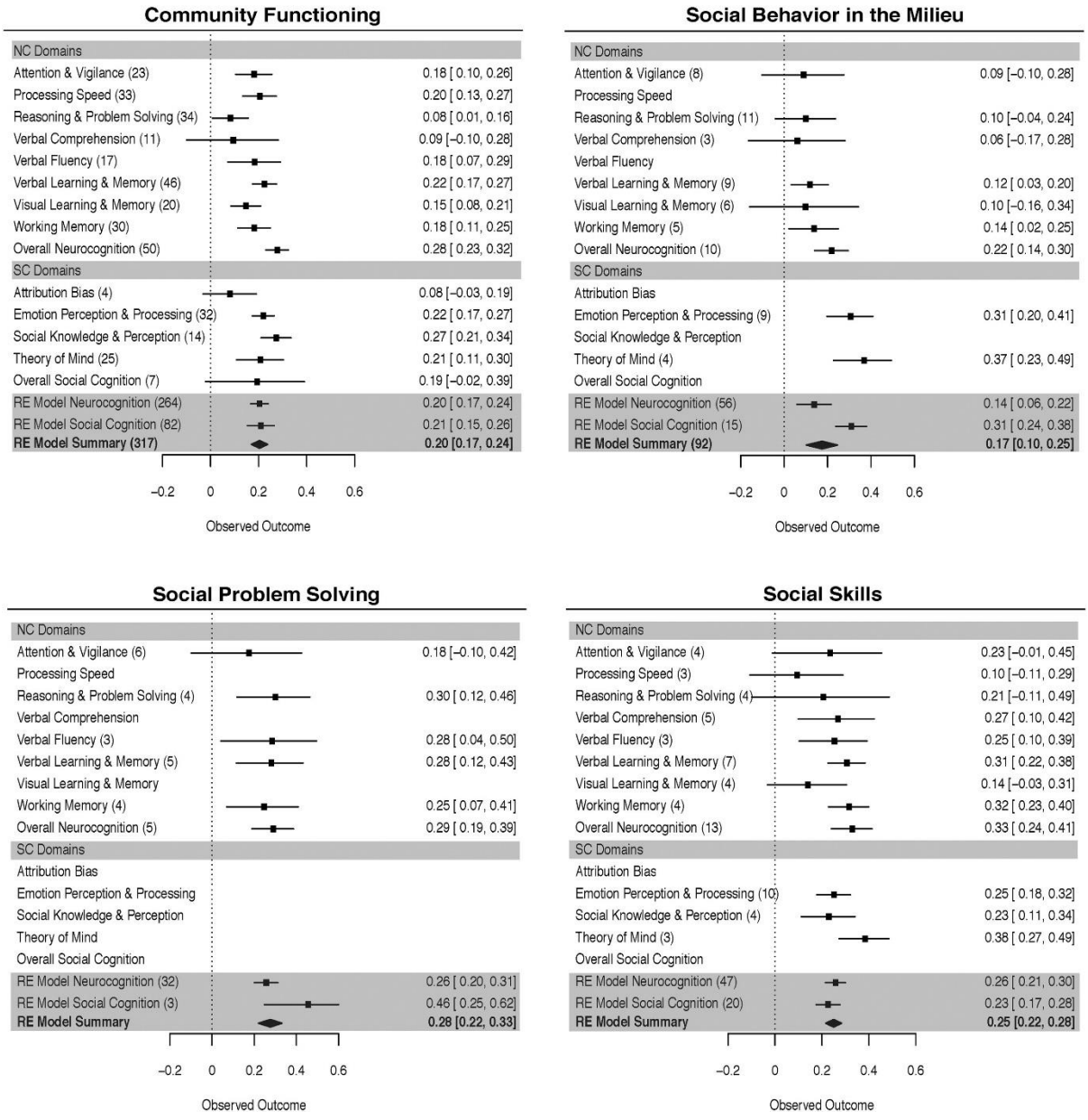

Figure 2. Meta-analyses conducted by Halverson et al. 2019, investigating how different neurocognitive and social cognitive domains affect functional capacity (social skills and social problem solving) and functional outcomes (community functioning and social behaviour in the milieu). Square points represent the average effect size of each domain to predict functioning, horizontal error bars represent 95% Confidence Intervals, and the lower diamond represents the averaged effect across subdomains. NC = Neurocognition, SC = social cognition.

Neurocognitive interventions

Currently, there are no clinically effective medications that improve cognitive deficits in schizophrenia. While there is evidence that antipsychotics may produce small improvements these are difficult to disentangle from practice effects in longitudinal studies[79] and the detrimental side-effects of the medication[80]. Overall, antipsychotics appear effective at reducing the positive symptoms alone. An alternative to medication is Cognitive Remediation (CR), an intervention focused on the massed practice of neurocognitive tasks to facilitate improvement (akin to 'brain training'). While CR is consistently linked to increased activation in prefrontal and thalamic regions[81], as well as functional, structural, and connectivity changes in the brain[82]; its efficacy in terms of improving functional capacity and outcomes is unclear. In the past two decades, there have been six meta-analyses of CR in schizophrenia that answer different questions. Together, they suggest that CR improves neurocognition at small to medium effect sizes, which is sustained at 6-month follow-up (d = 0.43)[83] and is effective in both in-patients (d = 0.28 – 0.48)[84] and FEP patients (d = 0.19 – 0.23)[85]. There are inconsistencies as to whether all neurocognitive domains are benefitted[83,86], but the most consistent improvements are in processing speed, working memory, and Verbal/Visual Learning and Memory. For symptoms, three meta-analyses suggest that CR produces small improvements to total symptoms both immediately post-intervention (d = -.18 to -.19)[83,85] and at 6-month follow-up (d = -.17). A larger review of 45 randomised-controlled trials also suggested that negative symptoms are reduced to a greater extent at both post-treatment (g = -.30) and follow-up (g = -.36)[87]. Finally, CR is effective at improving functioning at 6 month follow-up in chronic patients (d = 0.37 - 0.42)[83] and inpatients (g = 0.47)[84], but CR is relatively less effective in FEP patients (d = 0.18) and ineffective in Clinical High-Risk patients[88]. A specific example is that at one-year follow-up, CR increased full-time employment rates from 21% to 41%, the number of days worked by 19.5, and yearly income by $959[89]. This latter point is consistent with the aforementioned associations between neurocognition and vocational outcomes in schizophrenia[77].

Neurocognition summary

While neurocognition has helped to explain and improve both symptoms and functioning in psychosis, neurocognitive deficits appear to be generalised across domains and potentially not specific to schizophrenia[49]. It is also unclear why functional outcomes may further decrease with chronicity if neurocognitive deficits appear to plateau in adolescence. Critically, while neurocognition is associated with symptoms and functioning, these associations are not as strong as previously thought[52]. Indeed, neurocognition was initially suggested to explain 20 - 60% of the variance in outcomes[15], but recent meta-analyses put the figure at 4.4%[75]. While CR may improve

neurocognition in some patients this may only translate to vocational but not social functioning. Moreover, these improvements are again not clinically significant and reviews into CR have questionable quality[90]. Crucially, these perspectives cannot explain why patients with poor neurocognition do not necessarily have poor outcomes, nor how patients with intact neurocognition may present poor outcomes[91–93]; suggesting cognitive deficits are not necessary nor sufficient to cause poor functioning. Overall, 93.6% of the variance in outcomes was left unexplained by neurocognition. This finding was a major catalyst for the shift in focus to social cognition, as outlined below.

Is social cognition a better explanation of poorer functioning in schizophrenia?

Social Cognition

The start of the millennium saw a significant shift in focus to how social cognitive research in psychosis could improve symptoms and functioning, partially spurred on by the relative limitations of neurocognitive perspectives[94,95]. Social cognition is a set of mental processes responsible for the perception, decoding, interpretation, and regulating of our reactions to social stimuli and the minds of others[96] allowing us to communicate beyond basic language ability. Social cognitive processes are a very high-level set of skills that permeate nearly every aspect of complex modern living. Individuals must not only assess each social experience, but also understand that the same actions of others have different meanings depending on the social context, social norms, culture, and our pre-existing knowledge of that person. This section will first outline how social cognition is distinct from neurocognition, the relevant social cognitive domains and deficits in schizophrenia, and assess whether social cognition may improve explanations of symptoms and functioning.

How is social cognition distinct from neurocognition?

Social cognition is thought to rely on more the foundational neurocognitive and basic perceptual abilities[97], which themselves create a cascade effect from bottom-up processes to higher-level social cognitive processes[98]. However, social cognition is assumed to not simply be the application of neurocognitive processes to social stimuli, as the meaning of these perceptions is interpreted by social cognition. For example, an understanding of the mental states of others requires the abstraction of non-concrete concepts from concrete stimuli (e.g., intentions from facial expressions and body language)[99]. Indeed, every MATRICS domain has been associated with social cognition[100,101] and only 10% of patients with impaired neurocognition also have intact social cognition[102]. Moreover, both neurocognitive and social cognitive deficits share abnormal activation of the prefrontal cortex, temporal lobe, and hippocampus in patients. However, social cognition is specifically associated with abnormal limbic system activation (amygdala), the fusiform gyrus, and the parietal lobe[47]. Indeed, while the associations between neurocognition and social cognition are consistent, this association is relatively weak (e.g., 10% shared variance)[101] and factor analytical studies consistently suggest a two-factor model separating each construct better conceptualises the behaviour of patients[97,102,103]. Together, both neurocognition and social cognition are considered independent but semi-overlapping mechanisms[47,104].

Social Cognition deficits in schizophrenia

While social cognition has many varying definitions, for succinctness, this review focuses on social cognitive domains relevant to schizophrenia outlined by the Social Cognition Psychometric Evaluation (SCOPE) consensus: emotion perception/processing, social perception, Theory of Mind, and attribution style[105]. Other research may also include social knowledge[45,94], empathy[106], and emotional intelligence (EIQ) which overlap with social skills, Theory of Mind, and emotion processing respectively (see **Figure 1**). The most recent meta-analysis of social cognitive deficits in schizophrenia highlights large deficits in Theory of Mind (g = 0.96), social perception (g = 1.04), emotion perception (g = 0.89) and emotion processing (g = 0.88); moderate deficits in social knowledge (g = 0.54); but no evidence of attribution style biases (g = -0.02 to -0.17)[107]. Moreover, deficits in Theory of Mind specifically have been found to correlate with reduced medial front cortex activity and predict conversion to psychosis[108,109]. Further meta-analytical evidence also suggests deficits are greater relative to other psychiatric disorders such as bipolar[110], but smaller relative to Autism Spectrum Disorder (e.g., emotion recognition, g = 0.43)[111]. In psychosis specifically, neurological evidence suggests the under-recruitment of social cognitive related brain regions such as the bilateral amygdala, parahippocampal gyrus and fusiform gyrus and the over-recruitment of the left insula may play a role[112].

While neurocognitive deficits appear to be stable after initial diagnosis the course of social cognitive deficits is less clear; although current evidence suggests social cognition follows a similar trend to neurocognition. Specifically, chronicity and DUP are unrelated to emotion perception performance in some reviews[107,113] and studies[57], but tentatively related in others[114]. Social cognitive deficits are also observed both at FEP and chronic stages with minimal variability in performance between these groups[107,115,116]. The limited number of longitudinal studies also report that social perception, emotion processing, and ToM remain stable[117,118].

How does social cognition explain symptoms?

This increase in social cognitive research since the millennium largely aimed to explain symptoms and functioning in schizophrenia over and above neurocognition. The most recent meta-analysis suggests that both negative and disorganised symptoms are correlated weakly with Emotion Perception (r = -0.26 – 0.32), social perception (r = -0.20 – -0.22), and ToM (r = -0.25 – -0.32), while reality distortion was weakly related to Emotion perception (r = -0.22) and social perception (r = -0.21), but not to ToM (r = -0.08)[101]. Emotion processing has gained particular attention, with reviews suggesting deficits are moderate across all symptom categories[113], greater for inpatients[107,113], more severe in patients with persistent negative symptoms (d = 0.36 – d = 0.93)[62], and that negative

symptoms themselves are the most consistently associated with social cognitive deficits[96].

The associations to reality distortion may be explained through an emotion processing bias to interpret neutral face as negative in patients[119]; suggested to create persecutory delusions from an influx of negative information[47]. Disorganisation has also been suggested to be influenced by poor intention attributions (ToM), reduced integration of contextual social information and social knowledge[120] with normative behaviour, leading to seemingly incoherent behaviour (e.g., not understanding that explicit language is less acceptable at work than around friends). Finally, the experiential negative symptoms of amotivation and flattened affect have been suggested to be influenced by poorer learning of social information, meaning social reward salience is reduced (i.e., perceiving a happy face as neutral)[121]. Poorer social perception (e.g., unawareness of a friend's distress) may also be misinterpreted as being apathetic.

How does social cognition explain outcomes?

Social cognition has proved particularly relevant to functioning with clear implications for social outcomes. As the previous meta-analysis in **Figure 2.** summarises, the average correlation between social cognition and outcomes is $r = 0.24$ ($R^2 = 5.76\%$)[75] and is thus more closely related to functioning than neurocognition[122]. Dysfunctional social cognition has been suggested to underlie the inter-personal conflict, isolation, and social disengagement seen in schizophrenia[123]. For example, consider the social situation of meeting a friend at a café. While neurocognitive processes allow us to plan and organise the meeting, social cognition may inform us our friend is in distress which guides the conversation to support them, social knowledge allows us to understand the social dynamics of ordering a coffee (e.g., queuing, waiting in turn, politeness), and theory of mind informs us the baristas does not know our order if we do not communicate it. Disruptions to these processes may cause an over-emphasis on ourselves or perhaps conflict from jumping the queue. While this example is quite basic, this highlights the critical role that social cognition plays in actions taken for granted. A more severe example may be a patient's persecutory delusions suggesting their landlord intends to harm them - leading to housing issues. Moreover, poorer social cognitive learning may mean patients do not value socially appraised behaviours which are common functional outcome criteria (e.g., employment, stable relationships, or pro-social behaviour). Furthermore, poorer learning may lead to socially bizarre behaviour that inhibits the formation of strong social networks, social amotivation can lead to social exclusion, and incorrect mental state attributions may increase distrust in others. This may also lead to a positive feedback loop where negative social experiences lead to social withdrawal and subsequently reduced opportunities to find disconformity evidence for delusional or socially bizarre behaviour[124].

Social Cognition based interventions

If social cognition is more closely related to outcomes, it would be expected that improvements to social cognition would result in greater treatment gains relative to neurocognitive interventions. Currently, anti-psychotic medication has limited impact on improving social cognition[125] meaning the focus has shifted to psychotherapy. The social form of Cognitive Remediation (S-CR) is one such intervention, which may focus on the massed practice of social cognitive processes, such as facial affect recognition[126]. More commonly, social cognitive interventions tackle multiple social cognition domains and are commonly combined with other social interventions such as Social Cognitive Training (SCT) or Social Cognitive Interaction Training (SCIT), which also involve group-based role play, social practice, and increasing social knowledge[127]. The addition of S-CR to SCT has been suggested to improve treatment response[75,128], as poorer cognition may inhibit patient involvement through attentional deficits[129]. In terms of efficacy, some applications of S-CR have shown potential evidence for improvements in social cognition, symptoms, and functioning[130,131]. However, the current evidence base is limited, and few studies have separately administered S-CR without other social cognitive therapies such as SCT. While all of these interventions have been found to improve social cognition, only broad SCT which targets multiple domains is currently effective at improving social functioning (d = 0.41 - 0.82)[132]; consistent with increased neuroplasticity in related social-cognitive brain regions[133]. However, there is currently mixed evidence as to whether symptoms are improved at follow-up[95,132,134] and whether social cognitive interventions are more effective than neurocognitive Cognitive Remediation[83,86].

Social cognition as a mediator of outcomes

While the pattern of deficits in social cognition and subsequent associations to functioning appear similar to those of neurocognition, the primary value of social cognition in this context comes from its mediatory role between neurocognition and functioning. Indeed, a recent meta-analysis of 32 studies revealed several critical findings[75]. Firstly, neurocognition and social cognition together explained 9.2% of the variance in outcomes. Secondly, social cognition accounted for 7.3% of this total variance while neurocognition accounted for 1.9%. This pattern of findings suggests that social cognition: 1) partially mediates this relationship 2) explains incremental variance over neurocognition and 3) is more closely related to functioning than neurocognition (**Figure 3**). For example, included studies reported that facial emotion recognition partially mediated the association between vigilance and social skills, poor visual learning predicting poorer body language perception, and poor early visual perception leading to social perception difficulties and subsequently poorer social functioning[24,25]. That being said, these results are correlational and

therefore the direction is inferred from theory. Indeed, there is evidence Working Memory may predict functioning independently of social cognition[135]. In reality, these relationships are likely at least partly reciprocal, such as lack of employment, inpatient status, and poor community integration limiting opportunities to improve neurocognitive and social cognitive skills[136].

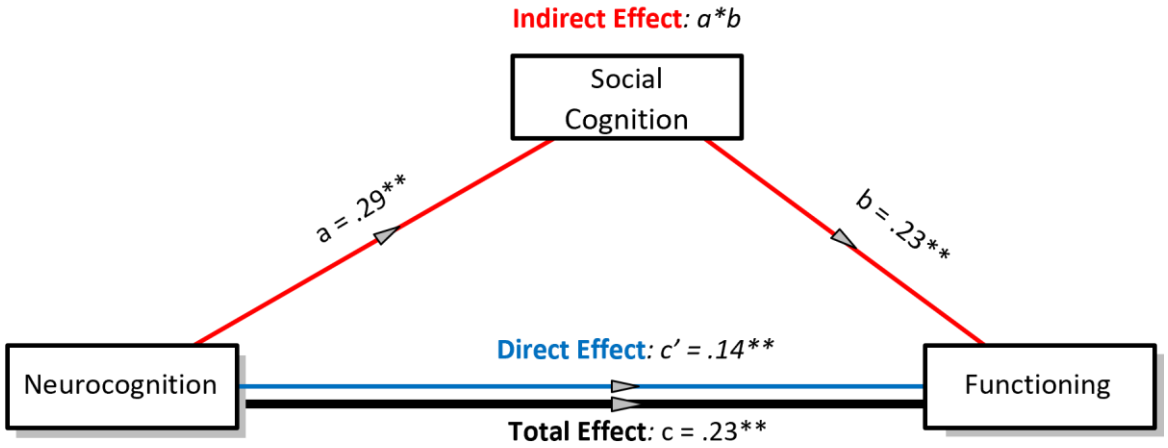

Figure 3. Mediation model from Halverson et al. 2019. Coefficients represent correlations. **Total effect**: simple regression of neurocognition predicting functioning; **Indirect effect**: the effect that neurocognition has on functioning through neurocognition's relationship to social cognition; **Direct effect**: the effect of neurocognition on functioning independently of the direct effect.

Aim of the current thesis

The first half of this Chapter has highlighted that cognitive research in psychosis has consistently supported broad deficits in both neurocognition and social cognition. While social cognitive research over the last few decades has improved our understanding of functioning in psychosis beyond neurocognition - it is still limited. Even when the effects of neurocognition and social cognition are combined, 90.8% of the variance in functioning is still left unexplained. While an understanding of outcomes may be incomplete without considering both neurocognition and social cognition[97], there is also a multitude of other factors to consider. Recently, there have been calls to identify additional variables, mediators, confounds, and moderators of both cognitive performances and functioning in psychosis[75]. This includes understanding why participants perform poorly on cognitive tasks, whether cognitive tasks truly capture cognitive abilities, and why cognition and cognitive gains from interventions may not translate to real-world functioning[137]. This thesis aimed to assess the potential influences of metacognition, Mood and Anxiety Disorder (MAD) co-morbidity, amotivation and clinical confounds on both cognitive performance and the translation of ability to functioning – as outlined below.

Does metacognition affect the translation of ability to functioning?

Metacognition

At the start of the previous section, it was explained that the rapid expansion of social cognitive research was influenced by the relative limitations of neurocognition. This section presents the same narrative, that the limitations of the social cognitive research of the mid-2000s saw a rapid increase in metacognitive research in psychosis. While metacognition is not a new concept, it has gained rapid momentum over the last 10 years; primarily as a mediator between cognitive ability and functional outcomes[137]. Partly due to this novelty, there is currently no universally agreed upon definition of metacognition[138], being described as overinclusive of "almost any cognitive process" (Reynolds & Wade, 1986, *pg.* 307–308). Unlike neurocognition and social cognition, metacognition does not have a consensus on its definition or domains relevant to schizophrenia (i.e., MATRICS and SCOPE). As a result, the below summary balances the detail needed to ensure consistency with past research with the conciseness necessary for this thesis. To do so, the concept of metacognition has been simplified with only the most relevant domains to psychosis and the following experimental chapters being outlined (for reviews see[140]).

What is metacognition?

Metacognition was first detailed in the education literature as "thinking about thinking"[141], stemming from observations that older but not younger children could accurately access their learning[142]. In psychology, metacognition is used to describe the reflexive monitoring of our cognitive processes and mental states, analysing their content, and applying these insights to adapt our cognitions and behaviour. There is also significant conceptual overlap between metacognition and cognition. For example, some metacognitive assessments include the integration of mental states of others, emotion identification, and consideration of events from another person's perspective; which all overlap with emotion recognition and theory of mind[143]. However, while metacognition relies on the accurate processing of discrete ('singular') pieces of social cognitive information, metacognition itself is the integration of reflections on these perceptions to form complex representations such as social norms, schemas, and how they go on to influence future perceptions in a reflexive cycle[144]. Social cognition is thus concerned with accuracy while metacognition is concerned with reflection and integration[140,145]. Put another way, recognising thoughts, feelings, or intentions does not equate to forming a global picture of who someone is.

Metacognition is also suggested to partially rely on neurocognition[146], but is distinct in that neurocognitive ability *determines* the rate of learning, whereas metacognition *assesses* the rate of learning. These distinctions are well described in the following example in the context of a medical exam[147]:

> "*A student completely absent of basic cognitions would not be able to learn the materials at all, but a student absent of meta-cognition would not be aware if they had learnt the material at all*".

Recently, the psychosis literature has begun to understand metacognition as a continuum of increasingly complex integrative behaviours that mutually interact with one another[148,149]. At the least integratively complex end are discrete metacognitive judgements, followed by moderately discrete judgements, and finally synthetic metacognition (**Figure 4**). Consequently, a breakdown of metacognition would cause a fragmentation of experiences (for reviews, see[148,149]). The remainder of this section will describe each level of metacognitive complexity, discuss assessment tools, outline the metacognitive disruptions in schizophrenia, and how metacognition may improve our understanding of functioning in patients. Of particular relevance are discrete and Moderately Discrete Metacognition (MDM) which are both assessed in the current thesis.

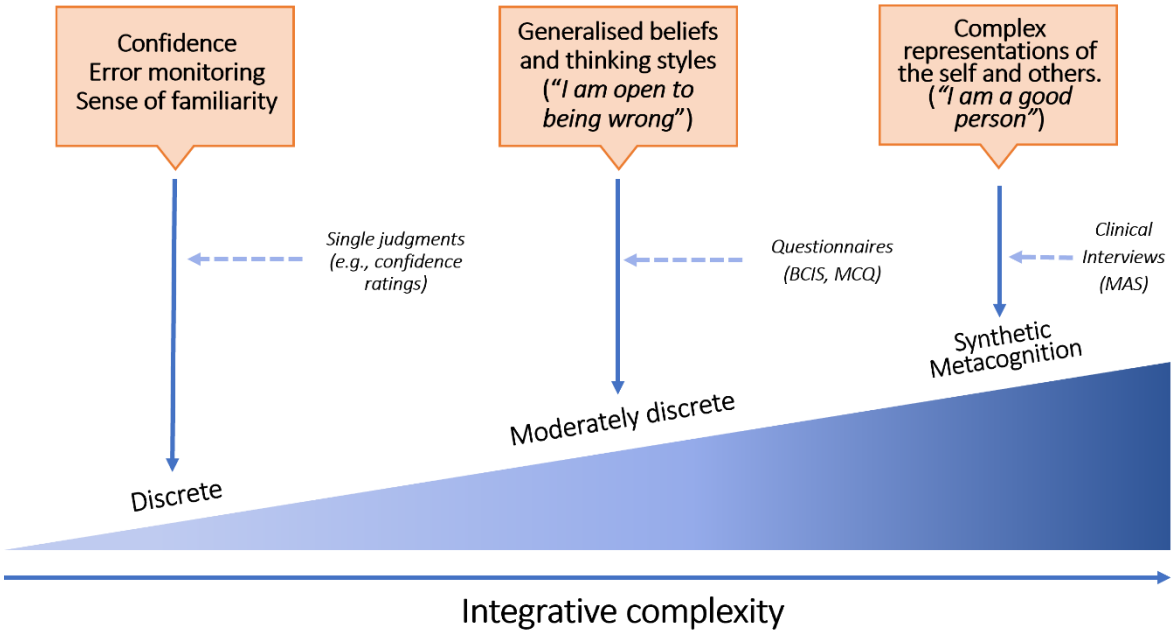

Figure 4. A conceptualisation of metacognition as the increasing integration of single (discrete) judgments to create higher order (meta) beliefs. Dashed lines represent potential measurement techniques. Adapted and expanded from (Lysaker, Hamm, Hasson-Ohayon, Pattison, & Leonhardt, 2018)

Discrete metacognition

The least 'integrative' form of metacognition is discrete metacognition: reflective judgments of single (discrete) experiences and perceptions[149]. Taking the example of the discrete experience of noticing the face of a passer-by, discrete metacognitive processes may assess our sense of familiarity with this person, the vividness of memory that we had met them before, our confidence in our judgement, and how this information is integrated to decide whether we should greet this person. A breakdown of discrete metacognition would create a lack of awareness of what information is known, what is unknown, if this experience may be a false memory or a thought that cannot be separated from reality, and potentially acting on faulty beliefs (e.g., greeting a stranger). These discrete metacognitive judgments are considered second-order (or meta-level) decisions, whereas neurocognitive and social cognitive judgments are considered first-order (object-level) decisions. While discrete metacognition has a multitude of potential assessment methods, of particular relevance to schizophrenia are confidence ratings and the use of knowledge, which are also referred to as metacognitive sensitivity[ii] (or 'monitoring') and metacognitive control. While metacognitive sensitivity refers to the appropriate allocation of confidence to correct and incorrect decisions, metacognitive control refers to the use of metacognitive insights to guide behaviour. These concepts are discussed due to their relevance to symptoms and outcomes, as outlined below and in both Chapter 3 and Chapter 4.

Metacognitive sensitivity and metacognitive

Dysfunctional confidence attributions and biases have long been reported in the general population. A well-known example is the Dunning-Kruger effect, where confidence follows an inverted U-curve with ability (**Figure 5**). One common assessment method in psychosis is to request confidence ratings in memory tasks ("meta-memory" research). These paradigms range from recalling previously presented images, false memory paradigms, or recall biases in autobiographical studies. In the psychosis literature, disrupted metacognitive sensitivity (aberrant confidence) is assessed through both raw confidence ratings and the indices of the Confidence Gap and the Knowledge Corruption Index (KCI). The Confidence Gap represents greater confidence for correct answers than incorrect answers (which is an adaptive metacognitive strategy), whereas the KCI is the proportion of highly confident incorrect responses, representing overconfidence in errors.

Generally, patients tend to be overconfident when incorrect and underconfident when correct relative to controls (Confidence Gap)[27,150], although overconfidence in errors is the most

[ii] This may also be referred to as metacognitive accuracy, monitoring resolution, and introspective accuracy.

consistent finding[27,151,152]. Across all current measures, metacognitive sensitivity is impaired at a medium effect size in schizophrenia (*g* = -0.57, K = 42)[153] and these deficits are present from the pre-clinical stages to the chronic stages of psychosis[150,154]. Moreover, deficits are not just found in retrospective memory assessments[155] but also found in perceptual[156], neurocognitive, and social cognitive tasks[157]; suggesting deficits are not solely due to memory biases and span different cognitive domains. Critically, the longitudinal deterioration of discrete metacognitive processes has been suggested to be critical for the pathogenesis of schizophrenia[150]. These deficits appear to progress with the disorder, with sub-clinical psychosis presenting overconfidence in errors, ARMS and FEP patients additionally showing a reduced Confidence Gap relative to controls, and chronic schizophrenia patients also presenting a significantly higher KCI[150].

Symptoms and outcomes

Aberrant confidence in psychosis has clear relevance for delusions - false beliefs held with exceptionally high confidence[2]. For example, high confidence in attributions of negative facial affect of others may lead to paranoia[157] or high confidence in a false memory of being watched may lead to delusions of surveillance. More general discrete metacognitive biases reported in schizophrenia, such as a Bias Against Disconfirmatory Evidence (BADE[158]), may also explain why delusions are held with high conviction, as opposing evidence is attributed less relative value. The underconfidence for correct decisions also likely contributes to delusion maintenance as correct information is likely not considered[150]. Indeed, this is supported by deficits in metacognitive sensitivity being greater in patients with current delusions[159,160] and those high in paranoid symptoms[156] at medium to large effect sizes. While cognitive deficits may not be specific to psychosis, the high confidence they are attributed may explain why delusions are specific to psychosis[155]. The associations to functioning are also wide-reaching as discrete metacognition affects how knowledge is interpreted and used (e.g., acting on erroneous beliefs). Critically, the underconfidence in correct decisions may explain the under capitalisation of preserved ability in patients and the poor transferral of new cognitive skills to functioning. This is supported by findings that patients tend to underestimate their cognitive ability[161], which may explain why patients with good neurocognitive ability continue to present poor outcomes. While promising, there have been further calls for discrete metacognitive research, especially in the early stages of psychosis[27,150]. Moreover, metacognitive control processes in psychosis (acting on metacognitive reflections) are not well understood.

Moderately Discrete Metacognition

Moderately Discrete Metacognition (MDM) describes the integration of discrete metacognitive reflections to create more general belief systems. They are akin to trait-like thinking styles, rather than 'in the moment' discrete metacognitive judgments. One clear example is how MDM integrates discrete metacognitive perceptions to form a concept of our abilities. For example, when baking, we may not feel confident that we know how long to whip meringue, or how much sugar to add to a conserve, but we feel confident in our ability to make a custard. These discrete confidence judgements are integrated to form the moderately discrete belief that "I am average at making desserts". This integration creates a novel insight that is greater than the sum of its discrete parts. These insights can in turn lead to adaptive behaviours such as buying a dessert recipe book, sharing these desserts with friends, or perhaps enrolling on a culinary course. If the Dunning-Kruger effect were applied to this example, the confidence for a person completely devoid of metacognition would follow a flat line (**Figure 5**). In psychosis, MDM is commonly assessed psychometrically through questionnaires, as outlined below and used in all subsequent Chapters.

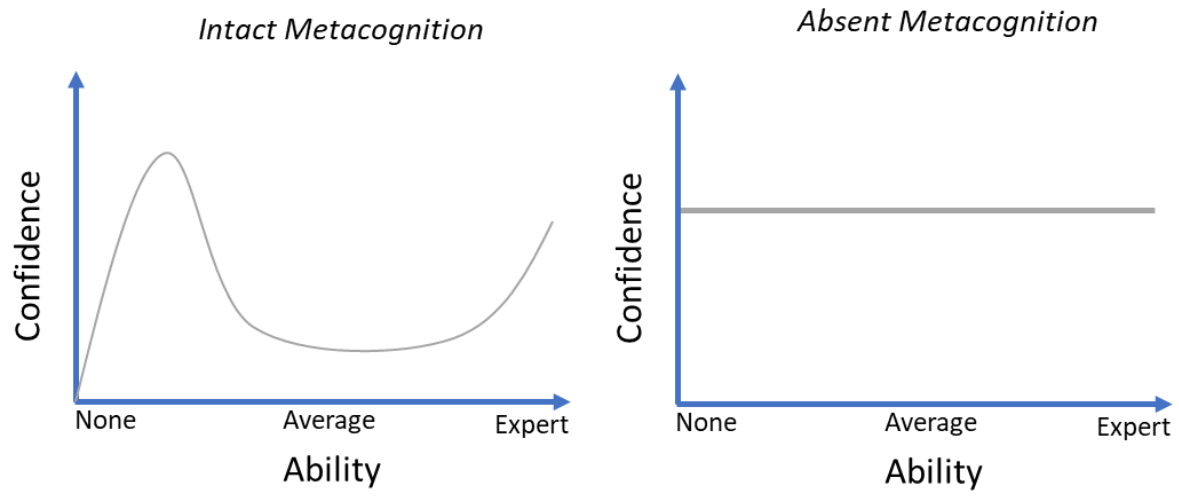

Figure 5. **Left**: proposed Dunning-Kruger effect of those with intact metacognition following an inverted U curve. **Right**: example of how a person without metacognition may report their confidence.

How is Moderately Discrete Metacognition in schizophrenia disrupted

Moderately Discrete Metacognition (MDM) in psychosis is most commonly assessed through metacognitive thinking styles (including in this thesis) by two instruments: the Beck's Cognitive Insight Scale (BCIS)[162] and the Metacognitions Questionnaire (MCQ)[163]. The BCIS was designed to extend assessments of clinical insight (i.e., unawareness of illness) to more general decision-making; assessing Self-Reflectivity and Self-Certainty. Respectively, these MDM thinking styles assess the openness to being fallible or to consider alternative hypotheses ("*Some of my experiences that have seemed very real may have been due to my imagination*") and pathological overconfidence and resistance to consider feedback ("*I can trust my own judgment at all times*"). Generally, psychosis patients show decreased Self-reflectivity and increased Self-Certainty[164], meaning they are less open to being wrong and are overconfident in their decision-making. The MCQ, on the other hand, is a broader MDM assessment with its roots in the Self-Regulatory Executive Function model of affective disorders[165]. This model proposes dysfunctional metacognitive processes maintain unhelpful thinking styles that produce worry, rumination, and attentional biases to control thoughts and events. For example, that rumination is beneficial, that constant over-analysis of thoughts is necessary, and that one's thoughts are uncontrollable, dangerous, and need to be controlled. The findings from two recent systematic reviews[166,167] have found psychosis patients present maladaptive metacognitive thinking styles across all of the five MCQ scales (k = 11, g = 0.49 – 1.31). The largest deficits appear in feelings that patients must control their thoughts (g = 1.31) and that their thoughts are uncontrollable and dangerous (g = 1.10). One interesting finding is that while chronic psychosis patients state they believe worry and rumination are beneficial, these metacognitive deficits are not present in ARMS[166] or mood disorders patients. Together, this may suggest this scale may relate to the transition to psychosis.

How does Moderately Discrete Metacognition relate to symptoms and functioning?

The clearest associations between MDM and symptoms are those between the BCIS and reality distortion. Reduced Self-Reflectivity has been suggested to cause a detachment from objectivity and a loss of perspective, while increased Self-Certainty can produce resistance to corrective information[162]. This is immediately relevant for delusions wherein poorer Self-Reflectivity diminishes the consideration of alternative hypotheses under the assumption one has perfect cognition. Delusions are then given exceptionally high confidence (Self-Certainty) making them both incredibly resistant to change and more likely to be acted upon. Indeed, both Self-Certainty and Self-Reflectivity correlate with positive symptoms both cross-sectionally and potentially longitudinally[164]. Moreover, ARMS patients present increased Self-Certainty (g = 0.45) and potentially reduced Self-

Reflectivity (g = -0.56, p = 0.14, k = 5); highlighting that a breakdown of metacognition may play a role in the transition to psychosis[168], which is also consistent with reviews of discrete metacognition[150]. There is also evidence that hallucinations may be specifically associated with high Self-Reflectivity but low Self-Certainty, while delusions are associated with low Self-Reflectivity and increased Self-Certainty[169]. However, there are conflicting self-reports of improved MDM in psychosis[27,164,170]. Currently, it is unclear whether this may represent either such severe MDM impairments that patients are completely unaware of their cognition[27], that MDM assessments are not reliable, or only specific subgroups of patients are impaired. This dispute is a primary aim of both Chapter 3 and Chapter 4.

In terms of functioning, the BCIS has been associated with independent living, psychosocial functioning, and Quality of Life even independent of negative affect[164]. This association is potentially explained by a lack of Self-Reflectivity meaning vocation or social skills are much harder to obtain, such as not allocating more study time to less-known information[171]. Moreover, real-world functioning not only depends on raw ability (cognition) but also on our appraisals of these abilities. For example, the metacognitive knowledge that we are an excellent cook may guide us to seek employment in this area, or the metacognitive insight that "I have poor memory" may lead to compensatory mechanisms (e.g., writing information down). Indeed, it has been suggested that metacognitive coping strategies for ignoring positive symptoms may explain the weak association between symptoms and community functioning[172]. This may also be relevant in explaining why poor cognitive abilities do not always lead to poor outcomes (e.g., coping mechanisms)[91] and why good cognition is not always translated to good outcomes (e.g., not acting on ability). However, again, there are inconsistencies in whether the BCIS is positively[173–175] or negative associated with functioning[176,177]. Moreover, higher Self-Certainty may offer a protective factor against poor self-esteem when symptoms are severe[178].

For the MCQ, the primary implication has been associations with positive symptoms[166,179] and predicting transition to psychosis[180–182]. Hallucinations have been suggested to be due to the misattribution of internal events to external sources (i.e., intrusive thoughts)[183]. Specifically, the maladaptive metacognitive belief that someone must constantly control their thoughts is proposed to create cognitive dissonance when intrusive thoughts appear. These difficulties to inhibit thoughts are thereafter attributed to external sources in an attempt to reduce distress[71]. While support for this proposal is limited in terms of distress reduction, there is wide support that hallucinations may be due to source misattributions[169,184–186], especially under uncertainty[184]. Associations to functioning are currently unclear as (at the time of writing) there are no investigations assessing how the MCQ relates to functioning in psychosis.

Synthetic metacognition

The most integratively complex form of metacognition is synthetic metacognition referring to the 'synthesis' of information. However, as synthetic metacognition is not the focus of the following Chapters the description below is relatively briefly. Synthetic metacognition involves bringing together any number of perceptions and reflections[187], differences between mental states, relationships between emotion and behaviour[16], along with social norms and the perceptions of others to create complex ideas of the self and others. An excellent example of synthetic metacognitive abilities is creating a dating profile - as a primary goal is to portray the self to others. To do so, the individual must synthesise their abilities with their emotional experiences, interests, memories, current state, and how these experiences relate to others. Our profile creator may integrate their high confidence in decisions for historical dates (*MDM*), pleasant emotional experiences when learning historical events (*affect*), and other people's relative disinterest in the subject (*comparing own experiences to those of others*) to form the synthetic belief that "I am a person who enjoys and is talented at studying history". They may also integrate enjoying meeting others, good emotion perception skills (*reflection on social cognition*), being perceived as a lively person, and the less prevalent experiences of enjoying being alone (*weighting of reflections*) into "overall, I am an extraverted person". Together, synthetic metacognition creates novel information that is greater than the sum of individual experiences – creating an identity. A person completely lacking synthetic metacognition may be unable to describe themselves accurately[148], perhaps only mentioning fragmented singular experiences such as "I visit museums" or "I feel sad now" on their dating profile.

How is synthetic metacognition assessed and disrupted?

Synthetic metacognition is almost exclusively assessed in psychosis by the Metacognition Assessment Scale (MAS) and its abbreviated counterpart (MAS-A). The MAS is a clinician-rated scale of patients' reflections from either interviews or recordings that rates people on four domains. The first two domains of Understanding One's Mind and Understanding the Other's Mind reflect integrations made about the self and others. The third domain of decentration refers to the ability to consider one's point of view as subjective and fallible, to recognise the difference between fantasy and reality, and to see the world from multiple perspectives (i.e., 'decentring' the self). Finally, the fourth domain of mastery involves using metacognitive insight to guide problem-solving (e.g., metacognitive control). People with schizophrenia commonly show deficits across all of the MAS domains[188] across all stages of clinical illness[140]. Moreover, these deficits are more severe than other

psychiatric disorders including substance abuse[189,190], bipolar disorder[191] and PTSD[192] suggesting medical adversity alone does not produce synthetic metacognitive deficits[193].

How does *synthetic* metacognition relate to symptoms and functioning?

Synthetic metacognition is associated with total symptoms with low heterogeneity (d = -1.07, I^2 = 0%, k = 15)[194], suggesting all dimensions of the MAS correlate with all symptom dimensions. Within current studies, the most consistent association is with negative symptoms both cross-sectionally[22,195–201] and longitudinally[202], with synthetic metacognition and symptoms potentially co-deteriorating[196,203]. There have been relatively few assessments concerning reality distortion, which is surprising, as reality distortion could also be considered a fragmentation between reality and internal experiences. In terms of functioning a recent meta-analysis has suggested that while synthetic metacognition is not related to overall outcomes (k = 32, d = .12) it is strongly associated with psychosocial functioning (d = 0.94)[194]. Some specific examples include increased social relations and frequency of social contact[204,205], improved vocational performance[143], help-seeking behaviour[199], and transferring skills to real-world functioning[206]. Moreover, synthetic metacognition post-intervention has been associated with response to psychosocial rehabilitation[207], subjective sense of recovery[208], and greater treatment gains[209–212], whereas poor synthetic metacognition may inhibit treatment gains through restricted abilities to understand their mind[140,199]. Recently, metacognition has also been suggested to be critical for motivation both generally and in psychosis[213,214], which may explain the associations to negative symptoms. As amotivation is an additional factor assessed by this thesis, further discussed can be found in the final section of this introduction.

What may metacognition add beyond neurocognition and social cognition?

The primary way that metacognition is relevant to this thesis is in how metacognition may explain symptoms and functioning above and beyond neurocognition and social cognition. This is not only the case as an independent predictor of outcomes, but also as a mediating variable between cognition and actual functioning (i.e., similar to how social cognition may mediate neurocognition). Indeed, there is recent initial support for this perspective[16,215] as well as the suggestion that metacognition may explain the "competence-performance gap" in psychosis - wherein functional capacity is not translated to actual functioning[16]. Indeed, neurocognition and social cognition are more strongly correlated with functional capacity (e.g., calculating a route to work or mock social interactions) relative to actual functioning in the real world[75]; suggesting other mediating variables may be more related to actual functioning. Metacognition may thus shed light on why some patients present intact cognitive ability[91–93] but continue to have poorer functioning[216].

Overall, metacognition is an extremely promising concept to explore in psychosis. However, metacognitive research as conceptualised here is in its relative infancy and requires further investigation. Indeed, while there have been reviews comparing the relative influence of cognition and metacognition[146] it is still unclear whether metacognition mediates this relationship or acts through separate but co-occurring pathways. Research into the earlier stages of psychosis is also needed as there is relatively limited literature available[146]. Indeed, recent reviews have called for an investigation into the interplay between neurocognition, social cognition, metacognition and functioning both generally and specifically at the earlier stages of illness[146]. Critically, it is also unclear:

a) Why is metacognition intact in some patients but impaired in others,
b) Why higher metacognition relates to improved outcomes in some patients but poorer outcomes in others, and
c) Whether self-reported metacognitive assessment tools give accurate representations of metacognitive ability.

These are some of the questions this thesis aims to address.

Metacognition summary

This thesis conceptualises metacognition as the increasingly complex integration of perception, emotional states, beliefs, memories, and our reflections on these experiences. Discrete metacognition involves reflective judgements on singular perceptions (e.g., “I am not confident I know this fact”), Moderately Discrete Metacognition (MDM) integrates these reflections further (e.g., “Overall, I have a poor memory”) and may be used to guide behaviour (e.g., “I should write this down or I will forget”), while synthetic metacognition syntheses these insights into complex representations of the self and others (e.g., “Compared to other people, I am forgetful”). A breakdown of metacognition may represent an unawareness of knowledge, poor coping mechanisms, and under capitalisation of preserved ability. The latter may be relevant to explaining the competence-performance gap in schizophrenia and this may help to improve functional outcomes. However, currently, there are limited investigations into the mediatory role of metacognition between both symptoms and cognition and cognition and functioning – which this thesis aims to address.

However, metacognition is not the only factor that requires further attention. The remainder of this introduction will focus on co-morbid Mood and Anxiety Disorders (MAD), amotivation, and confounds of patient status and their interactions with metacognition.

Do co-morbid Mood and Anxiety Disorders explain cognitive deficits and poor outcomes in schizophrenia?

Co-morbid Mood and Anxiety Disorders in schizophrenia

Schizophrenia patients are disproportionately diagnosed with a range of Mood and Anxiety disorders (MAD), which describe changes in the intensity, duration, or presentation of affective states[2]. Both psychotic symptoms and MAD symptoms are consistently associated[217,218], with FEP patients presenting a 23% co-morbidity rate with anxiety disorders such as social phobia, PTSD, OCD, Generalised Anxiety Disorder (GAD), panic disorder and specific phobia[11]. These MAD co-morbidity rates are elevated across all stages of psychosis: from childhood and adolescent Clinical High Risk (31.4% - 46.4%)[52], adult ARMS (8% - 34%), adult UHR (15% - 41%)[13], and FEP patients (23 – 26%)[11,218]. Critically, these prevalence rates are higher than those seen in healthy controls (11%)[219] and clinical controls (e.g., inpatients and outpatients, 12% - 27.0%)[220,221]. However, Major Depressive Disorder (MDD) is the most prevalent co-morbidity in schizophrenia, with a rate of 32.6% in outpatients[222].

There is a strong overlap between schizophrenia and MDD both conceptually and genetically[223]. Indeed, there is debate whether depressive symptoms are a core part of schizophrenia[224]. However, antipsychotic medication has limited effectiveness for depressive symptoms with over half of patients remaining in the acute stage[225]. The associations between psychosis and MDD may be due to the commonality of negative symptoms and depressive symptoms. However, recent reviews have found while both disorders overlap in anhedonia, anergia and avolition (social amotivation), negative symptoms are distinct in assessing observed sadness, alogia, poor attention, low concentration, blunted affect, and social withdrawal; whereas depressive symptoms cover low mood, pessimism, and suicidal ideation (k = 25)[226]. Moreover, while depression is often episodic and a deviation from 'typical' functioning, negative symptoms tend to be trait-like and enduring.

How do MAD disorders affect cognition, symptoms, and functioning in schizophrenia?

The relevance of MAD co-morbidity for this thesis is that MAD disorders have independent effects on schizophrenia symptoms, cognition, and functioning which may exacerbate or even explain observations in psychosis patients. For cognition, MAD independently predicts moderate deficits across neurocognitive tasks[227] even in the remitted stages of these disorders[228]. Social cognition is also impaired, including moderate impairments in Theory of Mind (*d* = -0.51 - -0.58)[229] and emotion recognition of all six basic emotions except sadness (g = −0.42 to −0.17)[230]. Together, this suggests

MADs present both global and relatively stable cognitive deficits that occur separately from episodes of low mood.

For symptoms, depressive traits have been found to pre-date psychotic onset[231–233] and exacerbate positive symptoms in a potentially reciprocal relationship[232,234]. The reverse is also true, where psychotic symptoms can predate depressive symptoms that subsequently increase depressive symptom severity[235–237]. However, it is currently unclear whether depressive symptoms predict later conversion to psychosis[13,224,237,238]. Although, it has been suggested that depressive traits may exacerbate delusions through self-critical beliefs and schemas and the anticipation of negative consequences[239]. These associations are further supported in that treatment of either depressive or psychotic symptoms can reduce symptoms of the other[240]. In terms of functioning, MDD itself is consistently associated with poorer functioning including lower employment, education[241], and Quality of Life[242], similar to schizophrenia. Within schizophrenia, MDD and depressive symptoms are also associated with reduced Quality of Life[243], functioning at long-term follow-up, rates of functional recovery[244], transferral of ability into functioning[245], and increased suicidality to an even greater extent than psychotic symptoms (k = 96)[246].

How is our current understanding of MAD disorders in schizophrenia limited?

While there is a good current understanding of how both psychosis and depression may relate to functioning, the interplay and separability between these disorders is not well understood. Clinically, this co-morbidity is sometimes overlooked in schizophrenia which reflects a form of hierarchical reductionism; where more 'severe' or readily recognisable disorders overshadow others[247]. Commonly, cognitive assessments do not consider the influence of MAD co-morbidity. Moreover, recent reviews have questioned if associations with reduced functioning are only evident at the initial stages of illness[218]. Moreover, it is also unclear to what extent MAD co-morbidity may contribute to the primary focus of this thesis – metacognition. MADs independently present consistent metacognitive deficits[248] and metacognition has also been found to mediate the relationship between depressive symptoms and distress[249,250]. Even before these models, metacognition was conceptualised to capture excessive self-reflection, rumination, and indecision:

> "*Think of the feckless obsessive, paralyzed by incessant critical evaluation of his own judgments and decisions*", pg. 910[141]

What is especially interesting is that while rumination in MDD is suggested to be due to excessive Self-Reflection, delusional behaviours in psychosis are suggested to be due to minimal Self-Reflection, yet both disorders are closely related. Overall, it is unclear what part MAD disorders may play in explaining cognition and functioning and how they may relate to metacognition in psychosis.

Does amotivation explain cognitive deficits and poor outcomes in schizophrenia?

Motivation

Poor motivation in schizophrenia is a long-standing observation. Kraepelin proposed avolition as a primary factor for functional deterioration[251] and Bleuler stated affective indifference was a key marker of the disorder[252]. In the current context, motivation is an internal state that directs and sustains goal-oriented behaviour. Much of the conceptualisation of motivation in psychosis comes from Self-Determination theory[253], which posits there are different types of motivation: intrinsic motivation (inherent reward e.g., eating or socialising), extrinsic motivation (external motivators e.g., financial incentives) and amotivation (lack of behaviour or intention to act). While motivation in schizophrenia can be assessed psychometrically or through cognitive tasks such as delay-discounting[254], it is most commonly assessed by clinician ratings of negative symptoms. There has been a recent surge in motivational research in the last 15 years[47], particularly due to its predictivity of cognition and functioning[255].

As explained earlier, negative symptoms can be categorised into expressive (restricted affect and alogia) and experiential dimensions (avolition, apathy, and asociality)[6], with amotivation relating to the latter experiential domain. There is a lack of consistent definition of motivation in schizophrenia with some considering motivation and negative symptoms identical[254]. Specifically, first-generation clinician measures of motivation (i.e., PANSS and SANS) were created before the five-domain categorisation of negative symptoms, whereas second-generation measures were created afterwards and are more encompassing of amotivation (i.e., CAINS and BNSS). This presents an issue as mediation models using motivation may be redundant and circular. This point was addressed in a recent a meta-analysis reporting that negative symptom assessments and motivation assessments (e.g., PANSS) are only weakly associated (r = -0.18, k = 46)[254] – suggesting that although the two constructs are related, they are distinct.

How may motivation explain cognition and functioning?

Increased levels of amotivation are seen across disorder chronicity[254,256]. While *responses* to rewarding or pleasurable stimuli are largely intact in schizophrenia[47], long-term memory for rewarding/pleasurable experiences, reward learning, prediction error, the representation of value in working memory, and anticipation of future rewards are all disturbed[47]. Overall, anticipatory

pleasure in schizophrenia is significantly impaired (*k* = 21, *g* = -0.42), although to a lesser extent than MDD (*g* = -0.87)[257]. This has been used to suggest patients have intact responsiveness to immediately present rewards, but impaired responsiveness to future rewards, implicating a diminution of anticipatory pleasure that impedes motivated behaviour[255]. Interestingly, while those with MDD also present deficits in consummatory pleasure (experienced pleasure) this may be intact in schizophrenia[255].

In the general population, motivation itself is clearly an extremely important variable for any action a person may perform. In schizophrenia specifically, amotivation has been used to suggest that patients may perform poorly on cognitive tasks not because they *cannot* perform well – but because they are not motivated to do so. Indeed, cognitive deficits are reduced from large to medium effect sizes when considering task-related motivation[258]. This appears to be stronger for post-task rather the pre-task motivation[258] suggesting patients become more de-motivated as tasks progress. This pattern of findings has also been replicated for MAD disorders[259,260] showing further overlap between MAD and psychotic disorders. For outcomes, poor motivation has been found to predict functional outcomes[261,262] independently of psychotic symptoms[263], such as reduced social engagement potentially due to both diminished interest and anxiety[264]. As one review outlines[254], negative symptoms may affect functional outcomes by increasing amotivation ('indifference'), while extrinsic motivation may not be a viable method to improve performance. Indeed, incentives do not enhance cognitive task performance nor does it relate to the increased activation of the dorsal-lateral prefrontal cortex seen in controls[265]; suggesting the issue is more so a failure to recruit cognitive resources rather than a lack of resources. Moreover, motivation may mediate the relationship between both symptoms[266] and cognition with real-world functioning in schizophrenia[267–271]. The size of these effects is also likely underestimated as poor motivation itself may cause a selection bias wherein patients with the lowest motivation are less likely to take part in clinical research.

How is our understanding of motivation in psychosis currently limited?

While there has been a significant increase in motivation research in schizophrenia there has been relatively little investigation of the influence of amotivation on cognitive performance. Moreover, if cognitive deficits are at least partially attributable to poorer motivation, then this finding has yet to be integrated into cognitive models of symptoms. This would have significant implications for current cognitive assessments in psychosis which currently involve large batteries of cognitive tasks (e.g., MCTB) that require significant time and attention from participants to be accurate. The tasks

themselves can involve monotonous, uninteresting, or (quite bluntly) boring tasks which may exacerbate this relationship. Indeed, although neurocognitive deficits are suggested to partially underlie negative symptoms, the causality may be the reverse (or more likely reciprocal). Moreover, our understanding of how MAD co-morbidity may influence motivation in psychosis is unclear[213].

Critically, metacognition (the primary aim of this thesis) has also been recently implicated in the development of motivational deficits in psychosis. Briefly, poorer synthetic metacognition has been associated with amotivation both cross-sectionally[214,272] and at 6-month follow-up, even independent of baseline motivation, anticipatory pleasure, and medication[213]. It is suggested that moderate levels of metacognition are necessary (but not sufficient) for high levels of motivation[273]. The integrated sense of self resultant from synthetic metacognition may influence goal-orientated behaviour because it supports the view of the self as a unique agent capable of initiating action[213]. Overall, a better understanding of how motivation may explain cognitive deficits, functioning, and competence performance gap[47,75,258] as well as how it may interact with MAD co-morbidity and metacognition is currently needed.

Do clinical confounds explain cognitive deficits and poor functioning in schizophrenia?

Confounds of patient status

The final factor investigated in this thesis is the potential clinical confounds of patient status unrelated to the core pathology of schizophrenia. A primary example is the longstanding criticism of anti-psychotic medication exacerbating cognitive deficits[99]. Common examples include parkinsonian motor-slowing and sedative side-effects worsening psychomotor slowness[274,275] and fatigue or confusion exacerbating poor concentration and decreased memory and attention[274,276–278]. This is immediately relevant to cognitive testing, as these side effects may artificially inflate poor performance (e.g., involuntary movements may be misinterpreted as poorer inhibition). Another common factor is institutionalisation, although this may be more accurately described as social exclusion considering more modern therapeutic approaches. Social exclusion, perhaps due to chronic inpatient status or mental health stigma, limits the ability of people to practice social skills, find fulfilling occupational work, and develop supportive relationships. Moreover, early cognitive decline may lead to social exclusion[279] perhaps again representing a reciprocal process. Mental health stigma is also relevant to how patients internalise this stigma, perhaps manifesting in poorer cognitive performance through defeatist beliefs (e.g., through the expectation of them to perform poorly) or a reduced tendency to transfer ability into functioning in the real-world[280]. These factors are critically important to understand when attempting to improve outcomes, however, in the context of understanding the pathogenesis of schizophrenia, they may also obscure and confound research.

Using schizotypy to remove the influence of clinical confounds

One approach to potentially circumvent these confounds is to assess individuals varying in psychometrically defined schizotypy. The "fully dimensional" conceptualisation of schizotypy suggests schizophrenia is not a truly categorical phenomenon (i.e., completely present, or completely absent). Rather, psychotic-like traits ('schizotypal traits') exist on a spectrum varying in intensity in the entire population. This viewpoint conceptualises schizotypy as a latent personality organisation that reflects "tendencies to behave and think in ways that are qualitatively similar to features seen in schizophrenia", pg. 454[281]). Around 5% of the general population experience psychotic experiences at some point, but the vast majority subside[282]. Although considered non-harmful and non-diagnostic, schizotypy traits are thought to represent a liability for developing

schizophrenia[283]. Other approaches also consider related disorders such as Schizotypal Personality Disorder (absence of reality distortion symptoms) under this umbrella. However, as the application of schizotypy in this thesis is to mitigate clinical confounds this approach is not applied here. This thesis does, however, consider more broad terms such as Psychotic-Like Experiences (PLEs) and social anhedonia under this umbrella, which is consistent with current perspectives (for a review, see[284]). Across approaches and definitions, schizotypy is considered pathologically neutral until combined with other risk factors[284]. Indeed, schizotypy represents a multi-finality when trying to explain the transition to psychosis[285], meaning schizotypy can not only relate to negative outcomes but also beneficial outcomes including artistic creativity, problem-solving, mathematical/scientific creativity, music, and painting[286,287].

Assessing schizotypy traits

Schizotypy personality traits are commonly assessed using self-report psychometric assessments that often assess psychosis-like behaviours according to diagnostic criteria[288]. For example, the Schizotypal Personality Questionnaire (SPQ) derives its items from the DSM-III-R criteria for Schizotypal Personality Disorder[289], while the Oxford-Liverpool Index of Feelings and Experiences (O-LIFE) partially derives its items from DSM-II criteria for schizophrenia[290]. Specifically, the SPQ models its constricted affect scale on the blunted affect symptom criteria of clinical schizophrenia. More broadly, schizotypy scales may ask questions such as “do you believe your accidents are caused by mysterious forces” (positive), " Do you feel that making new friends isn't worth the energy it takes?” (negative), or “Do you feel so good at controlling others that it sometimes scares you?” (disorganised). Factor analysis studies of psychometric schizotypy consistently replicate a positive, negative, and disorganised factor structure that maps, respectively, onto reality distortion, negative symptoms, and disorganised symptoms structure of schizophrenia, potentially supporting a psychosis-spectrum. Studies also suggest these traits are normally distributed in the general population[291], with the majority of people reporting at least one psychotic-like experience[284].

Schizotypy research either correlates these traits with outcomes of interest (‘continuous’ approach) or divides a sample into ‘high’ vs ‘low’ schizotypy (‘categorical’ approach). Categorical approaches commonly use either a median-split method[292] or compare individuals with extreme scores from a large pre-screening of participants to those low in schizotypy (e.g., > 95th vs 50th percentile). While the between-subjects approach is more digestible it has several limitations. Firstly, applications are inconsistent in terms of what dimension participants are split on or whether they are split by multiple dimensions; meaning comparison across studies is difficult. Secondly, because of the high intercorrelation between dimensions[293] splitting participants could obscure findings that are in fact due to association with another dimension. Consequently, it is unclear which cognitive

mechanisms would lead to these associations[284]. Moreover, there is a growing research base that suggest that schizotypy dimensions may not be directly related and thus splitting participants based on one domain may not represent schizotypy as a whole[294]. As a result, the continuous approach is applied in this thesis.

Application

The relevance of schizotypy research to diagnostic and treatment options is that inferences about behaviour in clinical patients can be made in the absence of clinical confounds[288]. In this case, schizotypy is used as a proxy to further assess genetic, etiological, cognitive, and functional divergences in clinical patents. Experimentally, if both schizotypy symptom traits in healthy controls and clinical symptoms in patients predict (for example) cognition, this would suggest this relationship is independent of confounds of patient status[284]. Findings from research can also be used to inform protocols for clinical studies that have a high probability of returning applicable findings and potentially saving resources (i.e., non-clinical 'pilot' studies). Assessments in psychometric schizotypy also have several other benefits. Firstly, assessments are resource-efficient relative to clinical samples; although this must be balanced with the smaller effect sizes expected according to the dimensional view (i.e., reduced deficits due to reduced 'symptom' intensity). These larger samples also mean moderation and mediation analyses are more likely to be adequately powered, however. Secondly, the recruitment of schizotypy samples also has the benefit of commonly being near the critical age of psychosis presentation[295] due to the reliance on undergraduate samples. Consequently, research is conducted in a critical period of change where interventions would be most effectively applied. Moreover, due to the higher rates of transition to psychosis and reduced functional outcomes in schizotypy[296], the construct itself is important to investigate as a potential screening tool for early interventions[297,298] and improve wellbeing.

However, a key consideration to caution inferences is that the disorganised scale of schizotypy is potentially more dissociated from the disorganised symptoms of schizophrenia, relative to the relationships between positive and negative traits to positive and negative symptoms[299]. Specifically, while clinical hallucinations may more clearly be reflected in schizotypy (e.g., vivid visualisations or a strong inner voice), the disorganisation of clinical patients does not always clearly extend to schizotypy. Disorganisation in schizotypy and schizophrenia is a complex and multifaceted phenomenon, characterized by a range of symptoms such as thought disorder, bizarre behaviour, and inappropriate affect. However, our understanding of disorganisation and its implications is still evolving. Indeed, there is disagreement on the measurement of disorganisation, with scales such as

the O-LIFE having items that are potentially more reflective of social anxiety[300]. More recent measures have sought to overcome this through the creation of novel measures, such as the Multidimensional Schizotypy Scale (MSS)[301], although the adoption of these scales is in its early stages. Consequently, it is important to caution the application of findings directly to schizophrenia, especially without careful consideration.

Validity

The proposed benefit of schizotypy in the current context is that research can be conducted in these low-intensity 'symptoms' and findings can be indirectly applied to psychosis in the absence of clinical confounds. Indeed, there is strong support for the utility of this approach. Firstly, as mentioned above, schizotypy traits follow the same latent structure as clinical traits and each domain are associated with their counterpart[302]; suggesting both tap into a similar latent construct. Each schizotypy dimension has also been relatively consistently associated with each respective schizophrenia symptoms cluster[303]. Secondly, schizotypy traits are relatively stable over time[285] and produce good test-re-test reliability[304]; suggesting they are a relatively enduring trait (i.e., personality) rather than a highly fluctuating state. In addition to individuals with stable schizotypy, a subgroup of people have been found to have increasing schizotypy longitudinally[285] which may reflect those transitioning to psychosis. Thirdly, schizotypy traits are a potential marker for the transition to psychosis[298]. Estimates have suggested 12–45% of participants in some longitudinal studies follow this path[305], which also supports the potential implication of environmental factors in combination with genetic pre-disposition being necessary to produce clinical psychosis. Fourthly, schizotypy is associated with similar biological abnormalities to schizophrenia including neurological differences (e.g., soft signs, structural differences, brain region activation levels, smooth eye pursuit movements), genetic risk overlap, neurochemical imbalances (e.g., dopamine), and environmental risk factors (e.g., trauma, cannabis use, birth complications)(for reviews see[284,306,307]). Finally, similarities also extend to behavioural deficits in associative learning[66,67], attention to irrelevant information[68,308], olfactory abilities[309], cognitive deficits, and physiological correlates according to reviews on genetic risk factors and neuroimaging studies (for reviews, see [307,310]).

However, while this approach has been applied to a wide array of neurocognitive and social cognitive tasks, it is currently unclear to what extent cognitive deficits are found in schizotypy[311] - meaning it is unclear whether this literature supports that cognitive deficits in clinical psychosis may be observed in the absence of clinical confounds. Moreover, while MAD co-morbidity has been

investigated in schizotypy (e.g., subclinical negative affect) there is little investigation of potential amotivation and metacognitive disruptions in schizotypy. This latter point is particularly critical, as if metacognition impairments may predict transition to clinical psychosis they would not be expected to be replicated in sub-clinical schizotypy.

Limitations

While schizotypy can be a useful research tool, it is crucial to acknowledge the inherent limitations and ongoing debates within this field of study. One such debate revolves around the existence of schizotypy in the general population versus its presence in a specific subgroup of individuals (the quasi-dimensional approach)[312]. Moreover, the application of schizotypy research to schizophrenia is inferential, necessitating a cautious interpretation of findings. It is essential to avoid overstating results and to recognize the importance of replicating these findings in clinical samples for validation. Another point of consideration is that not replicating deficits found in schizophrenia in schizotypy could be attributed to several factors beyond the mere removal of clinical confounds. Specifically, this could highlight a greater dissociation between schizophrenia and schizotypy than expected, or it could simply be a Type II error. On the other hand, these discrepancies could highlight mechanisms that deteriorate during the transition to psychosis, providing valuable insights for future research. The challenge lies in discerning which of these scenarios may be at play in each case. While schizotypy offers a valuable lens through which to study potential schizophrenia-like symptoms, it is crucial to approach this research with a nuanced understanding of its limitations and the complexities of the construct itself.

Summary

This literature review first outlined what schizophrenia is, what the typical trajectory post-diagnosis may encompass, and why schizophrenia is important to research. Next, it was summarised how research into both neurocognition and social cognition was conducted in an attempt to explain symptoms and improve functioning in patients. However, neurocognition and social cognition were described to both explain only 9.4% of the variance in outcomes, with interventions based on these principles presenting only moderate improvements in functioning. The current thesis aims to therefore contribute to reducing the 90.6% unexplained variance in outcomes through the investigation of four factors. The primary factor of this thesis is poorer metacognitive abilities in psychosis patients. The secondary factors include Mood and Anxiety disorder (MAD) co-morbidity, reduced motivation, and confounds of clinical status. These factors were chosen both for their independent influences on cognition and functioning and their potential interactions. This thesis will address these factors by considering the influence of metacognition, negative affect, and amotivation between psychometric schizotypy traits and cognitive performance in neurotypicals.

Chapter 2

Explaining neurocognitive performance in schizotypy.

The influence of metacognition, negative affect, and motivation on neurocognitive task performance in psychometric schizotypy

Introduction: The first experimental Chapter of this thesis aimed to assess the influence of metacognition, negative affect, and motivation on neurocognition (see Chapter 1 for details).

Methods: Two neurocognitive tasks were chosen: Probabilistic Reversal Learning and attentional set-shifting which represent a broad and specific measure of neurocognition, respectively. The influence of metacognition, negative affect, and amotivation were investigated by assessing these traits psychometrically and controlling for their influence. People varying in psychometric schizotypy were also assessed to mitigate the effects of clinical confounds (N = 128).

Results: Positive schizotypy ('hallucinations and delusions') was associated with poorer learning across both tasks, with evidence for a specific deficit concerning punishing stimuli. However, none of the potential mediators explained this relationship, but several methodological limitations of both the tasks and psychometric assessments meant this finding was not conclusive.

Discussion: Consequently, three further experiments were designed to investigate this further in subsequent Chapters. The first follow-up experiment adapted both neurocognitive tasks to include behavioural measures of metacognition, the second extended this adaptation to social cognition, and the third adapted the Probabilistic Reversal Learning task into an immersive Virtual Reality task to assess the influence of motivation.

The influence of metacognition, negative affect, and motivation on neurocognitive task performance in psychometric schizotypy

1.0: Introduction

1.1: Summary of aims

In Chapter 1, it was explained how the association between schizophrenia, cognition, and functional outcomes may be affected by confounds of negative affect, metacognition, motivation, and clinical confounds. This Chapter aimed to assess the influence of these mediators on neurocognitive performance using a sample varying in psychometric schizotypy. The first step of this investigation was to choose which cognitive domains to assess. However, as the questionnaire portion of this task was relatively long, a cognitive battery of multiple tasks would not have been practical. As a result, a task was chosen that measures neurocognition more broadly (Probabilistic Reversal Learning, PRL) and a well-researched domain that consistently produces large deficits in patients (set-shifting).

1.2: Probabilistic Reversal Learning

1.2.1: Description

Probabilistic Reversal Learning (PRL) tasks are a type of neurocognitive assessment that vary in implementation. However, in all designs participants are presented with one of two cues on each trial. One cue is more frequently (i.e., probabilistically) associated with reward while the other is more frequently associated with punishment. The aim of the task is for participants to learn these associations and to correctly predict which outcome will follow each cue. After a certain experimental condition is met (e.g., 60 trials) the relationships between the cues and reward/punishment are reversed and participants must adapt accordingly. These represent the two stages of the task: the initial learning stage and the reversal stage. PRL tasks assess visual learning, inhibition, and working memory ability to both positive (rewarding) and negative (punishing) stimuli. Each element of the PRL task also requires different but related cognitive processes for successful completion: the initial learning stage requires cumulative learning, the reversal stage requires the detection of error change and inhibiting responses to reinforced contingencies[313], and the probabilistic element requires participants to detect and ignore probabilistic errors within blocks, have confidence in estimated regularities[314], and understand the most effective strategy to capitalise upon the regularities. Overall, PRL tasks are broad assessments of neurocognition that allow some of these processes to be assessed separately.

For this Chapter specifically, PRL tasks are highly relevant due to their potential links to metacognition and functional outcomes. For outcomes, reinforcement learning facilitates an

understanding of the predictors of environmental outcomes to which we can adapt our behaviour, such as learning that some foods will make us sick ("taste aversion"[315]), or that specific social behaviours lead to social praise. Conversely, being less able to learn and re-learn outdated associations may present fixedness in responses. Indeed, PRL performance is associated with psychosocial functioning[316] which may be due to being less able to capitalise on environmental regularities[314]. Moreover, reversal learning represents adapting flexibly to changes in the environment. For example, a previously pleasant food may suddenly cause an allergic reaction, requiring us to unlearn the previously harmless association and adjust to the new harmful association. In terms of metacognition, PRL tasks have strong theoretical connections to the Becks Cognitive Insight Scale (BCIS). Specifically, the Self-Certainty subscale (overconfidence) may be expected to positively correlate with pre-reversal performance, but negatively correlate with performance after reversal. Moreover, higher Self-Reflectivity may improve reversal stage performance due to increased openness to fallibility in the face of contradictory evidence. It should be noted, however, that the PRL task is not considered a metacognitive task specifically and no investigation has currently associated psychometric metacognition with PRL performance.

1.2.2: PRL performance in Schizophrenia

The majority of PRL investigations in schizophrenia have reported deficits in both the initial learning and reversal stages[314,317,318,319–321], although some researchers have found intact initial learning[322]. A similar pattern of results is also observed in similar non-probabilistic reinforcement learning paradigms, suggesting patients learn less from rewarding[318,323]and punitive cues[316] relative to healthy controls. Deficits have also been found to be mitigated with sufficient trials[324] suggesting that rapid trial-by-trial learning is specifically disrupted. Although, again, other authors have failed to replicate these findings[325]. Indeed, the reasons for PRL task impairments have remained elusive, but theories point to impairments in representing the values of cues - supported through correlations with working memory[72,324,325], reduced flexible control[326], and both reduced decision confidence and updating confidence with feedback[314]. In terms of associations with symptoms, the initial learning phase has been associated with positive and negative symptoms[327,328] and the reversal stages with negative symptoms[317,322], but not all studies replicate these associations[317]. Overall, while PRL deficits are often found in patients, their specific nature and associations with symptoms remain unclear. Part of this heterogeneity has been attributed to poor statistical power and large variations between PRL task designs[321]. Specifically, PRL tasks vary in the number of reversals, trials, probabilistic reinforcement levels (e.g., 60% vs 80%), and the details of the task instructions[324]. Critically, such heterogeneity may also be influenced by negative affect, metacognitive ability, motivation, and confounds of clinical status which may have individual influences on performance[329].

1.2.3: PRL performance and Schizotypy

Only two grey literature abstracts have assessed PRL performance in schizotypy, for only limited details are available[330,331]. Consequently, the influence of clinical confounds from this perspective is unclear. As a result, the first aim of this study was to assess whether PRL performance deficits seen in clinical psychosis are replicated in psychometric schizotypy.

1.3: Set-shifting

1.3.1: Description

The second cognitive task aimed to measure a specific domain of neurocognition is attentional set-shifting. Set-shifting is a domain of cognitive flexibility/Executive Function that concerns shifting attention from one stimulus dimension to another (e.g., from a pink square to a white line). In the psychosis literature, three main tasks are used to assess set-shifting: the Trail Making Task (TMT), Wisconsin Card Sorting Task (WCST) and Intra/Extra-dimensional Set-Shifting task (IDED). The TMT is a two-stage task which involves connecting dots on a piece of paper with a pen. In stage A (TMT-A) the time taken to connect consecutively numbered dots is recorded (i.e., 1-2-3-4). In stage B (TMT-B) participants must connect dots that alternate in either numbers or letters in ascending order (i.e., 1-B-3-D-5). In this task, attention must shift between the letter and number dimensions and the completion time of TMT-B is taken as the outcome variable. The WCST presents participants with playing cards containing images differing in shape, colour, and quantity of shapes. Participants are tasked with correctly categorising newly presented cards on one of these dimensions chosen by the researcher (through trial and error). After a while (e.g., 10 sorts), the rule governing the correct response is changed and attention must shift from one dimension to another. Commonly, the number of sort errors is analysed. Finally Intra-Dimensional/Extra-Dimensional (IDED) set-shifting tasks[332] ask participants to select the 'correct' stimuli of two visual options across nine stages. In each stage, participants must discern through trial and error which of the presented shapes is 'correct' (arbitrarily chosen by the researcher). In the first seven stages, different pink shapes are reinforced as correct, until stage eight in which the target shifts to a different dimension of a white line. The number of errors in this Extra-Dimensional Shift (EDS) stage is the primary outcome.

1.3.2: Set-shifting performance in Schizophrenia

Set-shifting deficits in clinical patients are relatively consistent across tasks. For the TMT, a recent meta-analysis found all studies reported deficits at large effect sizes for both the TMT-A (SMD = -0.89) and TMT-B (SMD = -0.96[333]). These deficits appear to also be independent of IQ suggesting they are not a generalised, but specific deficit[334–340] (but see[341,342]). However, the TMT is confounded by other neurocognitive processes such as visual search, working memory, and

psychomotor/processing speed[343,344]. These latter two confounds are significant issues considering that psychomotor slowness can be exacerbated by antipsychotic medication. The WCST is perhaps the most commonly used set-shifting assessment in psychosis with deficits being a consistent finding[345–347]. Importantly, the WCST is not confounded by processing speed meaning the task gives a clearer assessment of set-shifting.

Arguably, the most relevant task to assess set-shifting is the IDED. Not only is the IDED unaffected by psychomotor speed, but it has the benefit of assessing other neurocognitive domains as part of the task structure including simple discrimination learning (visual learning), reversal learning, ignoring distractors, and rule abstraction. These metrics can subsequently be added as control variables when assessing set-shifting performance - which is not possible with the TMT or WCST. Studies using the IDED have reported that those with psychosis are more likely to fail (> 25 errors) the Extra-Dimensional Shift stage[334,340,342,348–351] and make more EDS errors[334,335,338,348,350–353]. These performance deficits are similar in size between both early onset[354] and late-onset psychosis[349] but are greater relative to bipolar disorder[355]. It is, however, unclear whether set-shifting deficits grow greater over time[340,356336,340,350,356–358], remain stable[341,346,348,359–369], or perhaps even improve[370,371]. Deficits in control participants are associated with cortical thinning[353] suggesting the prefrontal cortex is heavily involved in set-shifting. The most common explanation for deficits is perseverative responses: in which responding is sustained to the previously learned attentional set. This explanation may also highlight associations to functional outcomes through difficulties in transferring to new living situations, experiences[356], or generalising rules[340].

In terms of associations to symptoms, the most common relationship is between performance and negative symptoms[340,350,372] although this is inconsistent[336,354,358,365]. More consistent is the lack of association to positive symptoms[336,340,348,354,365,372]. Many studies do not consider the impact of disorganised symptoms[335,338,342,357], but those that have assessed disorganisation[354] and the related disorganised behaviour syndrome[356] and bradyphrenia[340] have found task deficits[336,358,372].

1.3.3: Set-shifting and Schizotypy

There have been limited investigations of set-shifting performance in schizotypy with the literature focusing exclusively on the TMT and WCST. A recent meta-analysis reported small deficits in both tasks[311] which may be associated with positive (deficits reported in[373–377], null findings[378]), negative (deficits reported in[373–375,379–382], null findings[378]), and total schizotypy (deficits reported in[383,384], null findings[385]), but potentially not disorganised schizotypy[378]. To the best of the writer's knowledge, only one study has used the IDED in schizotypy. This study found a trend level increase in EDS errors

for 6 to 12-year-old children with Schizotypy Personality Disorder (p = .068, d = .88)[386]. Consequently, the influence of clinical confounds from this perspective is unclear.

1.4: Aims and hypotheses

Overall, this study aims to replicate neurocognitive deficits in psychometric schizotypy using broad (PRL) and specific (IDED) neurocognitive tasks. Such a replication would suggest deficits in clinical patients may be independent of clinical confounds. Furthermore, this study also aimed to assess the roles of negative affect, metacognition, and motivation may play in explaining deficits by controlling for these factors psychometrically. The potential influences of these factors are described in Chapter 1, but to re-iterate: clinical negative affect has its own independent associations to poor cognition, cognitive tests may also be measuring poor motivation in clinical patients and not just cognition, and metacognition is critical for learning and the use of knowledge. These results may then be used to guide the understanding of cognitive deficits in clinical patients and subsequently the association between cognition and functional outcomes. It was hypothesised that:

1) Schizotypy would predict poorer performance on the Probabilistic Reversal Learning task measured by the percentage of correct responses. This poorer performance will be partially mediated by a) increased negative affect, b) poorer metacognition, and c) reduced motivation.
2) Schizotypy would predict poorer set-shifting performance in the Intra/Extra-dimensional set-shifting task as measured by errors in the Extra-Dimensional Shift stage. This poorer performance will be partially mediated by a) increased negative affect, b) poorer metacognition, and c) reduced motivation.

2.0: Methods

2.1: Participants

From an initial sample of 133 participants five were excluded (see **2.5: Data preparation**). The final sample consisted of 128 participants recruited through word of mouth in the UK. The sample was aged between 18 and 25 years old (*M* = 21.8*, SD* = 2.1) and had a relatively even biological sex split (54.6% female). The only exclusion criterion was a current diagnosis of schizophrenia or the use of antipsychotic medication. This study was approved by the University of Nottingham School of Psychology Ethics Committee, following the British Psychological Society's Code of Ethics and Conduct.

2.2: Materials and Apparatus

Schizotypy was assessed using the Oxford Liverpool Inventory of Feelings and Experiences (O-LIFE)[293] which is a 104-item dichotomous (yes/no) psychometric assessment. The O-LIFE consists of four sub-scales of which the first three map onto the same multi-dimensional structure as schizophrenia: assessing positive, negative, and disorganised schizotypy traits. These personality clusters are represented by unusual experiences ("*Do you believe in telepathy*"), introvertive anhedonia ("*Do you feel that making new friends isn't worth the energy it takes?")* and cognitive disorganisation ("*Are you easily distracted when you read or talk to someone?*"), respectively. The fourth subscale of impulsive non-conformity ("Do people who drive slow annoy you?") is not appraised due to it being considered non-central to schizotypy[290]. For consistency across chapters and other published work, the terms positive, negative, and disorganised schizotypy will be used rather than the O-LIFE subscale names. The O-LIFE was chosen due to its high internal consistency (α = 0.77 – 0.89), test-retest reliability over three months (*r* > .80)[387], and its prevalence in cognitive psychology enabling more direct comparisons. Negative affect was measured using the total score of the 21-item Depression, Anxiety, and Stress Scale (DASS-21) and Moderately Discrete Metacognition was assessed using the Beck's Cognitive Insight Scale (BCIS)[162]. As a reminder, the BCIS contains two subscales of Self-Reflectivity (monitoring one's thoughts and openness to fallibility) and Self-Certainty (pathological overconfidence). Finally, motivation was assessed using the Momentary Influences, Attitudes and Motivation Impact (MIAMI) for cognitive test questionnaire[258] which is given both pre-and post-task. The MIAMI and BCIS can be found in **Appendix A.** and **Appendix B.**, respectively. The PRL task, set-shifting task, and O-LIFE were administered using PsychoPy[388] using a 17-inch LCD laptop (60hz, 1366 x 768) whereas the remaining scales were pen and paper formatted.

2.3: Experimental Task Design

2.3.1: Probabilistic Reversal Learning Task

The aim of the PRL task was to gain as much virtual money as possible. At each trial, one of two cues were presented that were probabilistically reinforced with either the loss or gain of money. Initially, one cue was more commonly (i.e., "probabilistically") paired with reward (gaining money) and the other commonly paired with punishment (losing money). Participants respond by either betting 10p or £1 at each trial. To begin, participants do not know the contingencies and must randomly guess. However, with feedback after every trial (i.e., the loss or gain of money), participants should learn the contingencies and then use this information to make more accurate betting decisions. After every block of trials, these contingencies were reversed without warning to further increase task difficulty and to assess reversal learning. The optimal response pattern for the task is to bet £1 on the contingency related to reward on *every* trial (100%) and 10p on the contingency related to losing on *every* trial.

For this implementation, the two cues were two characters of the Agathodaimon font with no intrinsic meaning. Contingencies were reinforced in 80% of trials and therefore 20% of trials violated this contingency. Four blocks of 60 trials were used meaning there were three reversals. Participants began the task with £20 and this score was displayed throughout the task. Each trial started with a fixation cross for 200ms followed by a blank screen for 500ms. Next, one of the two cues was presented in the centre of the screen for 500ms. The cue was then replaced by a question mark which was displayed until a response was given. Participants used the "0" key to indicate a 10p response and the "1" key to indicate £1. After a response, feedback was given for 1000ms. This involved removing all other visual stimuli to only present "+ £1" or "+10p" for winning and "-£1" or "-10p" for losing, depending on response accuracy. Sound effects were also played that corresponded to the loss or gain of money. Reminders of response keys were presented at the response stage and the total score was updated at the start of the next trial. The task contained three equally spaced breaks in which participants pressed the spacebar to continue. One critical adaptation was that each bin of 20 trials within each block was pseudo-randomised to contain the 80% reinforcement levels; meaning all participants received the same trial order. A summary of the task structure can be found in **Figure 6**.

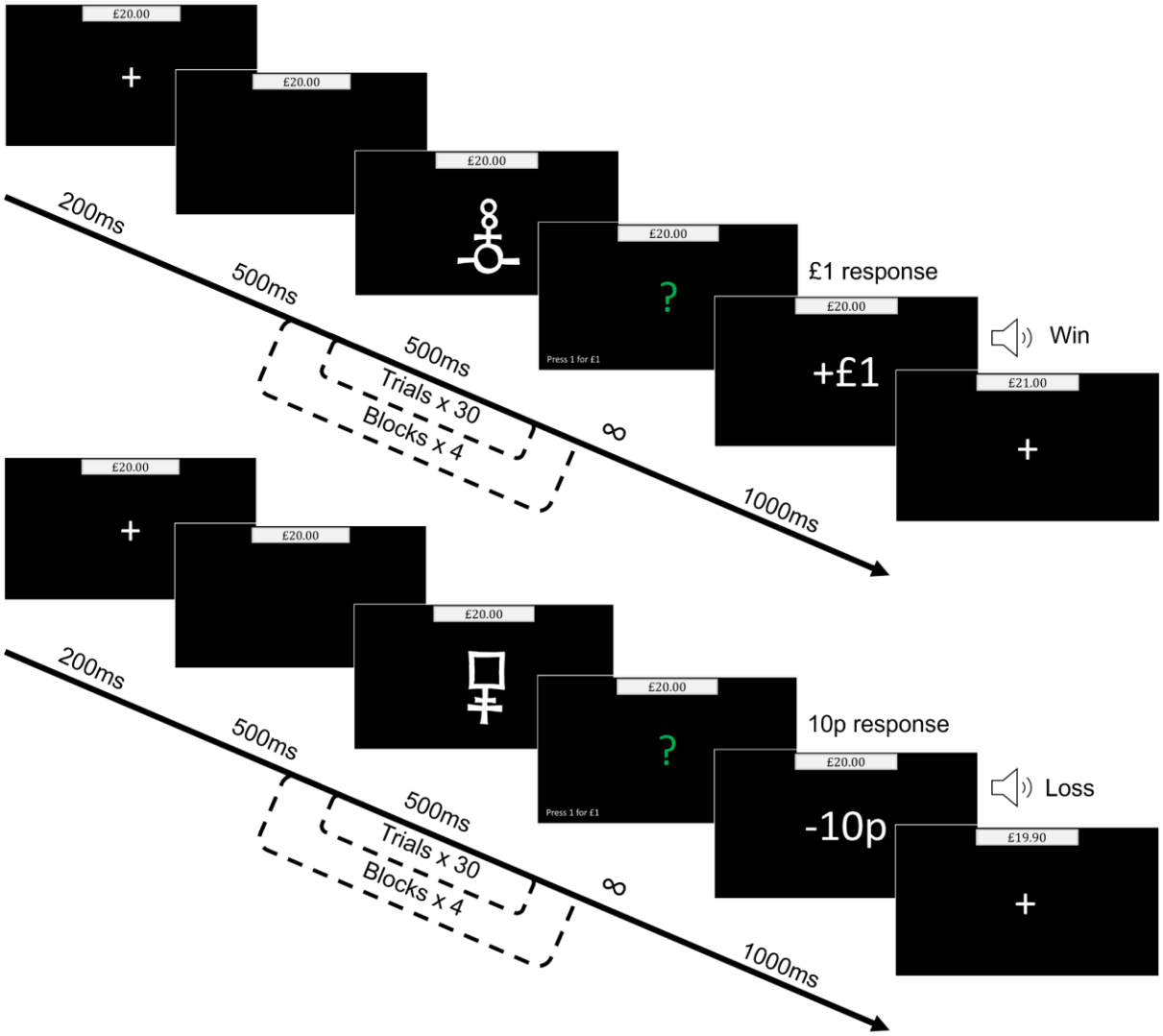

Figure 6. Trial structure of the Probabilistic Reversal Learning (PRL) task. Participants responded by betting either £1 or 10p at each trial. A total of 240 trials were presented (120 of each contingency). **Top**: rewarding trial example; **Bottom**: punishing trial example.

2.3.2: Set-Shifting Task

The IDED contains nine different experimental stages (see **Figure 8**). At each stage one of the presented shapes predicts 'winning' and the others predict 'losing'. The shapes themselves have no intrinsic meaning and these relationships are arbitrary. Initially, participants must guess at each stage and use feedback on their responses to learn the relationships. The first stage is the Simple Discrimination stage in which one of the two pink shapes is correct (SD, Stage 1). After five consecutive correct responses, the task proceeds to the Simple Discrimination Reversal stage in which the correct stimulus shifts to the other pink shape that was previously irrelevant (SDR, Stage 2). After another five consecutive correct responses the correct response remains the same, but distractor stimuli of a different dimension (white lines) are added (Compound Discrimination; C_D, Stage 3). In the next stage, the distractor white lines are superimposed over the same pink target shape (Compound Discrimination Superimposed; CDS, Stage 4) and the stage after reverses the relationship again back to the original pink shape (Compound Discrimination Reversal; CDR, Stage 5). The next stage introduces novel stimuli of both dimensions, but the correct dimension remains one of the two new pink shapes (Intra-Dimensional Shift; IDS, Stage 6) and then reverses to the other pink shape in the next stage (Intra-Dimensional Reversal; IDR, Stage 7). The critical stage is Stage 8 when the correct response shifts dimension to one of the white lines (Extra-Dimensional Shift; EDS). This then reverses again within the same dimension of white lines in the final stage (Extra-Dimensional Reversal, Stage 9). The EDS stage assesses attentional set-shifting, the IDS stage assesses rule-abstraction as previously learned rule is applied to new stimuli in the same dimension[340], the SD stage measures simple reinforcement learning, the reversal stages all assess reversal learning and inhibition, the C_D stage evaluates the impact of distractors, and the CDS stage also measures visual discrimination.

In this implementation, each trial began with a fixation cross in the middle of the screen along with four white borders at the top, right, left, and bottom of the screen. After 500ms the stimuli were presented within two of the four borders. The location was randomised to avoid response bias but is irrelevant to the task itself. Participants responded with the arrow keys to point to the shape they felt was correct with no response time limit imposed. All four borders would fill with a green or red colour if the response was correct or incorrect, respectively. Sound effects were also played that corresponded to correct or incorrect responses. Stage transitions were unannounced to participants. If participants did not complete a stage within 25 trials, then the task would skip to the end. A flow diagram of the task can be found in **Figure 7**.

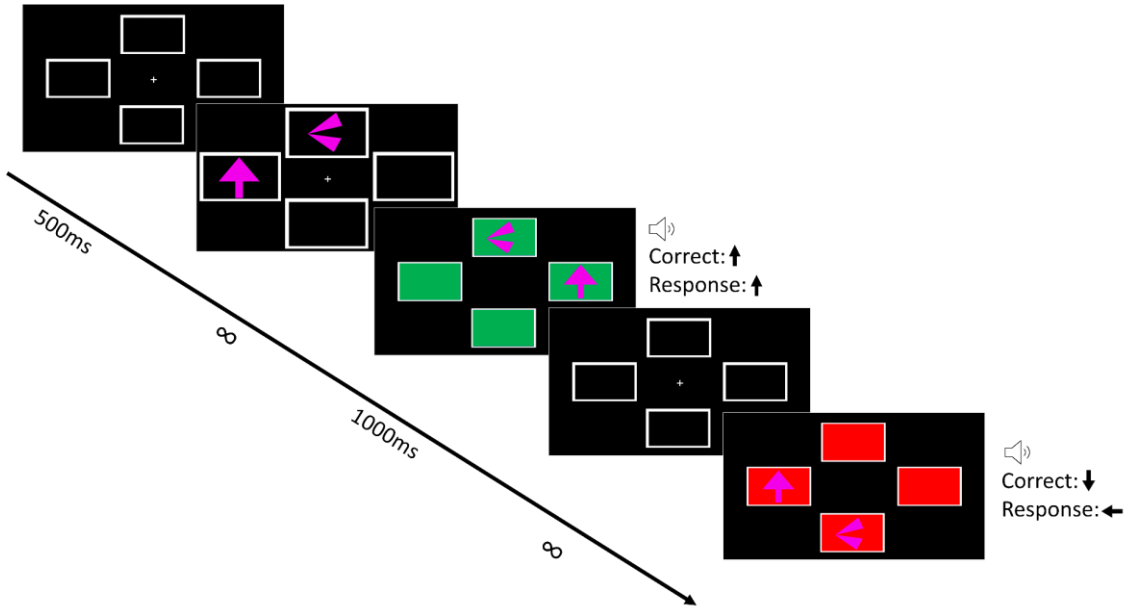

Figure 7. An example of a trial in the simple learning stage of the IDED task. The correct response is the Pac-Man shape and not the arrow. The location of the shapes was randomised to avoid response bias and did not relate to the correct response.

2.4: Procedure

The study began with participants being counterbalanced to receive the psychometric questionnaires or the experimental tasks first. If participants were allocated the questionnaires, they completed them in a randomised order with the experimenter nearby. Participants were told the latent construct measured by each questionnaire and told that they were not obliged to answer any questions. The experimental task section began with the completion of the pre-task motivation assessment, followed by the IDED, PRL and post-task motivation assessment. The start of the IDED began with an instruction screen explaining that different shapes would appear and pre-determined rules controlled which shape related to winning or losing. Participants were then told their task was to discover these relationships by trial and error and to use the arrow keys to select a shape. The PRL instructions explained that the task was a 'virtual betting game' in which participants should try and win as much money as possible. It was explained that at the start of each round one of two different symbols would be presented that could predict winning or losing the current round. Participants could respond with either 10p by pressing the "0" key or £1 by pressing the "1" key. Participants were also told they would receive a higher inconvenience allowance if they scored in the top 10% of previous participants to increase attention. Critically, participants were not told that the cue-outcome relationships may change. The final 44 participants completed a short Task Strategy Questionnaire (TSQ) asking for more general feedback (**Appendix C.**). The study ended with all participants being debriefed and receiving the higher inconvenience allowance of £5.

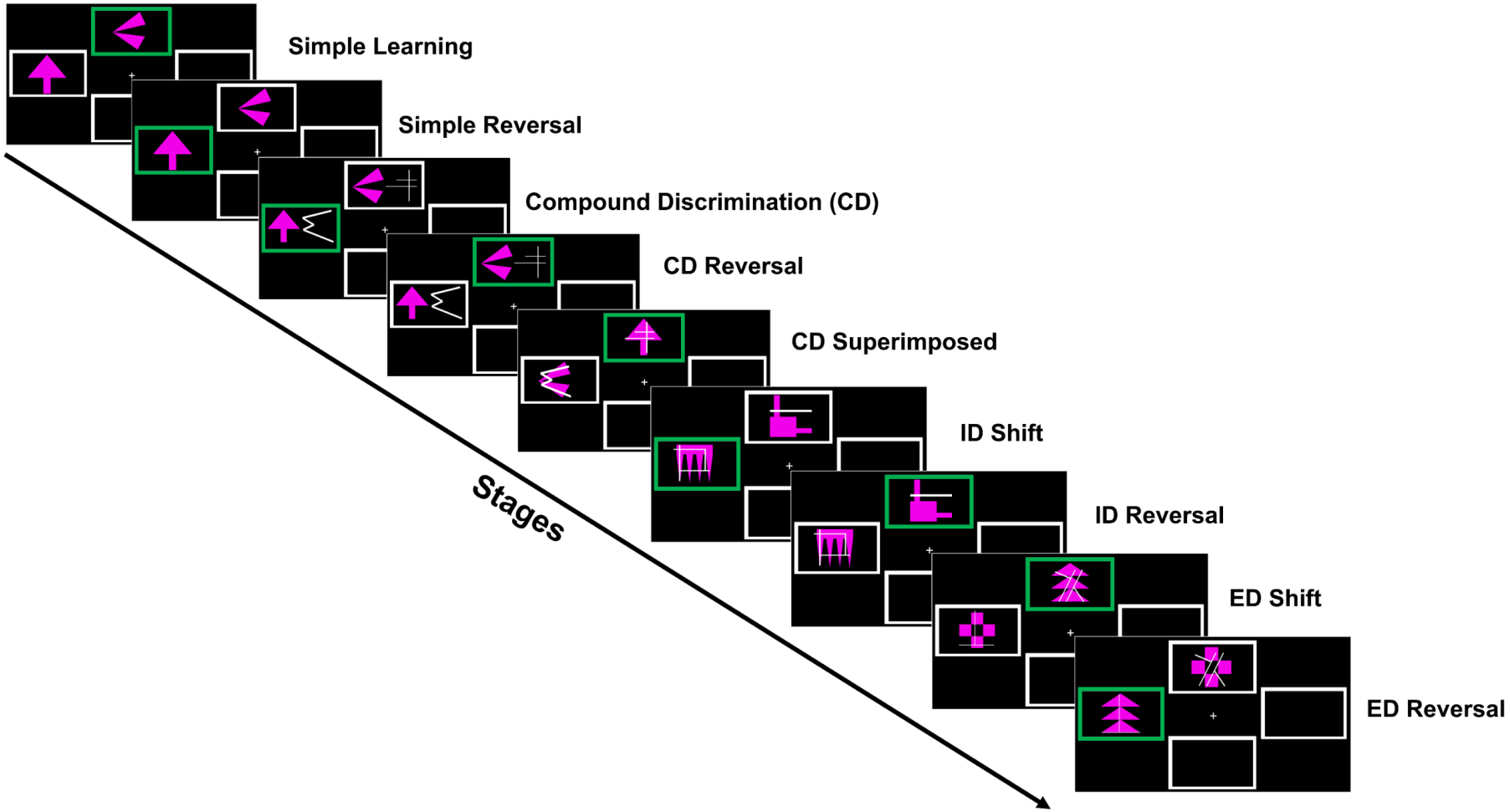

Figure 8. The nine stages of the IDED task. The critical stage is the Extra-Dimensional Shift (EDS) stage where the correct answer (highlighted in green) changes from the dimensions of a pink shape to a white line.

2.5: Data preparation

From the initial sample of 133 participants, five participants were excluded for failing to complete the PRL task. However, due to a coding error, the first eight participant's data for the IDED task was invalid. This meant that the sample sizes for the PRL and IDED tasks were 128 and 120 respectively. Outliers in the data for the tasks were not removed as inattention and low motivation were key aims of this study. Missing questionnaire data due to either participants withholding responses or missing items were imputed using the missForest R package[389]. This method is suitable for both ordinal and non-normal data and does not inflate Type I error rate, unlike mean replacement methods. Missing data are given in (percentage) and imputed error measured by [percentage falsely classified] were as follows: DASS-21 (0.21% missing cases)[13.4% PFC], BCIS (0.31%)[33.8%], pre-task MIAMI (0.23%)[10.2%], and post-task MIAMI (0.31%)[19.8%]. There are currently no agreed-upon cut-off criteria for a 'good' imputation using this method. However, simulation studies have found error rates between 5% and 20% which consistently outperformed other imputation approaches[390].

2.6: Analysis strategy

Validity checks were conducted including internal consistency, normative comparisons, and reasonable distribution of performance scores (i.e., ceiling effects, floor effects, and lack of variability). For the PRL task the primary outcome was the percentage of optimal responses (i.e., betting £1 for the rewarding cue on *every* trial including the 20% of trials that violate the contingency). Analysis of the PRL involved comparing increasingly complex Linear Mixed-Effects (LME) models, which are similar in conceptualisation to repeated-measures ANOVA[iii]. The analysis began firstly with estimating a null model which predicts overall performance from only the random-effect of participant (similar to an intercept alone regression model). Next, this null model is compared to a further model specifying a more complex structure, such as including the fixed-effects of block, contingency (rewarding vs punishing), or schizotypy traits. At each stage, a significant χ^2 test and lower AIC value indicate a superior fitting model. Significant fixed-effects ('main effects') were determined by using the *anova* function from the *car* R package. For the current study, a model specifying an interaction between block and contingency acted as a baseline model. To address Hypothesis 1, models specifying the additional effects of schizotypy and its interaction with Block and Contingency were calculated. As additional validation and to further investigate potential relationships between schizotypy and performance, errors in the SD (visual

[iii]The reason that different statistical tests were used for the PRL and IDED is that Linear Mixed-Effects models are only suitable for repeated-measures data (e.g., the EDS stage of the IDED is a single measurement).

learning), SDR (reversal learning), and EDS (set-shifting) stages of the IDED were also added in further models. To address Hypothesis 2, a Poisson logistic regression predicted the number of errors in the Extra-Dimensional Shift (EDS) stage from the three schizotypy scales[iv]. The effects of negative affect (DASS-21 total score), metacognition (BCIS), and motivation (MIAMI) were then added to both the PRL analysis models (Hypotheses 1a – 1c) and IDED Poisson regression models (Hypotheses 2a – 2c) – only if they correlated with schizotypy. This approach was used to reduce statistical complexity.

Bayes Factors (BF) were calculated where possible to differentiate between data insensitivity and a true null effect[391]. BFs were interpreted as follows for the alternate hypothesis (BF_{10}): $BF_{10} < 0.3$ supports the null hypothesis, BF_{10} between 0.3 and 3 is insensitive to detect effects (more data required), $BF_{10} > 3$ moderate evidence, $BF_{10} > 10$ strong evidence, $BF_{10} > 30$ very strong evidence, and $BF_{10} > 100$ decisive evidence for the alternate hypothesis[392]. In the following analyses, the term "parallel" indicates that a frequentist and Bayesian test were conducted and their results are considered together. Analyses were conducted in R studio[393], Jamovi[394] and JASP[395] using several statistical[396–400] and data visualisation packages[401,402].

[iv] The reason that different statistical tests were used for the PRL and IDED is that Linear Mixed-Effects models are only suitable for repeated-measures data (e.g., the EDS stage of the IDED is a single measurement).

3.0: Results

3.1: Descriptive statistics

Descriptive statistics for all psychometric data and task data can be found in **Table 1** and **Appendix D.**, respectively. In terms of internal consistency, the DASS-21 total score was excellent (ω_T = .92), the three schizotypy and post-task MIAMI were good (ω_T > .8), the pre-task MIAMI was acceptable (ω_T > .7), and both scales of the BCIS were poor (ω_T > .5). All psychometric data were non-normally distributed according to Shapiro-Wilk tests and so non-parametric tests were conducted where appropriate (all p_{FDR} < .031).

3.2: Normative comparisons

Normative comparisons were conducted for both the O-LIFE[293] and DASS-21 (N = 1794[403]) for which normative data were available. As data were non-normal, parallel One-Samples Wilcoxon Signed rank tests were used. For the O-LIFE a subgroup of the normative data was used that more closely matched the age characteristics of the current sample (N = 402). These normative comparisons found that both negative schizotypy (r_{rb} = .535, p_{FDR} = .002, BF_{10} > 999) and potentially disorganised schizotypy (r_{rb} = .222, p_{FDR} = .045, BF_{10} = 0.916) were higher in the current sample, whereas positive schizotypy did not significantly differ (r_{rb} = .005, p_{FDR} = .964, BF_{10} = .122). For the DASS-21, the total score was significantly high in the current sample at a large effect size (r_{rb} = .788, p = .002, BF_{10} > 999). The DASS-21 also provides clinical cut-off scores for responses of normal, mild, moderate, severe, and extremely severe categories. These proportions were: *Depression*: 59.4%, 8.59%, 17.2%, 3.90%, and 11.7%; *Anxiety*: 53.9%, 7.81%, 21.1 %, 3.91%, and 13.28%; *Stress*: 59.4%, 13.3%, 15.6%, 7.03%, and 4.69%. Overall, there were either high or typical levels of psychometric traits in the current study relative to the general population.

3.3: Psychometric relationships

A correlation matrix was created between all psychometric variables to identify potential mediators of PRL and IDED performance (**Table 2**). Spearman Bayesian correlations were used due to the violation of normality and to control for Type I error rate. All schizotypy scales were associated with greater levels of negative affect (r_s > .41, BF_{10} > 999), Self-Reflectivity (r_s > .38, BF_{10} > 999), and reduced pre-task MIAMI scores (r_s < -.20, BF_{10} > 10). Only disorganised schizotypy was associated with lower Self-Certainty (r_s = -.25, BF_{10} > 30). Finally, there was indecisive evidence whether schizotypy was associated with post-task MIAMI scores (r_s < -.19, 0.3 < BF_{10} < 3). Overall, each of the potential mediators was added in upcoming models as each correlated with at least one schizotypy scale.

Table 1 *. Psychometric descriptive statistics and normative comparisons of the current data.*

	Current								Normative							Comparison		
Scale	**Range**	**M**	**SD**	**Med**	**MAD**	**IQR**	**ω_T**	**α**	**N**	**Range**	**M**	**SD**	**Median**	**IQR**	**α**	**r_{rb}**	***p***	**Normality**
Unex	0-24	9.44	6.45	8.5	6.67	9.0	0.87	0.87	402	0-30	10.0	6.305	9	11	-	.005	=.964	< .001
Intan	0-20	6.54	4.61	5.5	4.45	6.0	0.81	0.81	402	0-27	5.44	4.00	4	5	-	.535	=.002	< .001
Cogdis	0-24	14.1	6.18	14.5	8.15	11.0	0.90	0.90	402	0-24	12.4	5.690	13	8	-	.222	=.045	= .002
Impnc	1-21	8.77	4.16	8.77	4.45	5.0	0.70	0.72	402	0-23	9.48	4.11	9	6	-	-.137	=.085	= .004
DASS Total	2-106	31.7	24.1	27.0	22.2	30.5	0.92	0.92	1794	0-122	18.7	19.32	14	-	0.88	.788	=.002	< .001
Depression	0-40	10.1	9.98	6.17	5.93	12.0	0.85	0.85	1794	0-42	5.66	7.74	2	-	0.82	.917	=.002	< .001
Anxiety	0-38	8.73	8.32	6.14	5.93	10.0	0.84	0.83	1794	0-42	3.76	5.90	2	-	0.90	.931	=.002	< .001
Stress	0-40	13.9	9.24	12.2	8.90	12.0	0.85	0.85	1794	0-42	9.46	8.40	8	-	0.93	.640	=.002	< .001
BCIS_R	11-31	19.2	3.95	18.6	4.45	5.25	0.64	0.60	-	-	-	-	-	-	-	-	-	= .002
BCIS_C	9-21	15.5	2.67	15.55	2.97	4.0	0.53	0.52	-	-	-	-	-	-	-	-	-	= .031
MIAMI Pre	49-78	65.6	6.47	66.0	5.93	7.25	0.77	0.75	-	-	-	-	-	-	-	-	-	= .004
MIAMI Post	43-72	62.0	6.41	62.5	6.67	9.0	0.83	0.83	-	-	-	-	-	-	-	-	-	= .002

Note: IQR – interquartile range, MAD = robust median absolute deviation, ω = McDonald's Omega total, r_{rb} = rank-biserial correlation. Comparison tests are Wilcoxon signed-rank tests and normality tests are Shapiro-Wilk tests. All *p* values are FDR corrected. [A] = Values from Mason & Claridge, 2005., [B] Henry & Crawford, 2005.

Table 2. *Bayesian Spearman correlation matrix of psychometric data*

	Schizotypy				**Negative Affect**			**Metacognition**		**Motivation**	
Variable	*Pos*	*Neg*	*Dis*	*Total*	*Depre*	*Anxie*	*Stres*	*S-Refl*	*S-Cert*	*Pre*	*Post*
Positive Schizotypy	—										
Negative Schizotypy	0.25^{**}	—									
Disorganised Schizotypy	0.48^{D}	0.43^{D}	—								
Negative Affect	0.41^{D}	0.45^{D}	0.61^{D}	—							
Depression	0.30^{***}	0.44^{D}	0.57^{D}	0.86^{D}	—						
Anxiety	0.37^{D}	0.27^{***}	0.44^{D}	0.75	0.52^{D}	—					
Stress	0.42^{D}	0.37^{D}	0.51^{D}	0.88^{D}	0.63^{D}	0.58^{D}	—				
Self-Reflectivity	0.42^{D}	0.38^{D}	0.39^{D}	0.36^{D}	0.29^{***}	0.34^{D}	0.36^{D}	—			
Self-Certainty	-0.10^{I}	-0.14^{I}	-0.25^{**}	-0.11^{I}	-0.14^{I}	-0.13^{I}	-0.02^{I}	-0.07^{I}	—		
Pre-task MIAMI	-0.20^{*}	-0.22^{*}	-0.34^{***}	-0.34^{D}	-0.29^{***}	-0.32^{D}	-0.32^{D}	-0.08^{I}	0.20^{*}	—	
Post-task MIAMI	-0.19^{I}	-0.19^{I}	-0.19^{I}	-0.20^{*}	-0.17^{I}	-0.23^{*}	-0.17^{I}	-0.17^{I}	0.23^{*}	0.47^{D}	—

Note: ^{N} = supports the Null, ^{I} = insensitive, * = $BF_{10} > 3$, ** = $BF_{10} > 10$, *** = $BF_{10} > 30$, ^{D} = >100 for 'decisive'.

3.4: Probabilistic Reversal Learning

3.4.1: Task validation

Task performance for each contingency and across each of the four blocks is **Figure 9**. The trends describe greater performance for the punishing contingency relative to the rewarding contingency. This effect is also stronger as the task progresses from Block 1 to Block 2. To support this description, a Linear Mixed Effects (LME) model predicting overall performance from the random-effect of participant alone was calculated (AIC: 8604). This model allows the estimation of the proportion of variance in performance explained by participants' different baseline neurocognitive abilities (i.e., their 'personal intercepts') which can be controlled for when estimating the effects of schizotypy. The random-effect of participant explained 29.0% of the variance in performance, suggesting substantial individual differences in baseline neurocognitive ability. Next, this model was compared to a further model adding both block and contingency as fixed-effects. This second model was a significantly better fit to the data ($\chi^2(4) = 37.4$, $p < .001$, AIC: 8574). A third model specifying an interaction term between block and contingency was a further better fit to the data ($\chi^2(3) = 27.1$, $p < .001$, AIC: 8553, $R^2 = 38.1$) and returned a significant Block x Contingency interaction effect ($F(3,874.8) = 9.095$, $p < .001$). Post-hoc t-tests with Holm correction for multiple comparisons (**Figure 9**) revealed that for the punishing cue, optimal responding increased between Block 2 and Block 3 only ($p = .004$), whereas for the rewarding cue performance decreased at Block 2 relative to Block 1, ($p = .015$) did not differ at Block 3 relative to Block 2 ($p = .271$), and then decreased again at Block 4 relative to Block 3 ($p = .006$). The Block x Contingency interaction effects were significant at Block 3 ($p = .007$) and Block 4 ($p = .001$). Overall, this suggests that performance was both greater for the losing cue relative to the winning cue and that the difference in performance between contingencies became greater as the task progressed. The model acted as the baseline model for further comparisons.

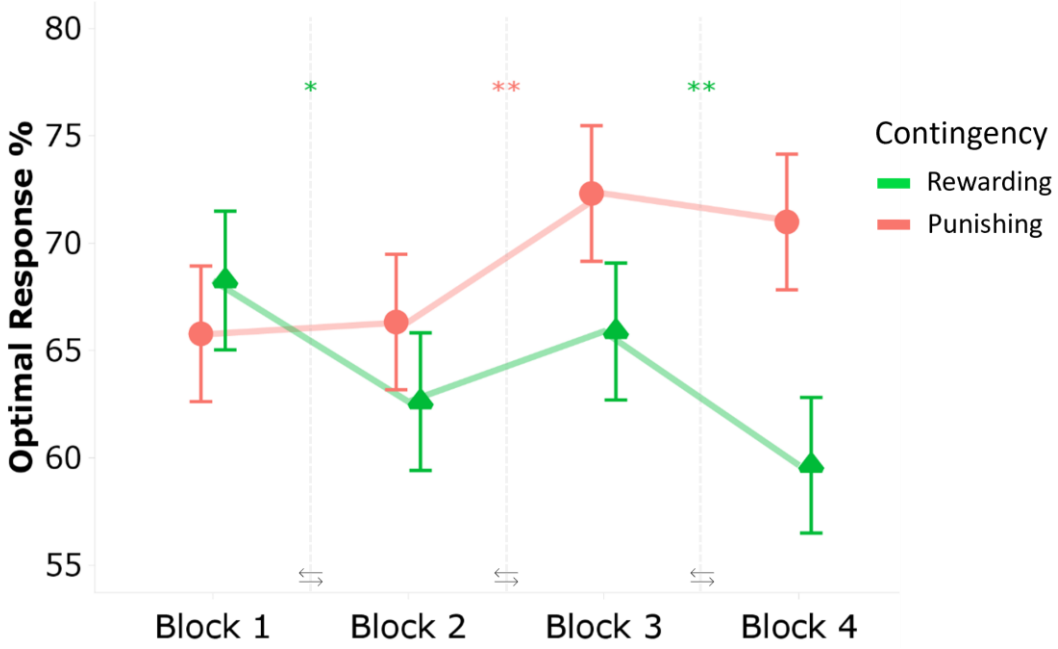

Figure 9. Plotted model coefficients of the Linear Mixed-Effects models predicting performance on the Probabilistic Reversal Learning task. Note: ⇆ represents contingency reversals, coloured significance values represent a significant change in optimal responses for punishing (*) or rewarding (*) stimuli relative to the preceding block.

3.4.2: Probabilistic Reversal Learning (PRL) performance and schizotypy

The next LME model added the three schizotypy scales simultaneously as fixed-effects to the Block x Contingency interaction model. However, this model was not a significantly better fit to the data ($\chi2(3) = 1.061$, $p = .787$, AIC: 8558, $R^2 = 39.0$). Next, two more models were specified that included interactions between the schizotypy and either Block or Contingency. While the Block x Schizotypy interaction model was not a significantly better fit ($\chi2(12) = 5.947$, $p = .919$, AIC: 8571, $R^2 = 39.3$) the Contingency * Schizotypy interaction model was ($\chi2(3) = 19.4$, $p < .001$, AIC: 8544, $R^2 = 41.6$). This meant that the strength of the relationship between schizotypy and performance differed between each contingency, but not between each block. As the fixed-effect of the interaction between negative schizotypy and contingency returned non-significant ($p = .670$), this model was re-estimated without this parameter (**Table 3**).

This final model returned a significant fixed-effect of positive schizotypy to predict poorer overall performance (β = -0.185[-0.323, -0.048], $p = .009$), but not negative schizotypy ($p = .729$) nor disorganised schizotypy ($p = .156$, **Figure 10**). There was also a significant interaction between both positive schizotypy and contingency (β = 0.256[0.138, 0.374], $p < .001$) and disorganised schizotypy

and contingency (β = -0.205[-0.362, -0.049], *p* = .010). To understand these interaction effects more clearly, the regression slopes within each contingency were compared against a null value of 0 (linear trend analysis, see **Figure 13**). This analysis found that positive schizotypy predicted poorer performance to the punishing contingency (B = -0.535[-0.935, -0.134], *p* = .009) but did not predict performance of the rewarding contingency (B = 0.192[-0.211, 0.595], *p* = .350). Translated to real-world units, for every additional item endorsed on the positive schizotypy scale the model predicts that participant accuracy of punishing cues decreases by 0.535% on the 0% to 100% scale (i.e., wagering £1 on punishing trials rather than 10p). This process was repeated for disorganised schizotypy which revealed that neither the linear trend for rewarding (B = -0.142[-0.585, 0.302], *p* = .530) nor punishing cues (B = 0.318[-0.123, 0.758], *p* = .156) were significantly different from 0. This means that disorganised schizotypy predicts a *difference* in performance between the two cues only. An unplanned parallel spearman correlation revealed that disorganised schizotypy did not correlate with the overall number of non-risky bets (r_s = .005, *p* = .957, BF_{10} = 0.173) suggesting this pattern of results is not a response bias.

Table 3. Linear Mixed-Effect model coefficients predicting response accuracy of the Probabilistic Reversal Learning task from the three schizotypy scales.

	Performance (Optimal %)					95% Conf Int	
predictor	**B**	**SE**	***t***	***p***	**β**	**LC**	**HC**
Intercept	65.8	3.092	21.269	< .001			
$Block^2$	0.547	1.877	0.291	= .771	0.013	-0.073	0.098
$Block^3$	6.536	1.877	3.483	= .001	0.153	0.067	0.239
$Block^4$	5.212	1.877	2.777	= .006	0.122	0.036	0.209
$Contingency^R$	2.126	2.918	0.729	= .466	0.057	-0.097	0.211
Positive schizotypy	-0.535	0.203	-2.636	= .009	-0.185	-0.323	-0.048
Negative schizotypy	0.086	0.248	0.347	= .729	0.021	-0.100	0.142
Disorganised schizotypy	0.318	0.223	1.424	= .156	0.105	-0.040	0.251
$Contingency^{R*} Block^2$	-6.201	2.689	-2.306	= .021	-0.109	-0.202	-0.016
$Contingency^{R*} Block^3$	-8.946	2.683	-3.334	= .001	-0.159	-0.252	-0.065
$Contingency^{R*} Block^4$	-13.8	2.676	-5.167	< .001	-0.248	-0.342	-0.154
$Contingency^{R*}$ Pos Scz	0.726	0.171	4.250	< .001	0.256	0.138	0.374
$Contingency^{R*}$ Dis Scz	-0.459	0.179	-2.572	= .010	-0.205	-0.362	-0.049

Note: $Block^2$ = the effect of Block 2 relative to Block 1, $Contingency^R$ = the effect of the rewarding contingency relative to the punishing contingency. $R^2_{fixed\text{-}effects}$ = 35.6%, $R^2_{random\text{-}effects}$ = 5.8%

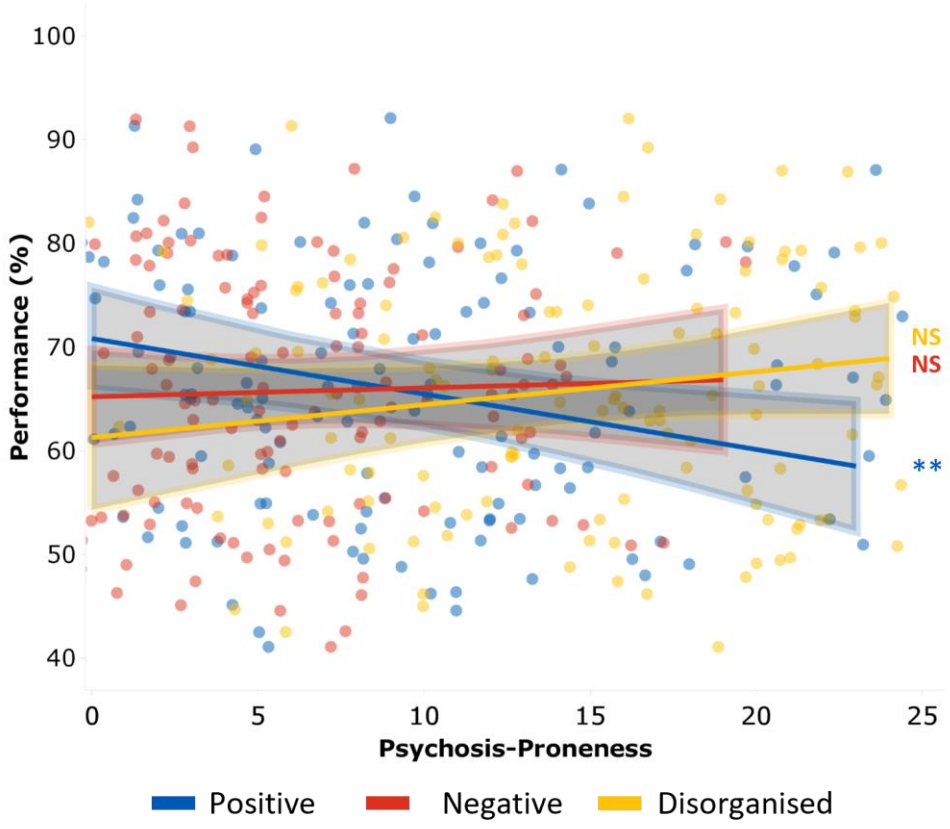

Figure 10. Plotted coefficients of the Linear Mixed-Effect model predicting response accuracy of the Probabilistic Reversal Learning task from the three schizotypy scales. Shaded regions represent 95% CIs.

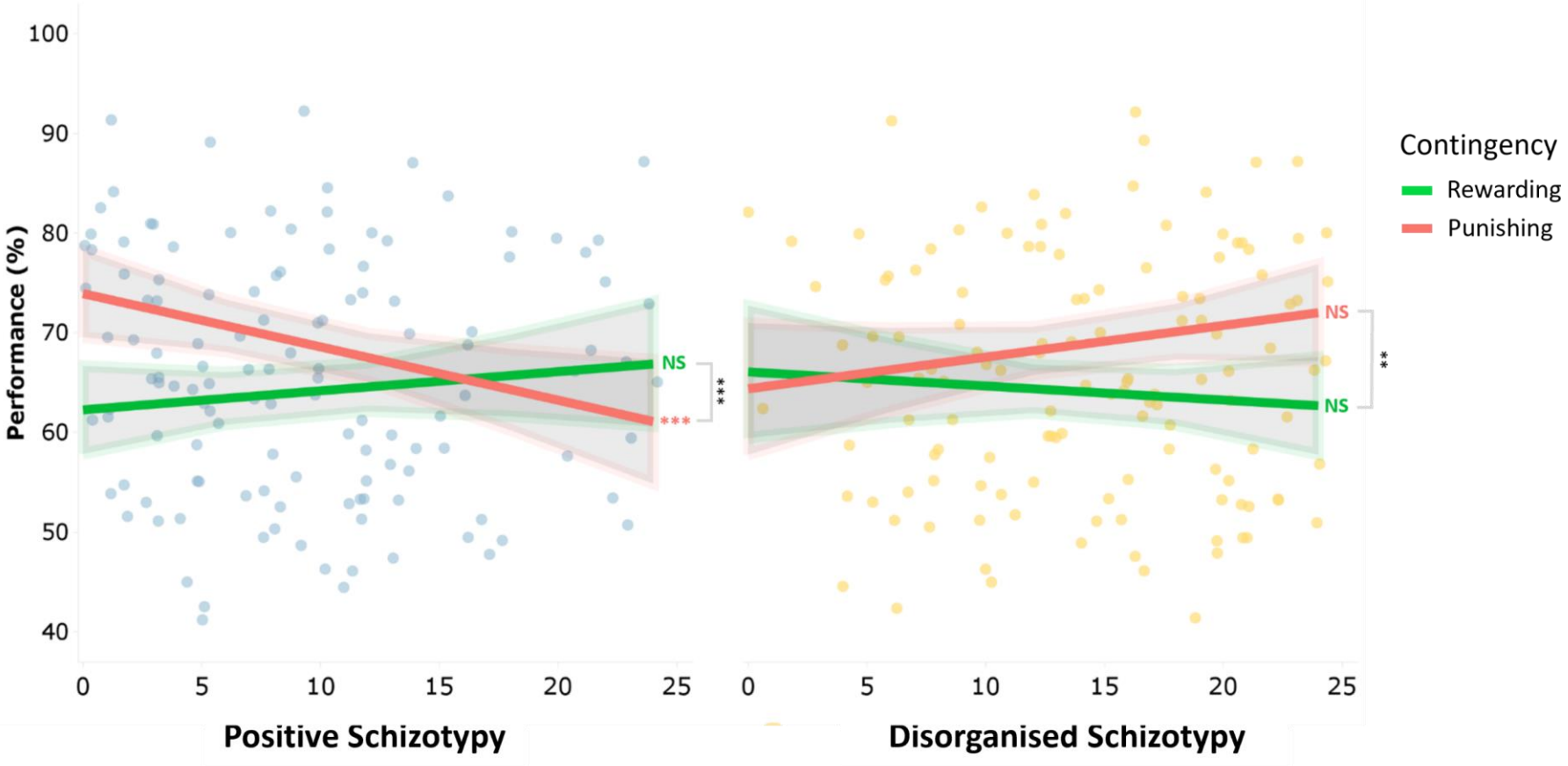

Figure 11. Plotted interaction effects of the Linear-Mixed Effects models predicting Probabilistic Reversal Learning (PRL) performance from schizotypy. **Left**: positive schizotypy predicted poorer performance to punishing but not rewarding cues. **Right**: disorganised schizotypy returned a significant interaction effect which came from the difference between ratings to the rewarding and punishing cue.

3.4.3: Mediators of PRL performance

Psychometric variables that were significantly correlated to positive or disorganised schizotypy were investigated as potential mediators of performance (see **3.3: Psychometric relationships**). The following analyses added these potential mediators as additional fixed-effects in additional LME models. The relationship between schizotypy and performance was then re-calculated to assess potential mediation. Overall, none of the models that added DASS-21 total score ($\chi2(1) = 0.110$, $p = .740$, AIC: 8546, $R^2 = 41\%$), Self-Reflectivity ($\chi2(1) = 1.829$, $p = .176$, AIC: 8512.0, $R^2 = 42.1\%$), Self-Certainty ($\chi2(1) = 2.576$, $p = .109$, AIC: 8511.2, $R^2 = 42.3\%$), nor pre-task motivation ($\chi2(1) = 0.817$, $p = .366$, AIC: 8513.0, $R^2 = 42.1\%$) were a significantly better fit to the data (Hypotheses 1a – 1c).

Additional mediation analyses also investigated the mediatory role of Extra-Dimensional Set-Shifting (EDS) errors and Simple Discrimination Reversal errors (**Supplementary Analysis A)**. The fixed-effect of EDS errors returned at trend level ($F(1, 114.3) = 2.883$, $p = .092$) and the fixed-effect of positive schizotypy also became trend ($F(1, 165.7) = 2.825$, $p = .095$) reducing by 35%. However, both the positive schizotypy x contingency ($F(1,817.8) = 12.0$, $p < .001$) and the disorganised schizotypy x contingency interaction effects remained significant ($F(1,818.0) = 6.716$, $p = .010$). A separate model reported that the fixed-effect of SDR errors returned significant ($F(1,116.4) = 4.133$, $p = .044$) and the effect of positive schizotypy returned as non-significant and reducing by 37% ($F(1, 166.2) = 2.655$, $p = .105$). However, again, the interaction with contingency remained significant ($F(1, 817.7) = 12.1$, $p < .001$). Overall, this may suggest that set-shifting and reversal learning explain a marginal proportion of the associations between schizotypy and performance, but they do not fully explain the association.

3.4.4: Self-reported task evaluation

The three schizotypy scales were correlated with the items of the Task Strategy Questionnaire (TSQ, **Appendix C.**) to provide further detail into performance differences (n = 44, **Table 4**). Positive schizotypy was associated with greater reports of using the 10p response due to miscomprehension ($r_s = .37$, $p = .012$, $p_{FDR} = .216$), negative schizotypy had a trend level associations of reporting no task strategy ($r_s = -.27$, $p = .070$, $p_{FDR} = .315$), and disorganised schizotypy presented trend associations to increased use of 10p responses due to miscomprehension ($r_s = .28$, $p = .066$, $p_{FDR} = .315$) and reduced ratings of task comprehension ($r_s = -.29$, $p = .056$, $p_{FDR} = .315$). However, none of these correlations survived correction for multiple comparisons. Open-ended comments were also collated into similar themes to understand how to improve comprehension of future studies which is discussed in Chapter 5.

Table 4. Frequentist Spearman correlation matrix between schizotypy and PRL task follow-up Task Strategy Questionnaire items.

	Schizotypy		
Task Strategy Questionnaire Item	**Positive**	**Negative**	**Disorganised**
There were relationships between cues and outcomes	0.08	-0.01	-0.19
I had a strategy	-0.08	-0.27†	-0.13
I understand how to respond optimally	-0.14	-0.18	-0.03
I chose 10p to avoid taking a risk	0.09	0.23	0.10
I chose 10p due to miscomprehension	0.37*	0.19	0.28†
I understood what my task was	-0.17	-0.10	-0.29†

Note: † = $p < .1$, * = $p < .05$, ** = $p < .01$, *** = $p < .001$. All associations do not survive correction for multiple comparisons. See **Appendix C.** for the full items.

3.5: Intra/Extra-dimensional (IDED) Set-Shifting Performance

3.5.1: Schizotypy

A multiple regression with Poisson distribution reported that positive schizotypy predicted an increase in EDS errors (OR = 1.026, $p < .001$), disorganised schizotypy predicted a reduction in EDS errors (OR = 0.969, $p < .001$), and negative schizotypy did not predict EDS errors (OR = 1.010, $p = .209$). These associations were also found to be independent of SD errors and SDR errors (all $p < .005$) suggesting the deficits are specifically due to set-shifting and not visual learning or reversal learning.

3.5.2: Potential mediators of Set-Shifting performance

The previous Poisson regression model was recalculated after adding each of the potential mediators to additional models. The DASS-21 total score significantly predicted a greater number of EDS errors (OR = 1.008, $p < .001$) but the associations between both positive schizotypy (OR = 1.020, $p < .001$) and disorganised schizotypy (OR = 0.960, $p < .001$) remained significant. Pre-task MIAMI scores did not predict EDS errors (OR = 1.016, $p = 0.298$) and the effects of schizotypy remained significant (both $p < .001$). Finally, Self-Reflectivity did not predict performance (OR = 1.009, $p = 0.352$) and Self-Certainty predicted an increase in set-shifting errors (OR = 1.033, $p = .006$). However, again, the associations between schizotypy and performance were unchanged. Overall, none of the potential mediators explained this relationship (see **Supplementary Analysis B** for details).

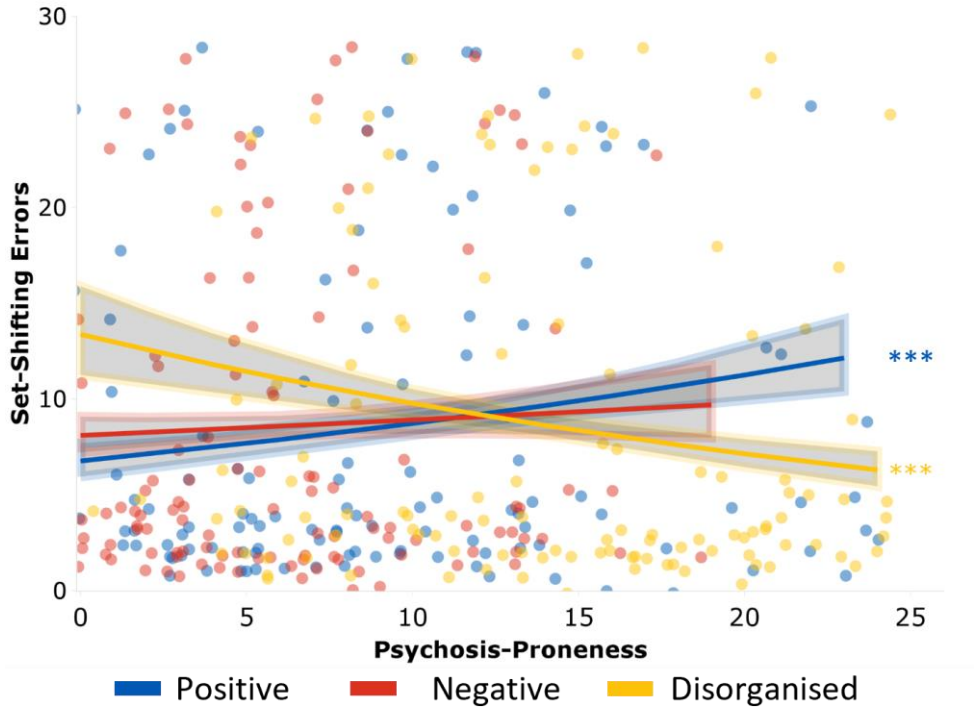

Figure 12. Plotted coefficients of the multiple Poisson regression predicting Extra-Dimensional Shift errors of the IDED task from the three schizotypy scales.

Table 5. Poisson regression predicting Extra-Dimensional Stage (EDS) Set-Shifting errors from the three schizotypy dimensions

	EDS Errors				95% Conf			
Variable	**Log(odds)**	**SE**	**Z**	***p***	**LC**	**HC**	**GVIF**	**OR**
Intercept	2.293***	0.079	29.192	<.001	2.138	2.446		
Positive Schizotypy	0.025***	0.006	4.490	<.001	0.014	0.036	1.369	1.026
Negative Schizotypy	0.009	0.008	1.257	=.209	-0.005	0.024	1.212	1.010
Disorganised Schizotypy	-0.031***	0.006	-5.070	<.001	-0.043	-0.019	1.480	0.969

Note: $X^2(3) = 32.174$, p <.001. GVIF = Generalised Variance Inflation Factor, OR = Odds Ratio.

4.0 Discussion

This chapter investigated potential explanations for neurocognitive performance deficits in psychometric schizotypy using a Probabilistic Reversal Learning (PRL) task and an attentional set-

shifting task (IDED). For the PRL, those higher in positive schizotypy exhibited poorer learning of punishing cues across all four blocks of the PRL task. There was also evidence that disorganised schizotypy may predict increased learning of punishing cues relative to the poorer learning of rewarding cues. For the IDED, positive schizotypy predicted poorer set-shifting performance and disorganised schizotypy predicted improved performance. Negative schizotypy did not predict any neurocognitive measure. These associations were not explained by negative affect, moderately discrete metacognition, and pre-or post-task motivation. The following discussion will first look at the effects of schizotypy in isolation before integrating the contributions of the potential mediators across both tasks. This Chapter will conclude with three study designs to further investigate these effects in Chapter 3, Chapter 4, and Chapter 5.

4.1 Schizotypy and Probabilistic Reversal Learning performance

The overall association between positive schizotypy and poorer PRL performance is consistent with the limited clinical literature[327,328]. More in-depth analysis revealed this effect emanated from poorer performance to the punishing cue only, which is consistent with more broad literature findings on clinical psychosis related to punishing cues[404]. Interestingly, this is the first application of a PRL task specifically in schizotypy, although schizotypy is associated with similar reasoning biases such as Jumping to Conclusions[405]. As a result, this study presents the novel finding that PRL deficits may span both schizotypy and schizophrenia and suggests that deficits in patients may be independent of clinical confounds. However, the explanation for this association in schizotypy is currently unclear. For positive schizotypy, it could be expected that poorer learning of punishing cues could be explained by avoidance behaviour. In the anxiety literature, there is evidence that negative affective states can capture attention from stimuli when arousal is low but divert attention when arousal is high[406]. This is also potentially consistent with findings in clinical psychosis patients who often attempt to repress or ignore negative affective experiences associated with the positive symptoms (e.g., persecutory delusions). In this task, the negative affective associations of the punishing stimuli may activate this mechanism. This may also explain why deficits are not found for rewarding cues, as hallucinations or delusions are rarely rewarding and would not require repression. Although, it is unclear whether the loss of money in the PRL task could be considered of such arousal to produce these effects.

Insights from the Task Strategy Questionnaire (TSQ) suggested positive schizotypy correlated with increased reports of using the 10p response due to miscomprehension (r_s = .37, p = .012). However, if this was the case, then it would be expected that performance to the punishing cue would increase due to a response bias - which is not the case. As a result, perhaps this association may in fact represent poor metacognitive insight into overall performance.

The TSQ may also explain the potential association between disorganised schizotypy and improved learning of the punishing cue. The TSQ tentatively suggested that those higher in disorganised schizotypy reported greater task miscomprehension and chose the 10p response due to this miscomprehension. Together, this may suggest that *poorer* neurocognitive learning of the punishing may produce greater miscomprehension, which manifests as a response bias to choose the 10p response. This response bias may then be misinterpreted in the statistical analysis as improved learning of punishing cues. This suggestion would be more consistent with the findings of positive schizotypy in the current study and the psychosis-spectrum literature more generally. However, what is currently inconsistent with past clinical literature[317,322] is the lack of association between negative schizotypy and performance. As negative schizotypy was higher in the current sample than in normative data, it is unlikely that this result is due to insufficient levels of this trait in the current sample. After summarising the results of the IDED, the influence of the potential mediators will be discussed across all three schizotypy scales.

4.2 Schizotypy and set-shifting performance

At the time of writing, this is the first assessment of set-shifting as measured by the IDED (a task without many of the confounds of other set-shifting tasks) in adult participants varying in psychometric schizotypy. The association between positive schizotypy and poorer performance is consistent with the schizotypy literature using the WCST or TMT[373–377] but contrasts with the clinical literature on positive symptoms[336,340,348,354,365,372]. While past clinical literature suggests perseverative responses may explain poorer performance, the poorer learning of punishing stimuli observed in the PRL task may also be relevant. Specifically, until the EDS stage participants are taught to respond to the pink shape dimension. When entering the EDS stage, participants initially continue to do so but the feedback now informs them that their responses of pink shape are now incorrect. It could be suggested that it is this learning from punishment that is disrupted, rather than participants failing to learn about the white line dimension (reward learning). This is also supported by previous research suggesting performance deficits are associated with reduced attentional disengagement from the previously reinforced dimension in schizotypy[407].

It is unclear whether the dissociation between positive schizotypy and schizophrenia is influenced by clinical confounds or is perhaps more simply a difference between schizotypy and schizophrenia. Although, a recent meta-analysis has replicated set-shifting deficits in drug naïve patients (SMD = -0.59 and -0.89, k = 8)[408]; suggesting medication confounds are an unlikely contributing factor. These inconsistencies are also seen in the lack of association between negative schizotypy and IDED performance in the current study relative to the clinical literature[373–375,379–382]. It could be argued these inconsistencies may be due to differences in the IDED relative to the TMT and

WCST, potentially suggesting deficits in the latter two tasks are influenced by other processes (i.e., visual learning and processing speed).

The most surprising finding is that disorganised schizotypy predicted improved set-shifting performance. The one study assessing disorganised schizotypy and set-shifting ability reported no associations[378] meaning the current literature is unclear. The majority of the clinical literature also does not separately assess disorganised symptoms from reality distortion[335,338,342,357]. However, this may explain the discrepancy between the current study and clinical literature: as perhaps reality distortion and disorganisation have opposing effects that are nullified when considered together. Indeed, there are examples of overall positive clinical symptoms masking the effects of sub-domains[101]. These findings could be suggested to be due to disorganised schizotypy presenting a bias to choose safer responses under uncertainty. However, the TSQ results found no support for this argument and this would not explain the current IDED results unless this uncertainty led to participants monitoring feedback more thoroughly (compared to other participants becoming complacent in the continued response of "pink shape"). Another, potential reason is perhaps disorganisation allows a greater openness to other decisions, riskier choices or illogical decisions which would be beneficial in this task. However, in this case it would be expected that performance on the other measures of the IDED would be impaired, which was not the case in the current study. It may simply be that disorganised schizotypy is beneficial to performance in both tasks, that schizotypy and schizophrenia are qualitatively different, or that these mechanisms are subject to change after the transition to psychosis.

4.3.0 The influence of potential mediators

4.3.1 Negative Affect

Despite the large associations between schizotypy and negative affect, no mediations were present suggesting schizotypy and negative affect may influence neurocognition through distinct rather than related pathways (e.g., both predicted set-shifting performance). Currently, there is mixed evidence on whether clinical negative affect in psychosis patients predicts set-shifting performance[337,352,356]. The current results may thus suggest that the associations between clinical symptoms and neurocognition may be at least partially independent of Mood and Anxiety Disorder co-morbidity. The lack of mediation is also insightful for the association between disorganised schizotypy and set-shifting performance. Specifically, the Cognitive Disorganisation scale of the O-LIFE also contains items related to social anxiety[303] – with the lack of mediation from negative affect suggesting the link is specifically due to disorganisation. In terms of mood disorders, the lack of association between

negative affect and PRL performance conflicts with a recent systematic review reporting that MDD patients present impairments in reward processing[409], but the finding of poorer set-shifting performance is consistent with impaired performance on the WCST and TMT[227]. Finally, the lack of mediation by negative effect may appear to conflict with the earlier suggestion of negative internal states producing deficit. However, the DASS-21 is a trait rather than state assessment of negative affect which may not be sensitive to detect this effect. Future studies should apply state measures such as the STICSA or apply Galvanic Skin Response (GSR) measures.

4.3.2 Metacognition

Unexpectedly, improved Self-Reflectivity was associated with greater levels of each schizotypy dimension contrasting the clinical literature. As outlined in Chapter 1, it is unclear whether this may reflect poor insight into cognition or, perhaps, that this greater metacognitive ability allows those high in schizotypy to function well. As this association is not the primary aim of this Chapter and research into metacognitive performance in schizotypy is relatively novel, these findings will be discussed in the general discussion (Chapter 6) where findings across all Chapters will be integrated more succinctly.

Moderately Discrete Metacognition as assessed by the Self-Reflectivity (*openness to fallibility*) and Self-Certainty (*pathological overconfidence*) scales of the BCIS also did not mediate performance on either task. Similar to negative affect, this suggests that metacognition, as measured by Self-Certainty, predicts poorer set-shifting performance independently of schizotypy. This finding is consistent with pathological overconfidence being associated with reduced consideration of new attention sets[410] and poorer set-shifting being associated with both ruminating metacognitive thinking styles[411] and poorer synthetic metacognition in clinical patients[412]. Moreover, that Self-Certainty but not Self-Reflectivity was associated with performance is consistent with recent systematic reviews in clinical patients[410,164]. However, while the results of the IDED are consistent it is unclear why this did not generalise to the PRL task. For example, it would be expected that Self-Certainty would be detrimental after the reversal as the trait inhibits consideration of alternative hypotheses. Perhaps the probabilistic nature of the PRL task raised levels of uncertainty to an extent that Self-Certainty did not become detrimental (unlike the non-probabilistic IDED). However, while the current results suggest metacognition does not explain cognitive deficits in schizotypy, it should be noted that the internal consistency of the BCIS was poor in the current study, which also replicates more recent reviews of the BCIS[164].

4.3.3 Motivation

Surprisingly, pre-task motivation did not predict performance on the PRL task which both involves goal-orientated behaviour and continued effort and is likely to cause fatigue at 240 trials. However, while pre-task motivation was associated with all three schizotypy scales consistent with clinical patients, disorganised rather than negative traits were the most closely associated. As the MIAMI also assesses task-related concerns and apprehensions more generally this may reflect anxiety-related items of the Cogdis scale.

However, the assessment of motivation in this study was limited. Specifically, one limitation is that the MIAMI was not given for each task, but rather across both. Moreover, responses to the pre-task motivation scale had a large a negative skew (skewness = -0.571, SE_{skew} = 0.214, **Appendix E.**). These unexpected high motivation reports may have been influenced both by the performance-based inconvenience allowance and all participants knowing the experimenter personally. Respectively, this may have raised motivation too high to detect amotivation-related deficits or have produced inaccurate motivation scores in some participants. As there is no normative data of the MIAMI for comparison, this suggestion cannot be further validated. Together, this may explain why the commonly found association between performance and negative schizotypy was not found, as it is suggested to be partially due to experiential negative traits such as amotivation. Alternatively, the current study was relatively short and so perhaps the long cognitive batteries applied in clinical research[258] are necessary to present deficits. Overall, the influence of motivation in the current study is inconclusive.

4.4 Strengths and limitations

A further limitation of this Chapter is the uncontrolled potential confound of Working Memory. Specifically, set-shifting performance has been suggested to be at least partially due to deficits in working memory in the WCST, TMT and IDED[341,350,372,413–418]. Although, set-shifting performance was found independent of visual learning and reversal learning which both require Working Memory, which may indirectly support independence. Regardless, this limitation would point to an alternative disrupted process rather than question the current findings. Some strengths of the current chapter include the validation analyses of the PRL task, the normative comparisons suggesting the null findings here are not due to sampling biases (i.e., levels of negative schizotypy were too low to detect response variation), and the collection of neurocognitive data across two tasks allowing direct comparisons to be drawn within the same sample. Finally, although the sample size of the TSQ analysis was small the analysis does give insights to guide future research.

4.5 Future Research

As outlined above, the results of the current study are somewhat inconclusive in terms of understanding the mechanisms behind these effects. The remainder of this Chapter will discuss the structure of the three upcoming experimental Chapters and how they aim to overcome these limitations.

4.5.1 Transition to Chapter 3

A primary limitation of the current research is the limited assessment of metacognition. Firstly, as outlined above the BCIS scale was found to be unreliable. One reason for this may be that the BCIS is designed for clinical samples and thus may not apply to neurotypicals. For example, the following item may be less reliable for those without hallucinations: "*Other people can understand the cause of my unusual experiences better than I can*". As a result, Chapter 3 will use a more application assessment of metacognition: the Metacognitions Questionnaires (MCQ-30), as outlined in Chapter 3. The second issue is that psychometric assessments of metacognition assume a person has the metacognitive ability to introspect on their poor metacognitive ability – which requires good metacognitive ability. Take, for example, the following item from the BCIS: "*Some of my experiences that have seemed very real may have been due to my imagination*". An individual who agrees with this statement would be considered to have good metacognition by collating cognitive experiences of correct and incorrect judgments, weighing the relative evidence of these situations, and reflecting on their experiences of the quality of their cognition. However, they may also be completely devoid of all metacognitive capacity and may simply overestimate their reflectivity or respond in a socially desirable way. Opposing cases such as these may not only explain the poor internal consistency of the BCIS but also the inconsistent metacognitive associations in clinical patients. Clearly, a more objective assessment of metacognitive ability is needed.

To address this issue Chapter 3 proposes to adapt the PRL task. In the introduction, it was explained that the PRL can also be considered a metacognitive task due to the use of confidence (metacognitive sensitivity) and acting on knowledge (metacognitive control). However, the issue is that these processes and neurocognitive processes cannot be separated through the single response dimension of 10p or £1. Specifically, the action of correctly betting £1 on the rewarding cue can be broken down into at least three decisions: a) correct learning of the contingency itself, b) allocating high confidence to this correct response and c) correctly deciding to act upon this knowledge to bet £1 rather than choosing the safer option of 10p. Interestingly, these decisions could represent neurocognitive ability, metacognitive sensitivity, and metacognitive control, respectively. Due to the

conflation of these processes, it is unclear from the current task design whether positive schizotypy predicts poorer learning, confidence, acting on ability, or any combination of these processes (and thus whether deficits are neurocognitive or metacognitive). Moreover, there is recent evidence that poor PRL performance may be due to impairments in confidence rather than neurocognitive ability[314] which the current design cannot validate. Furthermore, the current study reported that PRL deficits were independent of other neurocognitive abilities such as visual learning, reversal learning, and set-shifting which may suggest metacognitive deficits are the key cause. Consequently, Chapter 3 will create a Metacognitive-Probabilistic Reversal Learning (M-PRL) task and apply this method to also create a metacognitive IDED (M-IDED) task. Combined with the additional use of the MCQ-30, Chapter 3 will have both psychometric and behavioural metacognitive measures which can be compared (i.e., discrepancies between self-reported and actual metacognitive performance). This is particularly relevant considering recent calls for more objective metacognitive assessments[157] to help understand inconsistencies of self-report measures in patients[27].

4.5.2 Transition to Chapter 4

While Chapter 3 will apply behavioural metacognitive assessments to neurocognitive tasks, Chapter 4 will expand this into social cognition. As outlined in Chapter 1, social cognition is increasingly reported to be more closely related to daily functioning than neurocognition[75]. As the ultimate aim of this thesis is to improve daily functioning it may be more beneficial to expand the focus to social cognition. Moreover, this would allow an investigation of whether metacognitive processes are, for example, more relevant for social cognition than neurocognition. Chapter 4 will therefore address these aims by creating a metacognitive adaptation of a social cognitive task: the metacognitive adaptation of the Geneva Emotion Recognition Task short form[419] (M-GERT-S).

4.5.3 Transition to Chapter 5

The influence of motivation was unclear from the current findings potentially due to an inappropriate application of the psychometric measure. As the core principle of including motivation in this thesis is to ultimately understand how cognitive task realism may affect performance, adding another motivation measure was not pursued. Instead, Chapter 5 aimed the aim to make the task itself more realistic and motivating by adapting the PRL task into a Virtual Reality task (VR-PRL) using the open-ended comments from the Task Strategy Questionnaire. If task deficits are not replicated in the VR-PRL relative to the 2D-PRL it would suggest deficits in the current Chapter are (at least partially) attributable to motivation.

5.0 Conclusions

This Chapter aimed to assess the confounding effects of increased negative affect, poorer motivation, and poorer metacognition on neurocognitive performance in psychometric schizotypy. While similar deficits were replicated relative to clinical samples, including poorer learning of punishing stimuli and poorer set-shifting ability, these observations were not explained by any of the potential mediating variables. However, several methodological and psychometric issues meant that the assessment of motivation and metacognition may not have been successful and thus the implications of these results are unclear. The next three Chapters address the limitations of the current Chapter. Chapter 3 will expand the response dimensions of the PRL task to assess cognition and metacognition separately, collect objective indices of metacognition, and expand the psychometric metacognitive assessments. Next, Chapter 4 applies the same structure to social cognition which is more closely associated with daily functioning. Finally, Chapter 5 aims to re-assess the role of motivation by creating a Virtual Reality adaption of the PRL task to increase motivation relative to the current Chapter. Finally, Chapter 6 will involve a general discussion comparing performance across tasks and cognitive domains.

Appendices

Appendix A.

Pre- and Post-task motivation questionnaires

Participant number:_____ *Pre-test*

Momentary Influences, Attitudes and Motivation Scale (MIAMI-R)

In the following you will find **several questions which relate to the upcoming experiment as well as your current performance level**. Please read each statement carefully and decide how far/ to which extent this statement applies to you.

Please be as honest as you feel comfortable with

	fully agree	rather agree	rather disagree	fully disagree
1. I am only taking part in this research because someone else (e.g., therapist, partner) insisted.	1	2	3	4
2. I worry that the tasks will be too difficult for me.	1	2	3	4
3. I know what I can expect from this research.	1	2	3	4
4. I am aware of my capabilities and will be able to show them in the test situation.	1	2	3	4
5. I can hardly concentrate today.	1	2	3	4
6. I feel fit and capable.	1	2	3	4
7. Test situations like this are not for me.	1	2	3	4
8. I don't take psychological tests too seriously.	1	2	3	4
9. I experience the test situation as very unpleasant and would like to leave.	1	2	3	4
10. I fear that a bad test result will have negative consequences.	1	2	3	4
11. I am so nervous that my performance will not reflect my true capabilities.	1	2	3	4
12. I experience sensations/complaints which are particularly severe and distracting today	1	2	3	4
13. I don't care much about the assessment.	1	2	3	4
14. I fear that I will perform poorly.	1	2	3	4
15. I am not motivated at all to take part in the assessment.	1	2	3	4
16. Right now, I feel very tired and exhausted.	1	2	3	4
17. It is important to me to perform well today.	1	2	3	4
18. I am willing to do my best.	1	2	3	4
19. I feel comfortable and in good hands with the tester.	1	2	3	4
20. I don't worry about the outcome of this assessment	1	2	3	4

Thank you!

Participant number:_____ *Post-test*

Momentary Influences, Attitudes and Motivation Scale (MIAMI-R)

In the following you will find **several questions which relate to the previous assessment and your task performance**. Please read each statement carefully and decide, how far/ to which extent this statement applies to you.

Please be as honest as you feel comfortable with

	fully agree	rather agree	rather disagree	fully disagree
1. I pushed myself and gave my best.	1	2	3	4
2. I felt under pressure by the test situation.	1	2	3	4
3. The test situation was not as bad as expected	1	2	3	4
4. I felt very exhausted and worn out.	1	2	3	4
5. Many of the tasks were fun to do.	1	2	3	4
6. I was very nervous throughout the whole assessment.	1	2	3	4
7. I found many of the assigned tasks very difficult.	1	2	3	4
8. I could sufficiently concentrate on the tasks.	1	2	3	4
9. The assessment was way too long and in the end I lost my motivation.	1	2	3	4
10. I did not worry about the outcome of the assessment	1	2	3	4
11. There were too many things (e.g., noise) which disturbed my concentration.	1	2	3	4
12. During the assessment I repeatedly thought about how my performance would turn out.	1	2	3	4
13. I was not motivated and accordingly did not achieve my best performance.	1	2	3	4
14. I was fearful about the results while performing the tasks.	1	2	3	4
15. I felt being observed and uncomfortable, so I could not fully concentrate.	1	2	3	4
16. Different thoughts and concerns bothered and distracted me during the assessment.	1	2	3	4
17. I felt comfortable and in good hands with the tester.	1	2	3	4
18. I was distracted by bodily sensations (e.g., pain) during the assessment.	1	2	3	4
19. I did not really try hard	1	2	3	4
20. I was so focused on the tasks, I almost forgot that I was in a test situation	1	2	3	4

Thank you!

Appendix B.

Becks Cognitive Insight Scale (BCIS)

Please tick the box that most describes how you feel *in general*

	Do not agree at all	Agree slightly	Agree a lot	Agree completely
1. At times, I have misunderstood other people's attitudes towards me. (R)				
2. My interpretations of my experiences are definitely right. (C)				
3. Other people can understand the cause of my unusual experiences better than I can. (R)				
4. I have jumped to conclusions too fast. (R)				
5. Some of my experiences that have seemed very real may have been due to my imagination. (R)				
6. Some of the ideas I was certain were true turned out to be false. (R)				
7. If something feels right, it means that it is right. (C)				
8. Even though I feel strongly that I am right, I could be wrong. (R)				
9. I know better than anyone else what my problems are. (C)				
10. When people disagree with me, they are generally wrong. (C)				
11. I cannot trust other people's opinion about my experiences. (C)				
12. If somebody points out that my beliefs are wrong, I am willing to consider it. (R)				
13. I can trust my own judgment at all times. (C)				
14. There is often more than one possible explanation for why people act the way they do. (R)				
15. My unusual experiences may be due to my being extremely upset or stressed. (R)				

Appendix C.

Form given to participants explaining the rules of the PRL task and requesting insights into their responses.

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Task Strategy Questionnaire (TSQ) – V1

The University of Nottingham

Participant number.....

Instructions: In psychology we often do not ask participants why they acted the way they do, but rather assume it from their behaviour. Participants so far have been telling us their strategies/lack of strategy but we have not yet recorded this. We have selected 30 people to tell us their thoughts on this task as it can really help us understand any findings. Please be as honest as possible.

You may leave this blank if you do not wish to answer. There is no right or wrong answers; we are only interested in *your* strategy. **All the responses are anonymised**.

This is about the gambling game only

Did you think there was any relationship between the images and whether you won or lost?

Please describe: **YES/NO**

.....

.....

.....

.....

.....

Did you have a strategy? And if so could you describe it. **YES/NO**

.....

.....

.....

.....

Please turn over

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Please ask if the following is unclear

Explaining the main task: You saw two symbols (“♂”/“♀”). In the first 60 rounds “♂” was rewarded with money 80% of the time and punished 20% of the time. The opposite was true for “♀”. After 60 rounds, these relationships were reversed.

Optimum strategy: To gain the most virtual money as possible, people should bet £1, 100% of the time to “♂” and 10p 100% of the time to “♀”. This strategy should be reversed after each block of 60 trials.

1. If you knew the rules, would you know to respond in this optimum way? **Y/N**
2. Did you choose the less-risky response (10p) more often to avoid taking risk? **Y/N**
3. …. or due to *not* understanding the task? **Y/N**
4. Did you feel you understood what your task actually was? **Y/N**

Any general comments about the task?

……

……

……

……

……

……

Appendix D.

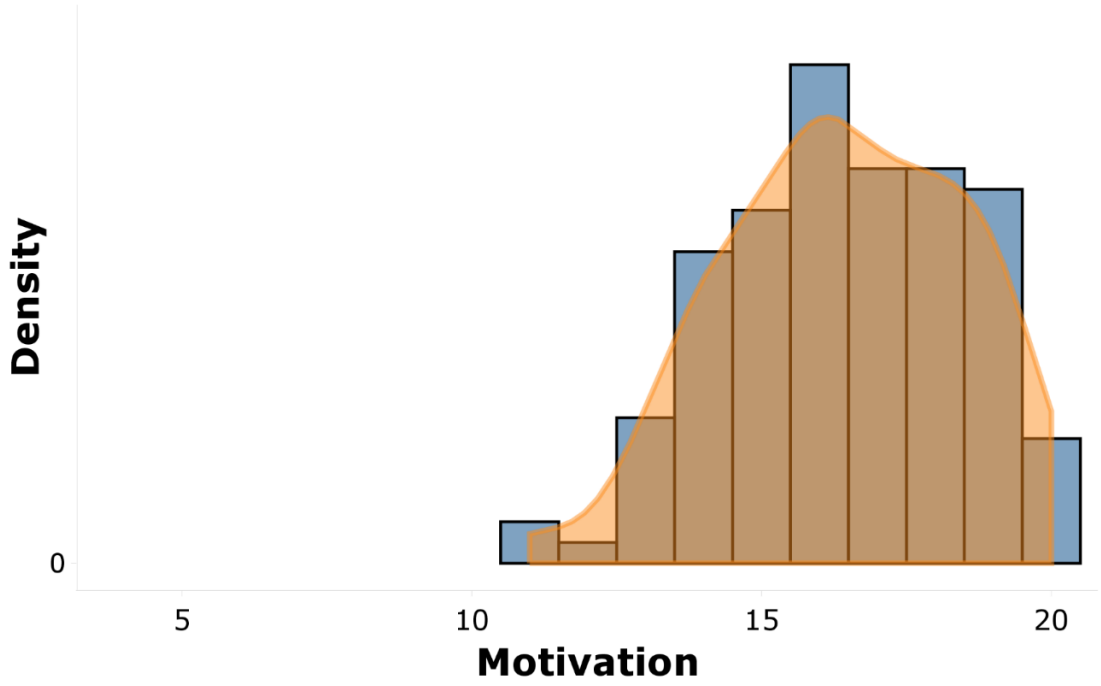
Descriptive statistics of performance in the Probabilistic Reversal Learning Task and Intra/Extra-Dimensional Set-Shifting task.

Task	*Metric*	Range	M	SD	Med	MAD	IQR
PRL (%)	*Block 1*	0 – 100	66.2	15.1	68.3	14.8	23.3
	Block 2	0 – 100	62.5	15.1	63.3	17.3	21.7
	Block 3	0 – 100	68	16.4	70.0	19.8	28.3
	Block 4	0 – 100	65.4	16.8	61.7	19.7	25.0
	Rewarding	24 – 92	63.9	14.6	63.3	16.1	22.1
	Punishing	37 – 95	68.9	14.4	70.4	16.7	22.0
IDED (errors)	*Simple Discrimination (SD)*	0 – 25	3.23	4.67	1.0	1.48	2.0
	SD Reversal (SDR)	1 – 19	2.64	3.38	1.0	0.00	1.25
	Compound Discrimination (CD)	0 – 38	3.25	6.54	1.0	1.48	2.0
	CD Superimposed (CDS)	0 – 40	1.60	5.39	0.0	0.00	1.0
	CD Reversal (CDR)	1 – 33	3.30	5.54	1.0	0.00	2.0
	Intra-Dimensional Shift (IDS)	0 – 34	1.23	4.24	0.5	0.74	1.0
	ID Reversal (IDR)	1 – 31	1.90	4.09	1.0	0.00	0.0
	Extra-Dimensional Shift (EDS)	0 – 35	8.74	9.11	4.0	2.97	12.0
	ED Reversal (EDR)	1 – 34	6.25	9.01	2.0	1.48	4.0

Note: Performance for the PRL is percentage of optimal responses and number of errors for the IDED. MAD = Median Absolute Deviation, IQR = Inter-Quartile Range.

Appendix E.

Distribution of the pre-task MIAMI scores. Blue bars represent a frequency histogram and the orange curve represents a density function. The figure shows that motivation scores were negatively skewed.



Supplementary analyses

Supplementary Analysis A

Linear Mixed Effects model controlling for the effects of set-shifting (top) and reversal learning (bottom) in the PRL analysis.

	Performance (Optimal %)					95% Conf Int	
predictor	**B**	**SE**	**t**	***p***	**β**	**LC**	**HC**
Intercept	66.590	3.453	19.282	< .001	0.000	0.000	0.000
$Block^2$	0.528	1.943	0.272	= .786	0.012	-0.077	0.101
$Block^3$	6.167	1.943	3.174	= .002	0.144	0.055	0.234
Block4	5.189	1.943	2.671	= .008	0.122	0.032	0.212
$Contingency^R$	3.706	3.074	1.206	= .228	0.100	-0.063	0.263
Pos Scz	-0.362	0.216	-1.681	= .095	-0.121	-0.263	0.020
Neg Scz	0.101	0.263	0.383	= .703	0.024	-0.100	0.148
Dis Scz	0.240	0.234	1.025	= .307	0.077	-0.071	0.225
Set-Shifting Errors	-0.199	0.117	-1.698	= .092	-0.098	-0.211	0.015
$Contingency^R * Block^2$	-6.001	2.787	-2.154	= .032	-0.106	-0.202	-0.010
$Contingency^R * Block^3$	-8.726	2.780	-3.139	= .002	-0.155	-0.252	-0.058
$Contingency^R * Block^4$	-13.891	2.771	-5.012	< .001	-0.250	-0.347	-0.152
$Contingency^R$ * Pos Scz	0.628	0.181	3.468	= .001	0.218	0.095	0.342
$Contingency^R$ * Dis Scz	-0.487	0.188	-2.592	= .010	-0.216	-0.379	-0.053

Note: $Block^2$ = the effect of Block 2 relative to Block 1, $Contingency^+$ = the effect of the positive contingency relative to the negative contingency. $R^2_{fixed\text{-}effects}$ = 35.6%, $R^2_{ranomd\text{-}effects}$ = 5.8%

	Performance (Optimal %)					95% Conf Int	
predictor	**B**	**SE**	**t**	***p***	**β**	**LC**	**HC**
Intercept	66.760	3.406					
$Block^2$	0.528	1.943	19.598	< .001	0.012	-0.077	0.101
$Block^3$	6.167	1.943	0.272	= .786	0.144	0.055	0.234
$Block^4$	5.189	1.943	3.174	= .002	0.122	0.032	0.212
$Contingency^R$	3.674	3.074	2.671	= .008	0.099	-0.063	0.262
Pos Scz	-0.350	0.215	1.195	= .232	-0.117	-0.258	0.024
Neg Scz	-0.009	0.265	-1.629	= .105	-0.002	-0.127	0.123
Dis Scz	0.267	0.231	-0.034	= .973	0.086	-0.060	0.233
Reversal Learning	-0.649	0.319	1.158	= .249	-0.117	-0.229	-0.004
$Contingency^R * Block^2$	-5.993	2.787	-2.033	= .044	-0.106	-0.202	-0.009
$Contingency^R * Block^3$	-8.732	2.780	-2.151	= .032	-0.155	-0.252	-0.058
$Contingency^R * Block^4$	-13.867	2.771	-3.141	= .002	-0.249	-0.347	-0.152
$Contingency^R$ * Pos Scz	0.631	0.181	-5.004	< .001	0.219	0.096	0.343
$Contingency^R$ * Dis Scz	-0.489	0.188	3.484	= .001	-0.216	-0.379	-0.053

Note: $Block^2$ = the effect of Block 2 relative to Block 1, $Contingency^+$ = the effect of the positive contingency relative to the negative contingency. $R^2_{fixed\text{-}effects}$ = X%, $R^2_{ranomd\text{-}effects}$ = X%

Supplementary Analysis B

Poisson regressions predicting set-shifting errors from positive, negative, and disorganised schizotypy while controlling for pre-task, post-task motivation, negative affect, and metacognition.

	EDS Errors				95% Conf Int			
Variable	**Log(odds)**	**SE**	**Z**	***p***	**LC**	**HC**	**GVIF**	**OR**
Intercept	2.031***	0.265	7.667	<.001	1.509	2.547		
Positive Schizotypy	0.025***	0.006	4.465	<.001	0.014	0.036	1.368	1.026
Negative Schizotypy	0.010	0.008	1.294	=.199	-0.005	0.024	1.208	1.010
Disorganised Schizotypy	-0.031***	0.006	-5.042	<.001	-0.043	-0.019	1.480	0.969
Pre-task motivation	0.016	0.015	1.040	=.298	-0.014	0.046	1.001	1.016

Note: $X^2(4)= 33.258$, *p* <.001. GVIF = Generalised Variance Inflation Factor, OR = Odds Ratio.

	EDS Errors				95% Conf Int			
Variable	**Log(odds)**	**SE**	**Z**	***p***	**LC**	**HC**	**GVIF**	**OR**
Intercept	2.917***	0.217	13.449	<.001				
Positive Schizotypy	0.025***	0.006	4.367	<.001	0.014	0.036	1.376	1.025
Negative Schizotypy	0.006	0.008	0.719	=.472	-0.010	0.021	1.244	1.006
Disorganised Schizotypy	-0.032***	0.006	-5.180	<.001	-0.044	-0.020	1.485	0.969
Post-task motivation	-0.036**	0.012	-3.070	0.002	-0.060	-0.013	1.050	0.964

Note: $X^2(4)= 41.482$, *p* <.001. GVIF = Generalised Variance Inflation Factor, OR = Odds Ratio.

	EDS Errors				95% Conf Int			
Variable	**Log(odds)**	**SE**	**Z**	***p***	**LC**	**HC**	**GVIF**	**OR**
Intercept	2.307***	0.078	29.764	<.001				
Positive Schizotypy	0.019***	0.006	3.349	<.001	-0.015	0.054	1.471	1.020
Negative Schizotypy	-0.004	0.008	-0.532	=.595	-0.052	0.043	1.380	0.996
Disorganised Schizotypy	-0.041***	0.006	-6.341	<.001	-0.043	-0.019	1.657	0.960
Negative Affect	0.008***	0.002	5.043	<.001	-0.002	0.017	1.675	1.008

Note: X2(4)= 55.831, *p* <.001. GVIF = Generalised Variance Inflation Factor, OR = Odds Ratio.

	EDS Errors				95% Conf Int			
Variable	**Log(odds)**	**SE**	**Z**	***p***	**LC**	**HC**	**GVIF**	**OR**
Intercept	1.600***	0.261	6.134	<.001				
Positive Schizotypy	0.023***	0.006	3.855	=.001	0.011	0.034	1.508	1.023
Negative Schizotypy	0.010	0.008	1.293	=.199	-0.005	0.025	1.301	1.010
Disorganised Schizotypy	-0.029***	0.006	-4.519	<.001	-0.042	-0.016	1.627	0.971
Self-Reflectivity	0.009	0.010	0.931	=.352	-0.010	0.027	1.427	1.009
Self-Certainty	0.033**	0.012	2.740	=.006	0.009	0.056	1.082	1.033

Note: $X^2(5)= 40.703$, *p* <.001. GVIF = Generalised Variance Inflation Factor, OR = Odds Ratio.

Supplementary Analysis C

Table 6. Multiple regression analysis predicting Self-Reflectivity scores of the BCIS from the three schizotypy scales.

	Self-Reflectivity						95% Conf Int	
predictor	**B**	**SE**	**t**	***p***	**BF_{10}**	**β**	**LC**	**HC**
Intercept	14.982	0.777						
Positive	0.190	0.055	3.474	< .001	125.1	0.311	0.134	0.488
Negative	0.179	0.074	2.426	0.017	6.508	0.209	0.038	0.379
Disorganised	0.089	0.061	1.456	0.148	0.704	0.139	-0.050	0.327

Note: F(3, 124) = 15.0, $p < .001$, $R^2 = 26.7\%$, $R^2_{adjusted} = 24.9\%$

Chapter 3

Metacognitive adaptations of neurocognitive tasks in psychometric schizotypy

High positive schizotypy predicts poorer processing of perceived negative stimuli and acting on these deficits.

Introduction: In the previous Chapter, deficits were observed in both a Probabilistic Reversal Learning (PRL) task and an attentional set-shifting task (IDED) in individuals high in schizotypy. However, due to the task designs, it was unclear whether poor performance was caused by cognition or metacognition. The current Chapter presents two novel metacognitive adaptations of these tasks: the metacognitive-PRL (M-PRL) and metacognitive-IDED (M-IDED) which separate cognitive ability from metacognitive sensitivity and metacognitive control. Additionally, more robust psychometric assessments of metacognition were taken.

Method: A total of 256 participants completed the M-IDED and 219 of these participants also completed M-PRL in an online study. Each of these tasks requested a neurocognitive judgment (total accuracy), confidence rating in this decision, and whether participants would like to volunteer this response for an appraisal (Koren accuracy). All participants completed assessments of schizotypy, negative affect, metacognition, and motivation.

Results: For the M-PRL, positive schizotypy was associated with poorer learning of punishing stimuli but not rewarding stimuli (total accuracy). The same pattern of results was also found for Koren accuracy. Positive schizotypy also predicted poorer confidence judgements when the outcome was perceived as negative. For the M-IDED, schizotypy was unrelated to accuracy, Koren accuracy, and confidence ratings. Despite schizotypy correlating with higher negative affect and poorer psychometric metacognition and motivation, none of these variables explained these associations.

Discussion: The current results replicated those of Chapter 2 and suggest schizotypy is associated specifically with neurocognitive deficits as measured by the M-PRL. Moreover, the association to Koren accuracy additional suggests participants also act upon faulty knowledge. This suggests participants also presented impaired metacognitive control which was further supported by higher self-reported metacognitive deficits. The current results highlight metacognitive control deficits as a key target for clinical samples.

High positive schizotypy predicts poorer processing of perceived negative stimuli and acting on these deficits.

1.0: Introduction

1.1: Summary of aims

In the previous Chapter, cognitive deficits were replicated in positive schizotypy (i.e., 'magical ideation') in a Probabilistic Reversal Learning (PRL) task and an attentional set-shifting task (IDED). It was expected that levels of negative affect, moderately discrete metacognition, and motivation would partially explain these deficits. However, this was not the case. Moreover, the PRL task itself confounded multiple cognitive and metacognitive processes within a single response dimension. Specifically, the act of betting £1 on the rewarding cue requires visual learning and working memory (*neurocognition*), high decision confidence (*metacognitive sensitivity*), and acting on this knowledge (*metacognitive control*). As a result, the current Chapter adapts both tasks to filter neurocognitive processes from metacognitive processes by expanding the task response dimensions. The aim of this Chapter is therefore to further explore the reasons for the deficits reported in Chapter 2.

1.2: Metacognitive task adaptations

1.2.1: Confidence – metacognitive sensitivity

The most common metacognitive adjustment to cognitive tasks is to assess response confidence. While many studies use overall confidence ratings, these only reflect an overall bias in responses (e.g., people's different 'anchoring points') rather than efficient allocation of confidence. What is more insightful is the association between cognitive accuracy and confidence: being highly confident *when* correct and reporting low confidence *when* incorrect (i.e., metacognitive sensitivity)[150]. As outlined in Chapter 1, patients with psychosis have been consistently found to be overconfident in errors and commonly underconfident in correct responses. However, while these associations have been found across the psychosis spectrum current evidence is limited[27,150]. Indeed, there have been many failed replications in clinical samples[420–426] and meta-analytical evidence has failed to find an association between metacognitive sensitivity and clinical symptoms[153]. Moreover, it is currently unclear *why* confidence is inappropriately attributed – especially underconfidence in correct responses and how this relates to functioning. There have been recent calls to assess overconfidence in wider contexts and with more precise confidence estimates beyond "low confidence" vs "high confidence"[27]. Considering the above, the current Chapter expands the response dimensions of the PRL and IDED to include ratings of confidence in cognitive task decisions.

1.2.2: Koren accuracy – metacognitive control

High confidence in knowledge does not necessitate acting on this knowledge. When we are uncertain in the real world we have the choice not to act or to seek out further information. However, almost all cognitive tasks do not consider this, forcing participants to respond to every trial - we cannot take notes in a working memory test, ask people about their internal states in emotion recognition paradigms, chose a cognitive task best suited to our skills, or simply respond "I do not know". While not allowing "I do not know" responses makes experimental and statistical sense, this disregards how decisions are made in the real world and the metacognitive insights this information represents. Indeed, people do not blindly act on all learning[216]. Whether information is acted upon is determined by a person's metacognitive sensitivity (confidence), the extent to which confidence determines behaviour (metacognitive control), and the relative cost-benefit of Type I or Type II (e.g., a false positive or false negative)[427]. For example, a surgeon may be highly confident in a diagnosis but request a second opinion if the potential consequences are severe. A lack of consideration of these processes assumes that participants are devoid of metacognition and act on their raw cognitive ability alone – which is not the case.

These considerations were implemented by Koren et al[427], who expanded the response dimensions of the WCST to also include metacognitive indices. Firstly, participants were told that their task score would increment for each correct decision. Next, in addition to requesting the participants' card sort decision, participants were also asked to rate their confidence in these decisions and, critically, whether this decision should affect their overall score (see **Figure 13**). If participants volunteered their response their score was updated accordingly, however, if the response was withheld their score was unaffected. From this paradigm two accuracy metrics can be calculated: total accuracy of the entire response set (i.e., number of correct responses ÷ total trials) and Koren accuracy (number of volunteered correct responses ÷ total volunteered responses). While total accuracy represents raw cognitive ability and acting on ability, Koren accuracy represents *acting* on ability more directly (metacognitive control). In their initial studies, Koren et al. reported that although psychosis patients presented total accuracy deficits in the WCST, no deficits in Koren accuracy were reported. Moreover, metacognitive indices were found to predict functioning over and above basic neurocognition; highlighting they tap into separate processes. These findings suggested that patients had poorer neurocognitive ability, but intact metacognitive abilities. Koren accuracy is thus immediately relevant for the current study's aims of separating cognition and metacognition and explaining how patients with poor neurocognition go on to have good functional outcomes.

However, there are limited assessments of these processes in the psychosis-spectrum. For example, patients have been found to ineffectively allocate greater encoding time to less-known

stimuli during learning tasks[428], relative to healthy controls, meaning cognitive deficits are exacerbated. Moreover, there are currently no assessments of Koren accuracy in psychometric schizotypy, which may highlight potential 'coping strategies' in individuals high in psychotic-like traits but with good functioning. Moreover, a breakdown of these strategies may be predictive of transition to psychosis.

1.3: Self-reported metacognition

Metacognitive research has become increasingly relevant for schizotypy, however, commonly this relies on subjective self-report methods. The use of Koren accuracy and confidence ratings in the current research provides a behavioural measure of metacognition, which is currently needed in the psychosis literature[27]. However, self-report measures are still highly useful and so they are still applied here. As the Beck's Cognitive Insight Scale (BCIS) may not be an appropriate measure of metacognition for non-clinical samples (see Chapter 2), the current Chapter adds a more applicable measure of metacognition – the Metacognitions Questions (MCQ-30). The MCQ-30 assesses metacognitive processes more generally, is relevant for non-clinical samples, and is generally more reliable than the BCIS (see **2.2: Materials and Apparatus** for details).

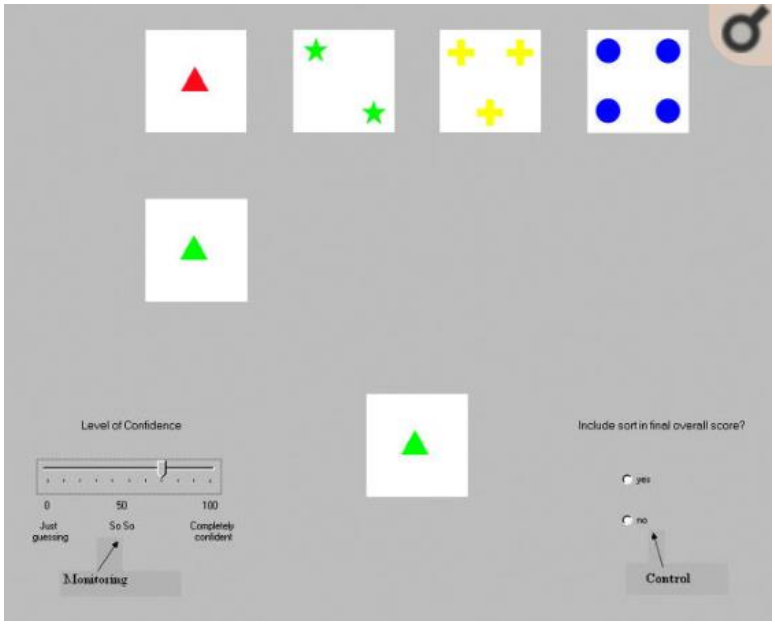


Figure 13. Trials from the metacognitive Wisconsin Card Sorting Task (WCST) reproduced from Koren et al., 2006. The authors separated neurocognitive ability from metacognitive sensitivity and metacognitive control.

1.4: Aims and hypotheses

This Chapter aims to investigate potential explanations for poor performance on Probabilistic Reversal Learning (PRL) and set-shifting tasks (IDED) associated with psychometric schizotypy. In Chapter 2, it was suggested that the single response dimension of accuracy conflated too many cognitive processes, meaning explanations for the association between schizotypy and performance became more difficult. Consequently, the response dimensions of the tasks were expanded to assess neurocognition and metacognition individually. Moreover, this chapter continues to assess the role of negative affect, psychometric metacognition, and motivation in neurocognitive and metacognitive task performance. It was hypothesised that:

1a) Positive schizotypy would predict poorer learning of punishing stimuli in the Metacognitive Probabilistic Reversal Learning (M-PRL) task as measured by total accuracy.

1b) Positive schizotypy would *not* predict poorer learning of punishing stimuli in the M-PRL task as measured by Koren Accuracy.

2a) Positive schizotypy would predict a greater number of Extra-Dimensional Set-shifting errors in the Metacognitive Intra/Extra-dimensional (M-IDED) set-shifting task.

2b) Positive schizotypy would predict a greater number of Extra-Dimensional Set-shifting errors in the Intra/Extra-dimensional (M-IDED) set-shifting task.

2.0: Methods

2.1: Participants

From an initial 348 participants that attempted the online experiment 259 were included (M-IDED = 256, M-PRL = 219). The sample was recruited through Prolific (45.9%), call for participants (12.3%), the University's recruitment system (20%) and word of mouth (21.6%). In this sample, 57% were biologically female, ages ranged from 18 – 65 years old (*M* = 25.6, *SD* = 8.4), 80% had or were studying for at least an undergraduate level qualification, 45% were current students, and 46% were currently employed. Of the 72.7% of participants that volunteered responses 11 participants reported taking anti-depressants, 1 participant reported beta-blockers, but no participant reported anti-psychotic medication. This study was approved by the University of Nottingham School of Psychology Ethics Committee, following the British Psychological Society's Code of Ethics and Conduct.

2.2: Materials and Apparatus

The O-LIFE, DASS-21, BCIS, MIAMI, and Task Strategy Questionnaire (TSQ) have been described in the previous Chapter and a full description of their content is omitted here. However, to quickly reiterate, the O-LIFE measures schizotypy, the DASS-21 measures negative affect, the BCIS contains the subscales of Self-Reflectivity and Self-Certainty that measure moderately discrete metacognition (see chapter 1, metacognition), the MIAMI is a cognitive test specific measure of pre-task and post-task motivation, and the TSQ assesses participant's different approached to the PRL task. Only the five motivation items of the MIAMI were used to reduce the experimental procedure time. In addition to these measures, the Metacognitions Questionnaire (MCQ-30**, Appendix A.**)[163] was used to assess moderately discrete metacognition. Higher scores represent a disruption of self-regulation and biased attention which exacerbate perseverative thinking styles[316]. The scale has five subscales: Cognitive Confidence (CC) which describes under-confidence in cognition i.e., "I have poor memory"; Positive Beliefs about worry (PB), meaning that reflection and worry are perceived as a beneficial strategy i.e., "Worrying helps me cope"; Cognitive Self-Consciousness (CSC) which is an over-awareness or preoccupation with thinking i.e., "I constantly examine my thoughts"; negative thoughts about Uncontrollability and Danger (UD) describing an inability to stop dysfunctional thinking, "My worrying could make me go mad"; and the Need to Control Thoughts (NC), "If I could not control my thoughts, I would not be able to function". The scale has been shown to have good to excellent reliability in its original conception (α = .72 - .93)[163] and in more recent investigations (ω_H =

0.85)[429]. Both experimental tasks were programmed using PsychoPy[388] and administered using Pavlovia and questionnaires were completed via Qualtrics.

2.3: Experimental Task Design

2.3.1: Metacognitive Probabilistic Reversal Learning Task (M-PRL)

The PRL from Chapter 2 was adapted to expand the response dimensions to include neurocognition, confidence, and acting on knowledge (**Figure 6**). Each trial began with a fixation cross in the centre of the screen for 200ms along with a running total of the participant's score at the top of the screen. Next, one of the two cues was presented along with the question "Do you think you will win?" (neurocognition). Participants could respond with the "Y" key for yes and the "N" key for no or by clicking the respective option with the mouse. After a response, a numerical keypad appeared to the right of the screen which asked participants how much they would like to bet on this decision (confidence) using the keyboard or mouse. Any value between 10p and £1 was valid. After entering their bet, a new screen was presented appeared asking participants if they would like to include this decision in their point total (acting on knowledge). The trial feedback was then presented on a new screen which displayed the wagered amount in green or red text (e.g., +84p or -15p) dependent on decision accuracy. If participants did not volunteer this response a subtext of "ignored" also accompanied the feedback. The next trial started with the fixation cross and an updated score total. The same sound effects of Chapter 2 were used for consistency. Critically, only two rather than four blocks of trials 60 trials were presented due to the increased task time. This design chosen is supported by the non-significant interaction between schizotypy and block in Chapter 2.

2.3.2: Metacognitive Intra/Extra-Dimensional (M-IDED) set-shifting task

The IDED task from Chapter 2 underwent similar adaptations to the M-PRL (**Figure 14**). The task design was identical to that described in Chapter 2 until a response was given. Rather than moving directly onto feedback, selecting a shape instead highlighted that option and a confidence scale was then presented. This asked participants to rate their confidence in this decision on a scale of 1 (low confidence) to 7 (high confidence). After participants used the keyboard or mouse to select a confidence rating a new screen was presented, asking participants if they would like to include this decision in their point total (identical to the M-PRL). After an option was selected, feedback began with all four borders changing to green or red depending on decision accuracy. If participants included the current response a scoreboard in the top right corner would flash in the same colour and with an updated score (i.e., Score 2, Score 1). This text would remain white if the response was excluded. The next trial then began with the fixation cross and the processes was repeated.

2.4: Procedure

2.4.1: Survey

The study began with participants being directed to a Qualtrics survey which explained the procedure. After giving informed consent and creating unique identifiers, demographic information was also collected including education, employment status, household income, and a voluntary question requesting current medication. Next, the survey randomised participants to complete either the questionnaires or the experimental tasks first. The questionnaire block presented each questionnaire and its respective items in a randomised order. For ethical reasons "Prefer not to say" response options were added to all questions. Inattention items were also added to the MCQ-30 and the O-LIFE which stated "*This is an attention check, please choose Prefer not to say*". The experimental task section began with the Geneva Emotion Recognition Task (the focus of Chapter 4), followed by the M-IDED and then the M-PRL. Participants opened a separate survey link for each of the tasks.

2.4.2: Metacognitive-IDED

The M-IDED began with similar instructions to the IDED (see **Appendix B.**), except clarifying the correct shape had been pre-determined and the location of the shapes did not correspond to the correct answers. The latter point was a common complaint of miscomprehension in Chapter 2 and is consistent with previous research[348]. Further clarification was also given stating the rules would change after a certain number of correct decisions. Therefore, their task was to discover the rules behind the game and that they would initially need to guess. Participants were then told they would gain a point for a correct response but lose a point for an incorrect response. After the confidence ratings were explained, participants were told they could choose to exclude the shape decision from affecting their score. Instructional videos (here) were then presented that guided participants through practice trials with other shapes. After the video, participants completed the pre-task MIAMI scale. As with Chapter 2, participants progressed through the nine stages or skipped to the end if they failed a stage (> 25 errors). After the M-IDED participants completed the post-task MIAMI and to proceeded to the M-PRL.

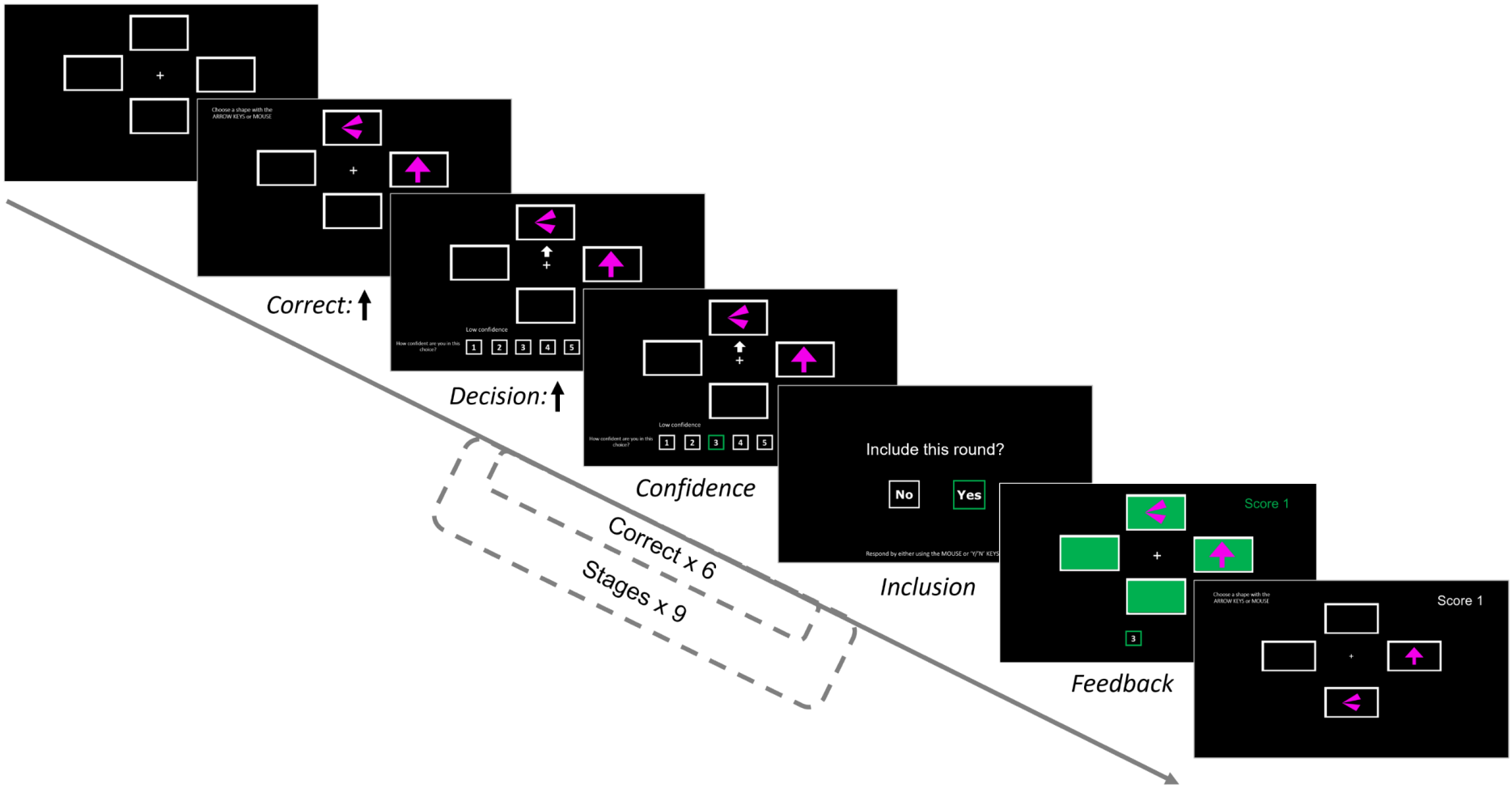


Figure 14. Trial structure of the metacognitive Intra/Extra-dimensional (M-IDED) set-shifting task. In addition to asking participants which shape they believed was correct, they were also asked to rate their confidence on a scale of 1 (low confidence) to 7 (high confidence) and whether to include this round in their points total.

2.4.3: Metacognitive-PRL

The M-PRL began similarly to the PRL task of Chapter 2, but with clearer instructions. The welcome instructions again stated the aim of participants was to win as much virtual money as possible (see **Appendix C**), but they additionally clarified that the task had already decided if participants would win or lose each round and that the symbols presented at each round may predict these outcomes. Specifically, participants were told they could not control *whether* they won or lost, only *how much* they won or lost. Participants should do this by choosing both how much money they wanted to wager and whether they would like to include the bet in their overall score. Further instructions were given explaining how to interact with the task and instructional videos (here) guided participants through several trials using different symbols. After this explanation, participants completed a second pre-task MIAMI questionnaire before moving on to the task. After the M-PRL, participants completed the second post-task MIAMI and the TSQ. Finally, participants were re-directed back to Qualtrics to continue the study. The entire study lasted between 55 and 65 minutes.

2.5: Data preparation

A total of 388 participants began the online study with 341 participants finishing the questionnaires. From this sample, four were excluded for excessive "Prefer not to say" responses, two requested the removal of all experimental data, and 15 failed at least one inattention item. This left 312 participants with complete questionnaire responses. Behavioural task data were retrieved for 256 participants who completed the M-IDED and 219 completing the M-PRL. The primary reasons for this were likely technical issues and participant fatigue. For data cleaning, outliers were not removed as low motivation was a key aim of all studies in this thesis. Missing data including "Prefer not to say" responses were imputed using the missForest R package[389]. Missing data are given in (percentage) and imputed error measured by [percentage falsely classified] were as follows: O-LIFE (0.46%[23.4%], DASS-21 (0.38%)[42.7%], BCIS (0.30%)[53.8%], MCQ-30 (0%).

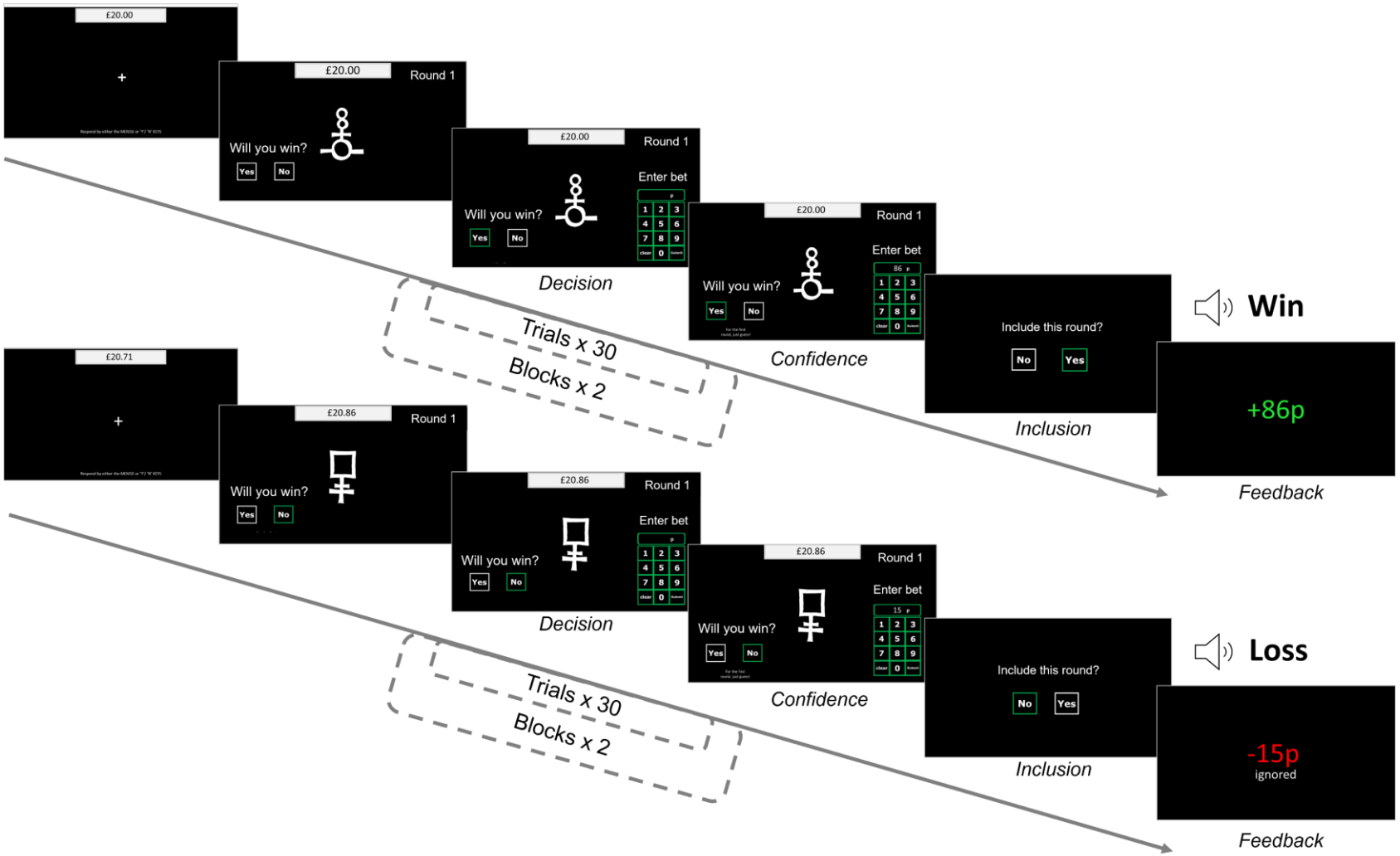


Figure 15. Trial structure of the Metacognitive Probabilistic Reversal Learning (M-PRL) task. **Top**: rewarding trial in which the participant correctly included the correct decision in their total. **Bottom**: punishing trial in which the participant correctly excludes this response from affecting their total

2.6: Analysis strategy

Pre-analysis checks were conducted including internal consistency, normative comparisons, and additional validation of the M-PRL due to the novel task adaptations to confirm that learning had occurred. For the M-PRL, the primary outcome variable was the accuracy of neurocognitive judgments (i.e., selecting "I will win" for the rewarding cue). The same method as Chapter 2 was applied for calculating increasingly complex Linear Mixed-Effects models (LME, *Hypothesis 1a*). For Koren accuracy, the adaptive response for the rewarding contingency was to include decisions (gain money), but to exclude decisions for the punishing contingency (avoid loss) and so performance on excluded rather than included punishing trials were analysed. The same LME model was then applied to Koren accuracy with a non-significant association between schizotypy and Koren accuracy being expected (*Hypothesis 1b*). A further LME model predicted confidence ratings (money wagered) from the three schizotypal scales. For the M-PRL, adaptive confidence allocation involved larger bets on the rewarding cue and lower bets on the punishing cue. For the M-IDED, the primary outcome variable was the number of errors (*Hypothesis 2a*) and Koren errors (*Hypothesis 2b*) of the EDS stage, which was again predicted by the three schizotypy scales using Poisson regressions. Metacognitive sensitivity was assessed by predicting confidence ratings for correct and indirect judgements using multiple regression. Finally, two Bayesian Spearman correlation matrices were created: the first to identify potential mediators of task performance by correlating schizotypy with other psychometric variables and the second to assess the associations between psychometric and behavioural metacognition measures. Analyses were conducted in R studio[393], Jamovi[394] and JASP[395] using several statistical[396,397,430] and data visualisation packages[401,402].

3.0: Results

3.1: Descriptive statistics

Descriptive summaries of psychometric data can be found in **Table 8** and task performance data in **Appendix D**. The O-LIFE, DASS-21, and all MCQ-30 scales except for the Need to Control Thought (NCT, $\omega_T = .78$) presented good to excellent internal consistency ($\omega_T > .80$), the pre-task PRL MIAMI, post-task IDED MIAMI, post-task IDED MIAMI and Self-reflectivity were acceptable ($\omega_T > .7$), and both Self-Certainty and pre-task IDED MIAMI were questionable ($\omega_T > .6$). All psychometric data were non-normally distributed (all $p_{FDR} < .001$). A 2 (continency) x 6 (bins of 10 trials) repeated measures ANOVA confirmed that learning occurred in the first Block of 60 trials before performance dropped after the contingency reversal (see **Appendix E.**). For the IDED, the greatest number of errors came from the EDS stage ($M = 6.7$, SD = 7.1). The pattern of data for the PRL task is described in **3.4.1: Baseline models**.

3.2: Normative comparisons

Normative comparisons of the O-LIFE[293] and DASS-21[403] were conducted using One-Samples Wilcoxon signed-rank tests. Positive schizotypy was significantly lower in the current sample relative to normative data at a small effect size ($p = .037$, $r_{rb} = 0.16$), negative schizotypy was significantly higher at a large effect size ($p = .001$, $r_{rb} = 0.78$), and disorganised schizotypy did not differ ($p = .407$). The DASS-21 total was significantly higher in the current sample at a large effect size ($p < .001$, $r_{rb} = 0.83$). The DASS-21 recommended cut-off criteria for normal, mild, moderate, severe, and extreme levels of each trait were as follows: *Depression*: 42.1%, 14.3%, 17.0%, 11.6%, and 15.1%; *Anxiety*: 53.7%, 8.1%, 15.8%, 6.9%, and 15.4%; and *Stress*: 56.4%, 15.1%, 9.3%, 16.6%, and 2.7%.

3.3: Psychometric relationships

3.3.1: Potential mediators

All three schizotypy dimensions were positive associated with negative affect ($r_s = 0.21 – 0.64$, $BF_{10} > 30$), but there was no evidence schizotypy was associated to any measure of motivation in either task ($r_s = -0.01 – -0.12$, $BF_{10} < 3$). For metacognition, greater Self-Reflectivity was associated with higher levels of disorganised ($r_s = 0.52$, $BF_{10} > 999$) and positive schizotypy ($r_s = 0.28$, $BF_{10} > 100$), but evidence for negative schizotypy was inconclusive ($r_s = 0.13$, $BF_{10} = 1.37$). There was no evidence that schizotypy was related to Self-Certainty ($r_s = 0.03 – 0.14$, $BF_{10} < 3$). For the MCQ-30, positive

schizotypy was associated with greater levels of maladaptive traits across all scale (r_s = 0.26 – 0.35, BF_{10} > 100) except Cognitive Confidence (CC, r_s = -0.14, BF_{10} < 3), negative schizotypy was related to increased Uncontrollability and Danger (UD, r_s = 0.18, BF_{10} > 3) and the Need to Control Thoughts (NCT, r_s = 0.25, BF_{10} > 100), and disorganised schizotypy was associated with all five maladaptive subscales (r_s = 0.31 – 0.60, BF_{10} > 100). Overall, negative affect, Self-Reflectivity, and all five MCQ-30 scales were potential mediators of performance (**Table 9**).

3.3.2: Psychometric and behavioural metacognition

Psychometric metacognition (BCIS and MCQ-30) was correlated with overall confidence, overall inclusion rates, and several metacognitive indices: Free-Response Improvement (FRI = Koren accuracy – Total accuracy), the Confidence Gap (CG = Confidence when correct – confidence when incorrect), and Control Sensitivity (Confidence when included – Confidence when excluded). Higher values on each of these indices indicate improved metacognitive performance[431]. Self-Certainty was associated with greater levels of overall confidence in the M-PRL (r_s = 0.18, BF_{10} = 5.3) but potentially not in the M-IDED (r_s = 0.11, BF_{10} = 0.715). The Need to Control thoughts scale of the MCQ-30 presented the same relationship to confidence ratings of the M-PRL (r_s = 0.16, BF_{10} = 3.1) and M-IDED (r_s = 0.12, BF_{10} = 0.935). All remaining associations either supported the null hypothesis (BF_{10} < 0.3) or the data were insensitivity to detect effects (0.3 > BF_{10} < 3). Overall, there was little evidence for associations between self-reported and behavioural metacognition **Table 7**.

Table 7. Bayesian Spearman correlation matrix of psychometric metacognition scales and behavioural metacognition scales.

	Confidence		**Inclusion**		**Sensitivity**		**Control**			
Variable	*PRL*	*IDED*	*PRL*	*IDED*	*CG IDED*	*CG PRL*	*FRI IDED*	*FRI PRL*	*KA IDED*	*KA PRL*
Self-Certainty	0.18*	0.11^{I}	0.05^{N}	0.05^{N}	-0.05^{N}	-0.05^{N}	-0.02^{N}	-0.01^{N}	0.01^{N}	-0.04^{N}
Self-Reflectivity	0.06^{N}	0.01^{N}	0.10^{I}	-0.01^{N}	0.04^{N}	-0.04^{N}	0.05^{N}	0.07^{N}	-0.04^{N}	0.01^{N}
Cognitive Confidence	-0.11^{I}	-0.05^{N}	0.07^{N}	-0.06^{N}	0.07^{N}	-0.02^{N}	0.03^{N}	0.01^{N}	-0.09^{N}	-0.04^{N}
Positive Beliefs	-0.05^{N}	0.04^{N}	0.01^{N}	0.01^{N}	0.03^{N}	-0.07^{N}	0.07^{N}	-0.04^{N}	-0.14^{I}	-0.06^{N}
Self-Consciousness	0.09^{I}	0.04^{N}	-0.02^{N}	0.06^{N}	-0.08^{I}	0.01^{N}	0.14^{I}	-0.05^{N}	-0.04^{N}	-0.08^{I}
Controllability + Danger	-0.05^{N}	-0.06^{N}	0.10^{I}	0.05^{N}	0.09^{I}	-0.01^{N}	0.04^{N}	0.03^{N}	-0.03^{N}	-0.04^{N}
Need to control	0.16*	0.12^{I}	0.10^{I}	0.03^{N}	0.01^{N}	-0.07^{N}	-0.01^{N}	-0.05^{N}	-0.03^{N}	-0.09^{N}

Note: FRI = Free-Response Improvement, KA = Koren Accuracy, CG = Confidence Gap. ^{N} = supports the Null, ^{I} = insensitive, * = BF_{10} > 3, ** = BF_{10} > 10, *** = BF_{10} > 30, ^{D} = >100 for 'decisive'.

Table 8. Psychometric descriptive statistics and normative comparisons of the current data.

	Current								Normative							Comparison		
Scale	**Range**	**M**	**SD**	**Med**	**MAD**	**IQR**	**ω_T**	**α**	**N**	**Range**	**M**	**SD**	**Median**	**IQR**	**α**	**ES**	***p***	**Normality**
Unex	0-25	8.4	5.9	7	5.9	8	0.87	0.87	402	0-30	10.0	6.305	9	11	-	-.159	=.037	< .001
Intan	0-24	8.61	5.7	8	5.9	8	0.86	0.86	402	0-27	5.44	4.00	4	5	-	.777	=.001	< .001
Cogdis	0-24	13.2	6.2	14	7.4	9	0.88	0.88	402	0-24	12.4	5.690	13	8	-	.061	=.407	< .001
DASS Total	0-108	37.5	26.5	32	26.7	40	0.94	0.94	1794	0-122	18.7	19.32	14	-	0.88	.831	=.001	< .001
Depression	0-42	13.9	11.1	12	11.9	18	0.91	0.91	1794	0-42	5.66	7.74	2	-	0.82	.954	=.001	< .001
Anxiety	0-40	9.1	8.7	6	5.9	12	0.83	0.82	1794	0-42	3.76	5.90	2	-	0.90	.868	=.001	< .001
Stress	0-42	14.4	10	14	11.9	14	0.85	0.85	1794	0-42	9.46	8.40	8	-	0.93	.651	=.001	< .001
BCIS_R	13-34	22.2	4.3	22	4.4	5	0.74	0.73	-	-	-	-	-	-	-	-	-	< .001
BCIS_C	6-22	13.3	3.1	13	3.0	4	0.61	0.66	-	-	-	-	-	-	-	-	-	< .001
MCQ_CC	6 -24	11.4	4.3	11	4.4	6	0.85	0.81	-	-	-	-	-	-	-	-	-	< .001
MCQ_PB	6 -24	10.2	4.5	9	4.4	7	0.90	0.90	-	-	-	-	-	-	-	-	-	< .001
MCQ_CSC	6 -24	16.8	5.1	18	5.9	8.5	0.91	0.91	-	-	-	-	-	-	-	-	-	< .001
MCQ_UD	6 -24	13.2	5.5	12	7.4	9.5	0.92	0.92	-	-	-	-	-	-	-	-	-	< .001
MCQ_NC	6 -24	12.0	4.1	12	4.4	6	0.78	0.77	-	-	-	-	-	-	-	-	-	< .001
MIAMI Pre IDED	9 - 20	17.0	2.4	17	3.0	4	0.69	0.68	-	-	-	-	-	-	-	-	-	< .001
MIAMI Post IDED	7 - 20	16.6	2.7	17	3.0	4	0.80	0.79	-	-	-	-	-	-	-	-	-	< .001
Understand IDED	1 - 4	3.2	0.9	3	1.483	2	-	-										< .001
MIAMI Pre PLR	5 - 20	16.1	3.4	16	3.0	5	0.79	0.79	-	-	-	-	-	-	-	-	-	< .001
MIAMI Post PRL	5 - 20	15.2	3.5	15	4.4	5	0.84	0.83	-	-	-	-	-	-	-	-	-	< .001
Understand PRL	1 - 4	2.1	1.0	2	1.483	1	-	-	-	-	-	-	-	-	-	-	-	< .001

Note: IQR – interquartile range, MAD = robust median absolute deviation, ω = McDonald's Omega total, *ES* = effect size which are rank-biserial correlations. Comparison tests are Wilcoxon signed-rank tests and normality tests are Shapiro-Wilk tests. All *p* values are FDR corrected. [A] = Values from Mason & Claridge, 2005., [B] Henry & Crawford, 2005.

Table 9. Bayesian Spearman correlation matrix of psychometric data

	Schizotypy			Neg Affect	Metacognition							Motivation		
Variable	*Pos*	*Neg*	*Dis*	*Total*	*S-Refl*	*S-Cert*	*CC*	*PB*	*CSC*	*UD*	*NCT*	*Pre-P*	*Pos-P*	*Pre-I*
Positive Schizotypy	-													
Negative Schizotypy	0.02^{N}	-												
Disorganised Schizotypy	0.41^{D}	0.19^{**}	-											
Negative Affect	0.30^{D}	0.21^{***}	0.64^{D}	-										
Self-Certainty	0.14^{I}	0.08^{I}	0.03^{I}	0.13^{I}	-									
Self-Reflectivity	0.28^{D}	0.13^{I}	0.52^{D}	0.46^{D}	0.01^{N}	-								
Cognitive Confidence	0.14^{I}	0.09^{I}	0.39^{D}	0.28^{D}	-0.08^{I}	0.36^{D}	-							
Positive Beliefs	0.26^{D}	0.02^{N}	0.31^{D}	0.27^{D}	0.09^{N}	0.29^{D}	0.33^{D}	-						
Self-Consciousness	0.30^{D}	0.06^{N}	0.31^{D}	0.31^{D}	0.16^{*}	0.28^{D}	0.09^{I}	0.23^{D}	-					
Controllability + Danger	0.35^{D}	0.18^{*}	0.60^{D}	0.55^{D}	0.01^{N}	0.48^{D}	0.38^{D}	0.35^{D}	0.39^{D}	-				
Need to control	0.29^{D}	0.25^{D}	0.31^{D}	0.35^{D}	0.26^{D}	0.39^{D}	0.17^{*}	0.23^{D}	0.33^{D}	0.47^{D}	-			
Pre-task Motiv M-PRL	-0.08^{I}	0.10^{I}	0.01^{I}	0.05^{N}	-0.04^{N}	-0.12^{I}	-0.09^{I}	0.03^{N}	-0.06^{N}	-0.11^{I}	0.06^{N}	-		
Post-task Motiv M-PRL	-0.01^{N}	0.12^{I}	-0.06^{I}	-0.05^{N}	0.08^{I}	-0.20^{**}	-0.10^{I}	-0.11^{I}	-0.01^{N}	-0.06^{N}	-0.02^{N}	0.45^{D}	-	
Pre-task Motiv M-IDED	-0.04^{N}	0.05^{N}	-0.11^{N}	-0.01^{N}	-0.02^{I}	-0.13^{I}	-0.13^{I}	-0.09^{I}	-0.05^{N}	-0.14^{I}	-0.04^{N}	0.53^{D}	0.48^{D}	-
Post-task Motiv M-IDED	-0.07^{N}	0.06^{N}	-0.12^{I}	-0.05^{N}	0.04^{N}	-0.13^{I}	-0.06^{N}	-0.04^{N}	-0.08^{I}	-0.10^{I}	-0.02^{N}	0.39^{D}	0.53^{D}	0.51^{D}

Note: ^{N} = supports the Null, ^{I} = insensitive, * = $BF_{10} > 3$, ** = $BF_{10} > 10$, *** = $BF_{10} > 30$, ^{D} = >100 for 'decisive'.

3.4: Metacognitive-Probabilistic Reversal Learning

3.4.1: Baseline models

The overall performance of all participants are plotted in **Figure 16** left, which visualized the optimal response percentage divided by Block (Block 1 and Block 2) and each contingency. Overall, participants performed more poorly after the contingencies were reversed (as expected). A null LME model was calculated estimating total accuracy from the random-effect of participants alone (AIC = 8024, R^2 = 14.3%). This was compared to further a baseline model adding Block and Contingency as fixed-effects, which was a significantly better fit to the data ($\chi^2(2) = 90.7$, $p < .001$, AIC: 7932.7, R^2 = 33.7%) and revealed a significant fixed-effect of both Block ($F(1, 652) = 86.6$, $p < .001$) and contingency and ($F(1, 652) = 10.4$, $p = .001$). The model revealed that performance for the rewarding cue was greater than that of the punishing cue ($d = 0.17$, $p < .001$) and performance decreased post contingency reversal between Block 1 and Block 2 (d = -0.55, $p < .001$, **Figure 16**). A further Block x Contingency interaction model was not a significantly better fit to the data ($\chi^2(1) = 2.7$, $p = .100$, AIC: 7928.0). The same Block + Continency model was then applied to Koren accuracy scores to assess changes in performance (metacognitive control). As a reminder, for the M-PRL Koren accuracy is the accuracy of volunteered responses for the rewarding cue ("I think I will win, and I want to include my bet"), but excluded responses for the punishing cue ("I think I will lose, and I want to exclude my bet"). This model revealed a significant fixed-effect of Block ($p < .001$) due to performance again dropping post-reversal. The effect of contingency ($p = .002$) was also significant, but unlike total accuracy, the performance was better for punishing rather than rewarding cues (R^2 = 57.2%, **Figure 16**). A separate within-participants t-test confirmed that Koren accuracy was greater than total accuracy across participants ($p < .001$, BF10 > 999, $d = 1.014$), meaning the responses participants included were more likely to be correct than all their responses.

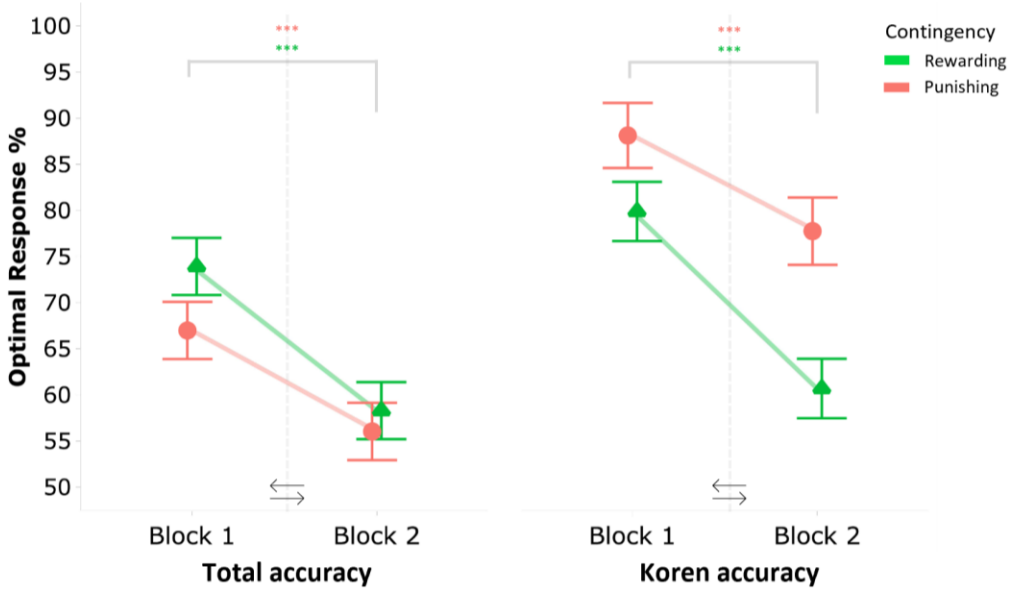


Figure 16. Linear Mixed-Effects models estimating M-PRL performance in the entire sample. ***Left***: total accuracy. ***Right***: Koren accuracy of included rewarding trials and excluded punishing trials. Note: ⇆ = reversal.

3.4.2 Total accuracy

Further models adding the three schizotypy scales to the baseline model were calculated. Neither the model adding the three schizotypy scales ($\chi^2(3) = 1.760$, $p = .624$, AIC: 7944, $R^2 = 34.3\%$) nor their interaction with Block ($\chi2(6) = 10.424$, $p = .108$, AIC: 7941, $R^2 = 35.8\%$) was a significantly better fit to the data. However, a further model adding Contingency x Schizotypy interactions returned as a better fit ($\chi2(6) = 13.714$, $p = .033$, AIC: 7938, $R^2 = 36.4\%$). As the interaction effect for negative schizotypy ($p = .213$) was non-significant, this model was re-calculated without this parameter (**Table 10**). In this final model, positive schizotypy predicted poorer overall task performance (β = -0.153[-263, -0.044], $p = .006$, **Figure 17**), which was due to poorer learning of punishing cues (B = -0.629[-1.080, -0.178], $p = .006$, Hypothesis 1a), but did not rewarding cues (B = 0.226[-0.225, 0.677], $p = .324$, **Figure 18**). There was a trend level interaction effect between disorganised schizotypy and Contingency ($p = .090$), however, disorganised schizotypy did not predict performance to the punishing cue (B = -0.241[-0.691, 0.210], $p = .294$) nor the rewarding cue individually (B = 0.201 [-0.249, 0.652], $p = .380$). Finally, neither the fixed-effects of negative affect ($p = .169$), Self-Reflectivity ($p = .367$), Self-Certainty ($p = .178$), pre-task motivation ($p = .595$), nor MCQ-30 total score nor any subscale ($p = .190$) significantly predicted or mediated total accuracy performance when added to this model.

Table 10. LME model predicting total accuracy from the three schizotypy scales

	Total Accuracy (%)					95% Conf Int	
predictor	B	SE	t	*p*	β	LC	HC
Intercept	69.1	3.364	20.5				
Contingency^{+}	5.409	3.790	1.427	= .154	0.111	-0.041	0.264
Block^{2}	-10.9	2.004	-5.463	< .001	-0.225	-0.305	-0.144
Contingency^{+} * Block^{2}	-4.679	2.834	-1.651	= .099	-0.083	-0.263	-0.044
Pos Scz	-0.629	0.229	-2.742	= .006	-0.153	-0.065	0.100
Neg Scz	0.079	0.193	0.408	= .684	0.017	-0.062	0.162
Dis Scz	0.201	0.229	0.879	= .380	0.050	-0.182	0.016
Contingency^{+} * Pos Scz	0.855	0.266	3.216	= .001	0.212	0.083	0.341
Contingency^{+} * Dis Scz	-0.442	0.260	-1.698	= .090	-0.144	-0.31	0.022

Note: Block^{2} = the effect of Block 2 relative to Block 1, Contingency^{+} = the effect of the positive contingency relative to the negative contingency. $R^2_{\text{fixed-effects}}$ = 26.8%, $R^2_{\text{ranomd-effects}}$ = 9.4%.

3.4.3 Koren Accuracy

The same Schizotypy x Contingency model was applied to Koren accuracy (**Table 11**). Positive schizotypy continued to predict poorer overall performance (B = -1.153, β = -0.259[-0.377, -0.141], *p* < .001, **Figure 17**), due to poorer performance of punishing cues (B = -1.152[-1.676, -0.626], *p* < .001) but not the rewarding cues (B = 0.229[-0.253, 0.710], *p* = .351, Hypothesis 1b). This suggests positive schizotypy predicts both poorer cognitive performance (total accuracy) and acting on faulty information (Koren Accuracy, **Figure 19**). Moreover, there was trend-level evidence (p = .051) that positive schizotypy presented stronger deficits in Koren accuracy than total accuracy – which would suggest participants are more likely to act on faulty information. Unexpectedly, disorganised schizotypy significantly predicted greater Koren accuracy (β = 0.171[0.051, 0.291], *p* = .005) due to increased performance of the punishing cue (B = 0.740[0.221, 1.259], *p* = .005), but not the rewarding cue (B = -0.200[-0.683, 0.282], *p* = .414). This meant that disorganised schizotypy presented intact neurocognition and an increased tendency to act on correct information (the opposite of positive schizotypy).

Table 11. LME model predicting Koren accuracy from the three schizotypy scales

	Koren Accuracy (%)					95% Conf Int	
predictor	**B**	**SE**	**t**	***p***	**β**	**LC**	**HC**
Intercept	88.044	3.835	22.959				
Contingency^{+}	-7.613	3.927	-1.939	= .053	-0.144	-0.289	0.002
Block^{2}	-10.281	2.212	-4.649	< .001	-0.195	-0.278	-0.113
Contingency^{+} * Block^{2}	-8.924	2.944	-3.031	= .003	-0.152	-0.251	-0.054
Positive Scz	-1.162	0.267	-4.353	= .000	-0.261	-0.378	-0.143
Negative Scz	0.019	0.220	0.085	= .932	0.004	-0.084	0.092
Disorganised Scz	0.733	0.263	2.786	= .006	0.169	0.050	0.289
Contingency^{+} * Pos Scz	1.391	0.280	4.974	< .001	0.327	0.198	0.456
Contingency^{+} * Dis Scz	-0.933	0.270	-3.456	= .001	-0.285	-0.446	-0.123

Note: Block^{2} = the effect of Block 2 relative to Block 1, Contingency^{+} = the effect of the positive contingency relative to the negative contingency. $R^2_{\text{fixed-effects}}$ = 17.1%, $R^2_{\text{ranomd-effects}}$ = 39.9%.

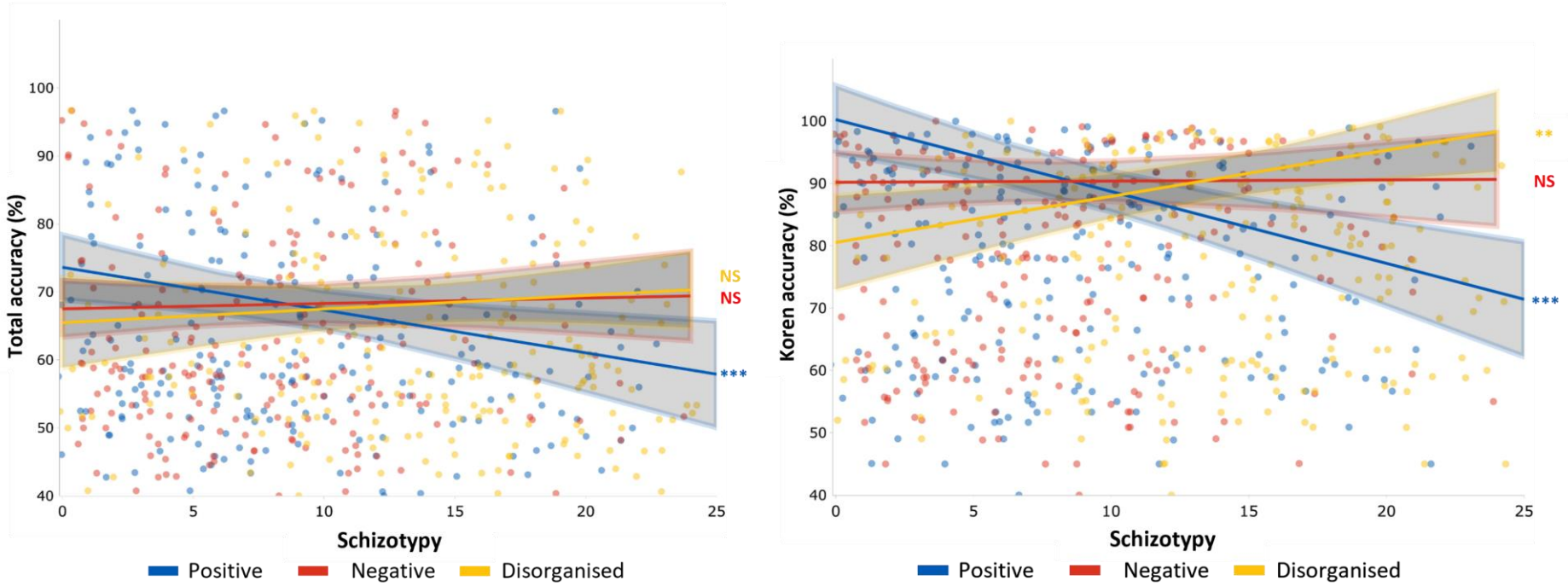


Figure 17. Plotted coefficients of the Linear Mixed-Effect models of the M-PRL. Positive schizotypy predicted poorer total accuracy performance (**Left**) and poorer Koren accuracy performance (**Right**). Disorganised schizotypy predicted improved Koren accuracy but not total accuracy.

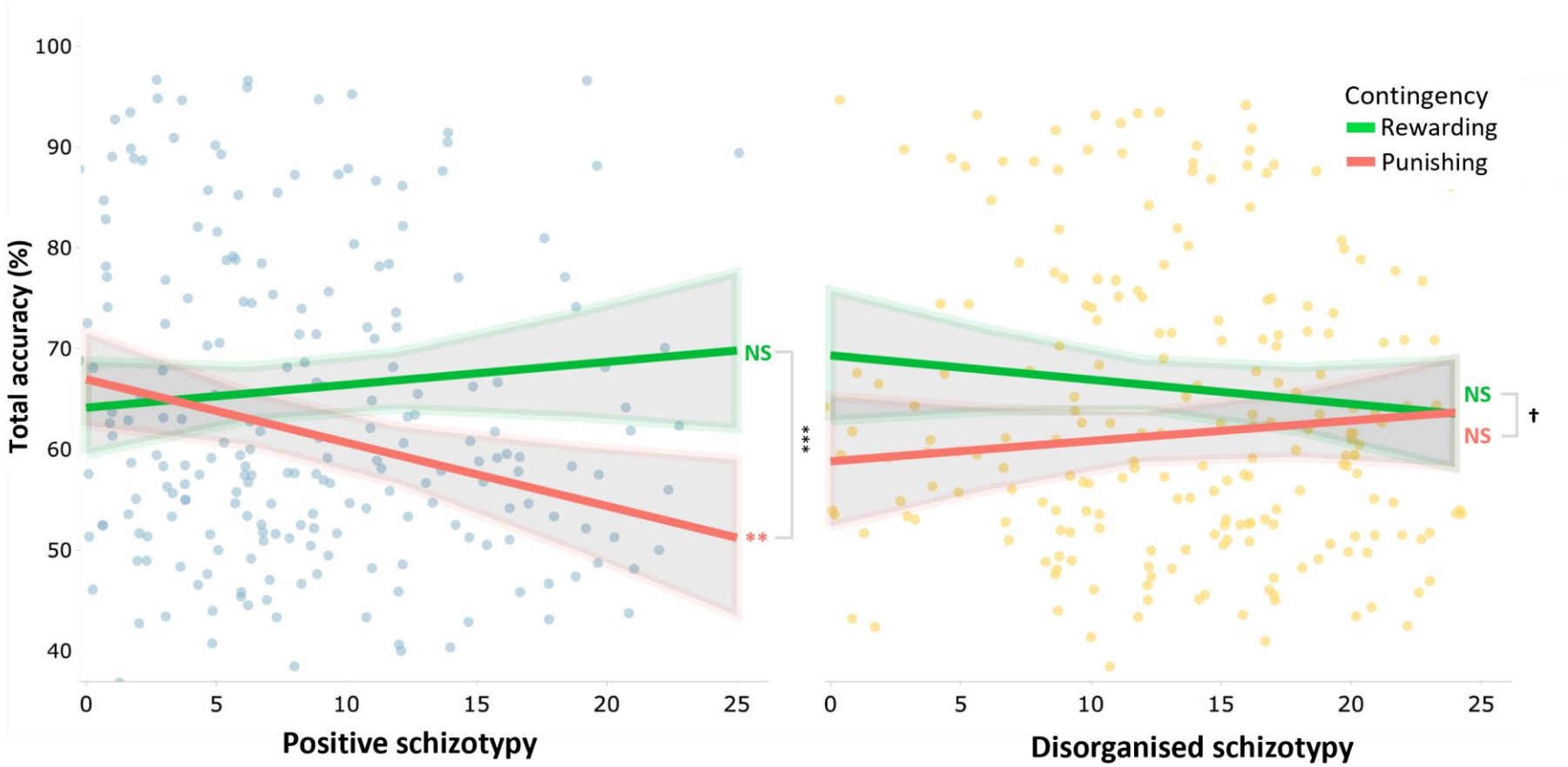


Figure 18. Plotted coefficients of the Linear Mixed-Effect models of the M-PRL divided by contingency. **Left**: positive schizotypy significantly predicted poorer punishing cue performance. **Right**: disorganised schizotypy did not predict performance.

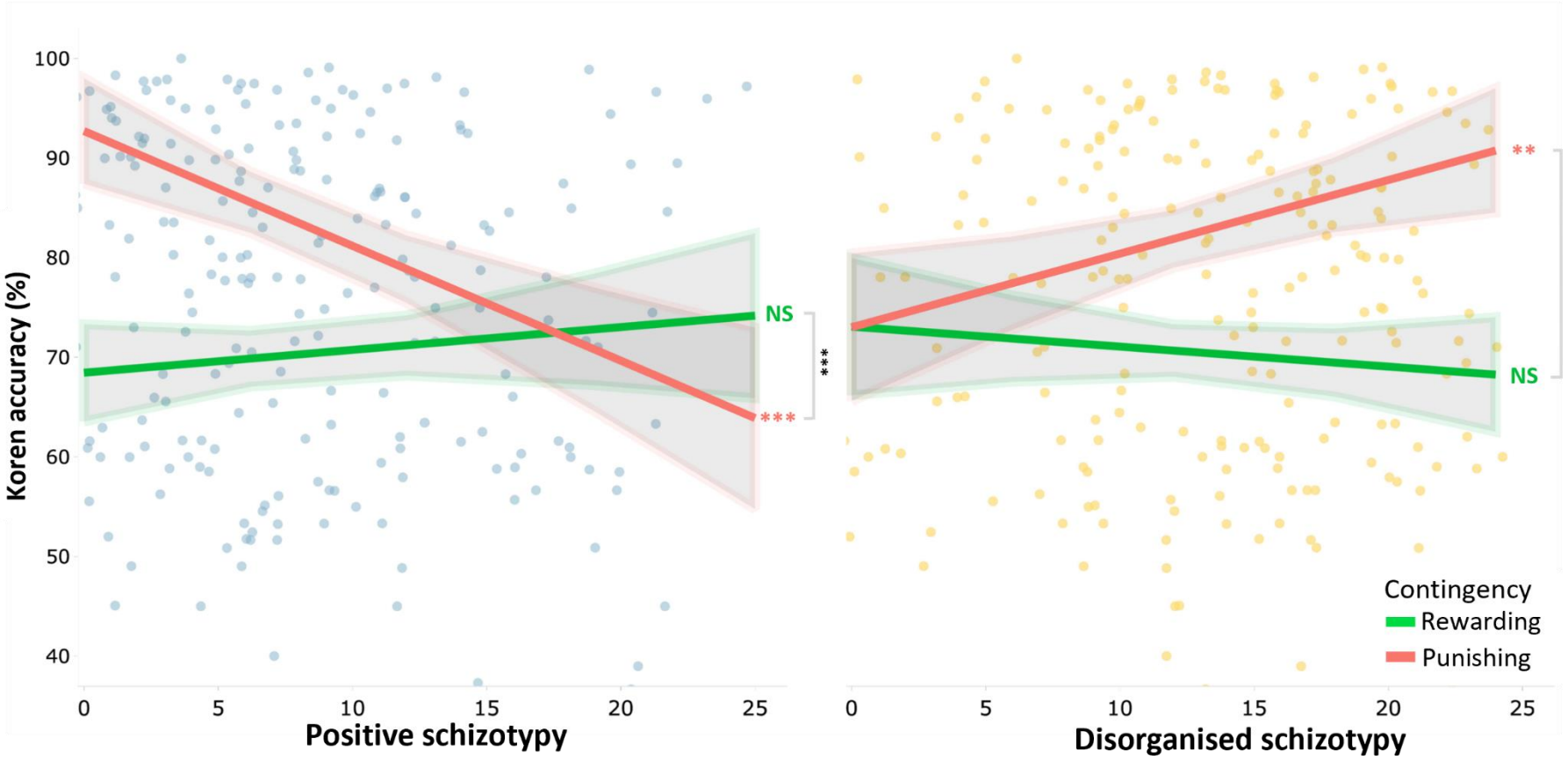


Figure 19. Linear Mixed Effect model results predicting Koren accuracy on the M-PRL. **Left**: Positive schizotypy predicted poorer performance to punishing cues but not rewarding cues. **Right**: disorganised schizotypy significantly predicted improved performance to punishing cues but not rewarding cues.

3.4.4 Confidence

Next, the outcome variable was changed to the amount of money bet on each trial (confidence). The analysis was divided into correct and incorrect judgements to avoid complex three-way interactions. As a reminder, the correct strategy is to bet more money on rewarding trials and less money on punishing trials. For both correct and incorrect judgments the baseline LME model was calculated as a Block x Contingency interaction model consistent with previous analyses. For correct judgments, this model revealed a significant fixed-effect of Contingency ($p < .001$) and Block ($p < .001$) which suggested more money was bet on the rewarding trials and (surprisingly) post-reversal (**Figure 20**). A Contingency x Block interaction effect ($p < .001$) further elaborated that the post-reversal increase was only significant for the punishing contingency, meaning when participants correctly knew they would lose they bet more money after the reversal than before the reversal. For incorrect decisions, participants bet significantly more money on the punishing cues relative to rewarding cues ($p < .001$, i.e., when they incorrectly thought they would win) and post-reversal ($p < .001$). The Block x Contingency interaction was not significant ($p = .112$, **Figure 20**). Overall, participants correctly bet more money on rewarding trials when they correctly believed they would win, relative to when they incorrectly believed they would win (Confidence Gap).

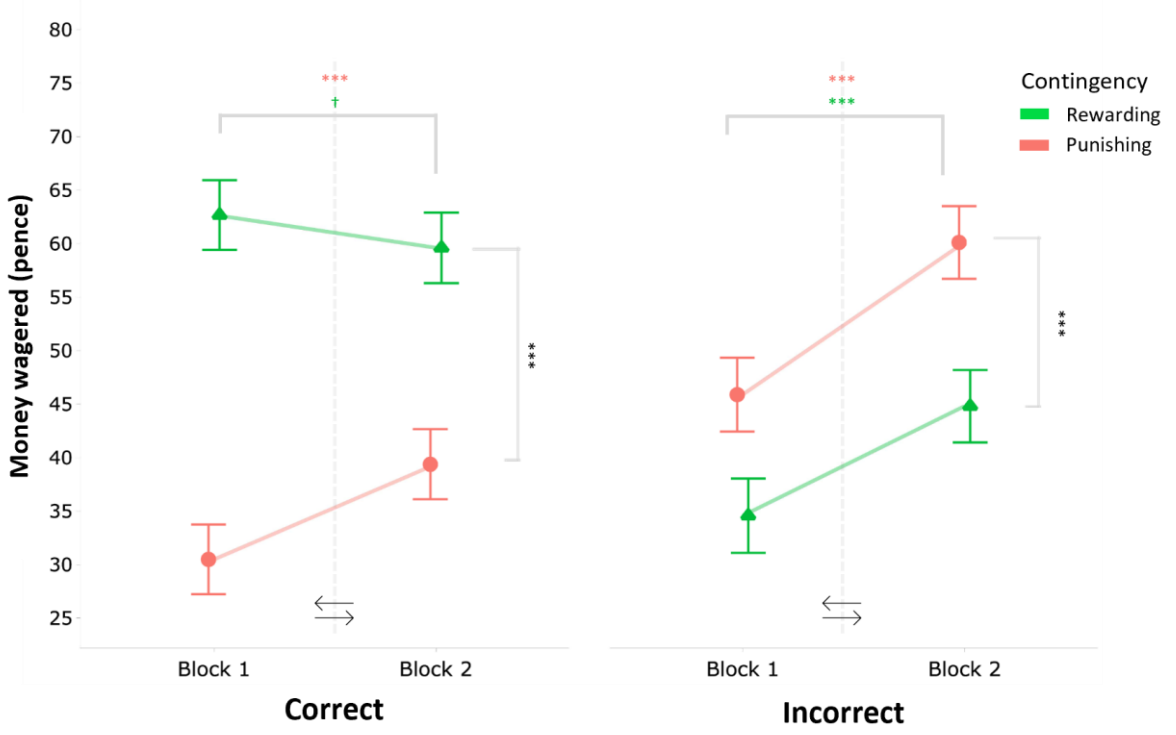


Figure 20. Linear Mixed effects models estimating confidence ratings (money wagered) in the M-PRL task. **Left**: correct responses. **Right**: incorrect responses. Note: ⇆ = reversal.

3.4.5 Confidence in correct decisions

Next, the three schizotypy scales were added to the correct decisions model. Neither a model adding schizotypy (p = .641) nor a schizotypy x Block interaction effect (p = .718) were significantly better fits to the data. However, a model specifying a schizotypy x Contingency interaction was a better fit, revealing a significant interaction effect of positive schizotypy alone (p < .001). After removing non-significant interaction effects the model was recalculated. However, this model was only a marginally better fit to the data ($\chi2(4) = 8.5$, p = .075, AIC: 7787). In this model, positive schizotypy predicted greater amounts of money bet overall (β = 0.134[0.023, 0.245], p = .019), due larger bets on the punishing cue (B = 0.631[0.105, 1.156], p = .019) but not the rewarding cue (B = -0.031[0.497, -0.115], p = .908). All other effects involving schizotypy were non-significant (p > .244, **Figure 21**).

3.4.5 Confidence in incorrect decisions

The process was repeated for incorrect decisions. The same model specifying a positive schizotypy Contingency interaction was not a significantly better fit to the data (p = .198), although there was a significant positive schizotypy x contingency interaction effect (β = 0.119[0.014, 0.225], p = .027), due to increased money wagered to incorrect rewarding cues (B = 0.612[0.057, 1.166], p = .031), but not incorrect punishing cues (B = 0.081[-0.472, 0.663], p = .774, **Figure 21**). No other effects of schizotypy were significant (p > .464, **Appendix F**). Overall, this suggested positive schizotypy predicts increased money wagered when they believed would lose money, but not when they believed they would win money.

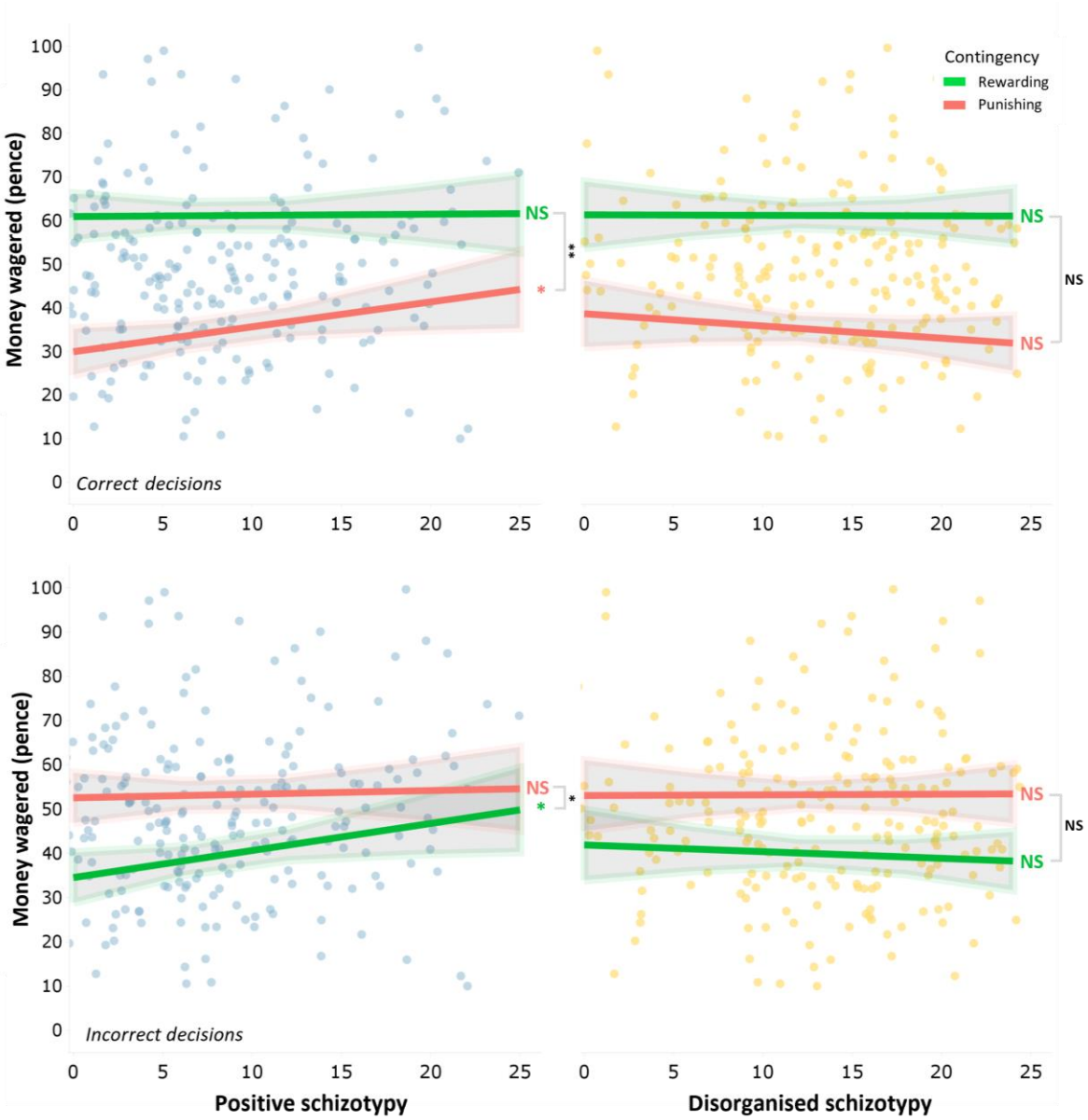


Figure 21. Linear-Mixed Effects models predicting confidence ratings (money wagered) on the M-PRL task. **Top**: correct decisions – those higher in positive schizotypy wagered more money when they correctly knew they would lose. **Bottom:** incorrect decisions – those higher in schizotypy wagered more money when they incorrectly thought they would win. Note: blue dots represent positive schizotypy (•) and yellow dots represent disorganised schizotypy (•).

3.5: Metacognitive-Intra/Extra-dimensional (M-IDED)

3.5.1: Total accuracy and Koren accuracy

The number of errors in the EDS stage of the M-IDED were much higher (M = 6.7, SD = 7.1) than errors in the other stages (< 2.5). A multiple Poisson regression predicting the number of EDS set-shifting errors found that no schizotypy scale predicted performance (p > .215, **Figure 22**). As schizotypy did not predict performance mediation analysis was not assessed. However, for completeness, neither negative affect (p = .156), Self-Reflectivity (p = .548), Self-Certainty (p = .202), nor pre-task motivation predicted performance (p = .467). However, the Positive Beliefs about Worry scale of the MCQ-30 (p = .021 OR = 0.986) and post-task motivation predicted fewer EDS errors (p < .001, OR = 0.929). The process was then repeated using 'Koren errors' (volunteered incorrect responses). There was trend-level evidence that disorganised schizotypy predicted fewer Koren errors (OR = 0.990, p = .051). However, neither positive schizotypy (OR = 0.995, p = .345) nor negative schizotypy (OR = 0.992, p = .113) were significant predictors (**Table 12**).

3.5.2 Confidence

For correct responses, a multiple regression revealed trend level evidence that disorganised schizotypy reduced correct decision confidence (β = -0.137, p = .063, BF_{10} = 0.615). Both positive schizotypy (BF_{10} = 0.186) and negative (BF_{10} = 0.187) were unrelated to confidence ratings. For incorrect responses, the analysis suggested both positive (BF_{10} = 0.217) and negative (BF_{10} = 0.190) schizotypy were unrelated to confidence. Disorganised schizotypy again returned as indecisive (BF_{10} = 0.407). Overall, there was limited evidence schizotypy was related to decision confidence in the M-IDED (**Table 13**).

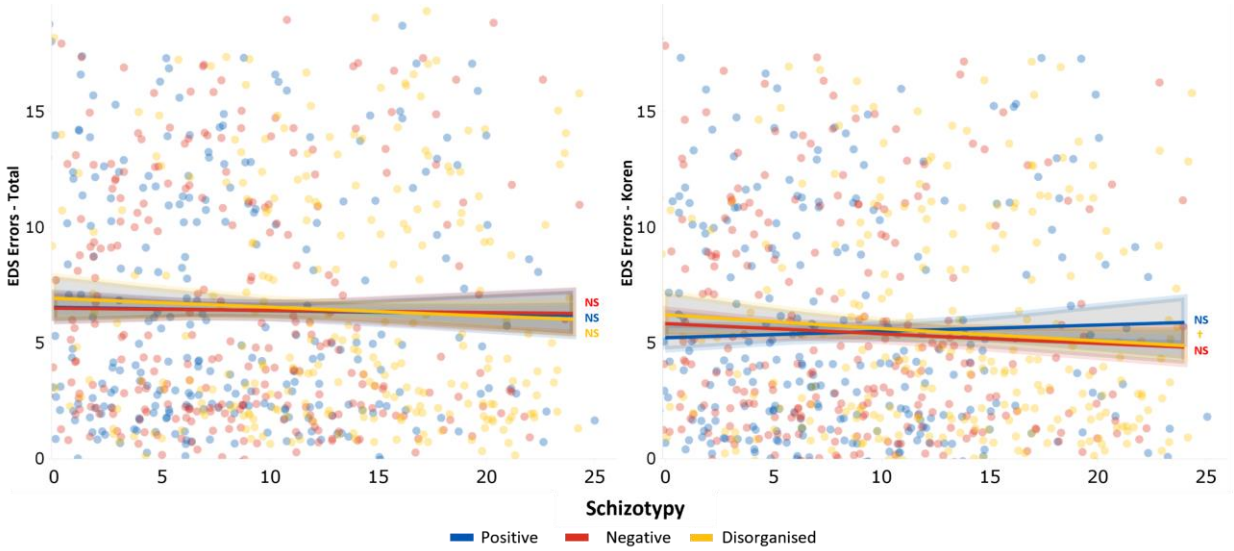


Figure 22. Plotted model estimations of Poisson regressions predicting total EDS errors (**left**) and Koren EDS errors (**right**, trials that participants volunteered for assessment) from the three schizotypy scales. No analysis was statistically significant. Shaded areas represent 95% Confidence Intervals.

Table 12. Poisson logistic regressions predicting the number of total errors (**top**) and Koren Errors (**bottom**) in the EDS stage of the M-IDED.

	EDS Errors				95% Conf Int			
Variable	**Log(odds)**	**SE**	**Z**	***p***	**LC**	**HC**	**GVIF**	**OR**
Intercept	2.0	0.065	30.6	< .001				
Positive Schizotypy	-0.002	0.005	-0.466	= .641	-0.012	0.007	1.288	0.998
Negative Schizotypy	-0.002	0.005	-0.316	=.752	-0.010	0.007	1.096	0.999
Disorganised Schizotypy	-0.006	0.005	-1.241	=.215	-0.015	0.003	1.384	0.994
	Koren EDS Errors				**95% Conf Int**			
Variable	**Log(odds)**	**SE**	**Z**	***p***	**LC**	**HC**	**GVIF**	**OR**
Intercept	1.9	0.069	26.7	< .001				
Positive Schizotypy	0.005	0.005	0.945	= .345	-.005	.015	1.321	1.005
Negative Schizotypy	-0.008	0.005	0.113	= .113	-.018	.002	1.088	0.992
Disorganised Schizotypy	-0.010	0.005	0.051	= .051	-.020	.000	1.413	0.990

Note: Total errors: $X^2(3) = 3.473$, $p = .324$, Koren Errors: $X^2(3) = 8.84$, $p = .031$. N = 253.

Table 13. Multiple linear regressions predicting positive and confidence ratings to correct (**top**) incorrect (**bottom**) decisions in the Extra-Dimensional Shift stage of the M-IDED.

	Confidence Correct								95% Conf Int	
predictor	**B**	**SE**	**t**	***p***	**BF_{10}**	**$R^2_{partial}$**	**VIF**	**β**	**LC**	**HC**
Intercept	5.133	0.247	20.8	<.001						
Pos Scz	0.011	0.018	0.635	=.526	0.186	0.160	1.281	0.045	-0.095	0.185
Neg Scz	0.012	0.017	0.696	=.487	0.187	0.193	1.094	0.046	-0.083	0.175
Dis Scz	-0.033	0.018	-1.868	=.063	0.615	1.371	1.374	-0.137	-0.282	0.007
	Confidence Incorrect								**95% Conf Int**	
predictor	**B**	**SE**	**t**	***p***	**BF_{10}**	**$R^2_{partial}$**	**VIF**	**β**	**LC**	**HC**
Intercept	4.590	0.293	15.7	< .001						
Pos Scz	0.023	0.021	1.079	0.282	0.217	0.477	1.257	0.077	-0.064	0.218
Neg Scz	-0.002	0.020	-0.095	0.925	0.190	0.004	1.085	-0.006	-0.138	0.125
Dis Scz	-0.033	0.021	-1.589	0.113	0.407	1.028	1.349	-0.118	-0.263	0.028

Correct: $F(3, 251) = 1.196$, $p = .312$, $R^2 = 1.4\%$, $R^2_{adjusted} = 0.2\%$. **Incorrect:** $F(3, 243) = 1.013$, $p = .387$, $R^2 = 1.2\%$, $R^2_{adjusted} = 0.0\%$. VIF = Variance Inflation Factor. Bayesian priors are full Cauchy (location = 0, scale = 0.354)

4.0: Discussion

The current Chapter expanded the response dimensions of a Probabilistic Reversal Learning (M-PRL) task and set-shifting task (M-IDED). These adaptations separated neurocognitive performance from metacognitive performance by requesting responses on knowledge, confidence, and acting on knowledge separately. For the M-PRL, the association between positive schizotypy and poorer learning of the punishing contingency ("I think I will win") was replicated. Moreover, this Chapter expands this replication by revealing participants also *acted* on poorer cognition to punishing stimuli ("I think I will win and I want to include this round", Koren accuracy), highlighting a deficit in metacognitive control. Moreover, those high in positive schizotypy incorrectly wagered more money when they thought they would lose, which also highlights a deficit in metacognitive sensitivity. Disorganised schizotypy significantly predicted increased Koren accuracy of punishing stimuli but not total accuracy, suggesting participants present intact cognition and improved metacognition. Surprisingly, schizotypy did not predict neurocognitive nor metacognitive deficits in the M-IDED which contrasts with Chapter 2. Finally, negative schizotypy was again unrelated to performance in either task.

4.1: Neurocognitive performance

The replication of poorer punishing cue accuracy in the M-PRL task ("Do you think you will win?") suggests that poorer performance in the previous PRL task, which conflated neurocognition and metacognition ("Do you want to bet £1 or 10p?"), is at least partially due to poorer neurocognitive performance. This supports the previous suggestion that positive schizotypy presents a deficit specifically in learning, such as through anxiety-inducing negative events producing avoidant processing styles. A future replication using eye tracking to assess punishing cue avoidant saccades would support this suggestion further. For disorganised schizotypy, the significant interaction of disorganised schizotypy and contingency reported in Chapter 2 was not replicated, potentially suggesting the possible performance benefits of disorganised schizotypy were not due to neurocognitive prosses, but perhaps rather metacognitive performance (discussed further in 4.2.2).

There is a clear contrast between IDED performance in the current study and Chapter 2. Neither the association between positive schizotypy and poorer performance nor disorganised schizotypy and improved performance was replicated. Interestingly, the median number of errors in both the IDED and M-IDED was 4 errors, suggesting lack of response variation is not the cause. While this may suggest deficits in Chapter 2 are metacognitive rather than neurocognitive, no associations

to confidence nor Koren errors were reported in the current Chapter. This is especially surprising 127 considering previous studies suggest metacognitive adaptations do not change the overall properties of the task[216]. While this could simply be a Type II error, consistent with the instability of previous set-shifting studies in schizotypy, this could be explained by the clearer task instructions. The M-IDED explained common misconceptions of Chapter 2 including "It is the shape and NOT ITS LOCATION that is important" and "once the game knows you have discovered the rule, the rule will change" (**Appendix B.**). This suggestion is consistent with clearer instructions mitigating reasoning deficits in schizotypy[432] and suggests miscomprehension may be a factor affecting cognitive task performance.

4.2: Behavioral metacognitive performance

4.2.1 Metacognitive Sensitivity

Metacognitive sensitivity is the efficient calibration of confidence and accuracy (i.e., highly confident when correct and underconfident when incorrect). For the M-PRL, adaptive confidence allocation meant larger bets on rewarding cues and lower bets on punishing cues. **For correct decisions**, positive schizotypy predicted increased money wagered on punishing cues - meaning that when participants saw the punishing cue and correctly knew they would lose, they bet more money and thus lost more money. **For incorrect decisions**, positive schizotypy predicted increased money wagered to rewarding stimuli - meaning when participants saw a rewarding cue but incorrectly thought they would lose, they wagered more money. Together, this suggests that the underlying effect is that when those high in positive schizotypy expect a negative outcome they are less able to effectively allocate confidence. This is partially consistent with the overconfidence in errors reported for clinical and non-clinical samples, although this study suggests the effect may be driven by expected outcomes rather than decision accuracy. These results are also consistent with the findings of impaired learning of punishment in both Chapter 2, extending this suggestion that it is the participant's perception of punishment that is key. While positive schizotypy predicted better performance for incorrect rewarding decisions, this was because the participant incorrectly perceived them as punishing and thus the underlying mechanism is maladaptive. This suggests that those high in positive schizotypy ineffectively modulate their confidence ratings to decisions they perceive as punishing, but not perceive as rewarding. This further strengthens the suggestion for the influence of negative emotional states affecting performance.

The M-IDED again delivered inconsistent findings similarly to Chapter 2. In contrast to the M-PRL, positive schizotypy was unrelated to confidence judgments and there was trend-level evidence that

disorganised schizotypy predicted decreased confidence ratings when correct. This latter point is likely a more general under confidence bias as the effect sizes for correct decisions (β = -0.137) and incorrect decisions (β = -0.118) are similar with overlapping confidence intervals. As the M-IDED does not allow for the separation of punishing and rewarding cues, it cannot be determined whether the influence of perceived punishment observed in the M-PRL extends to the M-IDED. Future studies should also adapt the M-IDED and IDED to separate the three distractor stimuli from the target stimulus, rather than pairing two stimuli under a single response dimension. Indeed, if rewarding and punishing cues were collapsed in the M-PRL, the current pattern of confidence ratings would not have been detected.

The suggestion that increased comprehension may have mitigate cognitive deficits may also extend to confidence ratings. Indeed, metacognitive sensitivity deficits have been suggested to only be present when perceived difficulty is high[157]. Perhaps another potential reason is that while the M-PRL used a continuous confidence scale, the M-IDED used a 7-point Likert scale which may not have been precise enough to detect effects. Future replications should use a 0% to 100% confidence scale consistently across tasks.

4.2.2 Metacognitive Control

Metacognitive control is the described top-down metacognitive influence over behaviour. In this study, Koren accuracy and its comparison to total accuracy were used to assess this. Positive schizotypy was found to predict poorer Koren accuracy to punishing stimuli. In other words, positive schizotypy predicted poorer cognitive ability to punishing stimuli (total accuracy) *and* predicted acting on that faulty knowledge. This does not replicate the initial findings that task deficits are mitigated when the participant can choose which information to act upon (at least as measured by the M-PRL)[216]. More broadly, these results may suggest clinical patients high in positive symptoms may have both neurocognitive deficits and metacognitive control deficits that allow faulty information to be acted upon. Surprisingly, while disorganised schizotypy was not associated with total accuracy, it did predict improved Koren accuracy of the punishing contingency; suggesting participants have intact neurocognition and are more likely to act upon correct neurocognitive judgments. Moreover, there was trend-level evidence for a similar finding in Koren errors of the M-IDED. This is consistent with other studies reporting intact neurocognition but metacognitive differences[433] and highlights the contrasting effects of positive and disorganised schizotypy, the utility of these metacognitive adaptions, and the separability of ability and acting on ability. Future research should investigate influences for this improvement, as this may highlight adaptive metacognitive control coping strategies that could be applied to clinical patients. Future research

should also assess whether total accuracy or Koren accuracy is more closely related to real-world functioning[216].

4.3: Potential mediators

A secondary aim of this Chapter was to again include potential psychometric mediators of performance including negative affect, motivation, and psychometric metacognition. Chapter 2 suggested several reasons for the lack of mediation previously, which were explored in the current Chapter. However, none of the potential mediators predicted nor mediated performance, but these null findings are still highly relevant. Firstly, it was suggested that the lack of mediation by motivation may be due to social desirability to over-report motivation and assess motivation overall rather than per task. This Chapter did not have these limitations, yet motivation still did not mediate performance. Further post-hoc exploration of motivation scores in the current Chapter suggested participants were more motivated at the start of the study but less motivated by the end of the study relative to Chapter 2 (**Supplementary Analysis**). The absence of a mediatory role of motivation in face of the higher variability of motivation scores may suggest motivation is not a relevant factor in schizotypy (or the MIAMI scale is not sensitive to detect effects). Secondly, it was suggested that the low internal consistency of the BCIS and its potential limited generalisability to non-clinical samples may have caused issues. The current study also employed the MCQ-30, however, despite the measure having excellent internal consistency and greater generalisability null findings were still found. This finding is especially surprising considering the significant associations between schizotypy and metacognitive sensitivity and metacognitive control; potentially highlighting a distinction between psychometric and behavioural measures of metacognition in schizotypy. This distinction is further supported by the general lack of associations between psychometric and behavioural metacognitive variables (**Table 7**).

4.4. Strengths and limitations

Primary strengths of the current Chapter include the moderate sample sizes across tasks, the validation between psychometric and behavioural metacognition, and the normative psychometric comparisons. A primary limitation of this study is that there is emerging evidence that metacognitive indices may be confounded by baseline neurocognitive performance[153]. Novel metrics such as meta d-prime or AUROC2 should be applied in future studies, which can give estimations of metacognitive performance independent of accuracy. However, this is not appropriate for the current design with

currently available statistical packages. A further limitation is that, ideally, the current study should have been conducted in person rather than online to be consistent with Chapter 2. However, this was not possible due to the COVID-19 pandemic.

5.0 Conclusions

This study supports that people higher in positive schizotypy present neurocognitive deficits in learning outcomes of punishing stimuli, as well as metacognitive deficits in both acting on this faulty knowledge and maladaptively attributing confidence to events perceived as negative. Conversely, those higher in disorganised schizotypy present intact neurocognition and elevated levels of metacognitive control: being more likely to act on correct information. These relationships were not explained by confounds of negative affect, amotivation, or surprisingly psychometric metacognition which was unrelated to performance. Moreover, psychometric metacognition did not correlate with behavioural measures suggesting the constructs are distinct. The next Chapter expands this methodology to the domain of social cognition.

Appendices

Appendix A.

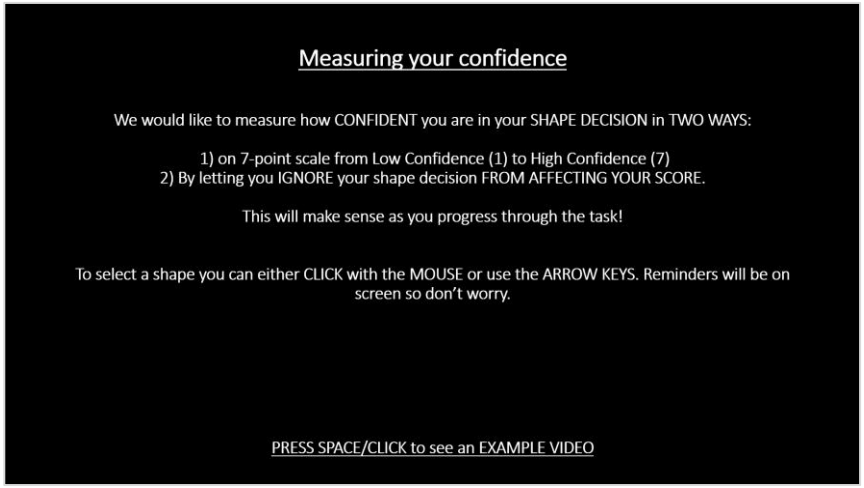
Metacognitions Questionnaire 30 item version (MCQ-30)

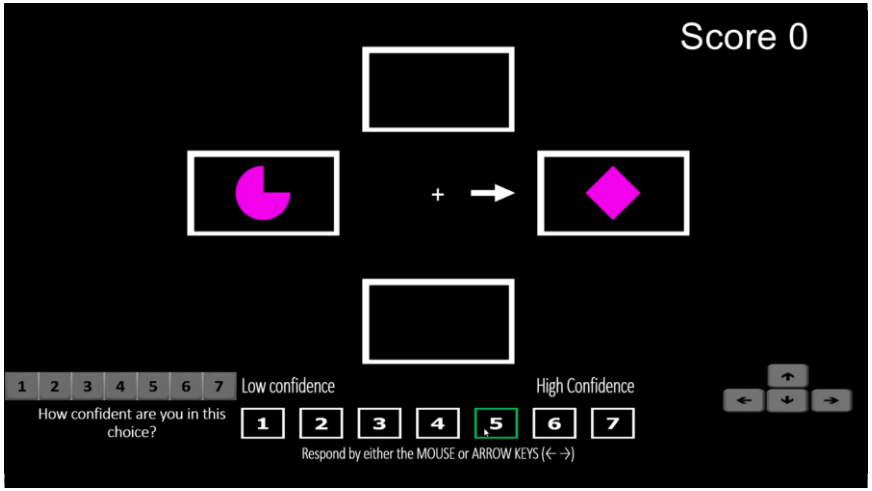
		Strongly Disagree	Disagree	Agree	Strongly Agree
1.	I do not trust my memory (CC)				
2.	I have a poor memory (CC)				
3.	I have little confidence in my memory for actions (CC)				
4.	I have little confidence in my memory for places (CC)				
5.	I have little confidence in my memory for words and names (CC)				
6.	My memory can mislead me at times (CC)				
7.	Worrying helps me to get things sorted out in my mind (PB)				
8.	Worrying helps me cope (PB)				
9.	I need to worry in order to work well (PB)				
10.	Worrying helps me to solve problems (PB)				
11.	I need to worry in order to remain organised (PB)				
12.	Worrying helps me to avoid problems in the future (PB)				
13.	I am constantly aware of my thinking (CSC)				
14.	I pay close attention to the way my mind works (CSC)				
15.	I think a lot about my thoughts (CSC)				
16.	I constantly examine my thoughts (CSC)				
17.	I monitor my thoughts (CSC)				
18.	I am aware of the way my mind works when I am thinking through a problem (CSC)				
19.	My worrying thoughts persist, no matter how I try to stop them (UD)				
20.	When I start worrying, I cannot stop (UD)				
21.	I could make myself sick with worrying (UD)				

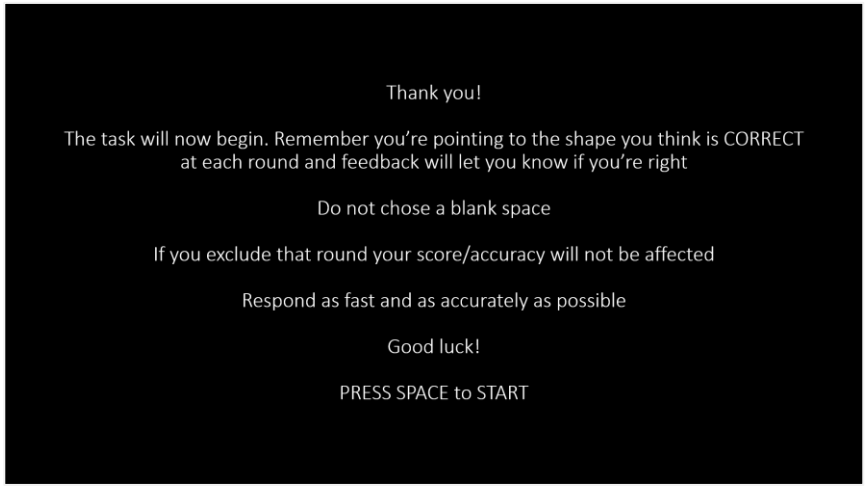
22.	I cannot ignore my worrying thoughts (UD)
23.	My worrying could make me go mad (UD)
24.	My worrying is dangerous for me (UD)
25.	If I could not control my thoughts, I would not be able to function (NCT)
26.	Not being able to control my thoughts is a sign of weakness
27.	I should be in control of my thoughts all of the time (NCT)
28.	It is bad to think certain thoughts (NCT)
29.	If I did not control a worrying thought and then it happened, it would be my fault (NCT)
30.	I will be punished for not controlling certain thoughts (NCT)

Appendix B.

Information screens presented as part of the Metacognitive-IDED

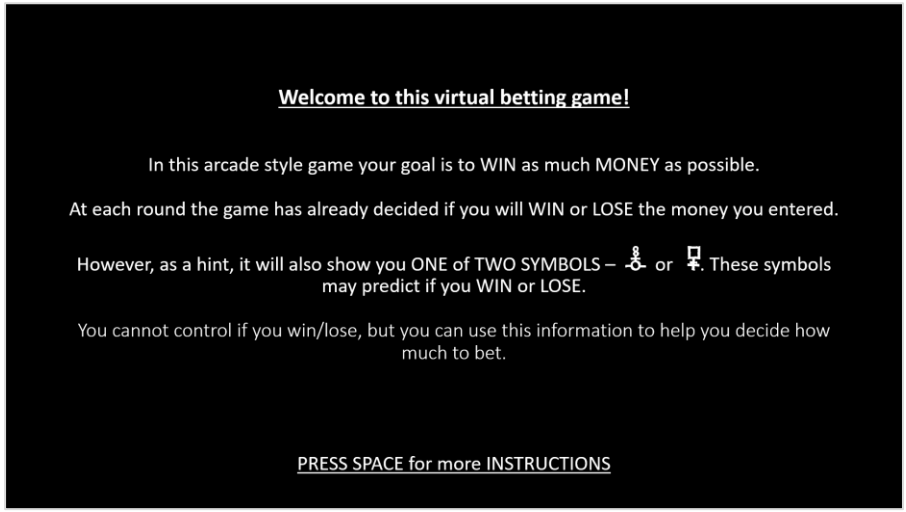


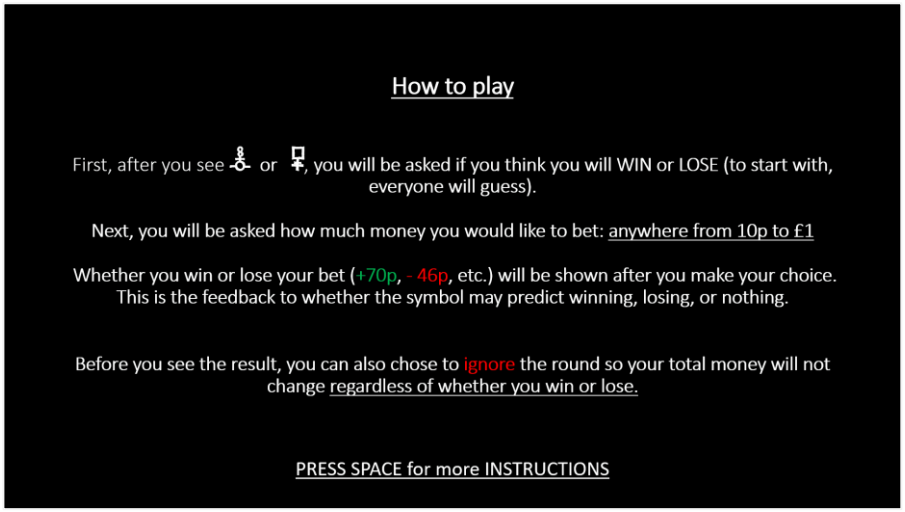


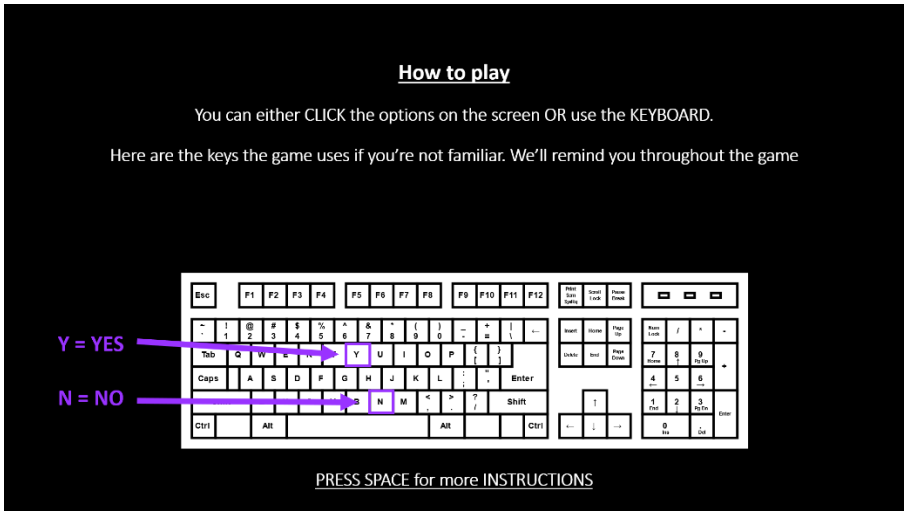


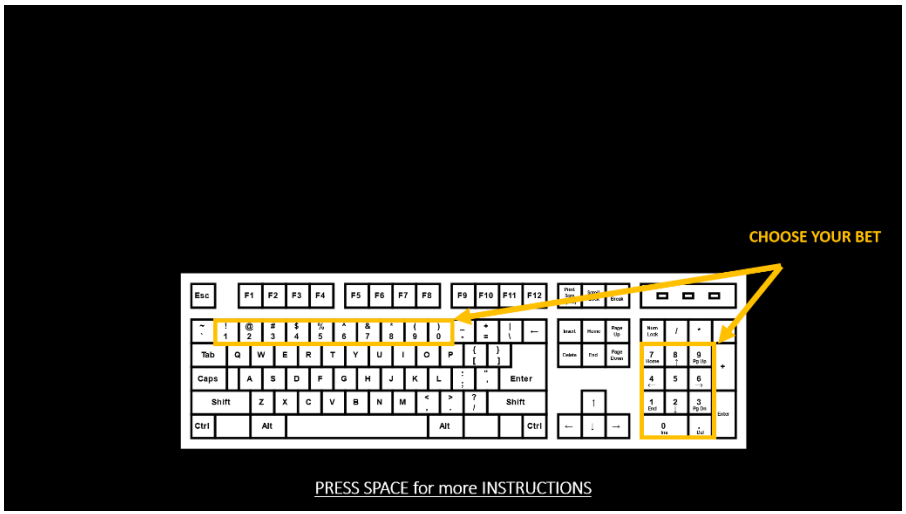
Appendix C.

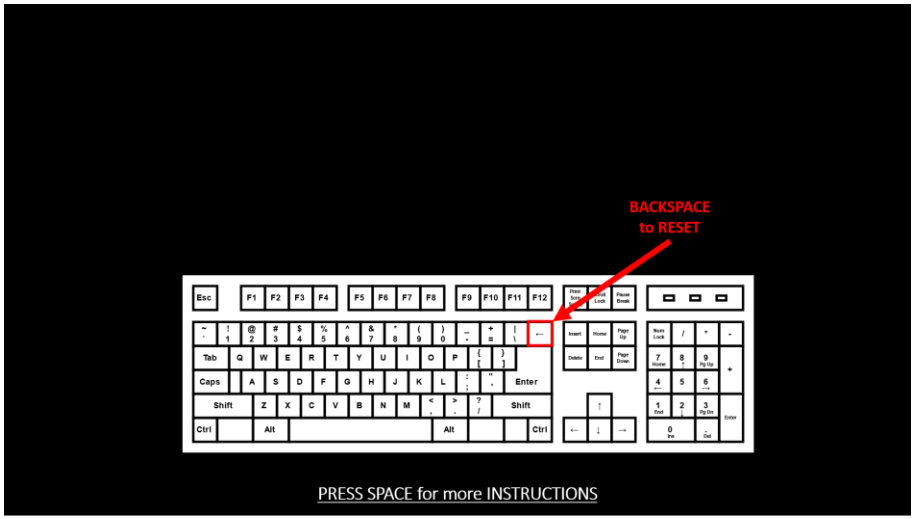
Information screens presented as part of the Metacognitive-PRL

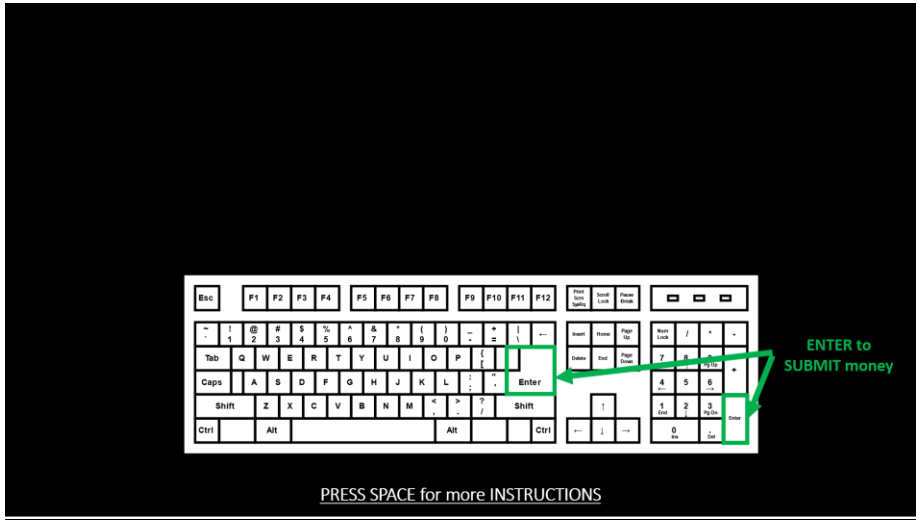


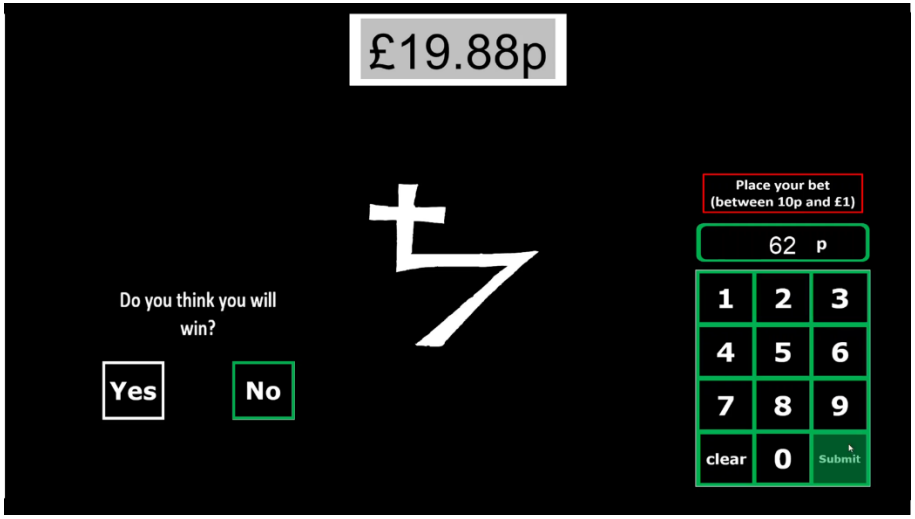







BACKSPACE
to RESET
PRESS SPACE for more INSTRUCTIONS


ENTER to
SUBMIT money
PRESS SPACE for more INSTRUCTIONS


£19.88p
Do you think you will win?
Yes
No
Place your bet
(between 10p and £1)
62 p
1 2 3
4 5 6
7 8 9
clear 0 Submit


In the first round the player lost -12p. In the second round, the player ignored the result so did not win the +64p and their total did not change.
Before we start the game, we'd like to ask how you're feeling right now.
PRESS SPACE to CONTINUE

Appendix D.

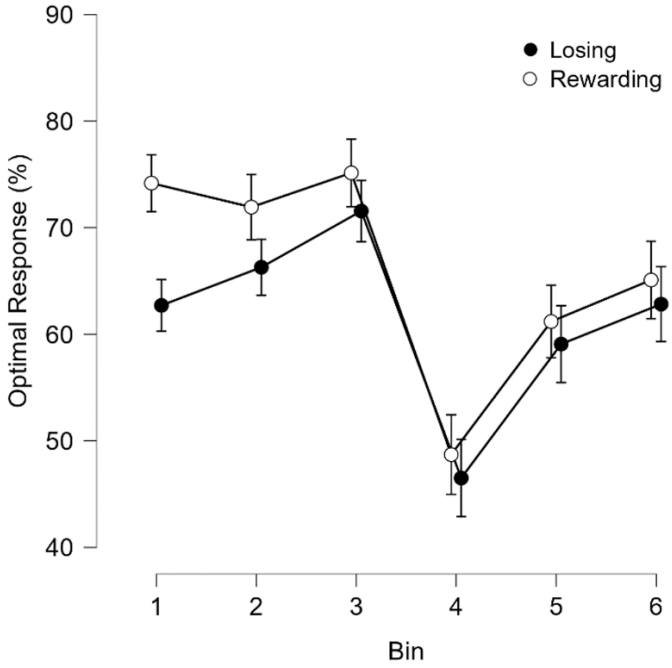
Descriptive statistics of performance on the Metacognitive-IDED task

Outcome	Stage	Range	M	SD	Med	MAD	IQR
Performance	SD	0 – 25	1.9	3.1	1	1.5	1
	SDR	0 – 22	2.0	2.7	1	0	1
	CD	0 – 24	1.9	3.1	1	1.5	2
	CDS	0 – 21	1.1	2.8	0	0	1
	CDR	0 – 25	2.5	3.8	1	0	1
	IDS	0 – 25	0.8	2.1	0	0	1
	IDR	0 – 24	1.9	3.0	1	0	0
	EDS	0 – 19	6.7	7.1	4	4.4	9
	EDR	0 – 20	4.8	5.1	2	1.5	7
Confidence	SD	1 - 7	5.0	1.2	5.3	1.1	1.7
	SDR	1 - 7	5.7	1.2	5.9	1.3	1.7
	CD	1.3 - 7	5.0	1.3	5.2	1.3	1.8
	CDS	1 - 7	5.3	1.4	5.5	1.5	2.2
	CDR	1 - 7	5.5	1.4	5.9	1.5	2.1
	IDS	1 - 7	5.1	1.2	5.3	1.0	1.5
	IDR	1 - 7	5.7	1.3	6.0	1.3	1.8
	EDS	1 - 7	4.8	1.4	5.0	1.5	2.0
	EDR	1 - 7	4.9	1.7	5.2	1.8	2.5
Inclusion	SD	0 - 100	90.2	16.5	100	0	15.5
	SDR	0 - 100	96.2	12.3	100	0	0.0
	CD	0 - 100	91.2	15.2	100	0	14.3
	CDS	0 - 100	93.4	14.8	100	0	0.0
	CDR	0 - 100	94.9	12.4	100	0	0.0
	IDS	0 - 100	91.7	12.9	100	0	16.7
	IDR	0 - 100	95.5	11.3	100	0	0.0
	EDS	0 - 100	87.7	17.4	100	0	22.2
	EDR	0 - 100	91.2	18.1	100	0	14.3

Note: IQR – interquartile range, MAD = robust median absolute deviation

Appendix E.

Top: Descriptive statistics of performance on the Metacognitive-PRL task. **Bottom**: A 2 (contingency) x 6 (Bin of 10 trials) repeated measures ANOVA with Greenhouse-Geisser correction reported a main effect of bin at a large effect size ($F(2.8, 613.8) = 65.603$, $p < .001$, $\eta^2_p = 0.232$) and significantly higher performance for the winning cue relative to the winning cue at a small effect size ($p = .010$, $d = -0.176$). Post-hoc comparisons with Holm correction for multiple comparisons found that performance pre-reversal increased from Bin 1 to Bin 3 only ($p_{holm} < .001$, $d = 0.209$) and, as expected, performance immediately decreased post-reversal at Bin 4 at a large effect size ($p_{holm} < .001$, $d = -1.094$). Performance increased at both Bin 5 ($p_{holm} < .001$, $d = 0.532$) and at Bin 6 ($p_{holm} = .033$, $d = -0.163$), but this did not return to the same level of performance as Bin 3 ($p_{holm} < .001$, $d = -0.399$). Overall, these results validated that learning had occurred during the task and the continency reversal was successful.



Outcome	Block	Valance	Range	M	SD	Med	MAD	IQR
Performance	Block 1	Positive	3 - 100	73.9	21.7	21.7	30.6	32.5
	Block 1	Negative	10 – 100	67.0	20.5	20.5	28.9	30
	Block 2	Positive	0 - 100	58.3	24.6	24.6	35.0	33.3
	Block 2	Negative	0 - 100	56.0	26.2	26.2	37.3	43.3
Confidence	Block 1	Positive	10 - 100	58.2	22.5	59.9	23.8	31.3
	Block 1	Negative	10 - 99	34.6	21.3	29.8	22.4	32.7
	Block 2	Positive	10 - 100	55.3	22.6	55.3	25.0	33.3
	Block 2	Negative	10 - 100	46.0	25.8	43.5	29.4	39.3
Inclusion	Block 1	Positive	33 - 100	88.4	13.9	93.3	9.9	20
	Block 1	Negative	0 - 100	57.8	33.1	58.3	46.9	65.8
	Block 2	Positive	3 - 100	81.1	19.8	86.7	19.8	23.3
	Block 2	Negative	3 - 100	63.8	32.6	71.7	42	63.3

Note: IQR – inter quartile range, MAD = robust median absolute deviation

Appendix F.

M-PRL model coefficients predicting confidence ratings (money) of correct decisions (top) and incorrect decisions (bottom).

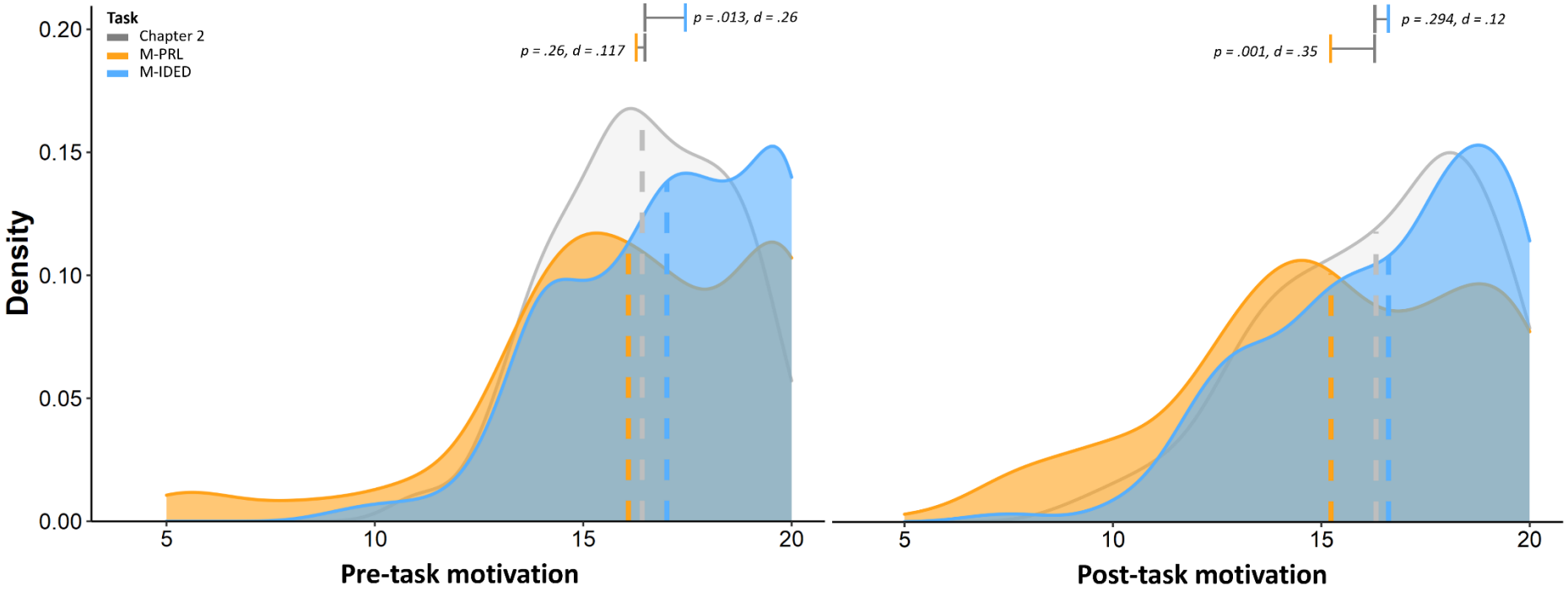
	Confidence Correct					**95% Conf Int**	
predictor	**B**	**SE**	**t**	***p***	**β**	**LC**	**HC**
Intercept	29.0	3.893	7.445	<.001			
Contingency^{+}	34.4	3.294	10.4	<.001	0.615	0.500	0.730
Block^{2}	8.9	1.739	5.116	<.001	0.159	0.098	0.220
Contingency^{+} * Block^{2}	-11.9	2.467	-4.843	<.001	-0.184	-0.258	-0.109
Positive Scz	0.631	0.267	2.360	= .019	0.134	0.023	0.245
Negative Scz	-0.031	0.249	-0.124	= .902	-0.006	-0.098	0.087
Disorganised Scz	-0.279	0.269	-1.039	= .300	-0.060	-0.174	0.054
Contingency^{+} * Pos Scz	-0.662	0.231	-2.858	= .004	-0.143	-0.241	-0.045
Contingency^{+} * Dis Scz	0.265	0.227	1.167	= .244	0.075	-0.051	0.201
	Confidence Incorrect					**95% Conf Int**	
predictor	**B**	**SE**	**t**	***p***	**β**	**LC**	**HC**
Intercept	46.4	4.122	11.261	<.001			
Contingency^{+}	-13.7	3.454	-3.963	<.001	-0.255	-0.381	-0.129
Block^{2}	14.2	1.806	7.883	<.001	0.265	0.199	0.331
Contingency^{+} * Block^{2}	-3.980	2.558	-1.556	= .120	-0.065	-0.147	0.017
Positive Scz	0.081	0.281	0.288	= .774	0.018	-0.104	0.139
Negative Scz	-0.180	0.263	-0.685	= .494	-0.036	-0.137	0.066
Disorganised Scz	0.020	0.283	0.07	= .944	0.004	-0.121	0.130
Contingency^{+} * Pos Scz	0.531	0.239	2.219	= .027	0.119	0.014	0.225
Contingency^{+} * Dis Scz	-0.171	0.234	-0.733	= .464	-0.051	-0.186	0.085

Note: Block^{2} = the effect of Block 2 relative to Block 1, Contingency^{+} = the effect of the positive contingency relative to the negative contingency. Correct: $R^2_{\text{fixed-effects}}$ =23.9%, $R^2_{\text{ranomd-effects}}$ = 58.5%., Incorrect: $R^2_{\text{fixed-effects}}$ =12.2%, $R^2_{\text{ranomd-effects}}$ = 54.2%.

Supplementary analyses

Supplementary Analysis A

Comparison of pre-task (left) and post-task (right) motivation scores as measured by the MIAMI between both tasks of Chapter 2 and the two tasks (M-IDED and M-PRL) of the current Chapter. The figures show that pre-task motivation was significantly greater for the M-IDED than Chapter 2 (suggesting that participants were more motivated in Chapter 2 before completing any assessments) and past-task motivation was lower for the M-PRL compared to Chapter 2 (suggesting participants were less motivated at the end of Chapter 3 relative to Chapter 2).



Chapter 4

Metacognitive adaptations of social cognitive tasks in psychometric schizotypy

Negative schizotypy predicts poorer emotion recognition but not acting on faulty knowledge

Introduction: The current Chapter expanded the response dimensions of a social cognitive task (Emotion Recognition) to include measures of social cognition and metacognition. The area of social cognition was chosen due to the closer association between social cognition and daily functioning relative to neurocognition.

Methods: A sample of 209 participants were recruited online and completed the short version of the Geneva Emotion Recognition Task (GERT-S) and the same questionnaires as Chapter 3 (O-LIFE, DASS-21, MIAMI, BCIS, and MCQ-30). Measures of total accuracy, Koren accuracy, and confidence ratings of Emotion Recognition decisions were the key outcome variables.

Results: Multiple regression analysis found that negative schizotypy predicted poorer total accuracy of negative emotions, but not Koren accuracy to negative emotions. This association was partially explained through increased maladaptive metacognition, as measured by the Need to Control Thoughts scale of the MCQ-30. Disorganised schizotypy traits predicted improved total accuracy and Koren accuracy of negative emotions. There was inconclusive evidence regarding positive schizotypy and accuracy. Positive schizotypy and disorganised schizotypy predicted an overconfidence and underconfidence bias in responses, respectively.

Discussion: These findings suggest that those high in negative schizotypy may not act on faulty social cognitive processes due to intact metacognitive processes, whereas those high in disorganised schizotypy act on their improved social cognitive abilities.

The results of the current Chapter without the influence of metacognition have been published in the following article: *https://www.frontiersin.org/articles/10.3389/fpsyt.2021.738344/full*

Negative schizotypy predicts poorer emotion recognition but not acting on faulty knowledge

1.0: Introduction

Social cognition refers to mental processes responsible for the perception, decoding, interpretation, and regulation of responses to social stimuli (see Chapter 1)[96]. In schizophrenia, theory of mind, social perception, attributional bias, and emotion processing have been identified as key domains[434] and are more closely related to functioning than neurocognition[75,94,95]. However, while the presence of social cognitive deficits is well established in schizophrenia, the mechanisms behind these deficits are not well understood. This research aims to identify potential explanatory factors in one important domain of social cognition: Emotion Recognition (ER). In clinical patients, ER performance is negatively associated with reality distortion, negative symptoms, and disorganised symptoms to a similar extent[101]. Generally, impairments are found in the perception of negative emotions (sadness and fear) and less consistently in positive emotions, although this may be due to a lack of more varied positive stimuli beyond happiness[96]. Deficits are also found in emotion recognition from body language suggesting deficits are not face-specific[435].

1.1: Social Cognition in schizotypy

The current understanding of ER deficits in schizotypy is inconsistent in terms of which dimensions predict performance. The most consistently implicated traits are negative[436–440] followed by positive[437,440,441], with fewer studies implicating disorganised traits[437,441]. However, other studies have reported no associations for these dimensions: negative[441–446], positive[436,438,439,443,444,446–448], and disorganised[436,438–440,443,444]. There is also less evidence relative to clinical studies as to whether deficits are specific to negative emotions[385,436,439]. Moreover, while more detailed assessments have found no evidence of deficits in disgust[441], there is mixed evidence for happiness[444,449], sadness[441,444], fear[441,444], surprise[441,449], and anger[441,444,449]. Unaffected relative studies similarly suggest deficits in the recognition of negative but not positive emotions[450], whereas polygenic risk reviews find inconsistent[451,452] and weak evidence (e.g., $R^2 = 0.001$[453]). These deficits have generally been found to be independent of more general face processing deficits[437], meaning it is emotion perception specifically that is disrupted. Irrespective of accuracy, there is evidence for slower processing speed[452] and altered activity and functional connectivity of the limbic system[442,454]. Despite the broad

array of research, ER tasks used in previous investigations are limited by the number of positive emotions presented (i.e., only happiness)[96] – which may explain literature inconsistencies through a lack of emotion variability. It is therefore important to include a wider variety of positive emotions (i.e., relief, pleasure, amusement, etc.) as implemented in the current study.

1.2: The relevance of negative affect for Emotion Recognition

As outlined in previous Chapters, negative affect, metacognition, and motivation may partially explain cognitive deficits in clinical patients. For this Chapter specifically, these potential confounds may also explain the emotion recognition literature inconsistencies. Specifically, Major Depressive Disorder (MDD) has been associated with poorer recognition of all six basic emotions except sadness (g = -0.42 to -0.17)[230], which may act as a moderating variable between schizotypy and ER performance (i.e., ER deficits occur only when negative affect is high). Previous research has suggested statistically controlling for negative affect when assessing both schizotypy and ER performance[437,441]. However, only one study at the time of writing has done so. This study found correlations between schizotypy and ER performance remained significant when negative affect was controlled for[436]. However, this methodological approach did not allow a comparison of the relative impact of schizotypy and negative affect on ER (e.g., by use of a mediation analysis or by comparing standardized effect sizes).

1.3: Metacognition and Social Cognition

While psychometric metacognition has so far been unrelated to neurocognitive performance, a closer relationship may be expected for social cognition. Metacognition has been suggested to relate to social cognitive judgements, both due to its potential to increase learning and its central role in synthetic metacognition through social self-concepts (see Chapter 1). However, at the time of writing, there have been no investigations of how psychometric metacognition may predict social cognitive performance in psychometric schizotypy. This also extends to behavioural metacognition, with no study assessing Koren accuracy of social cognitive judgments. As a result, it is unclear whether participants may act on faulty social cognitive judgments (at least in lab-based scenarios). Confidence ratings have received relatively more interest in clinical samples[156], with reports of overconfidence in errors for negative emotions and emotions of a weaker intensity[155], facial emotion recognition in non-immersive Virtual Reality[157], and an overconfidence bias in general emotion recognition[455] and ToM performance[456]. However, no study has assessed metacognitive sensitivity in

psychometric schizotypy. Consequently, it is unclear if metacognitive deficits in social cognition for clinical patients extend to non-clinical schizotypy.

1.4: Motivation

As outlined in the previous chapters, neurocognitive deficits are partially mediated by task motivation in schizophrenia[258]. Chapters 2 and 3 have failed to support that this relationship extends to schizotypy in the neurocognitive domain. However, there has yet to be a similar investigation into social cognition in the psychosis spectrum.

1.5: Aims and hypotheses

Considering the above, it is unclear whether a) negative affect may explain past literature inconsistencies in schizotypy, whether the null associations between b) psychometric metacognition and c) motivation with neurocognition reported in Chapters 2 and 3 extend to social cognition d) whether aberrant confidence ratings of social cognitive decisions found in schizophrenia extends to schizotypy, and e) how psychometric and behavioural metacognition may influence social cognition decisions in schizotypy. Therefore, this study aimed to replicate Emotion Recognition (ER) deficits in psychometric schizotypy and investigate negative affect, metacognition, and motivation as potential mediators of deficits. It was hypothesized that:

1a) Higher in negative schizotypy[436–440] will predict lower Emotion Recognition (ER) total accuracy.

1b) These deficits will be partially mediated by b) negative affect[437,441] c) motivation[258] and d) psychometric metacognition.

2) These ER deficits will not be replicated when assessing Emotion Recognition Koren accuracy.

3) Higher schizotypy will predict overconfidence in errors and underconfidence in correct decisions[150].

2.0: Methods

2.1: Participants

From an initial 232 participants, 23 were excluded (see **2.5: Data preparation**). The final online sample of 209 participants was recruited through the university's recruitment system (15.8%), Call for Participants (15.8%), social media (38.8%), and Prolific (29.7%). In this sample, 66% were biologically female, ages ranged from 18 – 69 years old (*M* = 27.4, *SD* = 10.2), 79.4% had at least an undergraduate level qualification, 44.0% were current students, and 45.9% were currently employed. Of the 148 participants that volunteered responses 51.6% reported no current medication, 10 participants reported taking anti-depressants, one participant reported taking lithium (a mood stabiliser)[v], and no participant reported anti-psychotic medication. This study achieved a power of 0.99 for a medium effect size in a multiple regression analysis[457] and exceed the recommendation of 180 participants for Structural Equation Modelling (SEM) used in this study design[458].

2.2: Materials

2.2.1: Psychometric materials

This Chapter used the same scales as outlined in Chapter 2 to measure schizotypy (O-LIFE), negative affect (DASS-21), pre- and post-task motivation (MIAMI), and Moderately Discrete Metacognition (BCIS and MCQ-30) and thus the details are omitted here.

2.2.2: Emotion Recognition

The Geneva Emotion Recognition Task - Short version (GERT-S)[419] is a 42-item Emotion Recognition (ER) task involving 14 emotions. Stimuli are one to three-second videos of 10 male and female actors. These actors speak non-sense syllables meaning ER is from dynamic facial expression, upper body language, and prosody (but not semantic meaning). The GERT-S includes high arousal positive emotions (*pleasure, relief, interest*), low arousal positive emotions (*joy, amusement, pride*), high arousal negative (*anger, fear, despair*), and low arousal negative items (*irritation, sadness, anxiety*). The GERT-S also includes disgust and surprise which are not categorised in the original conceptualisation. However, the current Chapter categorised disgust as negative and surprise remained uncategorised[vi], consistent with previous reports on schizotypy. In each trial, participants had to identify which one of the 14 emotions was being presented. A full description of the task can

[v] The following analyses did not differ in interpretation when excluding these participants.
[vi] The analyses were unchanged when adding surprise as a positive emotion.

be found in the original articles[419,459]. The GERT-S was selected from other ER measured due to the diversity of positive emotions, the scale's good internal consistency ($\omega_T = 0.89$[419,459]), the use of low-arousal emotions that may increase task difficulty and thus elicit deficits[155], and the greater representations of gender and skin colour. As with Chapter 3, additional scales were added requesting response confidence judgments (from 1 "low confidence" to 7 "high confidence") and whether participants would like to skip the current decision from affecting their performance (Koren accuracy). This created the Metacognitive-GERT-S (M-GERT-S) of which a recording can be found here.

2.3: Procedure

The data collection procedure was identical to that outlined in Chapter 3 except for the following additional information. Psychometric information was collected from one Qualtrics survey while the M-GERT-S was administered on a separate survey (**Figure 23**)[459]. The questionnaires were delivered first, followed by the M-GERT-S, M-IDED, and then the M-PRL. When participants opened the survey link to M-GERT-S, task instructions explained to participants that they would need to identify which emotion an actor was portraying from a list of 14 emotions. Next, two practice trials were presented with the option to repeat the practice. Importantly, participants were also presented with clear definitions of each of the 14 emotions before starting the task. As the M-GERT-S was delivered online, stimulus presentations depended on the time taken to load the next stage of the task. Finally, the confidence rating scale and Koren accuracy scale were explained to participants:

> "If you choose the CORRECT EMOTION you will GAIN A POINT and if you choose an INCORRECT EMOTION you will LOSE A POINT.
> We would also like to measure how CONFIDENT you are in your EMOTION DECISION in TWO WAYS
> 1) On a 7-point scale: from Low confidence (1) to High confidence (7).
> 2) By letting you SKIP this decision so it DOESN'T AFFECT your score.
> Your goal is to collect as many points as possible."

After the instructions, the pre-task motivation assessment was given. Due to licencing restrictions prohibiting amendments to the core task, a running total score was not displayed to update participants on their performance. The task on average took 15 minutes to complete.

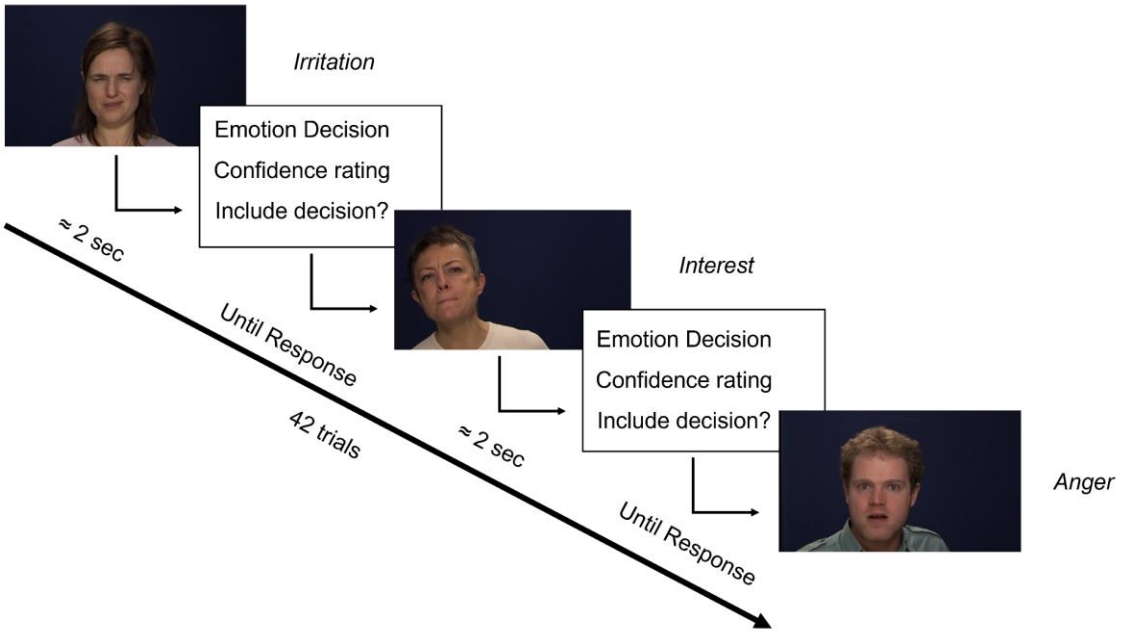


Figure 23. Trial design of the adapted M-GERT-S. Participants had to discriminate which emotion was portrayed from an option of 14 emotions, rate their confidence in this decision on a scale of 1 (low confidence) to 7 (high confidence), and where or not to include this decision in their point total.

2.4: Data preparation

From the original sample of 232 participants, 17 participants were excluded due to failing either awareness item (not selecting "Prefer not to say"), one participant withdrew their data, one participant was excluded due to responding "Prefer not to say" for all items, and six participants were removed for having performance below the median - 2.5 * Median Absolute Deviation (MAD, 37.8%). The exclusion of these latter six participants more readily satisfied the statistical assumptions of the following results but did not affect the overall conclusions. The Koren accuracy scale which asked if participants would like to skip a response was reversed to be consistent with the response inclusion scale of Chapter 3. Before reaction times were calculated, outliers were removed on a trial-by-trials basis according to median +/- 2.5 * MAD.

Seven participants did not respond to the inclusion items of the M-GERT-S before it was subsequently made compulsory after participant 10. Missing data except for these missing ratings and "Prefer not to say" responses were imputed using the missForest R package[389]. Missing data (percentage) and [percentage falsely classified] were as follows: DASS-21 (0.4%)[42.4%], BCIS (0.5%)[50.5%], MIAMI (1.2%)[31.4], MCQ-30 (0.1%)[45.9], O-LIFE(0.5%)[22.5%].

2.5: Analysis strategy

The primary outcome was recognition accuracy (0% - 100%) which was divided into positive and negative emotions. Surprise was not analysed due to the logistic regression required for analysis needing over 1000 participants. Validity checks were conducted including internal consistency, normative comparisons, and reasonable distribution of accuracy scores (i.e., ceiling effects, floor effects, and lack of variability). Two multiple linear regressions predicting positive Emotion Recognition accuracy and negative Emotion Recognition accuracy from the three schizotypy scales were conducted (*Hypothesis 1a*). Next, the analysis was repeated using Koren accuracy with the expectation that any performance deficits would not be replicated (*Hypothesis 2*). The potential mediation of performance by negative affect, motivation, and metacognition was assessed by adding all these variables simultaneous into a larger Structural Equation Model, predicting ER accuracy from the three schizotypy scales (SEM *Hypothesis 1b – 1d*). This approach reduces statistical complexity and gives a broader visual representation of these relations. Finally, multiple regressions predicted confidence ratings of correct decisions and incorrect decisions from the three schizotypy scales (*Hypothesis 3).*

All regression analyses passed the assumptions of linearity, homoscedasticity, lack of influential values (Cook's distance < 1), and no multi-collinearity (VIF < 5). Most analyses passed the assumption of normality. However, simulation studies have suggested violations of normality do not substantially influence standard error estimations when there are > 10 participants per variable[460] which this study achieved. Analyses were conducted in R studio[393], Jamovi[394] and JASP[395] using several statistical[396–400] and data visualisation packages[401,402].

3.0: Results

3.1: Descriptives

Descriptive summaries of psychometric variables and M-GERT-S scores can be found in **Table 15** and **Appendix A**, respectively. Overall, the ER scores of the entire sample presented a good range of difficulties and a lack of floor or ceiling effects. A follow-up paired samples t-test also suggested that accuracy scores were lower for negative emotions relative to positive emotions ($p < .001$, $d = .541$). The O-LIFE, DASS-21, and MCQ-30 presented excellent internal consistency ($\omega_T > .80$), the Self-Reflectivity scale of the BCIS and pre-task MIAMI scale was good ($\omega_T > .70$), the Self-Certainty scale of the BCIS were adequate ($\omega_T > .6$), and the M-GERT-S total scores were questionable to poor ($\omega_T < 0.5$). Due to the novel task adaptations and as a point total score was not displayed to participants, further analyses verified that the Koren accuracy and confidence rating scales were used in the intended manner. As summarised in **Appendix B**, accuracy and confidence ratings were higher for included responses and confidence ratings were higher for correct responses at large effect sizes (all $p < .001$, $BF_{10} > 999$, $r_{rb} > 0.760$) supporting this suggestion.

3.2: Normative comparisons

Non-parametric normative comparisons were conducted for the O-LIFE[293] and DASS-21[403] using One-Sample Wilcoxon signed-rank test. As normative medians were not available for the GERT-S[419], One-Sample t-tests were required (**Table 15**). The analyses reported that positive schizotypy was lower in the current sample ($p = .004$, $r_{rb} = -.27$), that negative schizotypy ($p < .001$, $r_{rb} = .76$), negative affect ($p < .001$, $r_{rb} = .80$), and total M-GERT-S score were higher in the current sample ($p < .001$, $d = 1.09$), and disorganised schizotypy did not differ ($p = .536$, $r_{rb} = .04$). The DASS-21 recommended cut-off criteria for normal, mild, moderate, severe, and extreme levels of each trait were as follows: *Depression*: 42.6%, 11.5%, 16.3%, 13.9%, and 15.8%; *Anxiety*: 53.1%, 7.2%, 12.9%, 9.1%, and 17.7%; *Stress*: 54.5%, 14.8%, 10.5%, 16.3%, and 3.8%.

3.3: Psychometric relationships

3.3.1: Identifying potential mediators

A Bayesian correlation matrix was calculated between potential mediating psychometric variables and schizotypy (**Table 16**). Negative affect was significantly associated with all three schizotypy scales ($r_{kendall} > .289$, $BF_{10} > 100$), Self-Reflectivity was associated with greater positive and disorganised schizotypy ($r_{kendall} > .221$, $BF_{10} > 100$), both reduced pre-task and post-task motivation were associated with cognitive disorganisation, and Self-Certainty was unrelated to schizotypy. As all the MCQ-30 scales had various relationships to schizotypy, all five MCQ-30 scales were entered as

predictors of positive, negative, and disorganised schizotypy in three separate multiple regressions to identify independent predictors. The full results can be found in **Appendix C**, which highlights that negative schizotypy remained related to only greater levels of Need to Control Thoughts (NCT, $p <$ .002) and disorganised schizotypy remains related to greater Uncontrollability and Danger ($p < .001$) and greater Self-Reflectivity ($p < .001$). The same analysis was conducted for the MIAMI scales which suggested that neither pre-task motivation ($p = .326$, $BF_{10} = 0.596$) nor post-task motivation returned as significant when added together ($p = .073$, $BF_{10} = 2.894$). However, as post-task motivation was the most predictive, this was taken forward. In summary, negative affect, Self-Reflectivity, Need to Control Thoughts, Uncontrollability and Danger, and post-task motivation were all assessed as potential mediators

3.3.2: Psychometric and behavioural metacognition

For completeness with Chapter 3, the psychometric and behavioural measures of metacognition were correlated. Beyond the association between Self-Certainty and increased response confidence overall, no correlation was significant (**Table *14***).

Table 14. Correlations between psychometric and behavioural metacognition measures.

	Overall		Sensitivity	Control	
Variable	*Confidence*	*Inclusion*	*CG*	*Koren Accuracy*	*FRI*
Self-Reflectivity	-0.04^{N}	-0.05^{N}	0.14^{I}	0.04^{N}	0.02^{N}
Self-Certainty	0.22**	-0.07^{N}	0.00^{N}	-0.09^{I}	-0.07^{N}
Cognitive Confidence	0.06^{N}	0.00^{N}	0.07^{N}	-0.02^{N}	-0.10^{I}
Positive Beliefs	-0.06^{N}	0.01^{N}	0.07^{N}	0.05^{N}	-0.10^{I}
Need to control	0.05^{N}	-0.02^{N}	0.04^{N}	-0.10^{I}	-0.06^{N}
Cognitive Self-Consciousness	0.03^{N}	-0.07^{N}	0.08^{IN}	0.07^{N}	0.05^{N}
Controllability + Danger	0.02^{N}	0.03^{N}	0.11^{I}	0.00^{N}	-0.04^{N}

Note: ^{N} = supports the Null, ^{I} = insensitive, * = $BF_{10} > 3$, ** = $BF_{10} > 10$, *** = $BF_{10} > 30$, ^{D} = >100 for 'decisive'. FRI = Free-Response Improvement, KA = Koren Accuracy, CG = Confidence Gap.

Table 15. Descriptive statistics of psychometric measures and the GERT-S including normative comparisons

	Current								Normative							Comparison		
Scale	**Range**	**M**	**SD**	**Med**	**MAD**	**IQR**	**ω_T**	**α**	**N**	**Range**	**M**	**SD**	**Median**	**IQR**	**α**	***ES* [Low, High]**	**p**	**Normality**
Unex	0-23	8.105	5.587	7.042	5.930	7.967	0.869	0.932	402	-	10.159	6.304	9	10	0.89	0.21[0.07, 0.33][S]	=.004	<.001
Intan	0-24	8.455	5.594	7.727	5.930	8.425	0.866	0.916	402	-	5.444	4.000	4.5	6.5	0.82	0.57[0.47, 0.65][L]	<.001	<.001
Cogdis	0-24	13.177	6.218	13.600	7.413	9.375	0.899	0.942	402	-	12.391	5.690	13	-	0.87	0.04[0.00, 0.14][S]	=.536	<.001
DASS Total	0-54	19.287	13.781	15.417	13.343	20.350	0.966	0.958	1794	0-61[B]	9.42	9.66	7	-	0.93	0.70[0.62, 0.76][S]	<.001	=.011
Depression	0-21	7.153	5.610	5.846	5.930	9.117	0.916	0.940	1794	0-21[B]	2.83	3.87	1	-	0.88	0.81[0.77, 0.83][L]	<.001	<.001
Anxiety	0-20	4.746	4.580	3.175	4.448	7.099	0.858	0.900	1794	0-20[B]	1.88	2.95	1	-	0.82	0.68[0.60, 0.74][L]	<.001	<.001
Stress	0-21	7.388	5.154	6.972	5.930	7.982	0.866	0.894	1794	0-21[B]	4.73	4.20	4	-	0.90	0.54[0.44, 0.63][L]	<.001	=.008
GERT-S Total	38 – 83	62.930	10.063	64.172	10.590	12.616	0.535	0.691	350	-	52	15.318	-	-	.81 - .83	1.09[0.91, 1.27 [L]	<.001	=.005
GERT-S Neg	29 - 90	60.059	13.748	61.499	14.120	19.294	0.503	0.628	350	-	-	-	-	-	-	-	-	=.002
GERT-S Pos	39 -100	68.979	13.079	71.871	16.473	17.108	0.400	0.562	350	-	-	-	-	-	-	-	-	=.001
BCIS_R	13-33	22.019	4.273	21.609	4.448	5.966	0.758	0.747	-	-	-	-	-	-	-	-	-	=.002
BCIS_C	6-21	12.919	2.971	12.783	2.965	3.999	0.654	0.694	-	-	-	-	-	-	-	-	-	=.007
MCQ_CC	6-24	11.593	4.230	10.729	4.448	5.946	0.850	0.886	-	-	-	-	-	-	-	-	-	<.001
MCQ_PB	6-24	10.172	4.388	8.607	4.448	6.324	0.900	0.934	-	-	-	-	-	-	-	-	-	<.001
MCQ_CSC	6-24	16.732	5.169	17.609	5.930	8.439	0.912	0.932	-	-	-	-	-	-	-	-	-	<.001
MCQ_UD	6-24	13.091	5.457	12.231	7.413	9.147	0.931	0.953	-	-	-	-	-	-	-	-	-	<.001
MCQ_NC	6-24	11.986	4.142	11.659	4.448	6.137	0.802	0.849	-	-	-	-	-	-	-	-	-	<.001
MCQ_Total	32-116	63.574	16.212	62.917	17.791	24.292	0.966	0.935	-	-	-	-	-	-	-	-	-	=.009
MIAMI Pre	10-20	17.392	2.244	17.621	2.965	3.281	0.739	0.793	-	-	-	-	-	-	-	-	-	<.001
MIAMI Post	8-20	16.264	2.643	16.561	2.965	3.918	0.797	0.858	-	-	-	-	-	-	-	-	-	=.014
Understand	1-4	3.570	0.699	3.745	0.000	0.942	-	-	-	-	-	-	-	-	-	-	-	<.001

Note: IQR – interquartile range, MAD = median absolute deviation, medians are interpolated medians (nearest integer is true median), **ω** = McDonalds Omega total. Distribution tests as Shapiro-Wilk tests and group comparison tests are one-sample Wilcoxon Sign Rank tests for OLIFE and DASS but one sample t-tests for MCQ and GERT-S (all *p* values are FDR corrected within each test), *ES = effect size which are rank-biserial correlations for Wilcoxon and Cohen's D for t-tests. Interpretation for effect sizes are i*n superscript. [A] = Values from Mason & Claridge, 1995., [B] Henry & Crawford, 2005. [C] Wells & Cartwright-Hatton, 2004.

Table 16. Correlations between the psychometric variables to identify potential mediators of task performance.

	Schizotypy			Neg affect	Metacognition							Motivation	
	Pos	Neg	Dis	DASS-21	Self-R	Self-C	MCQ_CC	MCQ_PB	MCQ_CSC	MCQ_UD	MCQ_NCT	Pre	Post
Positive	-												
Negative	0.083^{I}	-											
Disorganised	0.359^{D}	0.211^{D}	-										
Negative affect	0.289^{D}	0.217^{D}	0.516^{D}	-									
Self-Reflectivity	0.221^{D}	0.106^{I}	0.404^{D}	0.398^{D}	-								
Self-Certainty	0.055^{N}	0.018^{N}	-0.045^{N}	0.061^{I}	-0.03^{N}	-							
Cog Confidence	0.108^{I}	0.105^{I}	0.279^{D}	0.231^{D}	0.269^{D}	-0.017^{N}	-						
Positive Beliefs	0.215^{D}	0.065^{I}	0.225^{D}	0.235^{D}	0.183^{D}	0.086^{I}	0.278^{D}	-					
Self-Conscious	0.251^{D}	0.027^{N}	0.239^{D}	0.236^{D}	0.253^{D}	0.071^{I}	0.069^{I}	0.175^{D}	-				
Uncontrollability	0.279^{D}	0.169***	0.478^{D}	0.431^{D}	0.342^{D}	-0.007^{N}	0.279^{D}	0.271^{D}	0.334^{D}	-			
Need to Control	0.222^{D}	0.193^{D}	0.300^{D}	0.337^{D}	0.326^{D}	0.119*	0.161***	0.217^{D}	0.305^{D}	0.414^{D}	-		
Motiv Pre	-0.003^{N}	0.074^{I}	-0.118*	-0.115^{I}	-0.176^{D}	0.005^{N}	-0.169***	-0.105^{I}	-0.056^{N}	-0.084^{I}	-0.008^{N}	-	
Motiv Post	-0.050^{N}	0.094^{I}	-0.153**	-0.157***	-0.158***	0.072^{I}	-0.102^{I}	-0.013^{N}	-0.073^{I}	-0.076^{I}	-0.001^{N}	0.457^{D}	-

Note: M_ = Motivation, BCIS = Becks Cognitive Insight Scale, MCQ = Metacognitions questionnaire 30, DASS = total Depression, Anxiety, and Stress score. All correlations are Kendall's Tau with Bayes Factor, ^{N} = supports the Null, ^{I} = insensitive, * $BF_{10} > 3$, ** $BF_{10} > 10$, *** $BF_{10} > 30$, ^{D} = >100 for 'decisive'.

3.4: Emotion Recognition Accuracy

For negative emotions, negative schizotypy predicted poorer performance (β = -0.192[-0.333, -0.052], p = .007, BF_{10} = 3.238), disorganised schizotypy predicted improved performance (β = 0.256[0.096, 0.417], p = .002, BF_{10} = 4.387), and positive schizotypy returned as insensitive with a trend for impairment (p = .094, BF_{10} = 0.671, *Hypothesis 1*). All significant associations survived FDR correction for multiple comparisons (all p < .021) and adding age and sex as control variables. For positive emotions, no schizotypy scale significantly predicted performance (all p > .090) and all Bayes Factors suggested the data were insensitive to detect effects (**Table 2**). These effects are plotted in **Figure 24**.

3.5: Koren Accuracy

The above multiple regression analyses were repeated with Koren accuracy as the outcome. For negative emotions, negative schizotypy was not associated with Koren accuracy (β = -0.112[-0.256, 0.032], p = .127, BF_{10} = 0.417) with reduction a in effect size of 42% relative to total accuracy (*Hypothesis 2*). Disorganised schizotypy remained a significant predictor of improved performance (β = 0.231[0.066,0.396], p = .006, BF_{10} = 0.868) and positive schizotypy remained insensitive (BF_{10} = 0.429). For positive emotions, there was trend-level evidence that disorganised schizotypy predicted improved Koren accuracy (p = .071), but this did not survive correction and the Bayesian analysis supported the null (p_{FDR} = 0.219, BF_{10} = 0.239). Both negative and positive schizotypy were unrelated to Koren accuracy (p > .220, BF_{10} < .156, *Hypothesis 2*).

3.6: Individual Emotion Recognition Accuracy

A Spearman correlation matrix was calculated between the three schizotypy scales and individual ER accuracy. The effect of negative schizotypy for negative emotions may have come from reduced anger and fear recognition (both p < .074), although these analyses were both trends and did not survive FDR correction (both p < .395). The effect of disorganised schizotypy for negative items likely came from disgust (r_s = .254, p < .001, p_{FDR} = .007). To assess the latter, the multiple regression analysis was repeated with the exclusion of disgust. Disorganised schizotypy remained a significant albeit weaker predictor (β = 0.196[0.034, 0.357], p = .018), suggesting the effect was not carried by disgust alone. All other correlations were non-significant (**Appendix D**). Correlations between psychometric scales and reaction times are provided in **Appendix E**.

Table 2. Multiple linear regressions predicting positive and negative emotion recognition accuracy from positive, negative, and disorganised schizotypy.

	Pos Accuracy								95% Conf Int	
predictor	**B**	**SE**	**t**	***p***	**BF_{10}**	**$R^2_{partial}$**	**VIF**	**β**	**LC**	**HC**
Intercept	69.480	2.337	29.727	< .001						
Pos Scz	-0.106	0.187	-0.565	= .572	0.191	1.156	1.326	-0.045	-0.202	0.112
Neg Scz	-0.290	0.170	-1.706	= .090	0.477	1.399	1.105	-0.124	-0.268	0.019
Dis Scz	0.213	0.175	1.217	= .225	0.256	0.718	1.447	0.101	-0.063	0.266
	Neg Accuracy								**95% Conf Int**	
predictor	**B**	**SE**	**t**	***p***	**BF_{10}**	**$R^2_{partial}$**	**VIF**	**β**	**LC**	**HC**
Intercept	59.202	2.400	24.666	< .001						
Pos Scz	-0.323	0.192	-1.683	= .094	0.671	1.362	1.326	-0.131	-0.285	0.023
Neg Scz	-0.473	0.175	-2.706	= .007	3.238	3.449	1.105	-0.192	-0.333	-0.052
Dis Scz	0.567	0.180	3.152	= .002	4.387	4.622	1.447	0.256	0.096	0.417

Positive: F(3, 205) = 1.152, p = .329, R^2 = 1.7%, $R^2_{adjusted}$ = 0.2%. ***Negative:*** F(3, 205) = 4.469, p = .005, R^2 = 6.1%, $R^2_{adjusted}$ = 4.8%. VIF = Variance Inflation Factor. Bayesian priors are full Cauchy (location = 0, scale = 0.354)

Table 17. Multiple linear regressions predicting positive and negative emotion recognition Koren accuracy (included trials only) from positive, negative, and disorganised schizotypy.

	Pos Koren								95% Conf Int	
predictor	**B**	**SE**	**t**	***p***	**BF_{10}**	**$R^2_{partial}$**	**VIF**	**β_{std}**	**LC**	**HC**
Intercept	70.401	2.449	28.170	< .001						
Pos Scz	-0.135	0.199	-0.679	= .498	0.125	0.231	1.330	-0.055	-0.215	0.105
Neg Scz	-0.223	0.181	-1.231	= .220	0.156	0.756	1.110	-0.091	-0.237	0.055
Dis Scz	0.340	0.189	1.801	= .073	0.239	1.604	1.450	0.151	0.152	0.319
	Neg Koren								**95% Conf Int**	
predictor	**B**	**SE**	**t**	***p***	**BF_{10}**	**$R^2_{partial}$**	**VIF**	**β_{std}**	**LC**	**HC**
Intercept	61.147	2.750	22.233	<. 001						
Pos Scz	-0.359	0.219	-1.640	0.103	0.429	1.334	1.330	-0.131	-0.289	0.027
Neg Scz	-0.306	0.199	1.534	0.127	0.417	1.169	1.110	-0.112	-0.256	0.032
Dis Scz	0.572	0.207	2.758	0.006	0.868	3.682	1.450	0.231	0.066	0.396

Negative: F(4, 198)=2.06, p=.087, R^2 = 4.00%, $R^2_{adjusted}$ =2.06%, AIC: 1689, BIC: 1709. **Positive:** F(4, 198)=1.41, p=.233, R^2 =2.77%. = $R^2_{adjusted}$ =0.008%%, AIC: 1648, BIC: 1668. VIF = variance inflation factor, priors are full Cauchy (location=0, scale = 0.354)

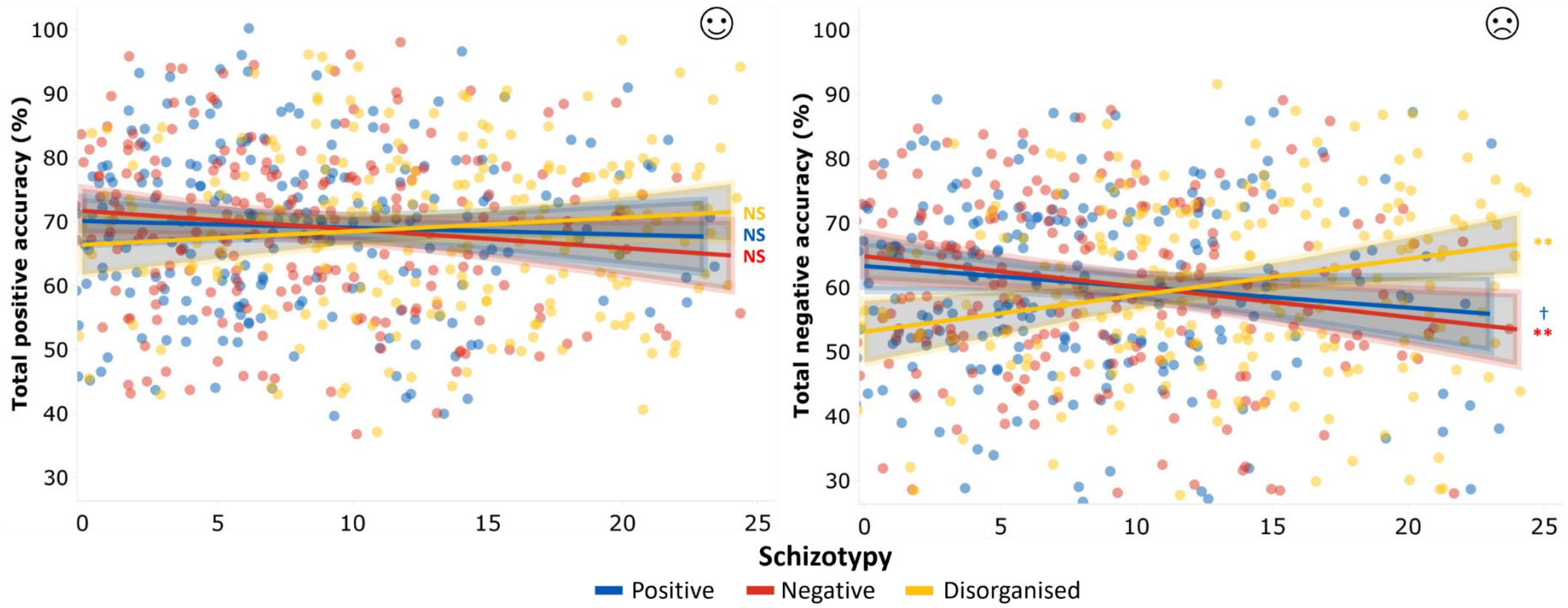


Figure 24. Multiple linear regression analyses predicting positive (**left**) and negative (**right**) emotion recognition accuracy. Negative schizotypy predicted poorer recognition of negative emotions and disorganised schizotypy predicted increased recognition of negative emotions.

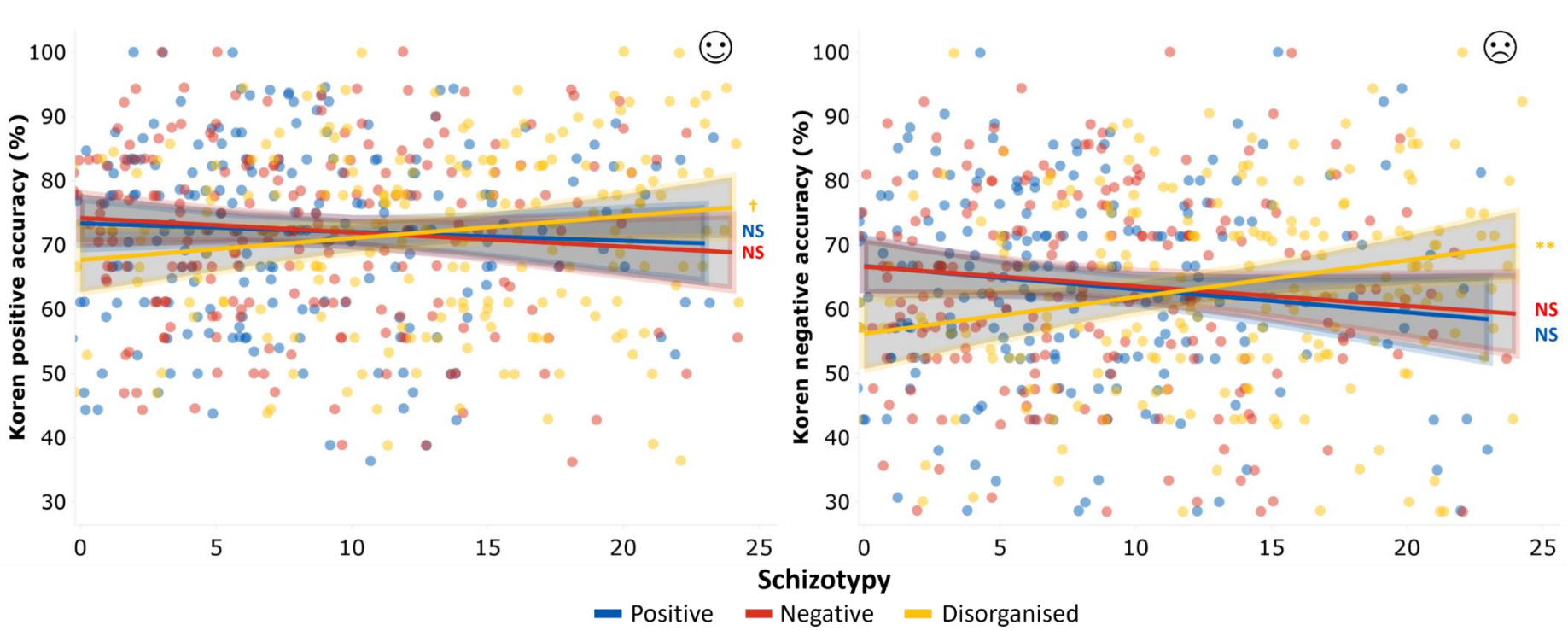


Figure 25. Multiple linear regression analyses predicting positive (**left**) and negative (**right**) emotion recognition Koren accuracy. Only disorganised schizotypy significantly predicted improved recognition of negative emotions.

3.7: Assessing potential mediators of performance using SEM

3.7.1: Explaining SEM modelling

An SEM was calculated to evaluate the potential mediation effects of psychometric metacognition and motivation. SEM was chosen over individual mediation analyses to a) give indices of overall fit to compare models b) suggest where effects are *not* present c) standardise all regression coefficients to allow comparability and d) be more succinct than multiple mediation analyses. The model coefficients can be interpreted the same way as multiple linear regression with the addition of some additional calculated effects: the total effect, direct effect, and indirect effect (**Figure 26**). The *indirect effect,* for example*,* is the amount of variance explained in M-GERT-S performance because of the relationship between schizotypy and metacognition, with metacognition in turn affecting M-GERT-S performance. The *direct effect* is the amount of variance explained in M-GERT-S performance by schizotypy controlling for the indirect effect. Finally, the *total effect* is the total of both the direct and indirect effects of schizotypy (a simple linear regression). A significant indirect effect is determined by Bias-Corrected accelerated (BCa) 95% Confidence Intervals which do not contain 0. A partial mediation requires only a significant indirect effect whereas a full mediation analysis additionally requires a non-significant direct effect. The size of the mediation effect can be assessed by the percentage change in coefficients between the total effect and direct effect. The SEM created here is a combination of many of these 'triads' (**Figure 26**).

The calculated SEM model predicted negative emotion recognition accuracy from the three schizotypy scales and entered metacognitive variables and post-motivation as potential mediators. To reduce statistical complexity negative affect was added as a control variable. Models were compared using AIC, BIC and ECVI values, with lower values representing a better model.

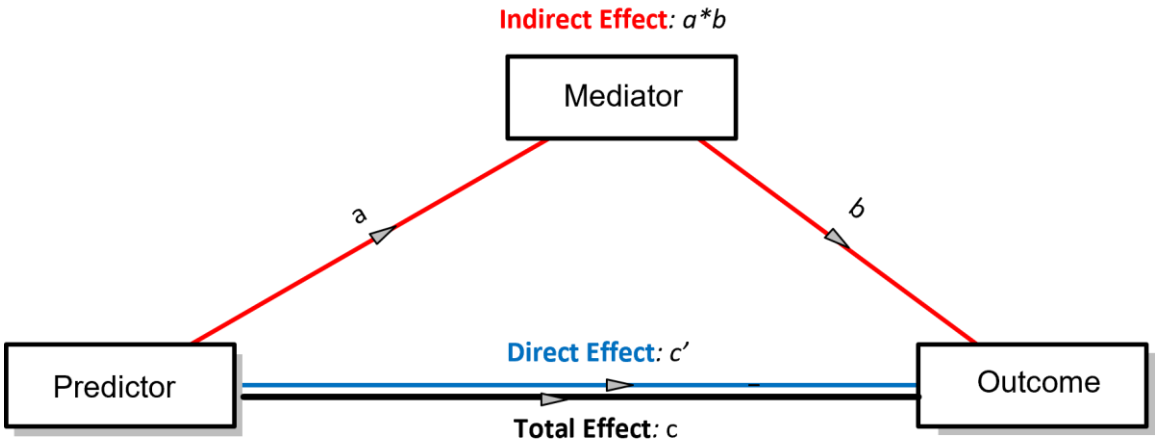


Figure 26. Diagram of a mediation analysis. Structural Equation Models can be understood as many of these analyses being calculated at once.

3.7.2: Negative Emotion Recognition Accuracy

An SEM was specified which predicted negative ER accuracy from negative and disorganised schizotypy with positive schizotypy added as a control variable. Next, Uncontrollability and Danger, Self-Reflectivity, and post-task motivation were added as mediators of disorganised schizotypy and Need to Control Thoughts was added as a mediator of negative schizotypy. As can be seen in **Figure 27***,* the direct effect of disorganised schizotypy remained significant when controlling for the mediators (β = 0.300[0.075, 0.503], *p* = .005). The respective indirect effects of disorganised schizotypy through Uncontrollability and Danger (β = -0.014, [-0.134, 0.108]), Self-Reflectivity (β = 0.036, [-0.040, 0.125]), and post-task motivation were all non-significant (β = -0.009, [-0.047,0.015]) suggesting no mediation was present. For negative schizotypy, the direct effect became trend with confidence intervals also containing zero (β = -0.140[-0.283, 0.008], *p* = .057). The respectively indirect effect of negative schizotypy through Need to Control Thoughts returned significant (β = -0.066[-0.136, -0.018], *p* = .026) and mediated 32% of the total effect of negative schizotypy. For clarity, these relationships have been isolated into a single 'triad' in **Figure 28**. While the direct effect was non-significant, this effect was still twice the size of the indirect effect. Consequently, partial mediation is a more appropriate conclusion. Together, these results suggest a) that disorganised schizotypy predicts improved ER accuracy independent of metacognition, motivation, and negative affect and b) part of the relationship between negative schizotypy and poorer ER accuracy is explained through poorer metacognition only (Hypotheses 1b - 1d). The results of this model are also described in **Appendix F**).

As a robustness check, a reverse model was also calculated which proposed schizotypy as the mediator of metacognition and motivation (**Appendix H**, **Appendix G**). Briefly, this reverse model suggested that negative schizotypy was also partially mediated by the Need to Control thoughts; suggesting this relationship may be reciprocal. However, Self-Reflectivity and Uncontrollability predicted performance indirectly through disorganised schizotypy, but not directly; suggesting disorganised schizotypy is theoretically more closely related ER performance. This latter suggestion is also supported by the reverse model being a better fit to the data (AIC = 4190, BIC = 4240, EVCI = 0.480, R^2 = 12.8%) relative to the original model (AIC = 6150, BIC = 6210, EVCI = 0.908, R^2 = 12.5).

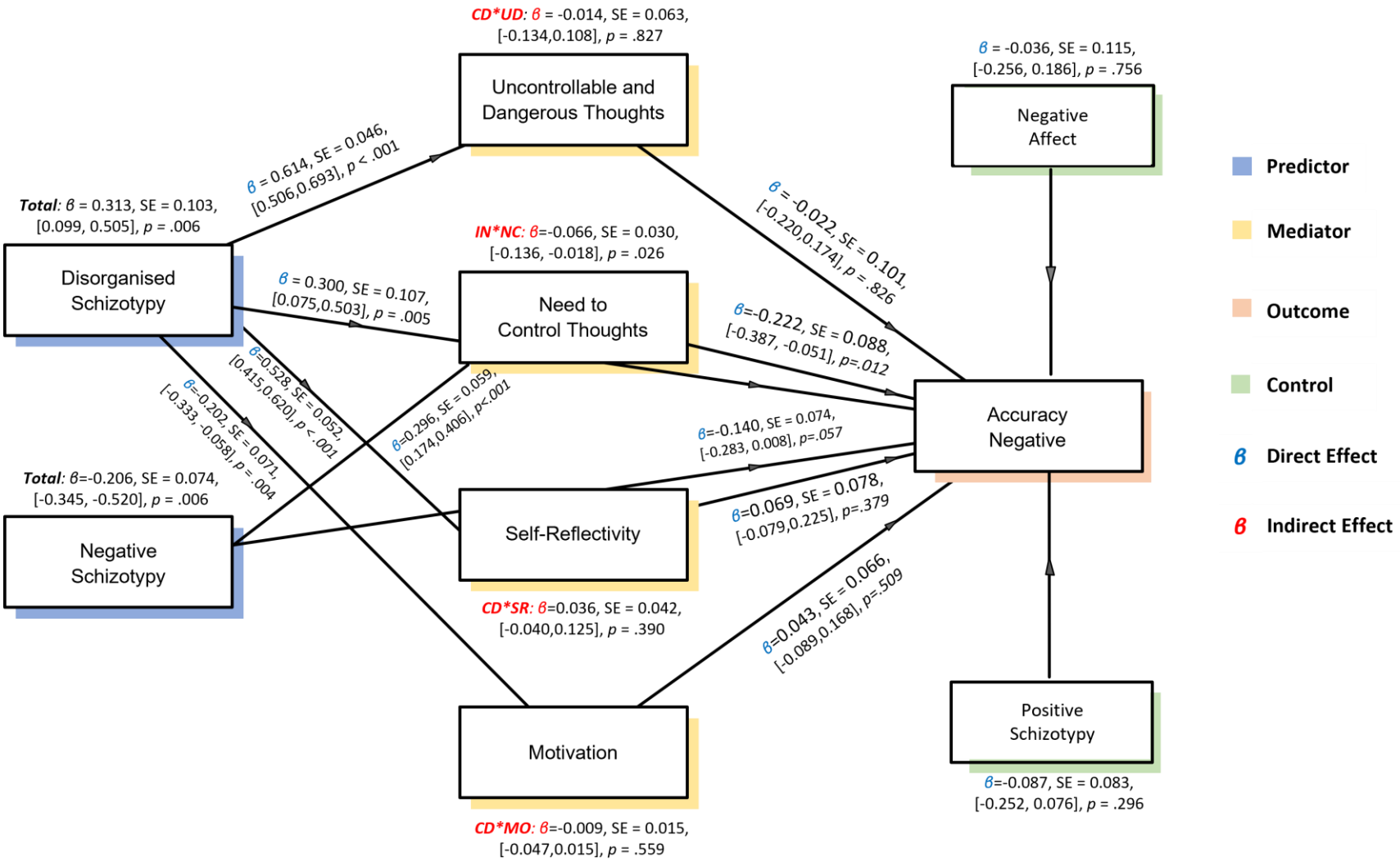


Figure 27. SEM model theorising metacognition and motivation as mediators of the effect between schizotypy and negative emotion recognition accuracy. The model suggests the effect of disorganised schizotypy is independent of metacognition and motivation, whereas the of negative schizotypy may be partially due to metacognition. Effects controlling for positive schizotypy and negative affect.

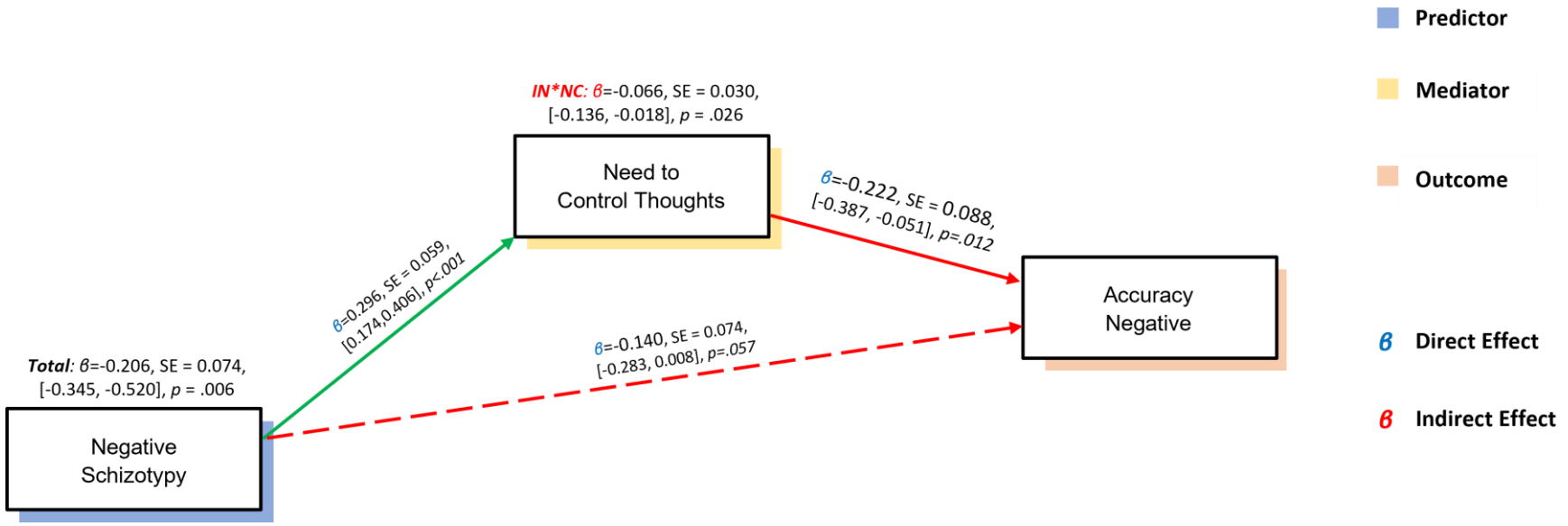


Figure 28. Reproduction of Figure 6 with only the partial mediation of negative schizotypy being highlighted for clarity. No changes to the model were made. Green arrows represent positive relationships, the solid red arrow represents a negative relationship, and the dashed red line represents the mediated relationship.

3.8: Response Confidence

The final analysis predicted confidence ratings of the M-GERT-S. Originally, these were divided by valence and accuracy in line with Chapter 3. However, the results did not differ by valence or accuracy (*Hypothesis 4*) only overall confidence ratings are shown for succinctness and to satisfy model assumptions more readily (**Appendix I**). A multiple regression analysis revealed that disorganised schizotypy significantly predicted reduced confidence ratings (β = -0.220[-0.381, -0.059], p = .008, BF_{10} = 3.892). Conversely, positive schizotypy marginally predicted greater confidence in decisions (β = 0.166[0.012, 0.320], p = .035, BF_{10} = 1.358). The results for negative schizotypy were insensitive (p = .290, BF_{10} = 0.497). The potential mediators for positive and disorganised schizotypy were also investigated, but only Self-Certainty significantly predicted increased confidence ratings when added to the model (β = 0.200[0.066, 0.333], p = .004). Disorganised schizotypy was largely unaffected by these additions (β = -0.186[-0.346, -0.026], p = .023) and positive schizotypy became trend (β = 0.131[-.023, 0.284], p = .095), although this likely reflects positive schizotypy presenting a marginal effect independently of Self-Certainty, rather than mediation. Together, this suggests positive schizotypy predicts an overconfidence bias and disorganised schizotypy predicts an underconfident bias independent of negative affect, metacognition, and motivation (**Table 18**).

Table 18. Multiple linear regression predicting response confidence ratings in the M-GERT-S from the three schizotypy dimensions.

	Confidence								95% Conf Int	
predictor	**β**	**SE**	**t**	***p***	**BF_{10}**	**$R^2_{partial}$**	**VIF**	**β**	**LC**	**HC**
Intercept	5.190	0.147	35.385	< .001						
Pos Scz	0.025	0.012	2.122	= .035	1.358	2.150	1.326	0.166	0.012	0.320
Neg Scz	-0.011	0.011	-1.061	= .290	0.497	0.546	1.105	-0.076	-0.217	0.065
Dis Scz	-0.030	0.011	-2.695	= .008	3.892	3.421	1.446	-0.220	-0.381	-0.059

***Note*:** F(3, 205) = 3.910, p = .010, R^2 = 5.4%, $R^2_{adjusted}$ = 4.0%. VIF = Variance Inflation Factor. Bayesian priors are full Cauchy (location = 0, scale = 0.354)

4.0 Discussion

4.1: Summary

This study assessed the potential mediatory roles of metacognition, negative affect, and motivation between schizotypy and Emotion Recognition (ER) performance. The first hypothesis that negative schizotypy would predict poorer ER performance was supported. However, these deficits did not extend to Koren accuracy (*Hypothesis 2*) suggesting participants do not act on faulty cognition. Disorganised schizotypy was unexpectedly associated with improved performance of negative emotions and improved Koren accuracy, meaning participants acted on their improved cognition. There was inconclusive evidence regarding positive schizotypy. In terms of the proposed mediations on *Hypotheses 1b* and *1d*, negative affect and motivation did not explain performance differences in schizotypy. However, while metacognition did not mediate the improved performance in disorganised schizotypy, it partially mediated poorer performance in negative schizotypy through increased levels of maladaptive "Need to Control Thoughts" (*Hypothesis 1c*). Finally, positive schizotypy presented an overconfidence bias and disorganised schizotypy presented an underconfidence bias in decisions which did not depend on valence or decision accuracy (*Hypothesis 3*).

4.2: Emotion Recognition Accuracy

4.2.1: Overall performance

The finding that schizotypy was associated with performance on negative emotions is consistent with reviews in patients with schizophrenia[96]. As the current study replicated these deficits in schizotypy this may suggest that the deficits in patients are independent of clinical confounds. However, the wider schizotypy literature now including this study is currently equivocal[385,436,439,441,442,444,446,449] and thus further investigation is still required. One reason why only negative emotions may have elicited effects is that they may activate unpleasant internal states in participants; producing excessive anxiety that can be detrimental to performance. This has been a consistent suggestion also in Chapter 2 and Chapter 3. The lack of significant association between positive emotion recognition and schizotypy is also consistent with some[385,439,446] but not all past investigations[436,440,442,449]. As there are currently no investigations in schizotypy or schizophrenia that compare performance to controls on the GERT-S, it cannot be ruled out that these findings are due to the ER instrument used. Due to the employment of the GERT-S, however, a lack of diverse positive stimuli is an unlikely explanation for these findings, which is a commonly cited limitation of previous emotion recognition research[96].

4.2.2: Negative schizotypy

These explanations are likely only applicable to negative schizotypy, which predicted poorer ER performance consistent with previous research in patients[101] and adds to equivocal research in schizotypy[436–440]. One clinical study reported that 20% of the variance in ER performance was explained by negative symptoms[461]. The current study found the effect of negative schizotypy explained 3.5% of the variance, which is in line with the dimensional view of psychosis as a spectrum (i.e., less severe deficits should occur with less severe schizophrenia-like experiences). The correlational analyses suggested these deficits were potentially due to poorer fear and anger recognition[vii], which is consistent with findings in patients[96]. Previous research has also found social anxiety items within negative schizotypy scales primarily drive these effects[438]. Specifically, poorer ER may increase social anxiety through reduced confidence in social cognitive abilities[437], perhaps leading to increased social withdrawal and negative traits[443]. However, in this study, negative schizotypy did not predict confidence in decisions which conflicts with this suggestion. Moreover, the O-LIFE conceptualises social anxiety under the disorganised dimension rather than the negative dimension[303].

Another explanation could be that this relationship is mediated through increased alexithymia, which is increased in clinical samples[462] and correlates with all three schizotypy trait dimensions[463,464]. This initially contradicts the current explanation being specific to negative schizotypy. However, without controlling for scale inter-correlation, it is unclear whether these associations are general or scale-specific. If this suggestion were accurate, the experiential rather than expressive negative traits would correlate with self-reported alexithymia. However, no study at the time of writing has controlled for alexithymia in this context. One study has assessed alexithymia, but because task performance was unaffected by schizotypy, further investigation was unnecessary[447].

4.2.3: Disorganised schizotypy

This study is the first to report a positive association between disorganised schizotypy and ER. This conflicts with previous research in schizotypy commonly reporting no associations[436–439,441,443,444] and patient samples finding negative associations[101]. This improved performance was driven primarily through disgust recognition, which further contradicts the impaired disgust recognition in patient samples[96] and schizotypy samples[437,441]. Previous studies have reported that schizotypy can exaggerate the perceived emotion expressed in ER tasks[465], which may lead to improved ER performance. However, performance benefits are commonly found in the paranoid subtype of patients[466] and paranoia-related (positive) schizotypy[465], rather than disorganised schizotypy.

[vii] These correlations did not survive correction for multiple comparisons.

Alternatively, perhaps participants who can more accurately identify negative emotions have a negatively biased perception of social interactions - leading to reports of disorganised thinking. As all schizotypy dimensions generally correlated with increased reaction time, increased deliberation time is an unlikely explanation. As the schizotypy ER literature has limited investigations of the disorganised aspects of schizotypy and no previous studies have used the O-LIFE or GERT-S, these findings are difficult to interpret.

4.2.4: Positive schizotypy

The current study was unable to determine the relationship between positive schizotypy and ER performance, as the Bayesian analyses suggested the data were insensitive to detect effects. Previously, it has been suggested that positive schizotypy traits such as paranoia may bias participants to expect negative facial emotions and that poorer ER may make individuals highly suspicious[440]. This is consistent with ER deficits correlating with positive symptoms in patient samples[101] but contrasts with the majority of non-clinical studies[436,438,439,443,444,446–448]. The disparity between clinical and non-clinical studies may be explained by very high levels of positive schizotypy traits being necessary to produce deficits. Indeed, negative schizotypy has been reported to only correlate with FER performance in those classified as being high in schizotypy[439]. In this study, the normative comparisons found that the levels of positive schizotypy were significantly lower in the current sample, whereas negative schizotypy and disorganised schizotypy were not and both predicted ER performance.

4.3: Koren Emotion Recognition Accuracy

While negative schizotypy predicted poorer total accuracy it was not associated with Koren accuracy. These findings replicate those of Koren et al.[216] and extend this relationship into the domain of social cognition for the first time. The results suggested nearly half (42%) of the detrimental effects of negative schizotypy on ER can be alleviated by asking participants which information they would like to act upon – suggesting good metacognitive control performance. However, the BF_{10} suggested there was only anecdotal support for the null hypothesis (BF_{10} = 0.417) and 58% of the original relationship remained, meaning deficits are not completely removed. This may suggest both clinical patients and people high in schizotypy can mitigate the impact of faulty social cognitive decisions by abstaining from responding (e.g., by potentially seeking out more information). This may help explain why the associations between social cognition and functional outcomes are only weak to moderate in patients[75], as the application of cognition is moderated by metacognition. Future research should aim to both replicate these findings in clinical patients and

more broad social cognitive tasks. If these findings are replicated, it may help highlight adaptive coping mechanisms used by some patients that can be applied to other people with schizophrenia. If these findings are not replicated, it may highlight a mechanism predictive of transition to clinical psychosis and a useful future therapeutic target.

4.4: Explanations for ER performance

4.4.1: Metacognition

Consistent with previous Chapters, psychometric metacognition was largely related to schizotypy[viii]. The SEM analyses suggested that greater Self-Reflectivity (i.e., "At times, I have misunderstood other people's attitudes towards me") and increased Uncontrollability and Danger (i.e., "I could make myself sick with worrying") were associated with disorganised schizotypy, which subsequently improved ER performance. However, these metacognitive variables did not directly affect ER performance. Greater Need to Control Thoughts ("I will be punished for not controlling certain thoughts") predicted reduced ER performance both directly and indirectly through negative schizotypy. This indirect relationship was found to be statistically reciprocal. This relationship could be explained through persecutory thinking styles increasing the avoidance of social contact which is a key aspect of negative schizotypy. This social withdrawal may thereafter further exacerbate persecutory thinking styles, due to the reduced exposure to disconfirmatory evidence and opportunities to develop social-cognitive skills (e.g., Emotion Recognition). This maladaptive psychometric metacognitive style, which is highly relevant to behavioural control, clearly conflicts with the finding of intact metacognitive control ability from the Koren accuracy analysis. The reasons for these contradictions are unclear, although this may suggest a dissociation between self-reported and behavioural measures of metacognition in schizotypy, or perhaps that these assessments measure different sub-domains of metacognitive control. This latter point is supported by the null associations between psychometric and behavioural metacognition in the overall sample. Previous clinical studies are inconsistent as to whether psychometric metacognition is beneficial or detrimental to ER performance[190,467,468]. Regardless, metacognition has clear relevance for both explaining social cognitive deficits and their transferral to real life which warrants further investigation.

[viii] These associations will be discussed in Chapter 6.

4.4.2: Motivation

Only disorganised schizotypy was related to pre- and post-task motivation, which partially conflicts with clinical studies implicating negative symptoms[258]. This lack of association to negative traits may potentially mean the associations to negative symptoms in patients are partially attributable to confounds of patient status (i.e., medication). Moreover, perhaps sub-clinical levels of negative traits do not replicate the clinical deficits in anticipatory pleasure[469,470], which is relevant for task-specific motivation. The novel association between motivation and disorganised schizotypy may be explained through the anxiety-related items of the Cogdis scale of the O-LIFE.

4.4.3: Negative affect

Although negative affect was found to be positively associated with schizotypy, it was unrelated to performance. This contrasts with previous research in clinical MDD[230] and is not explained by low levels of negative affect in the current sample (**Table 15**). An alternative explanation is that perhaps the GERT-S is not sensitive to detect deficits related to negative affect, as the use of multi-modal stimuli in the GERT-S (prosody, body language, facial expression) may provide adequate information for processing. This may be consistent both with smaller deficits in MAD disorders relative to schizophrenia[230] and only the more difficult to recognise negative emotions but not positive emotions being predicted by schizotypy. Irrespective of this, schizotypy was found to affect ER performance independently of negative affect, which may extend to the high MAD co-morbidity in clinical patients.

4.5: Response confidence

Clinical studies consistently report that patients are underconfident in correct responses and overconfident in errors[150], which may contribute to impaired functioning and delusion formation, respectively. However, the associations between schizotypy and confidence ratings in the current study were not dependent on accuracy, suggesting a divergence with most[150] but not all past research[420]. Positive schizotypy presented an overconfidence bias only, which is partially consistent with limited previous studies in schizotypy[433,471,472] and schizophrenia[154,156,160] reporting an overconfidence in errors. This lack of specificity to accuracy may also highlight a potential cognitive mechanism subject to deterioration at illness onset. As a clinical diagnosis is often the result of positive symptoms and is associated with a decline in social cognition, this overconfidence bias would now be applied to impaired cognitive performance. The underconfidence bias of disorganised schizotypy is a novel finding and may potentially explain the improved ER performance in the current study. Although deliberation time and motivation did not explain this relationship, underconfidence

may produce more effortful deliberation, which could be assess by measuring pupil dilation during deliberation. That being said, it is surprising disorganised schizotypy also predicts acting on correct knowledge (Koren accuracy) despite this underconfidence bias. This may represent a weaker link between confidence and acting on ability in disorganised (i.e., less confidence is needed to act). Finally, the fact that negative schizotypy was unrelated to confidence ratings is consistent with previous findings[473].

4.6: Strengths and limitations

The main strengths of this Chapter are the broad array of influences on ER performance considered, the in-depth information extracted from the metacognitive task adaptations, and the range of positive emotions asses. The first limitation is the cross-sectional nature of this study meaning that the SEM results are associative and so causality cannot be determined. Secondly, because only a single assessment of ER was used it is unclear if these results generalise to social cognition more generally, or if they are specific to the GERT-S. Finally, the internal consistency for the GERT-S was low which should caution interpretations.

5.0 Conclusions

This Chapter found that negative schizotypy was associated with deficits in the recognition of negative emotions, which may suggest this relationship in clinical patients is independent of confounds of patient status (i.e., anti-psychotic medication). Inconclusive evidence was found for an association with positive schizotypy, which may be explained by the low levels of positive schizotypy traits in the current investigation. Unexpectedly, disorganised schizotypy predicted improved recognition which may be due to under-confidence in decisions increasing in more effortful deliberation. All these relationships were independent of negative affect suggesting that impairments in clinical patients may be also independent of MAD co-morbidity. Similarly, task-related motivation also did not mediate deficits indicating this relationship may be specific to clinical samples. There was evidence that poorer self-reported metacognitive control processes may exacerbate ER deficits in negative schizotypy, whereas the intact behavioural metacognitive control processes may mean these deficits are not translated to the real world (Koren accuracy), highlighting a discrepancy between self-reported beliefs and actual behaviour. Future studies should expand these metacognitive adaptations to other social cognitive tasks.

Appendices

Appendix A

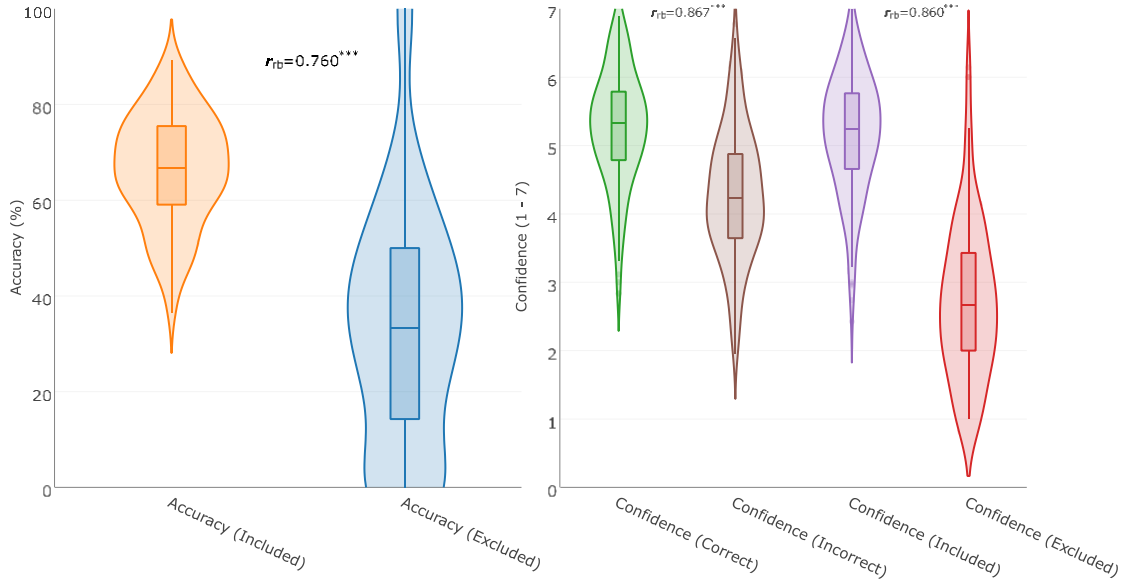
Performance descriptive statistics of the M-GERT-S.

	Accuracy (0% - 100%)					Confidence (1 – 7)					Inclusion (0% - 100%)				
Emotion	**M**	**SD**	**Median**	**MAD**	**IQR**	**M**	**SD**	**Median**	**MAD**	**IQR**	**M**	**SD**	**Median**	**MAD**	**IQR**
Interest	79.11	22.98	67.14	49.42	33.33	4.7	1.17	4.81	0.99	1.33	86.41	22.54	99.76	0	33.33
Amusement	76.87	28.16	99.51	0.00	33.33	5.6	1.04	5.89	0.99	1.33	94.50	17.17	99.93	0	0
Anger	76.08	25.14	67.04	49.42	33.33	5.6	1.05	5.94	0.99	1.33	96.28	15.17	99.96	0	0
Pleasure	76.08	27.18	67.12	49.42	33.33	5.32	1.07	5.42	0.99	1.67	92.72	19.64	99.91	0	0
Relief	73.21	26.45	66.94	49.42	33.33	5.01	1.13	5.1	0.99	1.33	90.78	20.98	99.88	0	0
Sadness	67.94	29.02	66.78	49.42	66.67	4.89	1.13	4.89	0.99	1.67	90.45	19.49	99.85	0	0
Joy	65.55	30.9	66.72	49.42	66.67	5.31	1.08	5.49	0.99	1.33	93.37	18.16	99.91	0	0
Irritation	65.55	30.38	66.71	49.42	66.67	4.45	1.19	4.25	1.48	1.67	83.82	24.80	99.71	0	33.33
Disgust	59.97	25.90	66.51	0.00	33.33	4.7	1.07	4.6	0.99	1.33	85.92	24.00	99.78	0	33.33
Despair	59.97	30.10	66.53	49.42	33.33	5.06	1.06	5.12	0.99	1.33	91.59	19.87	99.89	0	0
Fear	52.47	32.61	66.27	49.42	33.33	4.74	1.12	4.78	0.99	1.67	89.81	21.81	99.86	0	0
Surprise	46.73	24.92	33.7	49.42	33.33	4.35	1.22	4.66	1.48	2	79.61	28.22	99.64	0	33.33
Pride	43.06	28.79	33.57	49.42	33.33	4.61	1.07	4.6	0.99	1.33	85.28	24.94	99.76	0	33.33
Anxiety	38.44	28.60	33.48	49.42	66.67	4.35	1.09	4.38	0.99	1.33	82.85	26.49	99.73	0	33.33
Total	62.93	10.06	64.17	10.59	11.9	4.91	0.84	4.98	0.78	1.02	88.81	15.09	92.98	10.59	18.45
Negative Total	60.06	13.75	61.5	14.12	19.05	4.83	0.87	4.72	0.85	1.1	88.67	16.04	95.03	7.06	19.05
Positive Total	68.98	13.08	71.87	16.47	16.67	5.09	0.86	5.02	0.91	1.17	90.51	15.27	94.62	8.24	11.11

Note: IQR – interquartile range, MAD = median absolute deviation, medians are interpolated medians (nearest integer is true median).

Appendix B

Differences in accuracy and confidence ratings in the overall sample. Results suggest participants understood the confidence and inclusion scale ratings.



Appendix C

Multiple regressions predicting psychosis-proneness from metacognition

	Positive Schizotypy								95% Conf Int	
predictor	B	SE	t	p	BF10	R2partial	VIF	βstd	LC	HC
Intercept	-4.6941	1.9263	-2.437	0.016						
MCQ_PB	**0.1862**	**0.0853**	**2.182**	**0.030**	**3.313**	**2.292**	**1.183**	**0.1462**	**0.014**	**0.278**
MCQ_CSC	**0.1912**	**0.0779**	**2.453**	**0.015**	**8.166**	**2.878**	**1.370**	**0.1769**	**0.035**	**0.319**
MCQ_UD	0.1237	0.0842	1.469	0.143	1.271	1.051	1.784	0.1208	-0.041	0.283
MCQ_NCT	0.0522	0.1061	0.492	0.623	0.336	0.119	1.631	0.0387	-0.116	0.194
BCIS_R	**0.2480**	**0.0951**	**2.609**	**0.010**	**13.368**	**3.244**	**1.393**	**0.1897**	**0.046**	**0.333**
	Negative Schizotypy								95% Conf Int	
predictor	B	Robust SE	t	p	BF10	R2partial	VIF	βstd	LC	HC
Intercept	3.287	1.134	2.899	0.004						
MCQ_UD	0.111	0.082	1.361	0.175	0.487	0.886%	1.455	0.109	-0.0492	0.267
MCQ_NCT	**0.309**	**0.099**	**3.122**	**0.002**	**27.584**	**3.825%**	**1.455**	**0.229**	**0.0713**	**0.387**
	Disorganised Schizotypy								95% Conf Int	
predictor	B	SE	t	p	BF10	R2partial	VIF	βstd	LC	HC
Intercept	-5.07560	1.8048	-2.8122	0.005						
MCQ_CC	**0.17095**	**0.0856**	**1.9974**	**0.047**	**0.942**	**1.937%**	**1.300**	**0.03218**	**0.03218**	**0.231**
MCQ_PB	0.02018	0.0808	0.2496	0.803	0.147	0.031%	1.248	0.42426	0.42426	0.127
MCQ_CSC	0.03872	0.0724	0.5347	0.593	0.140	0.141%	1.389	-0.00277	-0.00277	0.151
MCQ_UD	**0.48347**	**0.0795**	**6.0791**	**< .001**	**>999**	**15.465%**	**1.868**	**0.28520**	**0.28520**	**0.562**
MCQ_NCT	-0.00415	0.0980	-0.0424	0.966	0.126	0.001%	1.633	0.03218	0.03218	0.126
BCIS_R	**0.41502**	**0.0900**	**4.6088**	**< .001**	**>999**	**9.515%**	**1.469**	**0.42426**	**0.42426**	**0.407**

Positive: *F(5, 203)=12.1, p=<.001, R^2 = 23.%, $R^2_{adjusted}$ =21.1%, AIC: 1301, BIC: 1325.* ***Negative:*** *F(2, 206)=11.565, p<.001, R^2 =9.22%. = $R^2_{adjusted}$ =8.33%, AIC: 1300 BIC: 1313.* ***Disorganised:*** *F(6, 202)=30.3, p<.001, R^2 =47.3%. $R^2_{adjusted}$ =45.8%, AIC: 1238, BIC: 1265. VIF = variance inflation factor, priors are full Cauchy (location=0, scale = 0.354). Robust SE calculated using the sandwich*[474] and lmtest[475] R packages.

Appendix D

Spearman correlations between the accuracy of each emotion and schizotypy.

			Schizotypy			
Valance	**Arousal**	**Scale**	**Total**	**Pos**	**Neg**	**Dis**
		Positive	0.718***	-		
		Negative	0.608***	0.122†	-	
		Disorganised	0.834***	0.490***	0.300***	-
Positive	High	Interest	-0.100	-0.106	-0.091	-0.071
		Pleasure	0.074	0.046	0.065	0.049
		Relief	-0.113	-0.047	-0.038	-0.139*
	Low	Amusement	0.034	0.035	-0.079	0.097
		Joy	-0.040	-0.011	-0.105	0.036
		Pride	0.035	-0.016	0.002	0.061
Negative	High	Anger	-0.018	0.058	-0.124†	0.050
		Fear	-0.024	-0.009	-0.131†	0.069
		Despair	0.009	0.000	-0.024	0.020
	Low	Anxiety	0.021	0.024	0.027	0.025
		Irritation	0.003	-0.076	0.001	0.083
		Sadness	-0.083	-0.001	-0.109	-0.050
	NR	Disgust[a]	0.167*	0.072	-0.012	0.254***
	NR	Surprise	-0.030	0.023	-0.037	-0.054

Note: [a] = Schlegel & Scherer (2016) did not suggest arousal of disgust, †= $p < .1$, * = $p < .05$, ** = $p < .01$, *** = $p < .001$. Trend-level correlations between negative schizotypy and anger and fear recognition and disorganised schizotypy and Relief accuracy do not survive correction for multiple comparisons ($p > .277$).

Appendix E

Spearman correlations between reaction time of each emotion and schizotypy

			Schizotypy				Negative Affect
Valance	**Arousal**	**Scale**	**Total**	**Pos**	**Neg**	**Dis**	**DASS Total**
		Pos	0.718***	-			
		Neg	0.608***	0.122†	-		
		Dis	0.834***	0.490***	0.300***	-	
		DASS Total	0.634***	0.404***	0.309***	0.681***	-
Positive	High	Interest	0.132†	0.075	0.070	0.139*	0.076
		Pleasure	0.083	0.069	0.058	0.078	0.055
		Relief	0.099	0.079	0.020	0.104	0.128
	Low	Amusement	0.158*	0.131†	0.101	0.135†	0.103
		Joy	0.115	0.015	0.124†	0.126†	0.052
		Pride	-0.005	-0.065	0.045	0.031	-0.013
Negative	High	Anger	0.071	0.015	0.057	0.051	0.055
		Fear	0.149*	0.042	0.180**	0.124†	0.032
		Despair	0.083	0.094	0.068	0.059	0.081
	Low	Anxiety	0.061	0.012	0.100	0.049	-0.042
		Irritation	0.042	0.031	0.012	0.018	-0.015
		Sadness	0.137*	0.101	0.091	0.118†	0.169*
	NR	Disgust[a]	0.173*	0.109	0.112	0.163*	0.175*
	NR	Surprise	-0.005	-0.001	0.026	-0.039	-0.077

Note: [a] = Schlegel & Scherer (2016) did not suggest arousal of disgust, †= $p < .1$, * = $p < .05$, ** = $p < .01$, *** = $p < .001$, DASS Total = Depression, Anxiety, and Stress Scale total score.

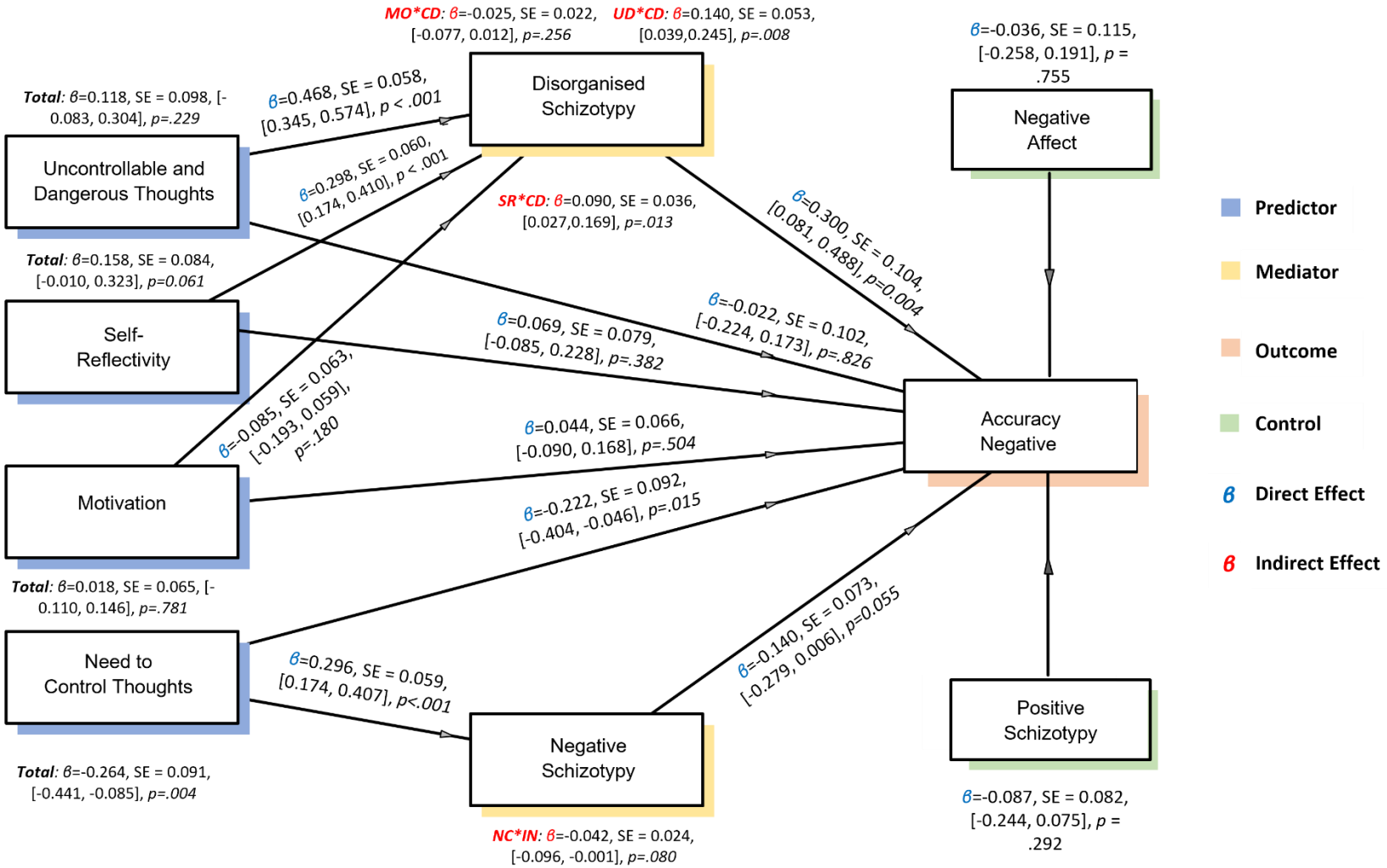
Appendix F

	Effect	Outcome	op	Predictor	β std.all	Robust Boot SE	Z	*p* value	BCa CI Lower	BCa CI Upper
1	Direct (C')	Accuracy Negative	~	Unex	-0.087	0.083	-1.045	0.296	-0.252	0.076
2		Accuracy Negative	~	Intan	-0.140	0.074	-1.900	0.057	-0.283	0.008
3		Accuracy Negative	~	Cogdis	0.300	0.107	2.790	0.005	0.075	0.503
4		Accuracy Negative	~	DASS	-0.036	0.115	-0.311	0.756	-0.256	0.186
5	(B)	Accuracy Negative	~	BCIS R	0.069	0.078	0.879	0.379	-0.079	0.225
6		Accuracy Negative	~	MCQ NCT	-0.222	0.088	-2.516	0.012	-0.387	-0.051
7		Accuracy Negative	~	MCQ UD	-0.022	0.101	-0.220	0.826	-0.220	0.173
8		Accuracy Negative	~	Motivation	0.043	0.066	0.661	0.509	-0.089	0.168
9	(A)	MCQ_UD	~	Cogdis	0.614	0.046	13.255	0.000	0.506	0.693
10		BCIS R	~	Cogdis	0.528	0.052	10.072	0.000	0.415	0.620
11		MCQ_NCT	~	Intan	0.296	0.059	4.981	0.000	0.174	0.406
12		Motivation	~	Cogdis	-0.202	0.071	-2.851	0.004	-0.333	-0.058
13	Indirect (A * B)	CD_UD	:=	Cogdis*MCQ UD	-0.014	0.063	-0.218	0.827	-0.134	0.108
14		CD_SR	:=	Cogdis * BCIS R	0.036	0.042	0.860	0.390	-0.040	0.125
15		CD_MO	:=	Cogdis * Motivation	-0.009	0.015	-0.585	0.559	-0.047	0.015
16		IN_NC	:=	Intan*MCQ NC	-0.066	0.030	-2.225	0.026	-0.136	-0.018
17	Total (C)	Cogdis	:=	Cogdis + 13 + 14 +15	0.313	0.103	3.037	0.002	0.099	0.505
18		Intan	:=	Intan + 16	-0.206	0.075	-2.759	0.006	-0.350	-0.056

Note: Model fitted with Robust Maximum Likelihood (MLR) with bootstrapped standard errors (10000 replications). *Fit indices: $\chi^2(18) = 154$, $p<.001$, CFI: 0.597, ACI: 6154, BIC: 6210, EVCI: 0.908, RMSEA: 0.191, SRMR: 0.146, GERT R^2: 12.8%*

Appendix G

Reverse model that considers schizotypy as a mediating variable.



Appendix H

Structural Equation Model results of the reverse model considering schizotypy as the mediator between metacognition and motivation with performance.

		Outcome	op	Predictor	β std.all	Robust Boot SE	Z	*p* value	BCa CI Lower	BCa CI Upper
1	Direct (C')	Accuracy Negative	~	Unex	-0.087	0.082	-1.054	0.292	-0.244	0.075
2		Accuracy Negative	~	Intan	-0.140	0.073	-1.921	0.055	-0.279	0.006
3		Accuracy Negative	~	Cogdis	0.300	0.104	2.890	0.004	0.081	0.488
4		Accuracy Negative	~	DASS	-0.036	0.115	-0.312	0.755	-0.258	0.191
5		Accuracy Negative	~	BCIS R	0.069	0.079	0.874	0.382	-0.085	0.228
6	(B)	Accuracy Negative	~	MCQ NCT	-0.222	0.092	-2.425	0.015	-0.404	-0.046
7		Accuracy Negative	~	MCQ UD	-0.022	0.102	-0.220	0.826	-0.224	0.173
8		Accuracy Negative	~	Motivation	0.044	0.066	0.660	0.509	-0.090	0.168
9	(A)	Cogdis	~	MCQ UD	0.468	0.058	8.121	0.000	0.345	0.574
10		Cogdis	~	BCIS R	0.298	0.060	4.974	0.000	0.174	0.410
11		Cogdis	~	Motivation	-0.085	0.063	-1.341	0.180	-0.193	0.059
12		Intan	~	MCQ NCT	0.296	0.059	4.981	0.000	0.174	0.407
13	Indirect (A*B)	UD_CD	:=	MCQ UD *Cogdis	0.140	0.053	2.653	0.008	0.039	0.245
14		SR_CD	:=	BCIS R * Cogdis	0.090	0.036	2.473	0.013	0.027	0.169
15		MO_CD	:=	Motivation * Cogdis	-0.025	0.022	-1.135	0.256	-0.077	0.012
16		NC_IN	:=	MCQ NC * Intan	-0.042	0.024	-1.750	0.080	-0.096	-0.001
17	Total (C)	BCIS_R_Total	:=	BCIS_R + 14	0.158	0.084	1.877	0.061	-0.010	0.323
18		UD_Total	:=	MCQ_UD + 13	0.118	0.098	1.203	0.229	-0.083	0.304
19		MO_Total	:=	Motivation + 15	0.018	0.065	0.278	0.781	-0.110	0.146
20		NC_Total	:=	MCQ NCT + 16	-0.264	0.091	-2.900	0.004	-0.441	-0.085

Note: Model fitted with Robust Maximum Likelihood (MLR) with bootstrapped standard errors (10000 replications). *Fit indices: $\chi^2(9) = 69$, $p<.001$, CFI: 0.720, ACI: 4190, BIC: 4240, EVCI: 0.480, RMSEA: 0.180, SRMR: 0.063, GERT R^2: 12.5%*

Appendix I

Response confidence analyses as a function of veracity and affect

	Pos Correct								95% Conf Int	
predictor	**B**	**Robust SE**	**t**	***p***	***BF*$_{10}$**	***R*$^2_{partial}$**	**VIF**	**β$_{std}$**	**LC**	**HC**
Intercept	5.662	0.166	34.167							
Pos Scz	0.025	0.014	1.790	.0749	0.975	1.929%	1.353	0.159	0.003	0.316
Neg Scz	-0.012	0.011	-1.091	.276	0.541	0.516%	1.126	-0.075	-0.218	0.068
Dis Scz	-0.025	0.015	-1.674	.096	1.720	1.627%	2.047	-0.180	-0.372	0.013
DASS	-0.002	0.006	-0.364	.716	0.375	0.067%	1.845	-0.034	-0.217	0.149
	Pos Incorrect								**95% Conf Int**	
predictor	**B**	**SE**	**t**	***p***	***BF*$_{10}$**	***R*$^2_{partial}$**	**VIF**	**β$_{std}$**	**LC**	**HC**
Intercept	4.968	0.187	26.633	< .001						
Pos Scz	0.037	0.015	2.456	.015	5.057	2.885%	1.358	0.190	0.037	0.342
Neg Scz	-0.019	0.014	-1.413	.159	0.563	0.974%	*1.125*	-0.099	-0.238	0.039
Dis Scz	-0.063	0.017	-3.785	< .001	188.1	6.593%	2.054	-0.360	-0.547	-0.172
DASS_Total	0.004	0.007	0.628	.531	0.214	0.194%	1.845	0.057	-0.121	0.234

Model Corr: *F(4, 204)=2.56, p=.040, R^2 = 4.78%, $R^2_{adjusted}$ =2.91%, AIC: 541, BIC: 561.* ***Model Incorr:*** *F(4, 203)=6.15, p<.001 R^2 = 10.8%, $R^2_{adjusted}$ =9.05%, AIC: 613, BIC: 633. VIF = variance inflation factor.*

	Neg Correct								95% Conf Int	
predictor	**B**	**Robust SE**	**t**	***p***	***BF*$_{10}$**	***R*$^2_{partial}$**	**VIF**	**β$_{std}$**	**LC**	**HC**
Intercept	5.358	0.154	34.802	< .001						
Pos Scz	0.024	0.016	1.502	.134	0.692	1.637	1.358	0.147	-0.010	0.305
Neg Scz	-0.003	0.011	-0.255	.799	0.263	0.037	1.124	-0.018	-0.162	0.126
Dis Scz	-0.029	0.015	-1.853	.065	1.427	1.966	2.054	-0.199	-0.393	-0.005
DASS	<0.001	0.006	-0.007	.995	0.289	0.000	1.845	0.001	-0.185	0.184
	Neg Incorrect								**95% Conf Int**	
predictor	**B**	**SE**	**t**	***p***				**β$_{std}$**	**LC**	**HC**
Intercept	4.625	0.175	26.362	< .001						
Pos Scz	0.033	0.014	2.319	.021	1.919	2.569	1.358	0.184	0.028	0.340
Neg Scz	-0.008	0.013	-0.639	.524	0.349	0.200	1.124	-0.046	-0.189	0.096
Dis Scz	-0.034	0.016	-2.187	.030	3.856	2.291	2.054	-0.213	-0.405	-0.021
DASS	-0.003	0.007	-0.441	.660	0.365	0.095	1.845	-0.041	-0.223	0.141

Model Corr: *F(4, 204)=1.84, p=.123, R^2 = 3.48%, $R^2_{adjusted}$ =1.59%, AIC: 550, BIC: 570.* ***Model Incorr:*** *F(4, 204)=3.01, p=.019, R^2 = 5.57%, $R^2_{adjusted}$ =3.71%, AIC: 591, BIC: 611. VIF = variance inflation factor.*

Chapter 5

Immersive Virtual Reality assessment of neurocognition in schizotypy

Can immersive Virtual Reality adaptations of cognitive tasks mitigate deficits in psychometric schizotypy? A 2D vs. Virtual Reality comparison

Introduction: Cognitive dysfunction in schizophrenia is associated with poorer daily functioning. However, other factors such as poorer motivation, engagement, and miscomprehension of cognitive tasks may partially explain poorer cognitive performance. The current Chapter compared previous findings in a 'traditional' 2D cognitive task in Chapter 2, with an immersive Virtual Reality (VR) adaptation of this task in individuals varying in psychometric schizotypy.

Methods: An additional 156 participants were recruited and completed the Oxford-Liverpool Index of Feelings and Experiences (O-LIFE), Depression Anxiety and Stress Scale (DASS-21), assessments of pre- and post-task motivation in additional to an immersive VR adaptation of the Probabilistic Reversal Learning task.

Results: The previous findings of Chapter 2 suggested that positive schizotypy predicted poorer performance to punishing stimuli only. In contrast, the current VR study revealed no associations between any schizotypy dimension and performance ($p > .375$). Both pre-task (Cohen's $d = 1.300$) and post-task motivation ($d = 0.506$) were significantly higher in the current VR study. The samples did not differ in terms of positive, negative, or disorganised schizotypy traits nor negative affect.

Discussion: This study presents preliminary evidence that immersive Virtual Reality environments may reduce cognitive task deficits in schizotypy; potentially through increased engagement, motivation, and comprehension. A replication of this effect in clinical patients may help to further understand contributory factors of poorer functioning.

1.0: Introduction

1.1: Summary of aims

Chapter 2 suggested that the association between schizotypy and poorer cognitive task performance was not explained by poorer motivation. However, methodological limitations included a positively skewed distribution, asynchronous application of the scale with each cognitive task, and the potential unsuitability of the MIAMI scale for non-clinical populations meant these conclusions were unclear. As a result, this Chapter aimed to manipulate rather than control for motivation statistically. To do so, an immersive Virtual Reality Probabilistic Reversal Learning (VR-PRL) task was created to increase levels of motivation and for results to be compared with the 2D-PRL task from Chapter 2.

1.2: What is Virtual Reality

The definition of Virtual Reality (VR) in the academic literature is often misaligned with that of the public, with research VR experiences lagging behind consumer-grade experiences. This is not surprising, considering the relatively high technical abilities required to produce convincing VR environments. As a result, research often settles for Computer-Generated environments based on real-life scenarios[157,476]. These environments are commonly displayed on computer monitors (similar to video games) or Head Mounted Displays (HMD) with only rapidly outdated 360-degree video playback (e.g., Samsung Gear VR)[477]. A more consistent literature term is *immersive* VR which describes a) the use of a stereoscopic HMD b) 6 degrees of freedom (room scale) tracking and c) an experience that creates a sense of presence in the user (the feeling of 'being there'). Another distinction is that VR creates a completely digital environment, whereas Augmented Reality (AR) overlays digital information on top of the real-world and Mixed Reality (MR) involves the manipulation of both digital and real-world environments. These Extended Reality (XR) environments are commonly interacted with through motion controllers, but this is not necessary; especially considering recent advances in hand tracking and voice control. The affordability of quality XR experiences has seen a rapid increase in adoption over the last five years with monthly active VR users in PC gamers growing by 446%[ix].

[ix] Steam is an online video game store which surveys the hardware of its users every month: Steam Hardware & Software Survey (steampowered.com)

1.3: Why use Virtual Reality?

Immersive VR offers several clear advantages for psychological research. Firstly, VR methodologies increase levels of motivation, engagement, embodiment, and enjoyment across educational[478,479], health and safety[480], motor skill[481], and exercise scenarios[482]. These benefits scale with the immersiveness of the experience[482], are sustained over multiple sessions (i.e., not simply a novelty effect)[479], and are not detrimental to core learning experiences (i.e., through distraction)[479]. Secondly, VR environments have the potential to be more ecologically valid meaning behaviour can be assessed more naturally, in real-time, and new information can be more readily applied to real life[480]. This contrasts with 'traditional' 2D tasks which are often visually unstimulating, simplistic, and removed from how they relate to the real world[483]. Thirdly, behaviour can be assessed in safe role-play environments without fear of negative repercussions, such as VR exposure therapy for anxiety disorders. Finally, there are clear practical benefits of creating potentially costly real-world environments with relative ease in XR. Moreover, every aspect of the experience is highly controlled and sharing these exact conditions simply involves sharing a data file. However, despite these advantages, there is a limited amount of current research in psychology that adopts VR methodologies.

1.4: Virtual Reality and psychiatric disorders

The current clinical literature primarily focuses on VR-based exposure therapies including acrophobia, arachnophobia, social anxiety[484], and hoarding disorder[477]. These VR therapies have presented similar effectiveness to typical interventions[484], but with the additional benefit of cost-effective scalability. Within the psychosis-spectrum, VR has been used to assess cognition and behaviour[485,486] and as an intervention medium. For example, one relevant study reported that metacognitive reflection in a false memory paradigm can be facilitated by viewing recordings of VR experiences which subsequently reduce symptoms[157]. In this scenario, delusional behaviour can be tackled more objectively by having a shared and recorded experience as a reference point for discussion. Moreover, initial VR-based interventions have been effective at reducing reality distortion and negative symptoms[487] and both improving cognitive[487–489] and social skills, all while being tolerable and safe[490]. These VR-based interventions offer a drug-free alternative to typical interventions, relevant for patients with significant medication side effects or drug resistance.

1.5 Aims and Hypotheses

Immersive VR offers the ability to create highly controlled, motivating, and realistic cognitive tests that may mitigate the motivational confounds of typical non-immersive cognitive tasks. The first aim of this chapter was to create an immersive VR adaptation of the Probabilistic Reversal Learning task outlined in Chapter 2 (VR-PRL). The second aim was to assess whether the negative association between schizotypy and poorer task performance in Chapter 2 would *not* be replicated using the VR-PRL task. Additional measures of negative affect and metacognition were also taken to be consistent with Chapter 2 and Chapter 3. It was hypothesised that:

1) Levels of both pre-task and post-task motivation will be greater for the VR-PRL task relative to the 2D-PRL task outlined in Chapter 2.
2) Schizotypy will *not* predict performance on the VR-PRL task.

2.0 Methods

2.1 Participants

Of an initial 163 participants, seven were excluded (see ***2.7: Data preparation***). The final sample of 156 participants was primarily recruited through the university's internal recruitment system (71%), with the remainder recruited through word of mouth. In the current sample, 80.8% were biologically female, 98.1% identified as cis-gender, and ages ranged from 18 – 29 years old (M_{age} = 20.5, *SD* = 4.6). In terms of vocation and education, 74.4% were students, 18% were employed, and 25.6% had at least an undergraduate level qualification. Of the 106 participants that volunteered their current medication, 50% reported no medication, 5.6% reported antidepressant medication and none of the final sample reported mood stabilisers, anti-anxiety medication, or anti-psychotic medication[x]. The only exclusion criteria were a current diagnosis of schizophrenia or epilepsy (due to the VR headset). This study achieved a power of 0.99 for a medium effect size (multiple regression analysis with three predictors)[457].

2.2: Materials

This Chapter used the same scales as outlined in the previous Chapters to measure schizotypy (O-LIFE), negative affect (DASS-21), pre-task and post-task motivation (MIAMI), and Moderately Discrete Metacognition (BCIS and MCQ-30) and thus the details are omitted here (see page 78 and 131 for details). In addition to these measures, a series of questions were added requesting feedback on the VR experience to assess the validity of the design. This included asking participants how present and immersed they felt, how interactive and alive the environment was, and any feelings of motion sickness. All these items were rated on a scale of 1 ("Not at all") to 5 ("Completely"). Participants were also asked to compare the current VR study to 'standard' psychological tasks in terms of enjoyment, motivation, attention, and willingness to return (on a scale of 1 = "Completely prefer 'standard'", 5 = "No preference", and 10 = "Completely prefer VR"). This was done under the assumption many participants would be psychology students and have taken part in other studies involving cognitive tasks. By 'standard', the question referred to 'typical non-VR tasks'.

2.3 Apparatus and setup

The physical testing environment was partitioned into two areas: the first for completing the surveys and the second for the VR task without physical obstructions. The VR area was 11.47m^2 (3.7m x 3.1m) with the Oculus guardian boundary (a virtual safety boundary) positioned 20cms away from

[x] These results were unchanged with the exclusion of these participants.

the physical walls; leaving a total explorable area of 10.2m^2 (3.5m x 2.9m). A mat was placed in the centre of the VR area as a reference point for participants and a pulley system was used to suspend the HMD's power and display cables (see **Figure 29**). The VR HMD used was an Oculus Rift S (2019) with two Oculus Touch controllers. The Rift S has an LCD display pane with a resolution of 1280 x 1440 pixels per eye, a field of view of 115 degrees, and a refresh rate of 80Hz. The computer hardware included an Octa-core i7-11700 and an NVIDIA GTX 2060 which ensured sufficient frame rates were displayed to maintain display fluidity and reduce potential motion sickness. The virtual environment was developed using the Unity Game Engine (version 2019.4.17f1) and the Oculus integration package (Version 1.6.1).

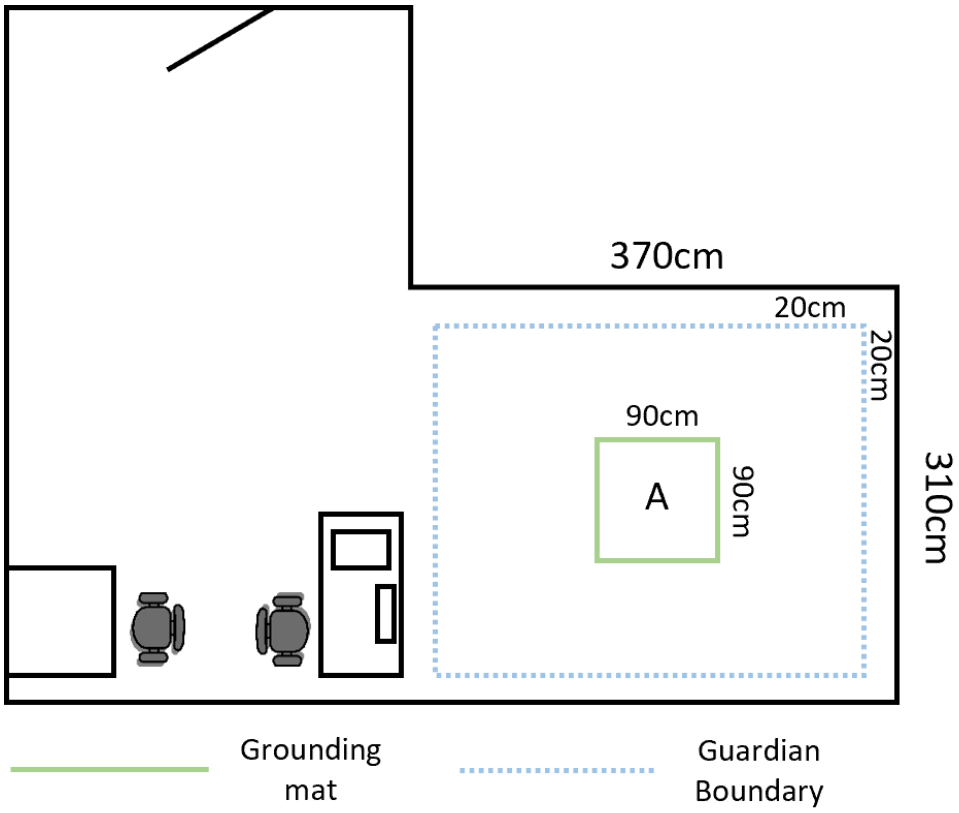


Figure 29. Diagram of the Virtual Reality setup. Participants completed the questionnaires at the bottom left desk and then moved to the VR area and stood at Point A, where there were no physical obstructions.

2.4 Virtual Reality Probabilistic Learning Task Design

The VR task design was created based on open-ended feedback from the Task Strategy Questionnaire (TSQ) of Chapter 2. Briefly, common feedback suggested it was not clear 1) how participants should respond on initial trials without prior knowledge of the cues 2) why participants could not abstain from responding and 3) why money was being bet. As a result, the task itself was changed to an "alien symbol decryption game" wherein participants must translate alien radio transmissions in a virtual arcade scenario (see the procedure for details). This design addresses these concerns as 1) the symbols were of alien origin which explained why participants had no prior knowledge 2) allowing the withholding of (likely incorrect) responses is not in an arcade business' interest of making money and 3) betting money is common in arcades.

The VR-PRL task itself was displayed on the screen of a virtual arcade machine (**Figure 34.** *D*) and was almost identical to Chapter 2, with the exceptions of the alien narrative, stimulus timings, and interaction through motion controllers. At each trial, a fixation cross was presented in the centre of the screen for 500ms, followed by an alien UFO image for 100ms, and then one of the two cues below the UFO. After a response of either 10p or £1 was given participants received feedback with a 1000ms delay. This delay allowed participants time to move their gaze to the display. Consistent with Chapter 2, contingencies were reinforced at an 80% probability, participants learned these contingencies through trial and error, a running total of participants' money was positioned on the screen, and the same pseudo-random trial order and sound effects were used. However, the task differed from Chapter 2 by including two blocks of 60 trials (one reversal), added sounds effects of a 'coin falling' and 'UFO' when participants inserted money and a cue appeared, the UFO visualisations (**Figure 30**), and more details instructions (see Procedure).

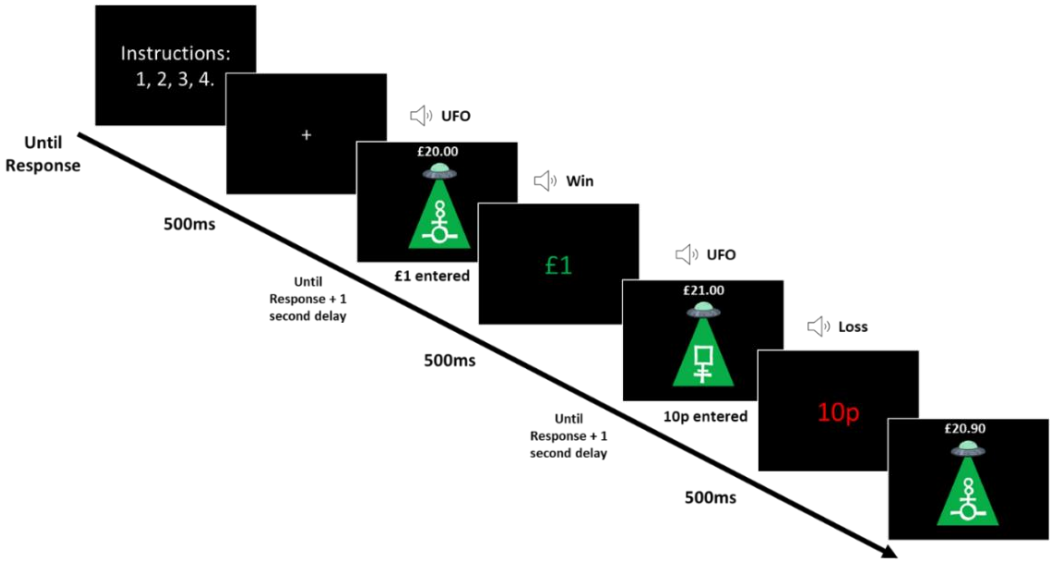


Figure 30. Trial structure of the Virtual Reality adaptation of a Probabilistic Reversal Learning task.

2.5 Virtual environment design

A virtual environment was created to resemble a typical urban town in the United Kingdom (**Figure 32**). Roads, architecture, brickwork, and virtual crossings also corresponded to standards in the UK. A central park and several large apartment complexes were added to increase the visual variety and suggest the presence of a larger town. The virtual testing environment was in one of these buildings which was modelled after a UK arcade (or "amusements"), often found at touristic coastal destinations. The environment included pool tables, arcade machines, a reception with an ice cream shop, and a bowling alley which are typical of these attractions. The VR-PRL task was displayed on an arcade machine distinguished by its green colour (**Figure 32**, D). Several other machines displayed videos of alternative games to increase immersion. The environment also included 3D spatial audio of urban sound effects in the street and arcade sounds effects in the arcade. The outside explorable area of the environment was 972m^2 and the interior arcade area was 361m^2. The environment was iteratively developed from earlier prototypes (**Appendix A**) and according to pilot participant feedback (**Appendix B**). A video of the virtual environment and the task can be found here and here.

Figure 31. Participant perspective of the exterior Virtual Reality environment. A) participant's start location, B) park entry, C) centre view of the park and the exit gate, and D) side view of arcade exterior.

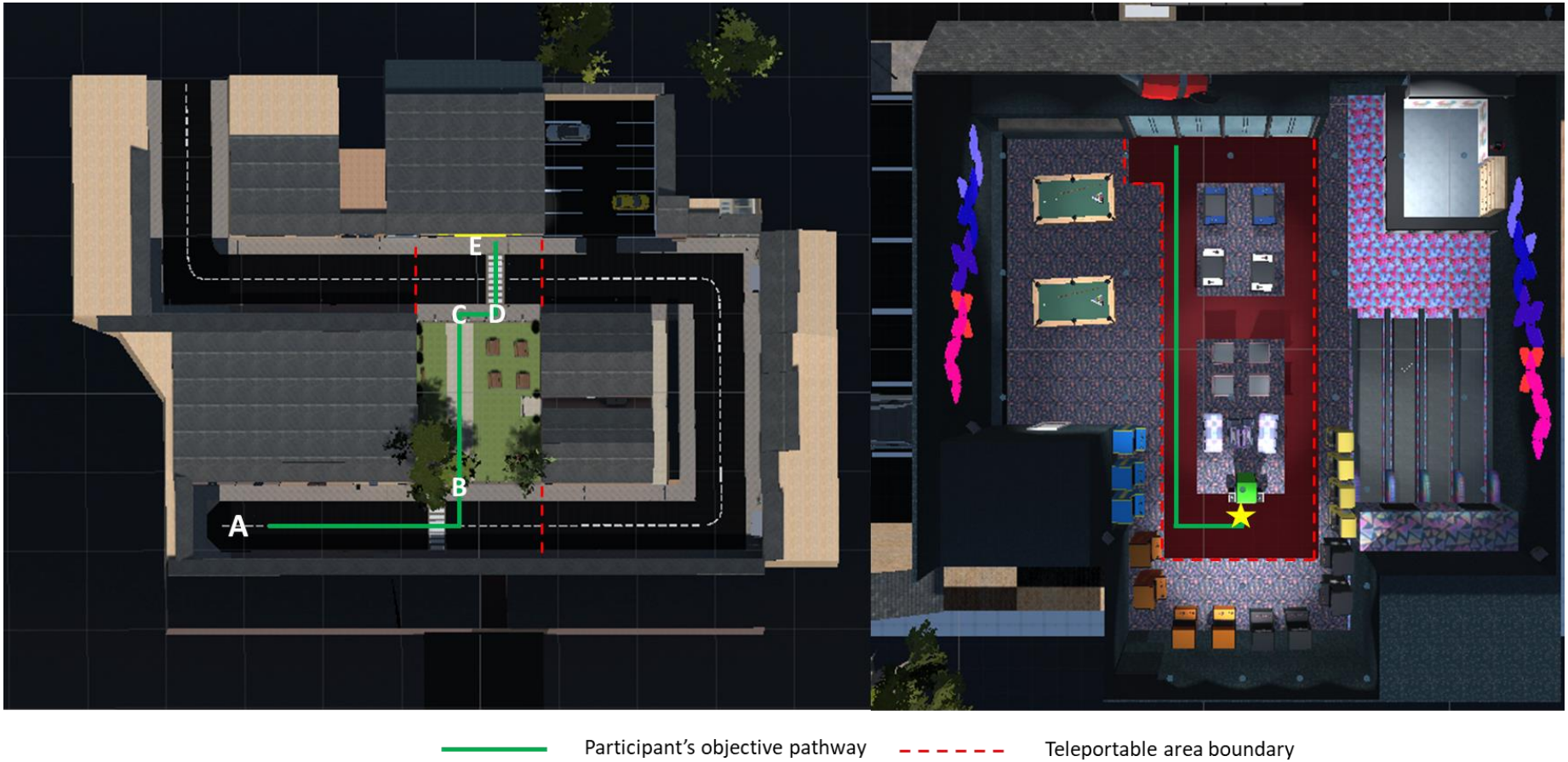


Figure 32. Aerial view of the VR testing environment. **Left**: A) participant start location, B) and C) interactable gates, D) interactable crossing, E) interactable arcade front doors. **Right**: arcade interior. The yellow star represents the end goal position of the participants.

2.6.0 Procedure

2.6.1 Questionnaires

Each session began with participants being seated at the primary computer and completing a Qualtrics survey. This included study information, informed consent, and collecting demographic information such as the voluntary disclosure of current medication. After completion, participants were randomised to complete either complete the questionnaires or the VR task first. If participants were randomised to questionnaires, these were completed at the participant's own pace and in a randomised order (see *Materials*) in the same manner as Chapter 3 and Chapter 4. After the questionnaires, the survey informed participants that the VR section would now begin and to inform the experimenter.

2.6.2 Virtual Reality setup

The VR portion of the study began with participants being asked to stand on the grounding mat (**Figure 29***, A*). It was explained to participants that this mat could be used as a reference point for their location within the room. Next, participants were told how to hold the controllers and the purpose of each button for this study (**Figure 33**). Specifically, the button under their index finger was used for teleportation, their middle finger for grabbing objects, and the joystick under their thumb was used to control the direction they faced. Participants were told they could also walk and turn in real life and these movements would be tracked in the VR environment. Participants were then asked to demonstrate these mechanics several times before beginning the task to aid comprehension.

Figure 33. The Oculus Rift S was used as the HMD with two motion controllers to track hand movement. Copyright belongs with Oculus.

2.6.3 Task instructions

Participants put on the HMD and watched three instructional videos to slowly introduce them to the VR environment, procedure and task instructions (see link). Firstly, the guardian boundary system was shown to participants with a guided demonstration. Participants then practised this themselves by walking up to the edge of the guardian boundary and feeling the real-world walls. Next, it was explained how participants should explore the environment using the controls to teleport and grab objects. Participants were told that their first objective was to reach a green arcade machine at a nearby amusement (**Figure 32***, Right*) and that they should complete several milestones on their journey. Participants began their journey on a nearby street (**Figure 32***.* A) and were required to navigate through a park, over an interactive road crossing, and then to enter the arcade. Participants moved by both walking in real life and the teleportation mechanic. The grabbing mechanic was used to open the park gates and arcade entrance doors. These milestones were implemented to increase the sense of interactivity and exploration and to let participants practice the controls.

Finally, the task instructions were explained in detail. This began with showing participants a scoreboard of previous attempts and stacks of 10p and £1 coins used to place bets. Coins were picked up with the grabbing mechanic that participants had just used to open the gates and doors and then dropped into the machine. Next, the video played practice rounds of the main task and a voiceover gave additional insights. The voiceover outlined the alien narrative along and task instructions which were also later shown on the arcade itself (see **Appendix C**). Briefly, these instructions explained that participants: a) would need to initially guess and learn from feedback, b) could find their winnings in the coin return tray below the display and that these winning could be re-entered as new bets, c) must bet every round even if they believe they will lose, d) could press the grey “refill” button if they ran out of coins, and f) must learn the contingencies and use this information to bet more optimally in the following rounds. After these videos, participants removed the headset and then completed the pre-task motivation assessment at the computer. Participants’ questions then were answered before returning to the virtual environment to start the VR task.

2.6.4 Virtual Reality task

The VR task started with participants navigating through the environment to the arcade entrance (approx. 90 seconds). The arcade machine presented a scoreboard of previous high scores taken from Chapter 2, which aimed to increase motivation and reiterate the primary aim of gaining money. Participants pressed the virtual “Go” button on the arcade machine to start the task and progressed through the displayed instructions (**Appendix C**). A 10p or £1 coin tray was positioned at either side of the arcade machine for participants to use (**Figure 34***,* D). Although participants started the task with an initial £20 displayed on the running total, only £11 was spawned in the coin trays so that an

equal number of coins could be presented to reduce response biases. Each trial began with the image of a UFO and an 'alien' sound effect being played. Next, one of the two cues was presented. Participants bet either 10p or £1 by grabbing the virtual coins with their virtual hands and releasing the coin into the coin insert slot on the arcade machine. This coin would then disappear and after 1000ms feedback would be given dependent on the accuracy of the bet (e.g., +£1, -10p). At the end of each trial participant's running total was updated and any winnings (new coins) were spawned at a coin return tray below the machine. To ensure participants always had access to coins these were replenished on 75% of trials. This also meant that coins were slowly depleted - encouraging participants to use the coins in the return tray to increase immersion. After the final trial, the machine displayed "GAME FINISHED", participants removed the headset, and then returned to the computer. The VR task took around 20 minutes to complete.

2.6.3 Post-task questionnaire

After the VR task, participants immediately completed the post-task motivation assessment. They were then presented with the VR evaluation questionnaire which asked them to rate their experience in terms of presence, immersion, enjoyment, interactivity, the environment being alive, and general feedback (see *Materials*). If participants had not completed any other psychological tasks, they were requested to skip the section comparing the current VR experience to other research. Finally, participants were fully debriefed and either received course credit or monetary compensation for their time. The entire study took approximately 60 minutes.

Figure 34. Interior of the amusements from different perspectives (A – C). Participants completed the task on a green arcade machine at the back of the environment (D).

2.7: Data preparation

From an initial sample of 163 participants, five were excluded for failing awareness checks in the MCQ-30 or O-LIFE (not selecting "Prefer not to say" when requested), one was removed due to accidentally exiting the VR task, and one further participant did not complete the VR task (N = 156). One participant's data for the pre-task motivation assessment was removed as this was unintentionally completed before instructions were given. Aside from these missing data, all other missing data including "Prefer not to say" responses (between 0.06% and 0.5% of psychometric data) were imputed using the missForest R package[389]

2.8: Analysis strategy

Validity checks were conducted including psychometric internal consistency, normative comparisons, and reasonable distribution of performance scores (i.e., ceiling effects, floor effects, and lack of variability). The primary outcome was the percentage of optimal responses (i.e., betting £1 on the rewarding cue on *every* trial). To address whether schizotypy was associated with task performance, each of the schizotypy scales was added simultaneously into increasingly complex Linear-Mixed Effects (LME) models (*Hypothesis 2*) consistent with Chapter 2. As it was expected that task deficits would only be reduced but not completely mitigated in the current study, the potentially mediatory roles of negative affect, metacognition, and motivation were also considered. Finally, post-task evaluations and feedback were explored to assess the validity of the VR environmental design. Where possible both frequentist and Bayesian analyses were conducted to differentiate between data insensitivity and a true null effect[391]. Analyses were conducted in R studio[393], Jamovi[394] and JASP[395] using several statistical[396–400] and data visualisation packages[401,402].

3.0 Results

3.1: Descriptive statistics

Descriptive summaries of psychometric data can be found in **Table 15**. Non-parametric normative comparisons of the O-LIFE[293] and DASS-21[403] revealed that none of the three schizotypy scales significantly differed from the normative data (all $p > .068$, $r_{rb} < 0.11$), but the DASS-21 total score was significantly higher in the current study at a medium effect size ($p < .001$, $r_{rb} = 0.61$). The current data were also compared against those of Chapter 2. In terms of demographics, the current sample had a higher proportion of males ($\chi 2 = 21.1$, $p < .001$) and was significantly older ($t(225.7) = -3.223$, $p = .002$, $d = 0.371$). As a result, age and sex were added as control variables in the upcoming analyses. In terms of personality traits, the current sample did not differ in levels of positive schizotypy ($p = .057$), negative schizotypy ($p = .166$), disorganised schizotypy ($p = .695$), nor negative affect ($p = .582$) relative to Chapter 2. Finally, performance at the end of Block 2 was consistent between the current study and the 2D-PRL of Chapter 2 ($t(282)$, $p = .481$, $BF_{10} = 0.17$, $d = .08$).

3.2: Reliability and validity

The O-LIFE, DASS-21, and all MCQ-30 scales except for the Need to Control Thought ($\omega_T = 0.769$) presented good to excellent internal consistency ($\omega_T > .80$), post-task motivation was acceptable ($\omega_T > .7$), both Self-Reflectivity and pre-task motivation were questionable ($\omega_T > .60$), and Self-Certainty was poor ($\omega_T = .57$). A 2 (contingency) x 6 (bins of 10 trials) repeated-measures ANOVA confirmed that learning had occurred in the initial learning block and performance decreased post-reversal (**Supplementary Analyses**).

3.3 Psychometric relationships

A Bayesian Spearman correlation matrix was created between all psychometric variables to identify potential mediators of performance (**Table 20**). Briefly, schizotypy again correlated strongly with negative affect, Self-Reflectivity (but not Self-Certainty), and while positive and disorganised schizotypy correlated with the MCQ-30 broadly only negative schizotypy correlated only with Uncontrollability and Need to Control thoughts. Schizotypy was unrelated to motivation across all analyses.

Table 19. Descriptive statistics of psychometric measures and normative comparisons

	Current								Normative							Comparison		
Scale	**Range**	**M**	**SD**	**Med**	**MAD**	**IQR**	**ω_T**	**α**	**N**	**Range**	**M**	**SD**	**Median**	**IQR**	**α**	***ES* [Low, High]**	***p***	**Normality**
Unex	0-27	10.904	6.408	11	7.413	10	0.885	0.880	402	-	10.159	6.304	9	10	0.89	0.08[-0.26, 0.10]	=.718	0.004
Intan	0-19	5.782	4.506	4.5	3.707	7	0.818	0.815	402	-	5.444	4.000	4.5	6.5	0.82	0.11[-0.08, 0.29]	=.259	< .001
Cogdis	0-24	13.853	6.136	14	8.154	11	0.893	0.892	402	-	12.391	5.690	13	-	0.87	0.01[-0.17, 0.19]	=.068	< .001
DASS Total	0-55	17.16	12.309	14	10.378	15	0.931	0.929	1794	0-61[B]	9.42	9.66	7	-	0.93	0.61[0.49, 0.72][M]	<.001	< .001
Depression	0-21	5.378	5.06	3	2.965	6	0.899	0.898	1794	0-21[B]	2.83	3.87	1	-	0.88	0.75[0.77, 0.89][L]	<.001	< .001
Anxiety	0-19	5.077	4.602	4	4.448	6.25	0.859	0.898	1794	0-20[B]	1.88	2.95	1	-	0.82	0.84[0.77, 0.87][L]	<.001	< .001
Stress	0-19	6.705	4.372	6	4.448	5.5	0.817	0.808	1794	0-21[B]	4.73	4.20	4	-	0.90	0.34[0.17, 0.49][S]	<.001	< .001
BCIS_R	15-32	22.885	3.791	23	4.448	5	0.655	0.633	-	-	-	-	-	-	-	-	-	0.022
BCIS_C	6-20	12.571	2.731	13	2.965	3	0.565	0.558	-	-	-	-	-	-	-	-	-	0.086
MCQ_CC	6-24	11.532	3.967	11	4.448	5.25	0.819	0.817	-	-	-	-	-	-	-	-	-	< .001
MCQ_PB	6-24	10.436	4.48	9	2.965	5	0.897	0.896	-	-	-	-	-	-	-	-	-	< .001
MCQ_CSC	7-24	16.808	4.757	17	5.93	8	0.900	0.895	-	-	-	-	-	-	-	-	-	< .001
MCQ_UD	6-24	13.006	5.23	12	5.93	8	0.903	0.900	-	-	-	-	-	-	-	-	-	< .001
MCQ_NC	6-24	10.583	3.532	10	2.965	4	0.769	0.754	-	-	-	-	-	-	-	-	-	< .001
MCQ_Total	36-108	62.365	13.367	60.5	13.343	18	0.868	0.876	-	-	-	-	-	-	-	-	-	0.005
MIAMI Pre	9-20	17.323	2.117	18	1.483	3	0.622	0.603	-	-	-	-	-	-	-	-	-	< .001
MIAMI Post	10-20	17.542	2.291	18	2.965	4	0.782	0.775	-	-	-	-	-	-	-	-	-	< .001
Understand	1-4	2.896	0.909	3	1.483	2	-	-	-	-	-	-	-	-	-	-	-	< .001

Note: IQR – interquartile range, MAD = median absolute deviation, medians are interpolated medians (nearest integer is the true median), **ω** = McDonald's Omega total. Normality tests are Shapiro-Wilk tests and group comparisons are one-sample Wilcoxon Sign Rank tests for OLIFE and DASS. *ES = effect size which is rank-biserial correlations. Interpretation for effect sizes are in superscript*. [A] = Values from Mason & Claridge, 1995., [B] Henry & Crawford, 2005.

Table 20. Bayesian Spearman correlation matrix of psychometric data to identify potential performance mediators

	Schizotypy			Neg Affect	Metacognition							Motivation	
Variable	*Pos*	*Neg*	*Dis*	*Total*	*BCIS_R*	*BCIS_C*	*MCQ PB*	*MCQ UD*	*MCQ CC*	*MCQ CSC*	*MCQ NCT*	*Pre*	*Post*
Pos Schizotypy	-												
Neg Schizotypy	0.15^{N}	-											
Dis Schizotypy	0.57^{D}	0.44^{D}	-										
Negative Affect	0.37^{D}	0.38^{D}	0.68^{D}	-									
Self-Reflectivity	0.39^{D}	0.20^{*}	0.50^{D}	0.36^{D}	-								
Self-Certainty	0.13^{I}	0.08^{I}	0.10^{I}	0.10^{I}	0.07^{N}	-							
Pos Beliefs about Worry	0.20^{*}	0.15^{I}	0.28^{***}	0.23^{**}	0.26^{***}	0.16^{I}	-						
Uncontrollability	0.40^{D}	0.23^{**}	0.64^{D}	0.55^{D}	0.44^{D}	0.10^{I}	0.22^{**}	-					
Cognitive Confidence	0.25^{***}	0.12^{I}	0.32^{D}	0.22^{**}	0.33^{D}	-0.08^{I}	-0.02^{N}	0.27^{***}	-				
Cog Self-Cons	0.31^{D}	0.03^{N}	0.30^{***}	0.21^{*}	0.19^{*}	0.13^{I}	0.14^{I}	0.32^{***}	0.04^{N}	-			
Need to Control	0.22^{*}	0.21^{*}	0.27^{***}	0.27^{***}	0.13^{I}	0.22^{*}	0.22^{**}	0.21^{*}	0.16^{I}	0.16^{I}	-		
Pre-task MIAMI	0.04^{N}	0.00^{N}	-0.06^{N}	-0.12^{I}	0.01^{N}	-0.07^{N}	0.05^{N}	-0.01^{N}	-0.05^{N}	0.01^{N}	-0.06^{N}	-	
Post-task MIAMI	-0.02^{N}	0.03^{N}	-0.12^{I}	-0.17^{I}	-0.06^{I}	0.01^{N}	-0.03^{N}	0.04^{N}	-0.12^{I}	0.07^{N}	-0.04^{N}	0.36^{D}	-

Note: ^{N} = supports the Null, ^{I} = insensitive, * = $BF_{10} > 3$, ** = $BF_{10} > 10$, *** = $BF_{10} > 30$, ^{D} = >100 for 'decisive'.

3.4. Virtual Reality Probabilistic Reversal Learning task performance

3.4.1 Baseline model

An LME model predicting task performance from the fixed-effects of Block and contingency was a significantly better fit to the data than the null model ($\chi2(2) = 116.9$, $p < .001$, AIC: 5316, $R^2 = 48.9\%$); returning a significant effect of both Block ($p < .001$) and Contingency ($p < .001$). However, a further model adding a Block x Contingency interaction effect was not a significantly better fit to the data ($\chi2(1) = 0.027$, $p = .869$, AIC: 5346), suggesting changes in performance from Block 1 to Block 2 was consistent across contingencies (**Supplementary Analysis B**). However, to be consistent with Chapter 2, this Block x Contingency model was used as the baseline model for comparison in all upcoming analyses.

3.4.2 Schizotypy

Next, the three schizotypy scales were added as additional fixed-effects. However, neither this model ($\chi2(3) = 0.867$, $p = .834$, AIC: 5322.0, $R^2 = 49.4\%$) nor models specifying a schizotypy x block interaction ($\chi2(6) = 3.632$, $p = .726$, AIC: 5274, $R^2 = 49.8\%$) or a schizotypy x contingency interaction ($\chi2(6) = 2.128$, $p = .907$, AIC: 5226, $R^2 = 49.5\%$) were significantly better fits to the data. As the aim of this Chapter was a comparison to the 2D-PRL task of Chapter 2, the original best fitting model was fitted and assessed. As detailed in **Table 21**, the fixed-effects of positive ($p = .564$), negative ($p = .924$), and disorganised schizotypy ($p = .375$) as well as their interactions with Contingency were all non-significant ($p > .428$). Linear trend analysis confirmed positive schizotypy did not predict performance of the rewarding cue (B = -0.113[-0.546, 0.320], $p = .609$) nor punishing cue (B = -0.018[-0.451, 0.415], $p = .934$, **Figure 35**). The same pattern of null findings was found for disorganised schizotypy (*rewarding*: B = 0.067[-0.423, 0.557], $p = .787$; *punishing*: B = 0.198[0.688, 0.794], $p = .428$). Overall, these results firmly suggest that schizotypy was unrelated to performance in the VR-PRL task (*Hypothesis 2*). As a result, the potential mediatory roles of negative affect, metacognition, and motivation were not investigated.

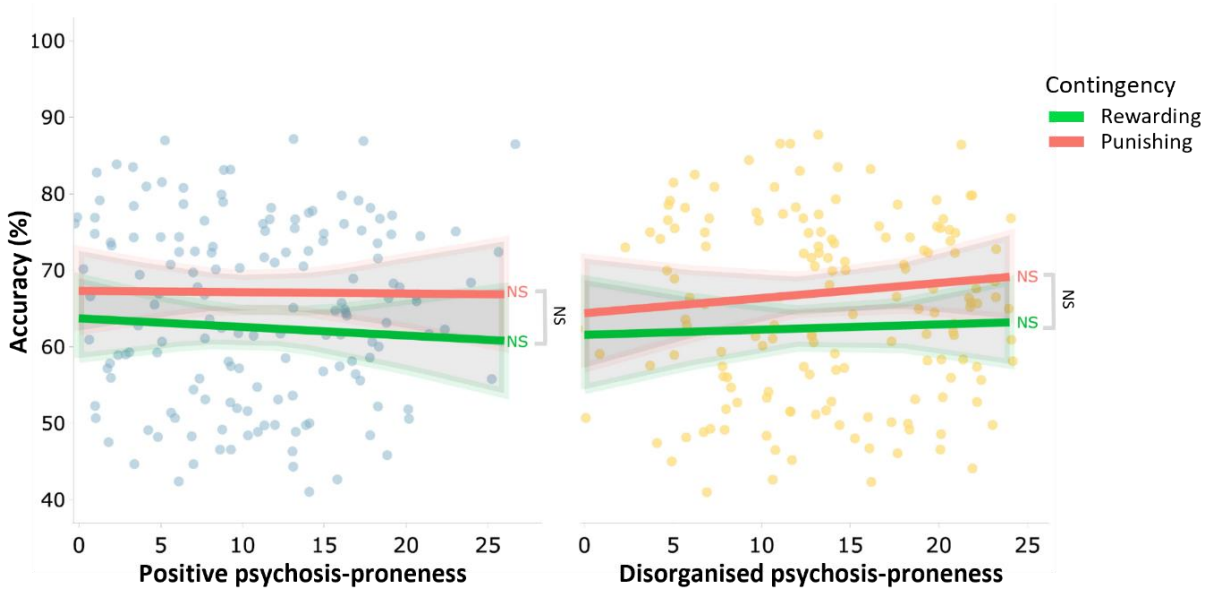


Figure 35. Results of Linear-Mixed Effects (LME) model predicting performance on the VR-PRL task. Plots show the null interaction effects between positive schizotypy (left) and disorganised schizotypy (right) and contingency.

Table 21. Linear Mixed-Effects (LME) model predicting VR-PRL performance. Schizotypy was unrelated to task performance.

	Performance (Optimal %)					95% Conf Int	
predictor	**B**	**SE**	**t**	***p***	**β**	**LC**	**HC**
Intercept	72.5	2.916	24.9				
Contingency^{+}	-1.790	3.097	-10.9	< .001	-0.047	-0.207	-0.113
Block^{2}	-13.3	3.097	-0.578	< .001	-0.351	-0.415	-0.288
Pos Scz	-0.018	0.220	-0.083	= .564	-0.006	-0.151	0.139
Neg Scz	-0.213	0.240	-0.889	= .924	-0.050	-0.162	0.061
Dis Scz	0.198	0.249	0.794	= .375	0.064	-0.093	0.221
Contingency^{+} * Pos Scz	-0.096	0.238	-0.397	= .428	-0.035	-0.209	0.139
Contingency^{+} * Dis Scz	-0.130	0.249	-0.524	= .600	-0.056	-0.266	0.153

Note: Block^{2} = the effect of Block 2 relative to Block 1, Contingency^{+} = the effect of the positive contingency relative to the negative contingency. $R^2_{\text{fixed-effects}}$ = 35.6%, $R^2_{\text{ranomd-effects}}$ = 14.0%.

3.5.0 Virtual Reality task feedback

3.5.1 Validation and evaluation

Levels of motivation of the current VR-PRL task were significantly higher than the 2D-PRL task of Chapter 2 for both pre-task ($p < .001$, $d = 1.300[1.015, 1.581]$, $BF_{10} > 999$) and post-task motivation ($p < .001$, $d = 0.506[0.263, 0.748]$, $BF_{10} = 563$) (*Hypothesis 1*). Descriptive statistics of the VR evaluation survey also suggested participants felt highly immersed ($M = 4.416$, $SD = 0.790$) and present ($M = 4.169$ $SD = 0.934$); supporting the fidelity of the environment (see **Table 23**). Participants also stated the environment was highly interactive ($M = 4.357$, $SD = 0.830$), that the environment felt alive ($M = 3.838$, $SD = 1.000$), and that they generally did not feel motion sick ($M = 1.617$, $SD = 0.923$). Finally, exploratory correlations suggested schizotypy was not associated with any VR evaluation metric (**Table 22**).

3.5.2 Participant preferences and task feedback

Further evaluation explored participant experiences of the VR task relative to typical 2D tasks (**Table 23**). The 110 participants who had taken part in other psychological research reported preferring the current VR environment in terms of enjoyment, motivation, attention, and willingness to return at large effect sizes and decisive levels of evidence (all $p < .001$, $r_{rb} > 0.944$, $BF_{10} > 999$). The percentage of participants preferring the current VR task over non-VR tasks (i.e., scoring > 5) on each dimension were as follows: enjoyment (93.6%), motivation (88.2%), attention (90.9%), and willingness to return (92.7%). Schizotypy was not correlated with these preferences ($r_s = - .16$, all $p > .10$). Finally, open-ended feedback was used to highlight areas for improvement of the current design (see **Appendix D**). Participants primarily commented on motivational aspects of the task, such as enjoying the experience and the clarity of instructions. Specific recommendations included expanding the real-world explorable area (guardian boundary), adding other humans, and using a higher definition HMD. Of the ten participants who reported cybersickness, five of these stated this was due to the instructional videos and not the main task. Overall, these findings support that the VR environment was of sufficient quality and the manipulation of increased motivation relative to the 2D study was successful.

Table 22. Bayesian correlations between personality variables and evaluation of the VR environment.

	Schizotypy			Affect	Metacognition			Motivation			
Variable	**Pos**	**Neg**	**Dis**	**DASS-21**	**SR**	**SC**	**MCQ-30**	**Pre**	**Post**	**Under**	**Sickness**
MIAMI Pre	0.059^{N}	-0.015^{N}	-0.022^{N}	-0.072^{N}	-0.003^{N}	-0.071^{N}	-0.012^{N}	—			
MIAMI Post	-0.022^{N}	-0.026^{N}	-0.128^{I}	-0.158^{I}	-0.078^{N}	-0.020^{N}	-0.035^{N}	0.331^{D}	—		
Understand	-0.155^{I}	0.121^{I}	0.007^{N}	0.077^{N}	-0.040^{N}	-0.167^{I}	-0.002^{N}	0.195^{I}	0.217^{M}	—	
Presence	-0.059^{N}	-0.010^{N}	-0.144^{I}	-0.188^{I}	-0.112^{I}	0.042^{N}	-0.090^{N}	0.165^{I}	0.304^{D}	0.006^{N}	-0.084
Immersion	-0.008^{N}	0.023^{N}	-0.030^{N}	-0.107^{I}	-0.073^{N}	0.024^{N}	-0.024^{N}	0.156^{I}	0.319^{D}	-0.040^{N}	-0.067^{I}
Interactivity	-0.029^{N}	-0.044^{N}	-0.029^{N}	-0.051^{N}	-0.104^{I}	-0.002^{N}	-0.083^{N}	0.114^{I}	0.355^{D}	-0.08^{N}	0.026^{N}
Alive	-0.062^{N}	-0.073^{N}	-0.099^{I}	-0.074^{N}	-0.063^{N}	0.012^{N}	-0.066^{N}	0.138^{I}	0.357^{D}	-0.091^{N}	-0.068^{N}
Sickness	-0.009^{N}	0.077^{N}	0.027^{N}	0.110^{I}	-0.046^{N}	-0.020^{N}	0.060^{N}	-0.148^{I}	-0.173^{I}	-0.071^{N}	—

Note: M- = Motivation, All correlations are Pearson's Rho with Bayes Factor, N = supports the Null, I = insensitive, M = >10 for 'moderate', D = >100 for 'decisive'.

Table 23. Descriptive statistics of comparisons between the current VR task and experience of standard 2D tasks

		Descriptive statistics						Comparison	
	item	**N**	**M**	**SD**	**Med**	**MAD**	**Range**	**Test**	**Effect size**
General	*Presence*	154	4.169	0.934	4.0	1.000	*1 - 5*	-	
	Immersion	154	4.416	0.790	5.0	0.000	*2 - 5*	-	
	Interactive	154	4.357	0.830	5.0	0.000	*1 – 5*	-	
	Alive	154	3.838	1.000	4.0	1.000	*1 – 5*	-	
	Sickness	154	1.617	0.923	1.0	0.000	*1 - 5*	-	
VR vs. 2D	*Enjoyment*	108	8.809	1.975	10.0	0.000	*1 -10*	$p < .001$	$r_{rb} = .953$
	Motivation	108	8.300	2.075	9.0	1.000	*1 - 10*	$p < .001$	$r_{rb} = .944$
	Attention	108	8.691	1.948	10.0	0.000	*1 - 10*	$p < .001$	$r_{rb} = .966$
	Comeback	108	8.909	1.966	10.0	0.000	*1 - 10*	$p < .001$	$r_{rb} = .975$

Note: VR vs 2D scale as follows: 1 = completely prefer standard, 5 = no preference, 10 = completely prefer VR. Tests are One-samples Wilcoxon Signed-rank test comparing the median value against 5 (no preference).

4.0: Discussion

The current study assessed whether amotivation may partially explain cognitive deficits in psychometric schizotypy. To do so, an immersive Virtual Reality implementation of a Probabilistic Reversal Learning task (VR-PRL) was created. The results supported that the VR-PRL task was more motivating than the 2D-PRL task of Chapter 2 (*Hypothesis 1*) and that the task deficits reported in Chapter 2 were not replicated (Hypothesis 2); suggesting that task adaptations completely mitigated these deficits. Implications for future cognitive assessments in both non-clinical and clinical samples are discussed.

4.1: Schizotypy and VR-PRL performance

Previous clinical research in schizophrenia[258] and other disorders[259] has reported that motivation partially mediates cognitive deficits. As a result, it was expected deficits in the current study would be reduced, but not completely mitigated. The full mediation of the current study is particularly surprising, considering previous studies using VR in the psychosis spectrum have reported cognitive task deficit - suggesting motivation is not the sole cause. Moreover, motivation was not significantly associated with schizotypy in the current sample, which further conflicts with past clinical research. While this may suggest a clear disconnect to clinical samples, as deficits in schizotypy are more closely tied to motivation, the MIAMI did not prove to be a reliable tool in the current study.

4.2: Explanations for task deficits not being replicated

While the core task was identical between the 2D-PRL and VR-PRL (further supported by no differences in overall performance), the contexts surrounding the tasks were markedly different. Considering this, another potential explanation is that the increased task instructions, clarity, and alien narrative of the VR-PRL may have increased task comprehension. This is supported by comprehension difficulties partially explaining probabilistic reasoning deficits in schizotypy[432]. As task comprehension was not assessed in the current study, future studies could investigate this possibility. Specifically, comprehensive psychometric measures could be applied or a 2D-PRL task could be created using the alien narrative. Another possibility is that the VR task may have been perceived more as a game, which may reduce levels of task-related anxiety. Indeed, both Chapter 2 and Chapter 3 reported a negative association between positive schizotypy and learning of punishment. Perhaps when participants are in a real-world testing scenario (Chapter 2 and Chapter 3) they become more concerned with their performance and the punishing trials in particular lead to

distraction. However, when they are immersed in a virtual world this effect may be removed through the gamification of the experience (e.g., being immersed may mean anxiety caused by a nearby experimenter watching is not an issue). If this were true, these findings should also be replicated in other cognitive domains (e.g., working memory, emotion recognition, etc.). If replicated, this may imply deficits in clinical patients may partially be due to task-related anxiety, which is consistent with the suggestions of the previous Chapters. As personality traits did not differ between samples and demographic variables were statistically controlled for, sample variations between Chapters is an unlikely explanation for these findings.

4.3: Explanations for task benefits not being replicated

This explanation may also explain the lack of improved performance concerning disorganised schizotypy found in Chapter 2. Specifically, disorganised schizotypy may increase attention to 'threat-related' cues which may increase learning. Indeed, anxiety has been found to increase attention to low-arousal threats[406] which the punishing cue of the PRL task may fall under. While negative affect did not mediate performance here, the DASS-21 is a trait rather than a state assessment of negative affect; meaning this relationship is unlikely to be captured. This could be validated through both eye-tracking provided by more recent VR HMDs (e.g., longer dwell time for punishing cues) and state anxiety measurements such as the STICSA. Future research should also consider that task adaptations may not only remove the detrimental effects of positive schizotypy, but also the beneficial effects of disorganised schizotypy.

4.4: Implications for Virtual Reality in cognitive research

While it is unclear exactly what manipulation may have produced the current results, this study highlights the utility of VR in cognitive research. A novel contribution is the lack of association between schizotypy and the appraisals of the VR experience (**Table 22**). Critically, while some psychiatric disorders are related to elevated cybersickness[491], this was not the case for sub-clinical schizotypy traits. Currently, no such assessment has been conducted in clinical patients. As a result, these findings present the first indirect evidence that VR experiences may be tolerable to clinical psychosis patients to the same extent as controls (although medication side effects may pose an additional challenge in patient). Similarly, the lack of association to VR appraisals (e.g., immersion) may mean similar VR design principles are applicable across the psychosis-spectrum. The results here have also not only emphasised VR as a method to reduce task-related anxiety, but also its clear

preference over standard psychological assessments. A critical finding was that participants were much more willing to return for future tasks. Currently, around 30% of clinical patients do not adhere to medication[492], therapeutic drop-out rates vary drastically (e.g., Cognitive Remediation, 16.6%[84]; online education, 48%[493]), and half of patients disengage with Cognitive Behavioural Therapy[494]. Virtual Reality may be a suitable method that novel therapeutic interventions could use to increase adherence and ultimately improve treatment gains (e.g., VR social skills training). This suggestion is further supported by the lack of association between schizotypy and willingness to return, which may suggest that the positive overall evaluations of VR may extend to clinical patients. While resource intensity is the biggest limitation of VR research in psychology, this may be offset in clinical interventions through adherence and in psychological research through more efficient participant recruitment. However, it should be noted the current sample was comprised primarily of younger adults, which may not have captured issues relevant to older adults. Indeed, VR requires a non-trivial amount of effort to correctly position the headset, understand interactions, and become accustomed to the setup – all of which younger adults are likely to have greater foundational knowledge of. This is especially important considering psychosis patients are often older than the current sample. Furthermore, the current VR study also required participants to have intact mobility, which would not be suitable for other samples.

4.5: Strengths and limitations

The primary strength of this study is the relatively high ecological validity provided by the immersive Virtual Reality task. Other strengths include the pseudo-matched design between this Chapter and Chapter 2 on personality traits, the high internal consistencies of most scales, and the modest statistical power. The primary limitation of this study is the mixed design and thus a replication should be conducted using a within-participants design. The PRL task is not appropriate for this design (i.e., participants *learn* sets of rules), but a working memory task may be appropriate. Secondly, the current study may have manipulated too many variables at once (e.g., realism, motivation, and comprehension) meaning it is difficult to pinpoint the reasons behind the current findings. Future research should address these separately for comparison (e.g., the alien narrative in a 2D scenario).

5.0: Conclusions

The current study created an immersive, more ecologically valid, and highly motivating VR version of a neurocognitive task. It was found that the current task adaptations mitigated the cognitive deficits associated with positive schizotypy, although which specific aspect of the environment may have produced this change is unclear. It was suggested the VR task may have reduced task-related anxiety through gamification and high immersion which may have removed attentional changes to punishing stimuli. If this is supported in future replications, this may suggest that while schizotypal individuals may present cognitive deficits in lab-based assessments (i.e., 2D tasks), this may not extend to more realistic scenarios (i.e., VR tasks). Replications in clinical patients may also suggest cognitive deficits have been overestimated and therapeutic interventions should perhaps target defeatist self-beliefs.

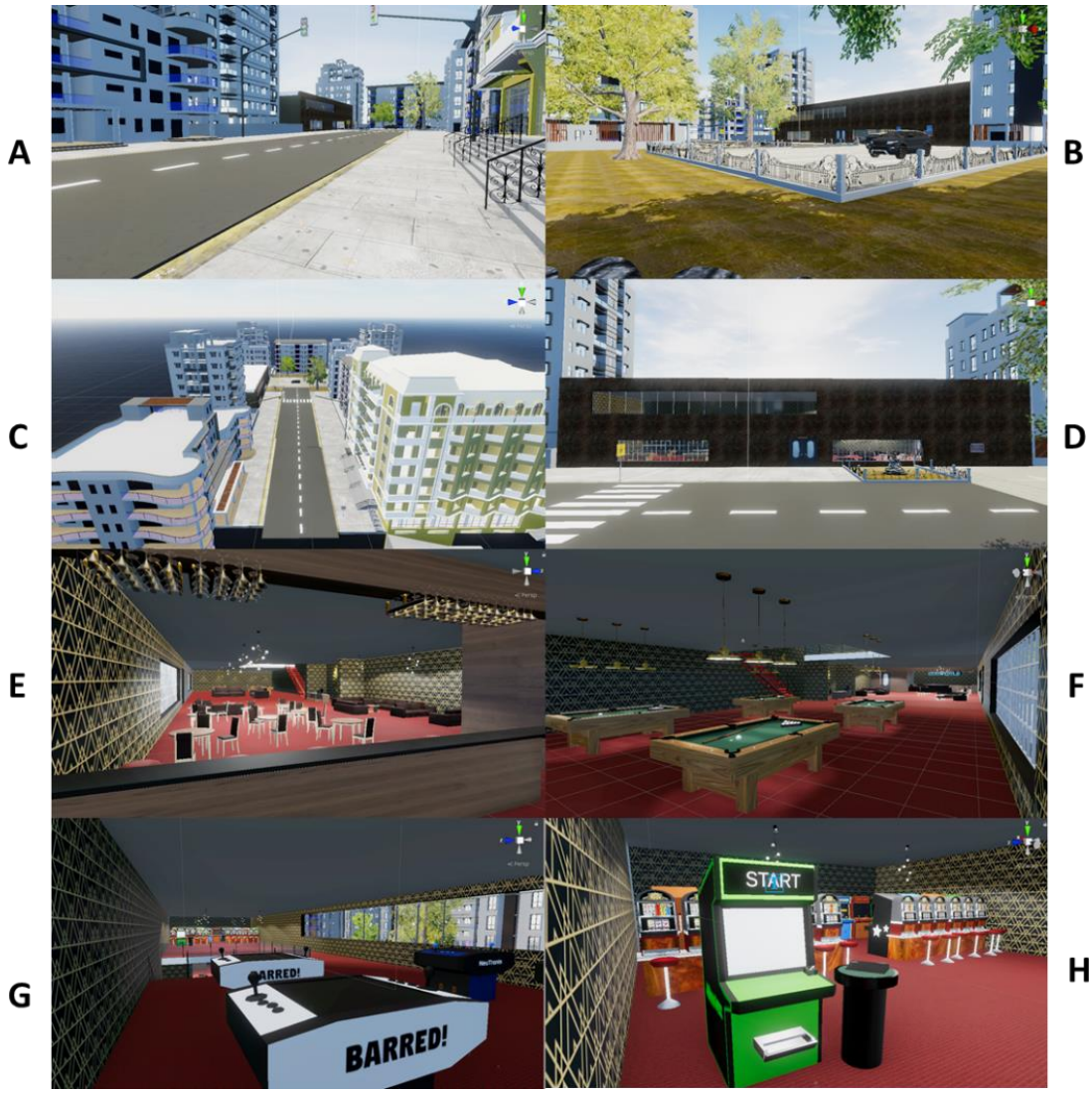
5.1: Acknowledgements

I would like to thank Sabina Beganovic and Madeline Humber for their help in collecting the data for this experiment.

Appendices

Appendix A

Previous virtual environments and design process.



Appendix B

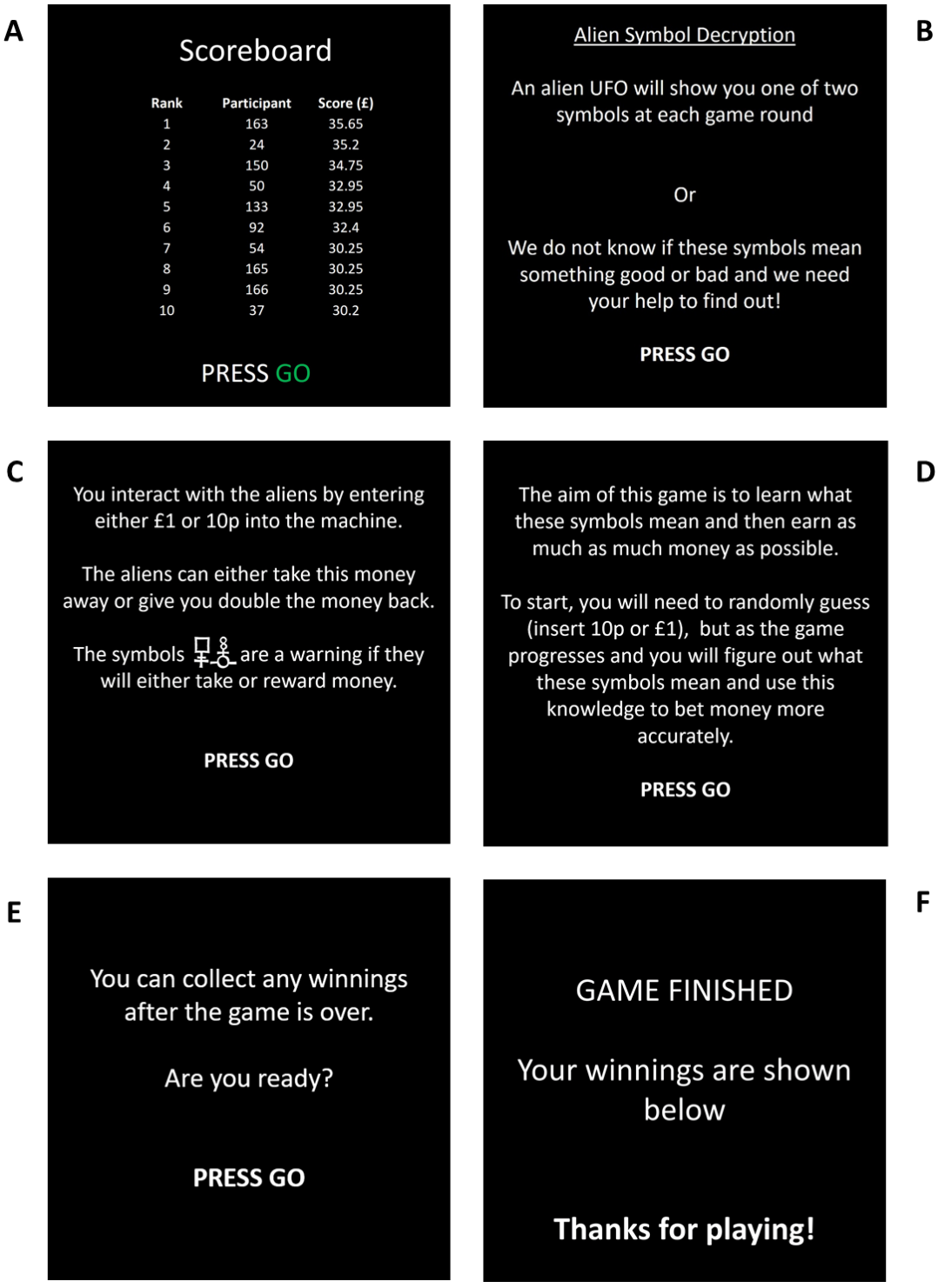
Task adaptations made from piloting feedback

Due to the novel nature of the task a pilot sample of 10 participants was taken to provide feedback. This included comments on the overall task, areas of the VR introductions, environment, or PRL task that were not clear, and to assess levels of motion sickness. Data from these participants were not used in the analysis as the task dynamics changed considerably. These changes included:

1. Participants often did not follow the instructions of how to navigate to the arcade (specifically, to open the gates, use the crossing, and open the arcade doors). This was likely a mixture of a large amount of instructional information and distraction from the novelty of the experience. The following was implemented as a result: a) all verbal instructions were given before entering the virtual environment b) reduced the teleportable distance to 10 meters so participants were required to take their time, c) imposing navigational restrictions until objectives were completed d) only allowing participants to teleport to a certain portion of the arcade.
2. Participants occasionally had difficulty positioning themselves in front of the green task machine due to not understanding the teleportation controls or that they could walk in the real world. Two keyboard controls were implemented whereby the experimenter could manually position the participants and then disable the teleportation ability.
3. Participants the coin insert was placed on the right-hand side of the machine along with the £1 coin tray. However, participants tended to favour the £1 coin tray, likely as this was in sight more often or being unaware of the 10p coin tray. As a result, the coin insert was move to the centre of the screen and the lateralisation of the coin trays were randomised.
4. Due to the limited field of view for the Rift S participants were originally not able to see the money display, main task display, and coin trays at once. This led to participants being unaware of their current total. Consequently, the money display was re-positioned from the top of the machine to now be on the arcade screen, so it was always visible.
5. Originally, the instructions did not differ from those of Chapter 2. However, from the open-ended feedback of Chapter 2 and comments from the current pilot sample suggesting the task was unclear changed were made. This led to the narrative of alien symbol decryption.
6. All videos were subtitled and re-recorded to aid understanding after participants stated there was a significant amount of information to understand.

Appendix C

Task instructions presented on the virtual arcade machine. A) Virtual scoreboard with real participant data from Chapter 2. B) – E) task instructions presented to participants. F) End screen shown to participants before they removed the headset.



Appendix D

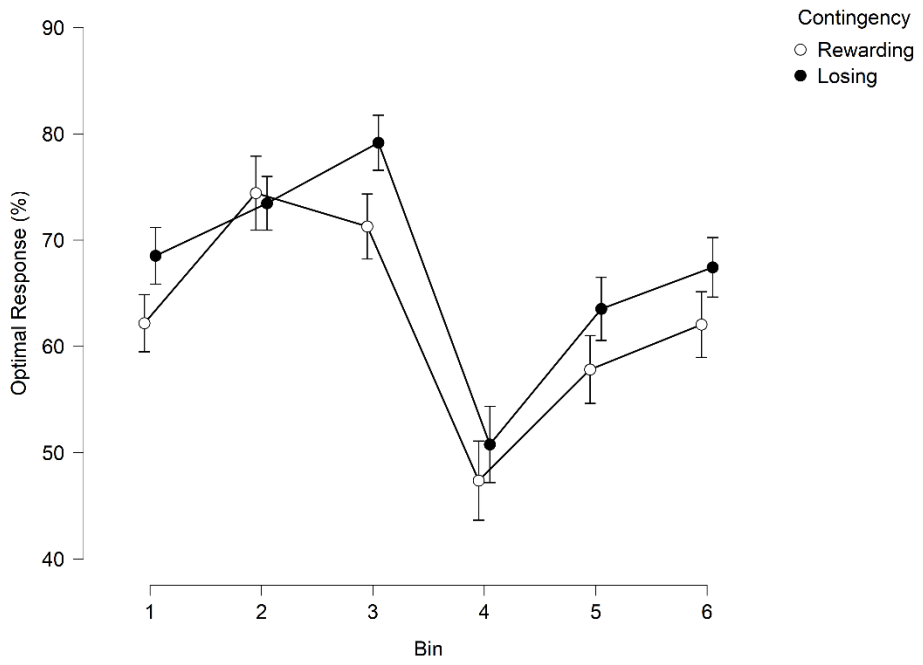
Summary of open-ended feedback of the VR paradigm

Category	**Comments**									
Motivation	Total	Enjoyed	Realistic	Interactive	More inter-activity	Immersive	Not Enjoyable	Too long	Progress bar	Engaging
	44	*25*	*8*	*3*	*2*	*2*	*1*	*1*	*1*	*1*
Instructions	Total	Instructions clear	Unsure of task	While to under-stand	Instructions unclear	Instruction too fast	Practice good	Clear Procedure	Practice controls good	
	30	*13*	*8*	*2*	*2*	*2*	*1*	*1*	*1*	
Environment/ Mechanics	Total	Concerned walking into walls	Need other humans	Glitches	Add body collisions	Larger room	Dislikes hand physics	Add social sounds	More exploration	Poor graphics
	14	*3*	*2*	*1*	*1*	*1*	*1*	*1*	*1*	*1*
Hardware	Total	Headset uncomfortable	More control practice	Controls confusing	Controls easy	Visual Distortion				
	13	*2*	*1*	*2*	*2*	*6*				
Motion sickness	Total	During task	During videos	No motion sickness						
	11	*5*	*5*	*1*						

Supplementary Analyses

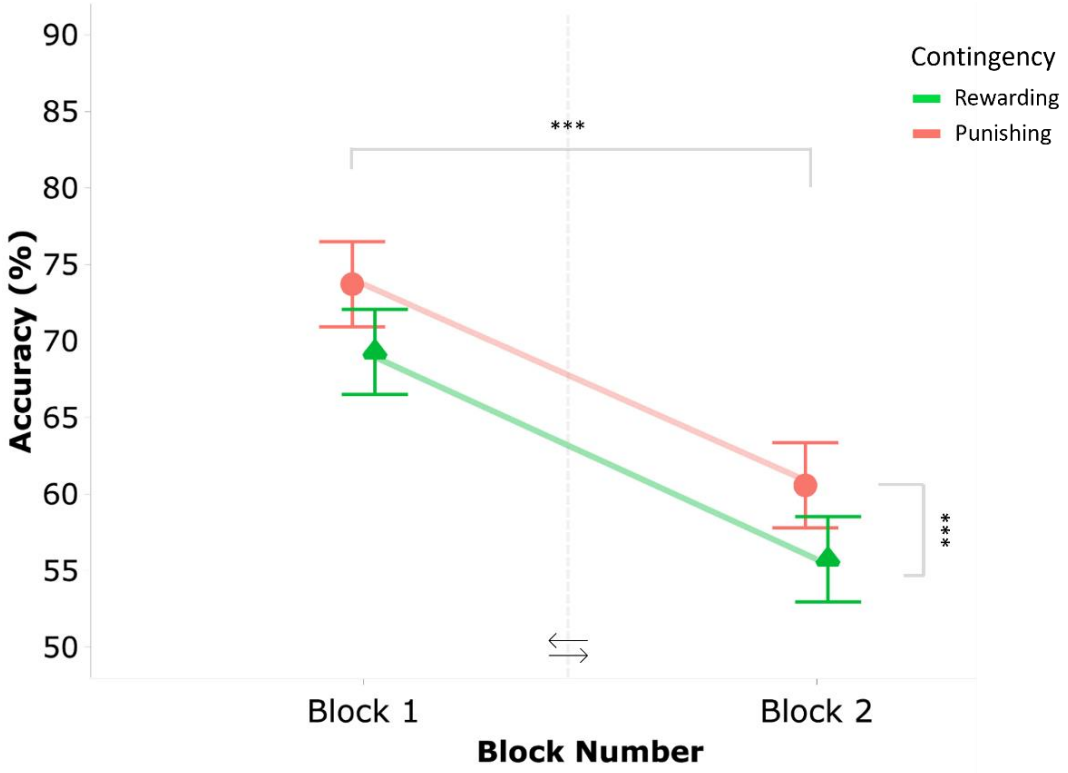
Supplementary Analysis A

A 2 (contingency) x 6 (bins of 10 trials) repeated-measures ANOVA with Greenhouse-Geisser correction reported a main effect of bin at a large effect size ($F(3.224, 499.7) = 62.766$, $p < .001$, $\eta^2_p = 0.288$) To validate learning had occurred in the PRL task, performance was divided both into responses to the rewarding and losing and into bins of 10 trials. A 2 x 6 repeated-measures ANOVA with Greenhouse-Geisser correction reported a main effect of bin at a large effect size ($F(3.224, 499.7) = 62.766$, $p < .001$, $\eta^2_p = 0.288$) and significantly higher performance for the losing cue relative to the winning cue at a small effect size ($p < .001$, $d = -0.285$). Post-hoc comparisons compared average performance between each bin o 20 trials across both contingencies. Post-hoc t-tests with Holm correction for multiple comparisons found that performance increased from Bin 1 to Bin 2 ($p_{holm} < .001$, $d = 0.510$), but this did not increase further from Bin 2 to Bin 3 ($p_{holm} = .639$, $d = 0.080$); suggesting maximum learning was achieved by trial 40 of 60. As expected, performance immediately after the contingency reversal at Bin 4 produced and significant decline at a large effect size ($p_{holm} < .001$, $d = -0.958$). Performance increased at both Bin 5 ($p_{holm} < .001$, $d = 0.620$) and at Bin 6 ($p_{holm} = .006$, $d = 0.259$), but this did not return to the same level of performance as Bin 3 ($p_{holm} < .001$, $d = -0.510$).



Supplementary Analysis B

Performance was significantly lower in Block 2 (post-reversal) relative to Block 1 ($p < .001$) and significantly lower for the rewarding cues relative to punishing cues ($p < .001$).



Chapter 6

General Discussion

Summary

This thesis aimed to better understand cognitive abilities and daily functioning in people with schizophrenia. While cognitive deficits have consistently been found in past research, cognition only explains around 10% of the variance in daily functioning. Moreover, recent research has proposed factors other than the core symptoms of schizophrenia may influence this relationship. As a result, this thesis evaluated how the influence of confounds of patient status, negative affect, amotivation, and metacognition may affect cognitive abilities in psychometric schizotypy. To do so, four studies were designed. The first assessed the influence of these variables psychometrically in relation to neurocognition (Chapter 2), the second adapted these neurocognitive assessments to include behavioural measures of metacognition (Chapter 3), the third then expanded this into the domain of social cognition (Chapter 4), and the final study manipulated motivation levels by creating an immersive Virtual Reality neurocognitive task (Chapter 5). While the findings of these Chapters have been discussed in isolation, this concluding discussion highlights themes between Chapters and discusses the wider implications of this research.

Confounds of patient status

All Chapters in this thesis collected data in samples varying in psychometric schizotypy. This approach may allow indirect inferences to people with schizophrenia to be made in the absence of clinical confounds (e.g., medication side effects, social exclusion, and stigma). All Chapters consistently supported that positive or negative schizotypy were associated with poorer cognitive ability. These findings replicate those of clinical patients, including recent reviews reporting cognitive deficits persist in drug naïve patients[408]. Together, this supports that cognitive deficits in clinical patients are not solely due to these confounds. Throughout this thesis, a key finding is that schizotypy predicted performance towards punishing stimuli (**Table 24**). This finding is consistent with previous clinical studies[316] . Across Chapters, it was suggested that punishing stimuli may produce negative internal states in participants that interfere with cognitive decision-making. This was further supported by the finding that the underlying mechanism may be *perceived* negativity and that reducing task-related anxiety (VR-PRL) may mitigate deficits. As trait negative affect was unrelated to performance, state-level (in the moment) assessments of negative affect or physiological data (e.g., GSR) are needed to validate this argument in future studies. While deficits were not found in regards to rewarding stimuli in contrast to the clinical literature[318,323], recent investigations suggest that while anticipation pleasure may be specifically disturbed[47], responses to rewarding stimuli may be intact. Therefore, perhaps the current tasks here do not tap into anticipatory pleasure. Alternatively, perhaps reward learning may be intact in schizotypy relative to clinical schizophrenia.

The one exception to these findings was the lack of association to set-shifting performance in the M-IDED. This was particularly unexpected considering deficits were found in the IDED, in the wider set-shifting literature, and because previous research has suggested metacognitive task adaptations do not affect the neurocognitive properties of tasks[216]. It is unclear why this finding was observed, but future studies that adapt the IDED to separate punishment and reward learning would give greater insight into this issue.

Another unexpected finding was the beneficial effects of disorganised schizotypy across neurocognitive and social cognitive tasks; especially considering effect sizes were larger than the detrimental effects of positive schizotypy. Again, this was specific to punishing stimuli only. These findings highlight how critical it is to assess of each of the three primary schizotypy scales individually, as these findings would not be observed by measuring total schizotypy traits. However, separating disorganised traits or symptoms is relatively less common and so explanations for this pattern of findings are especially unclear. Within the current cognitive literature, the beneficial effects of schizotypy are rarely reported. The majority of this research considers increases in divergent thinking styles and creativity[495,496] but this is mostly tied to positive schizotypy. While this conflicts with much of the literature the fact these findings are highly consistent in four separate studies warrants further investigation. Regardless, the current findings strongly emphasise that schizotypy is not a pathological phenomenon. It is likely that whether the effects of schizotypy are expressed detrimentally or beneficially is dependent on moderating factors that were not currently assessed[497,498]. The identification of these factors may be key to understanding risk factors for transition to frank psychosis in some. To do so, future research should collect samples with a wide array of functional abilities to be more able to identify those who may adapt and those who may not adapt to schizotypy traits. Moreover, as the majority of the explanations proposed in this thesis relate to attention both eye tracking and pupil dilation methods would give much-needed insights. This pattern of findings suggests that sub-clinical disorganised traits can be adaptive, but clinical levels of disorganised symptoms tend to be maladaptive. It is essential to underscore that despite the abundance of evidence endorsing the use of schizotypy, such findings should not be directly transferred to clinical samples without additional scrutiny. The idea that schizotypy and schizophrenia exist on a continuum does not necessarily imply that one is merely a milder form of the other. Schizophrenia involves deficits across numerous different domains, and even if there is consistency between deficits in schizotypy and schizophrenia, this does not necessarily mean that the causes of these deficits are identical. Therefore, it should not be concluded that the interventions derived from these insights should be the same. Further discussion on disorganised schizotypy and its associations to cognition and metacognition are expanded upon in the

Metacognition section.

Table 24. Summary of significant relationships between schizotypy and cognitive task performance.

	Performance					
	Rewarding/Positive			Punishing/Negative		
Task	*Positive*	*Negative*	*Disorganised*	*Positive*	*Negative*	*Disorganised*
2D-PRL	-	-	-	Poorer	-	*Potentially improved*[2]
M-PRL	-	-	-	Poorer	-	-
GERT-S	-	-	-	*Potentially poorer*[1]	Poorer	Improved
VR-PRL	-	-	-	-	-	-

Note: [1] = p = .07, [2] = significant interaction effect

Motivation

To summarise the potential role of amotivation more concisely further analyses were conducted. Multiple regressions were calculated predicting pre- and post-task motivation from the three schizotypy scales (N = 570 participants, **Table 25**). Overall, there was moderate evidence that schizotypy was *unrelated* to motivation (all $p_{Bonf} > .458$, $BF_{10} < 0.056$) which contrasts with the clinical literature[254 256]. A common explanation in this thesis was that the MIAMI may not be suitable for non-clinical research, as the MIAMI has not been validated and did not present suitable internal consistency in this thesis. Moreover, across all Chapters, the expected associations between negative affect and metacognition with motivation were also largely not replicated. Together, this perhaps points to an issue with instrumentation rather than a disconnect between schizotypy and schizophrenia. In patients, motivation levels are predictive of treatment response[499] and treatment gains[83,500] and so a greater understanding of these processes are critical. The use of other psychometric assessments cross-validated with physiological measures such as pupil dilation would verify these suggestions.

The potential exception to this pattern was the null associations between schizotypy and neurocognitive performance as measured by the VR-PRL. It is unclear how to balance the low reliability of the MIAMI with large increases in motivation for the VR-PRL. As a result, it cannot be concluded that motivation was or was not a deciding factor in mitigating cognitive deficits. However, these findings do strongly emphasise that cognitive tasks do not only tap into cognitive ability. Specifically, the findings here suggest those high in schizotypy may only present deficits in lab-based

assessments, but not more realistic, understandable, and enjoyable VR tasks. Indeed, these findings suggest cognitive task deficits may not necessarily stem from poor cognitive ability, but perhaps from factors unrelated to cognition (i.e., task-related anxiety). This is an important and novel contribution to the literature that highlights the importance of considering simpler alternative explanations. Cognitive tests in schizotypy and wider psychology should consider potential confounding interaction with the general testing environment and consider that clearer task instructions and the gamification may help gain a clearer picture of cognitive abilities.

To support these claims each of the suggested factors should be assessed individually. Firstly, motivation would be better evaluated by replicating the VR-PRL task with the original instructions of Chapter 2 and measuring motivation more objectively through pupil dilation. Secondly, comprehension could be assessed by applying the alien narrative to the 2D-PRL task. Finally, task realism may be difficult to manipulate individually in Virtual Reality without also increasing motivation. Perhaps an In Real Life version of the PRL task (IRL-PRL), akin to the Wisconsin Cart Sort Task, may be appropriate. Pinpointing these influences would have significant implications for clinical patients. If supported, this would advocate for interventions to address the influence (for example) of comprehension or defeatist beliefs to take advantage of preserved abilities – rather than aiming to improve potentially intact cognition (i.e., Cognitive Remediation).

Negative Affect

In contrast to the inconsistent influence of amotivation, negative affect presented a clear lack of influence. As in the previous section, the results were summarised across all participants (**Table 25**). Across all 617 participants the strongest predictor of negative affect was disorganised schizotypy (β = .519, $p_{Bonf} < .001$), which was primarily due to the depression subscale (β = .334, $p_{Bonf} < .001$). This is somewhat surprising, considering that negative schizotypy includes affect-related items and the Cognitive Disorganisation scale of the O-LIFE includes anxiety-related items. Although, perhaps this association is due to an indirect association through maladaptive cognitive thinking styles (e.g., rumination). The relatively weak association between negative schizotypy and negative affect also supports the separability of apathetic states and negative emotional states. This is further replicated in the differential associations to cognitive performance. Overall, these findings provide further support for the continuity between schizotypy and schizophrenia relating to clinical Mood and Anxiety Disorders (MAD) co-morbidity. However, these findings do not support that subclinical levels of negative affect impact cognitive performance in schizotypy.

Table 25. Meta-regression results of the total data of this thesis. Each row represents the standardised coefficients of a multiple regression. All analyses controlled for age and metacognitive analyses additionally controlled for negative affect.

		Schizotypy (β coefficients)		
Variable	*Scale*	*Pos*	*Neg*	*Dis*
Negative affect (N = 613)	*Total*	.100*	.176**	.519**
	Depression	-.026**	.260**	.509**
	Anxiety	.166**	.102**	.399**
	Stress	.146**	.086*	.455**
Metacognition (BCIS) (N = 613)	*Self-Reflectivity*	.160**	.050	.166**
	Self-Certainty	.074	.025	-.211**
Metacognition (MCQ-30) (N = 447)	*Pos Beliefs about Worry*	.110	.015	.154
	Uncontrollability	.065	.017	.371**
	Cognitive Confidence	-.034	-.010	.390**
	Cognitive Self-Cons	.180**	-.036	.069
	Need to Control	.149*	.235**	-.042
Motivation (N = 570)	*Pre-task MIAMI*	.025	.037	-.047
	Post-task MIAMI	-.034	-.066	.033

Note: * = $p < .05$, ** = $p < .01$. All p values are Bonferroni corrected for multiple comparison.

Metacognition

Psychometric metacognition

The influence of metacognition was assessed psychometrically through the use of the BCIS and MCQ-30, which both measure Moderately Discrete Metacognitive thinking styles. In the current literature, both clinical schizophrenia and schizotypy research are inconsistent in terms of whether psychometric metacognition is impaired or improved. The results of all participants were again summarised (**Table 25**), as discussed below.

Positive schizotypy

Positive schizotypy was related to increased ratings of Self-Reflectivity (β = .160, $p_{Bonf} < .001$), Cognitive Self-Consciousness ('pre-occupation with thoughts', β = .180, $p_{Bonf} < .01$), and the Need to Control Thoughts (β = .149, $p_{Bonf} < .05$). The findings of Self-Reflectivity are particularly surprising, as it would have been expected that those high in positive schizotypy would have reduced Self-Reflection, replicating the clinical literature. For example, delusional behaviour has clear links to reduced ratings of being fallible and accepting alternative explanations. However, this view is from the perspective of positive symptoms which are by definition maladaptive, whereas positive schizotypy traits are not defined as such. In non-clinical samples, participants may have perceptual aberrations and atypical thinking styles, but they are likely to have intact daily functioning. Considering this, perhaps increased Self-Reflection is an adaptive strategy to overcome positive traits. Put another way, it could represent a protective factor that limits the impact on daily functioning. This suggestion would also be consistent with associations between positive schizotypy and Cognitive Self-Consciousness and the Need to Control thoughts; potentially representing that participants recognise their positive schizotypy traits through constant thought examination and believe they must exert control over their cognitions. Indeed, other investigations have suggested this heightened awareness could be a protective factor in some people[497], but produce harmful consequences in others[498]. For example, synthetic metacognition has been implicated in stress management strategies[200], more active rather than passive coping styles[501] and greater illness insight[195,196], but high self-reflectivity predicts depressive symptoms in schizophrenia[502].

Negative schizotypy

Negative schizotypy was related to an increased Need to Control Thoughts only (β = .235, $p_{Bonf} < .01$). This finding was suggested to be part of a positive feedback loop in Chapter 4: wherein persecutory thinking styles may increase the avoidance of social contact, leading to higher levels of introvertive anhedonia, which further reduce social contact and opportunity to collect evidence against these

persecutory thinking styles. Under this suggestion, perhaps social contact could be improved in clinical patients by tackling metacognitive thought control schemas (e.g., Metacognitive Therapies[503–506]). The null findings also have significant utility. For example, the lack of association between negative schizotypy and Self-Reflectivity may suggest excessive self-doubt may not impact social withdrawal. Moreover, negative schizotypy appeared generally unrelated to self-reported metacognition. This may have implications for clinical studies wherein negative symptoms are the strongest predictor of cognition and functioning, as perhaps improving self-reflective processes would not be beneficial.

Disorganised schizotypy

It may be expected that those with disorganised thinking may have worse metacognitive abilities, as fragmentation of thinking may mean it is harder to accurately understand their own cognitive processes and regulate their behaviour. However, the opposite was consistently found across these studies. Disorganised schizotypy was related to higher ratings of Self-Reflectivity (β = .166, p_{Bonf} < .001), but reduced Self-Certainty scores (β = -.211, p_{Bonf} < .001). For the MCQ-30, poorer Cognitive Confidence (β = .390, p_{Bonf} < .01) and increased ratings of Uncontrollability and Danger was reported in those high in disorganisation (β = .371, p_{Bonf} < .01). The reduced ratings of Cognitive Confidence are in stark contrast to the improved cognitive ability in disorganised schizotypy observed. Previous studies have similarly reported poor confidence in cognition despite intact ability[507], which together highlight a metacognitive disconnect between perceived and actual performance. However, cognitive confidence did not mediate the relationship between disorganised schizotypy and improved performance, which suggests increased effortful deliberation in an attempt to compensate for perceived poor ability is an unlikely explanation. While this, unfortunately, leaves the explanation for improved performance unclear, it does suggest that improving confidence in cognition would not negate cognitive gains in disorganised schizotypy.

These findings do raise an issue with the MCQ-30, however, as the MCQ-30 assumes higher scores represent a maladaptive thinking style. However, without behavioural data for reference, this relationship cannot be determined. Furthermore, these associations are also likely to differ in meaning dependent upon sample characteristics. For example, student samples tend to be high-functioning meaning poorer Cognitive Confidence scores may be maladaptive. However, for those with low functioning, reduced Cognitive Confidence may be adaptive. Indeed, metacognition could be considered a key moderating factor in both the adaptive or maladaptive expression of schizotypy traits. For example, it could be suggested that over-active self-reflection and doubt may protect those high in schizotypy, but a deterioration of these processes and cognitive ability at the onset of

psychosis may mean schizotypal traits are not controlled and poorer cognition is not reflected upon. This may be a key reason for previous literature inconsistencies.

Interpreting this pattern of associations and the stark contrast to clinical patients is particularly challenging given the existing body of literature on this topic. In clinical patients, there is a consistent pattern for decreased self-reflectivity, increased self-certainty, and poorer metacognitive capacity (e.g., MAS scores). Most research in schizotypy focuses on metacognitive beliefs (i.e., self-reported thinking styles) rather than metacognitive capacity. To date, two studies have assessed how metacognitive capacity, as measured by the clinically-rated MAS-A, is associated with schizotypy (as measured by the SPQ). In the first study[508], negative traits predicted reduced awareness of others' minds and decentration (understanding that others are independent of the self) but not self-reflection or mastery (use of insights). No associations with positive or disorganised traits were found. However, a subsequent study did not replicate these findings using the same measures[509]. Instead, negative traits were related to poorer mastery, and disorganised traits were related to improved self-reflection and awareness of others - the reasons for the latter are not discussed. Nevertheless, both studies are consistent with the current findings on disorganisation, which collectively provide initial support for the idea that subclinical disorganisation is associated with an increased capacity for reflection and heightened reflective thinking patterns. Future research would benefit from measuring both metacognitive capacity and beliefs within the same study. Specifically, while the expected mediation of performance differences between schizotypy and cognition by metacognition was not observed, this may be because measures such as the BCIS and MCQ-30 do not capture metacognitive capacity, which may be more relevant to task performance.

Another explanation for the seemingly inconsistent findings of disorganised schizotypy lies in the extent to which its measurement truly reflects the disorganised symptoms of schizophrenia. Recent studies have not only supported the claim that positive and negative schizotypy traits are more distinct than first thought[294] (a suggestion that this thesis supports in terms of their different relationship to cognition and associations with metacognition), but also that disorganised schizotypy may mediate the relationship between positive and negative schizotypy[510]. A recent large network analysis of schizotypy supported these findings[510] and suggested that cognitive disorganisation held a central role in the experience and psychometric structure of schizotypy. While disorganisation is a core feature in both schizotypy and schizophrenia, its manifestation in schizotypy may not be as disruptive as in schizophrenia, which may explain differences in associations to cognition and metacognition between the two. Few studies have directly compared disorganised schizotypy and schizophrenia beyond factor analytical designs, which makes interpretation particularly difficult. However, one recent study using the PANSS and O-LIFE[300] found that while positive and negative

schizotypy dimensions correlated with corresponding symptom ratings in patients, disorganised schizotypy and symptoms were unrelated. This suggests that these measures may capture different aspects of disorganisation, with cognitive disorganisation in schizotypy potentially more related to social anxiety and neuroticism than to disorganised schizophrenia. This is crucial when interpreting the relationship between cognitive disorganisation, cognitive ability, and metacognition, and the contrast with past clinical literature. It may be more appropriate to consider disorganisation's indirect relationship to cognition and metacognition through its higher-order relationship to positive and negative traits, rather than direct associations. While there is growing evidence for these dissociations, a qualitative understanding of the lived experiences of disorganised symptoms and traits is lacking. Qualitative data could provide insights into the content, experience, and severity of these traits, as well as differences in coping strategies, which could affect trait presentation and daily functioning.

Behavioural metacognition

The limitations of psychometric assessments of metacognition were the primary rationale for Chapter 3 and Chapter 4 – both of which adapted cognitive tasks to allow the behavioural assessment of metacognitive sensitivity (confidence) and metacognitive control (Koren accuracy). In terms of sensitivity, the M-GERT-S results again reported deficits in allocating confidence specifically to punishing stimuli, while the results of the M-PRL further validated that it was specifically the *perceived* negativity of stimuli. This may be in partial conflict with the wider confidence literature that emphasises errors as the source of overconfidence. Although, as those high in schizotypy are more prone to make errors on negatively valence trials, this may conflate accuracy and confidence. One way to test this hypothesis would be to control for baseline accuracy levels when assessing confidence ratings (e.g., meta-d prime)[153]. However, this was not possible with the current study design. If supported, perhaps delusional beliefs may be more associated with attributing exceptionally high confidence to *perceived* negative events, rather than delusions being attributed high confidence because they are erroneous beliefs. That being said, these processes represented a general confidence bias rather than a specific deficit in metacognitive sensitivity.

In contrast, associations between schizotypy and metacognitive control presented interesting findings. In Chapter 4, negative schizotypy was found to predict poorer recognition of negative emotions when considering all emotion recognition decisions, but not when only responses volunteered by participants were included (Koren accuracy e.g., “I think this emotion is sadness and I want this decision to count towards my points total”). This discrepancy suggested that although participants presented faulty social cognition (total accuracy), their intact metacognitive processes

meant they did not act on these deficits (Koren accuracy). This finding is the first to be seen in the schizotypy literature and in the wider social cognition literature, replicating the limited neurocognitive studies in patients[216]. Future studies should assess whether total accuracy or Koren accuracy is most predictive of actual behaviour to highlight potential implications for functioning. If this is supported, the current findings may suggest clinical patients high in negative symptoms and intact metacognitive control may present a subgroup of participants with good functioning. Although, this may only apply to the interplay between negative schizotypy, social cognition, and social functioning, as deficits between positive schizotypy and neurocognitive performance persisted and may represent an alternative pathological pathway. Overall, these results provide novel contributions to the psychosis literature by grounding metacognitive responses in actual behaviour, rather than retrospective subjective questionnaires.

Implications

The current research has promising potential in terms of its applications to experimental psychology and the psychosis-spectrum. A consistent message has been that critical reflection on exactly what our assessments measure is needed and to consider that a multitude of process are involved in simple actions in experimental tasks, or perhaps a much simpler process (such as amotivation) may be at play. The novel applications of Koren accuracy to neurocognitive and social cognitive tasks has highlighted this approach, in which ability does not necessitate acting on ability. Koren accuracy offers a simple modification that could be applied more broadly in psychology, allowing a richer understanding of cognitive processes and changing the nature of tasks to be more reflective of daily tasks. Critically, these insights are more theoretically located more closely to actual behaviour, which may offer novel targets for further investigation and coping strategies for those with maladaptive cognition.

More broadly, Koren accuracy may offer an insight into the relatively weak link between functional capacity (ability) and functional outcomes in schizophrenia. The current research offers preliminary evidence to conduct replications in clinical samples. Specifically, further investigation into the adaptive coping strategies in negative schizotypy to inhibit poor emotion recognition translating to real life could be applies to other scenarios. Perhaps clinical patients present a similar pattern of behaviour which could inform treatment targets and highlight potential coping strategies for other patients. Critically, if the effects of Koren accuracy are replicated in clinical patients, this would caution the acceptance of cognitive battery scores as being reflective of community

functioning. This would warrant a deeper assessment of patient abilities in clinical practice before therapeutic recommendations could be made.

An exciting implication of this research is the further integration of Virtual Reality technology in both experimental psychology and potentially clinical practice. In psychological research, it offers an exciting method to assess cognitive abilities in a naturalistic, controlled, and efficient manner. Moreover, if the increased motivation caused by VR does indeed reduce task deficits, Virtual Reality may prove to an assessment engagement tool in schizophrenia to tackle amotivation. In terms of research, this could increase engagement of patients with experiments and paint a more accurate picture of their abilities, perhaps also helping to disentangle the inconsistent findings in previous research. The start of these applications can be seen in VR assessments of functioning and therapeutic interventions[490]. Indeed, VR may prove to be a more resource efficient method to engage service users in their own healthcare as a larger focus on digital healthcare. The amplified engagement induced by Virtual Reality may serve as a key instrument in addressing the prevalent disparity between the currently limited effectiveness of existing treatments and the profound need for optimized therapeutic outcomes.

Conclusions

This thesis has provided methodological and theoretical suggestions for the wider psychosis literature. Firstly, cognitive deficits found in schizophrenia can be replicated in psychometric schizotypy suggesting deficits in patients are not solely due to clinical confounds. However, this thesis suggests *sub*-clinical positive traits are the most closely related, in contrast to negative clinical symptoms. Moreover, deficits found in schizotypy and patient samples may be impacted by the *perceived* negativity of stimuli, rather than the actual associations between stimuli and outcome. However, disorganised schizotypy appears to be distinct in its relationship to improved cognition and potentially metacognition, although current evidence may point towards disorganised schizotypy as measured by the tools in this thesis as more representative of social anxiety and neuroticism. Thus, these findings may not be applicable disorganised symptoms in clinical patients. Secondly, subclinical disorganised traits in schizotypy may uniquely result in improvements in cognitive testing, highlighting the non-pathological nature of schizotypy and the dissociation to likely detrimental disorganised symptoms. Thirdly, future cognitive research should not consider cognitive tests an exclusive measure of cognition performance, as these tests tap into other factors. In schizotypy, task-related anxiety and task comprehension may play a significant role. In wider psychology, the gamification of cognitive tasks may give a clearer window into actual cognitive ability. Fourthly,

while metacognition may play a key role in the psychosis-spectrum, current psychometric assessments cannot differentiate between adaptive self-reflection from maladaptive naivety. These psychometric assessments must be interpreted in collaboration with behavioural data, such as cognitive tasks, metacognitive indices of performance, or daily functioning. Initial findings of this approach in the current thesis suggest that intact metacognitive control processes in negative schizotypy may mitigate poorer cognitive ability from affecting behaviour. Metacognitive control may be a key therapeutic consideration in clinical samples high in negative symptoms. Finally, while trait negative affect does not appear to contribute to cognitive deficits in schizotypy, it is unknown if this is the case for state negative affect. Overall, the role of metacognition and task factors (i.e., motivation, defeatist beliefs, etc.) are promising areas for future schizotypy research that may have impactful implications for people with schizophrenia.

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