# Individual Differences in Schizotypy and Affect-Related Learning

Siri Srinivasa Damathmari

*This thesis is submitted in partial fulfilment of the Honours Degree of Bachelor of Psychological Science*

School of Psychology

University of Adelaide

October 2019

Words: 9100

## Table of Contents

## List of Figures

## List of Tables

## List of Abbreviations

The following list outlines abbreviations used in this thesis.

Schizotypal Personality Questionnaire …….. SPQ

Schizotypal Personality Questionnaire- Brief Revised Updated …… SPQ-BRU

Schizotypal Personality Disorder …….. SPD

Conditioned Stimulus …….. CS

Unconditioned Stimulus …….. US

True Discrimination …… TD

Pseudo-Discrimination …… PD

Relative Validity Effect …… RVE

**Abstract**

Schizophrenia has been widely linked to cognitive impairments and disruptions in learning. Studies have suggested that poor facial affect processing may underlie the social and interpersonal deficits observed in individuals with the disorder. However, clinical studies have been confounded by the effects of medication and hospitalization. Hence, the current study examines individual differences relating to schizotypy (a personality measure that measures predisposition to schizophrenia-spectrum disorders) and learning about positively and negatively valanced faces. Additionally, relationships between the subscales of schizotypy (as measured by the Schizotypal Personality Questionnaire- Brief Revised Updated) and learning about the affective outcomes is examined. 62 participants completed a battery of tasks which assessed overall learning as well as the relative validity effect, a selective learning paradigm. Although there was insufficient evidence for selective learning and its relationship with schizotypy, it was found that cognitive-perceptual and disorganized dimensions of schizotypy related to greater overall learning about positively valent outcomes whereas the interpersonal dimension of schizotypy was associated with greater overall learning about negatively valent outcomes. Theoretical and clinical implications of these findings are discussed.

*Keywords*: schizophrenia, schizotypy, affect-related learning, facial affect processing

## Declaration

This thesis contains no material which has been accepted for the award of any other degree of diploma in any University, and, to the best of my knowledge, this thesis contains no material previously published except where due reference is made. I give permission for the digital version of this thesis to be made available on the web, via the University of Adelaide's digital thesis repository, the Library Search and through web search engines, unless permission has been granted by the School to restrict access for a period of time.

Siri Srinivasa Damathmari
1st November 2019

**Acknowledgements**

Firstly, a huge thank you to Irina for being an absolute savior with your guidance throughout the year. It has been an incredible rollercoaster of events and I definitely could not have been able to cope without your patient mentorship and immeasurable support. I truly could not have asked for a better supervisor.

To my parents and family; none of this would be possible without your blessings. Thank you for your faith in my pursuits and for your confidence in my decisions. I know this year has not been easy for you either but thank you for standing with me.

Chinmaye, thank you for our daily therapy sessions; you have been by my side at every step of this grueling journey and together, (I hope) we have made the most of the lessons this year has taught us.

To Pranav, although I cannot thank you enough for all you have done for me, I really appreciated your calm reassurances after tolerantly listening to my many stressful rantings.

Nidhi, Hemakshi, Anya, Ojas, Pramodh and the gang – thank you for being there for me whenever I needed you and for encouraging me with your kind words. I am also grateful to all of my friends who took part in the study simply because I requested them to and asking for nothing in return for your two hours of effort.

I would like to thank my co-researchers, Andy and Bart, not only for your cooperation with my hectic life this year, but also for your much-needed advice and friendship.

Thank you also to Dr Matthew Dry, my teachers and course coordinators for your understanding and support throughout the year.

# CHAPTER 1

# INTRODUCTION

## 1.1 Overview

Recent advances in the study of schizophrenia have exposed cognitive deficits, particularly relating to the processing of affective information, as a central cause of the interpersonal deficits that are characteristic of the illness (Bediou et al., 2004). Difficulties with facial affect identification and recognition (Kohler, Walker, Martin, Healey & Moberg, 2009; Bediou et al., 2005), and brain abnormalities in emotional processing areas (Kosaka et al., 2002) have been identified. It follows then, that social symptoms such as affective blunting may be related to impaired affect processing abilities. Studies looking at the personality dimension of schizotypy, which shares commonalities with schizophrenia, overcomes many of the confounds associated with studies conducted with participants in the clinical range of schizophrenia (Morrison et al, 1988) and similar results have been found, relating affect processing impairments with higher levels of schizotypy (Poreh, Whitman, Weber & Ross, 1994; Dickey et al., 2011). Moreover, there are divergent results when correlating the subtypes of schizotypy with affect processing. To clarify these relationships and to explore affect-related learning, the present study examines the relationships between the subtypes of schizotypy and learning about positively and negatively valent outcomes.

## 1.2 Schizophrenia

Even before Kraeplin's (1896) conceptualization of dementia praecox, query about the origins of psychosis had occupied a large part of psychological literature. Later named 'schizophrenia' by Eugene Bleuler (1911, 1950), it became the most characteristic of all psychotic disorders, with a lifetime prevalence of 0.4% in the general population (Bhugra, 2005;

McGrath, Saha, Chant & Welham, 2008). Schizophrenia subjects one to a distorted perception of reality (McReynolds, 1960) and the resultant symptoms include delusions, hallucinations, disorganized thinking and speech, grossly disorganized or abnormal motor behaviour (including catatonia), and negative symptoms such as anhedonia, avolition and social withdrawal (American Psychiatric Association, 2013).

While the heterogeneity of the disorder makes it difficult to classify the entire syndrome of schizophrenia (Andreasen, 1997), two prominent typologies emerge in the literature. The first involves grouping symptoms into 'positive' or 'negative' subtypes (Andreasen & Olsen, 1982) by utilising the Positive and Negative Syndrome Scale (Kay, Fiszbein & Opler, 1987). Positive symptoms include hallucinations, delusions and persistently bizarre behaviour. On the other hand, negative symptoms including affective flattening, alogia, avolition, anhedonia, and attentional impairment can be described as a deficit from normal functioning (Lang et al., 2015). In this method of classification, people with schizophrenia either have a dominance of positive symptoms or negative symptoms or have mixed symptomology where both or neither subtype is dominant. Alternatively, the Bern Psychopathology Scale (Strik et al., 2010) divides the symptoms of schizophrenia into language, affect and motor subtypes. Both typologies, however, have been shown to be complementary; the language-dominant subtype is more strongly associated with positive symptoms and the motor-dominant subtype is more strongly associated with negative symptoms (Lang et al., 2015). Problematically however, there were significant overlaps in the occurrence of these subtypes, resulting in ambiguous assessments; this begs for a more dimensional approach to the diagnosis and conceptualization of psychiatric disorders in general.

### 1.3 Schizotypy

Schizotypy is a multidimensional personality trait that demonstrates vulnerability to schizophrenia and related disorders (Raine, Lencz & Mednick, 1995). The continuity model of schizotypy (Meehl, 1962, 1990) proposes that all members of a population can be assessed for their level of schizotypal traits and it can be measured on a wide range; from normal variance to personality psychopathology and even psychosis, thus allowing for assessment of individual differences (Kwapil, Barrantes-Vidal & Silvia, 2007; Mason, Claridge & Jackson, 1995). While sub-clinical levels of schizotypy can play a part in the experience of daily life events (Kwapil, Brown, Silvia, Myin-Germeys & Barrantes-Vidal, 2012), a longitudinal study reveals that schizotypy assumes strong predictive power for diagnosis of clinical symptoms as well (Kwapil, Gross, Silvia, & Barrantes-Vidal, 2013). In particular, breaking down the construct into 'positive schizotypy' and 'negative schizotypy' in a similar approach to that mentioned previously in relation to schizophrenia, reveals divergent patterns of impairment at the 10-year follow-up. While both subtypes expectedly predict schizotypal personality ratings and psychosis-like traits, the positive dimension is associated with a greater occurrence of mood disturbances and substance abuse, whereas the negative dimension is strongly associated with interpersonal deficits and schizoid personality traits. Evidently, examining by subtypes or dimensions allows for a more comprehensive correspondence with schizophrenia-spectrum symptomology than considering schizotypy a unitary construct (Vollema & van den Bosch, 1995).

The Schizotypal Personality Questionnaire (SPQ) was originally developed by Raine (1991) as a tool to assess levels of schizotypy for diagnosis of Schizotypal Personality Disorder (SPD). It is based on the DSM-III-R definition of SPD which prescribes diagnosis based on nine symptoms: ideas of reference, social anxiety, magical thinking, unusual perceptions, eccentric

behaviour, no close friends, odd speech, constricted affect, and suspiciousness. It is not surprising then, that a nine-factor lower-order structure has the best fit for the SPQ as well as for the most recent Schizotypal Personality Questionnaire-Brief Revised Updated (SPQ-BRU) (Davidson, Hoffman & Spaulding, 2016; Montague, 2016). However, three higher-order factors can also be distinguished: cognitive perceptual, interpersonal and disorganised.

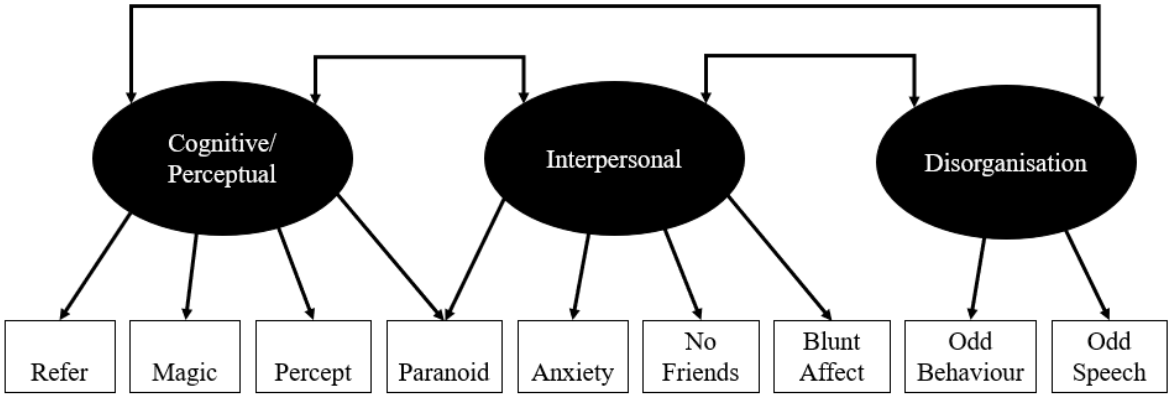



*Figure 1.1.* The Three-Factor Model of Schizotypy. Higher-order factors are represented by circles and the variables that they relate to are represented by squares (adopted from Raine et al., 1994).

The three-factor model of schizotypy (Figure 1.1) relates the cognitive perceptual dimension of schizotypy with the variables: ideas of reference, magical thinking, unusual perceptions and paranoid symptoms. The interpersonal dimension is also associated with paranoid symptoms, as well as anxiety, no close friends and blunt affect. Finally, the disorganised dimension relates to eccentric behaviour and odd speech. The CP and DO dimensions are considered part of positive schizotypy but are assumed to represent distinct traits in the three-factor model, whereas the IP dimension remains strongly linked with negative schizotypy (Bowman & Turnbull, 2009). This model, comparative to all others, most reliably captures the individual differences in schizotypal traits – at both normal and clinical ranges of the spectrum

(Rossi & Daneluzzo, 2002) and is invariant across age and gender (Fossati, Raine, Carretta, Leonardi & Maffei, 2003).

While it is important as a personality trait in and of itself, schizotypy can also be useful in understanding the aetiology of schizophrenia and related disorders by acknowledging the 'fully-dimensional' approach to psychopathology (Barrantes-Vidal, Grant & Kwapil, 2015; Nelson, Seal, Pantelis & Phillips, 2013). As seen in Figure 1.2, this approach defines schizotypy as the underlying personality trait of psychotic disorders, imagined on a continuum applying to all members of the population, instead of a focus only on those in the clinical range (as in the quasi-dimensional model). It conceptualizes how higher levels of schizotypy can increase the vulnerability to develop disorders in the clinical range such as schizotypal personality disorder and schizophrenia, when genetic and environmental contributors are present.

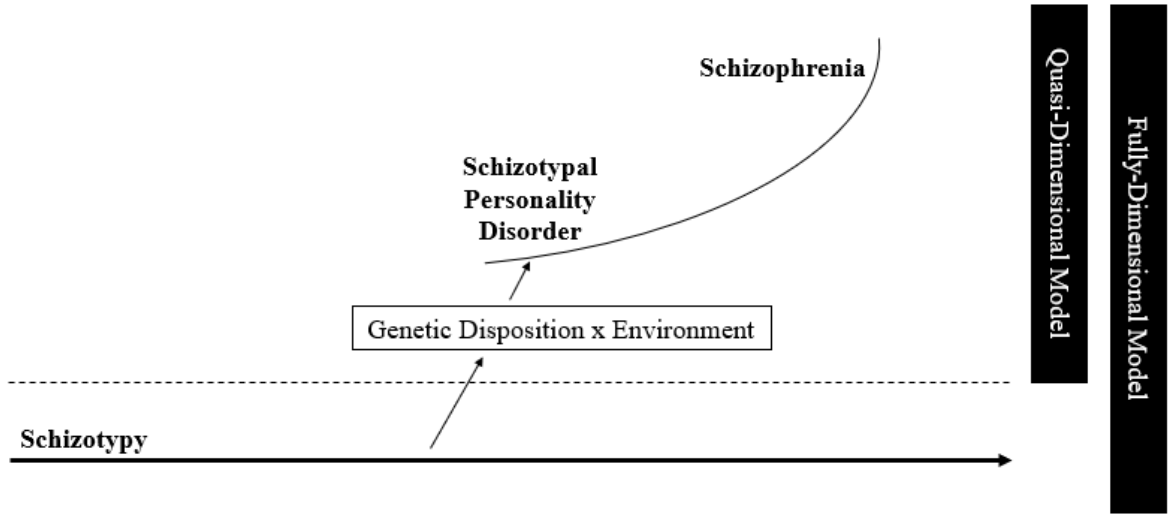



*Figure 1.2.* An illustration of the quasi-dimensional and fully-dimensional models of schizophrenia as they relate to the underlying personality trait, schizotypy (adapted from Claridge & Beech, 1995)

By considering the full spectrum of schizotypal traits, from normal variances to psychotic disorders, the fully-dimensional perspective better captures the heterogeneity of schizophrenic symptomology as well, and enables for identification of mechanisms and factors that can advance movement along the continuum (Barrantes-Vidal et al, 2015). For example, genetic epidemiological research suggests that certain biomarkers contribute to the inheritance of schizotypy but expression of symptoms, particularly in the clinical range, only result if they interact with environmental factors (such as fetal hypoxia) as well (Cannon, van Erp & Glahn, 2002). Dysfunction at the frontal lobe, temporal lobe and sub-cortical areas which can be observed at varying levels along the schizotypal spectrum, have all been linked to a greater risk for the development of schizophrenia (Raine & Lencz, 1995). Vollema, Sitskoorn, Appels & Kahn (2002) further demonstrates that a higher score for the positive dimension of schizotypy as defined by the SPQ is related to a greater genetic predisposition to the development of schizophrenia. Understanding this continuation between schizotypy and schizophrenia can also bring to light some protective factors such as frontal lobe reserve or general intelligence which can allow individuals with high schizotypy to be more resilient to temporal dysfunction (Rosell, Futterman, McMaster & Siever, 2014). This interaction between genetic and environmental factors to produce clinical symptoms can be elaborated within Meehl's (1962) framework which integrates schizophrenia, schizotaxia (a biological vulnerability for schizophrenia) and schizotypy. Figure 1.3 (adapted from Lenzenweger, 2006) illustrates this framework, which is consistent with the 'fully-dimensional' view of schizotypy.

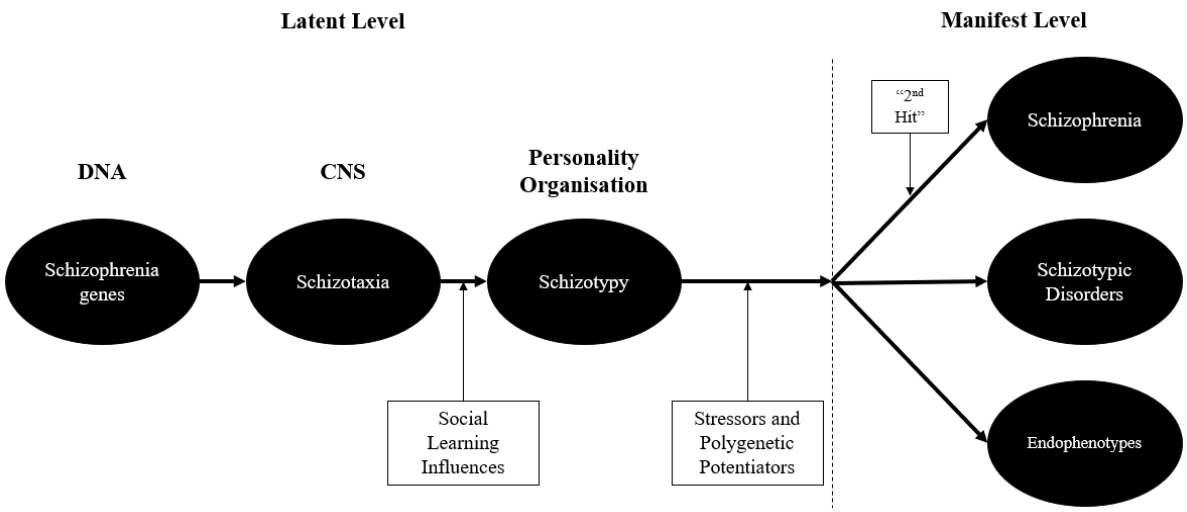



*Figure 1.3.* The Developmental and Genetic Diathesis for Schizophrenia. Genetic vulnerabilities can lead to dysfunction in the neural networks which signal a biological vulnerability for schizophrenia, named 'Schizotaxia'. Environmental factors (in particular, social learning influences) can then then lead to increased Schizotypy; a personality trait-related vulnerability to schizophrenia. Psychosocial stressors and polygenetic potentiators can interact with schizotypy to produce symptoms in the clinical range, manifesting in a diagnosis of schizophrenia (following a '2nd hit' such as in utero exposure to maternal influenza), SPD or endophenotypes (e.g. working memory and attention impairments). CNS = Central Nervous System. (Adapted from Lenzenweger, 2006).

Study of the pre-clinical range of schizotypy can provide a window into the genesis of schizophrenia and related disorders better than a study with subjects already in the clinical range. This is because it overcomes a multitude of confounds associated with clinical subjects relating to hospitalisation, medication, severity, chronicity, comorbidity and distress (Barrantes-Vidal et al, 2015). Hospitalization is a common part of treatment in chronic or acute cases of schizophrenia and prolonged hospitalization is associated with heightened distress and what is

known as the ‘Social Breakdown Syndrome’; a deterioration of social communication capacities (Upadhyay, 1987). Consequently, hospitalisation renders subjects inappropriate for experimental research since the symptoms cannot be separated from social deficits due to schizophrenia itself.

Neuroleptics have been commonly used to treat the psychotic symptoms of schizophrenia since the 1950s (Blanchard & Neale, 1992). Not only do they dampen the dopaminergic system, they also interfere with other systems including alpha-adrenergic and serotonergic receptors. Changes in the functionality of these neurological pathways can alter cognitive and motor abilities that are distinct from the disorder itself, thus making medicated subjects unsuitable in these domains of research (Weickert et al., 2003). While there are identified methods (for example, a factorial manipulation of dosage levels) to cope with these confounds, utilising non-clinical participants who are not required to take medication for psychotic symptoms overcomes the issue altogether.

Furthermore, there is potential for broader implications of schizotypy research as studies have shown it may share an axis with autism – spectrum disorders (Dinsdale, Hurd, Wakabayashi, Elliot & Crespi, 2013) as well as anxiety disorders and depression (Lewandowski et al., 2006).

### 1.4. The Aetiology of Schizophrenia

Several accounts have been put forward to explain the aetiology of schizophrenia. Early theorists attributed the symptoms of schizophrenia to childhood disturbances, particularly relating to problems with mother-infant interactions, and drew links to Freudian theory (Arieti, 1955; Goldfarb, 1961). Around the same time, Kurt Schneider’s philosophy lent way to a phenomenological perspective, where schizophrenia could be classified as arising from a distortion of self-perception (Parnas & Handest, 2003). By developing a list of ‘first-rank

symptoms' – what he believed to be fundamental to the diagnosis of schizophrenia, he has provided root to subsequent diagnostic criteria (Walker, Kestler, Bollini & Hochman, 2004). Then, with the growing popularity of neuroscientific approaches, the dopamine hypothesis of schizophrenia proposed that the disorder was due to hyperactivity of certain dopaminergic pathways, based on the development of neuroleptic treatments (Madras, 2013; Howes & Kapur, 2009). More recently, it has been suggested that dysfunction of NMDA receptors in GABAergic pathways may also play a role in the pathophysiology of schizophrenia (Coyle, 2006). Behavioural genetic studies suggest multiple genes may heighten the risk for the development of schizophrenia, and that prenatal and postnatal environmental factors (such as stress, infections and brain damage) can play a large part in the expression of symptoms (Walker et al., 2004). High comorbidity rates of 15-20% of individuals with schizophrenia also experiencing social anxiety means it is unsurprising that anxiety has been proposed as a pre-eminent factor in the aetiology of schizophrenia (McReynolds, 1960; Lecomte & Théroux, 2017).

More than all of these, however, deficits in cognitive functioning have been brought to light as a fundamental and reliable part of the schizophrenic syndrome (Frith, 2014). Disruptions can be found in a wide range of cognitive abilities; from basic sensory processing (Braus, Weber-Fahr, Tost, Ruf & Henn, 2002) to higher-order capacities like memory, language, attention and planning (Kuperberg & Heckers, 2000; Jentsch, 2003). Furthermore, poor performance on social cognition and neurocognition tasks can predict the onset of psychosis in high-risk individuals (Kim et al., 2011).

### 1.5. Facial Affect and Interpersonal Deficits

These generalised impairments in cognitive function have been shown to underlie many of the diagnostic symptoms of schizophrenia, particularly relating to interpersonal deficits

(Walker et al., 2004). While numerous studies have attempted to explain the poor social competencies though the lens of language dysfunction (Andreason, Hoffman & Grove, 1985) or speech abnormalities (Murphy & Cutting, 1990), the problem may be more fundamental – an impairment in affect processing. Since emotions are intimately associated with interpersonal communication, it is necessary to be able to construe and reciprocate affective cues to be able to communicate effectively in social situations (Morrison, Bellack & Mueser, 1988).

Symptoms such as blunted or inappropriate affect can be traced to difficulties in recognising, understanding and expressing facial emotions (Bediou et al., 2005); which are an important form of non-verbal communication, capable of both expressing information and regulating social behaviour (Morrison et al, 1988). As it is such a crucial aspect of human functioning, the ability to recognise facial affect is gained as young as 3 years old and by 10 years old, it is on-par with adults (Ekman and Oster 1979). It is unsurprising then, that disturbances in facial affect processing mechanisms have been associated with a number of antisocial behaviours which can be traced to maladaptive judgements of social cues (Marsh & Blair, 2008).

A multitude of studies suggest that people with schizophrenia have dysfunctions in emotional processing areas such as the amygdala (Kosaka et al., 2002) and the right prefrontal cortex which has been identified to be responsible for facial affect recognition (Blonder, Bowers & Heilman, 1991; Daskalakis et al., 2002). In particular, there appears to be dissimilar processing for differently valent stimuli in people with schizophrenia compared to the general population. Kosaka et al. (2002) found that while happy faces bilaterally activated the amygdala in both patients and healthy controls, fearful faces bilaterally activated the amygdala only in patients whereas only the right amygdalae were activated in the control group. Facial affect

identification is considerably poorer in people with schizophrenia (Kohler et al., 2009) and it was found that patients performed worse than controls in labelling facial expressions of sadness and anger but not happiness, disgust and fear (Bediou et al., 2005). Taken together, these studies suggest that neurological deficits may form the basis of impairments in facial affect processing in people with schizophrenia and there appears to be differential processing of faces based on the valence of the expressed affect.

When examining facial affect recognition over the course of schizophrenia, some studies suggest chronically ill patients had the most severe generalised impairment in facial perception in comparison to acutely ill patients (Mueser, Penn, Blanchard & Bellack, 1997), while other studies state no relationship between facial affect recognition and state-dependency (Wölwer, Streit, Gaebel & Polzer, 1996). The implications of clinical studies are hence obfuscated by these conflicting conclusions as well as medication or hospitalisation confounds (Morrison et al, 1988).

As explained previously, studying schizotypy (and to an extent, SPD) instead of schizophrenia can overcome many of those confounds. By comparing people diagnosed with SPD to healthy controls, it was found that higher schizotypy scores was related to poorer facial recognition and facial affect recognition (Poreh et al., 1994) as well as problems in interpreting and generating facial affect (Dickey et al., 2011). People diagnosed with SPD were found to be significantly worse than matched controls at labelling positive facial affect compared to other emotions (Waldeck & Miller, 2000). Self-face recognition relating to the right cortical hemisphere has also been found to be impaired with high levels of schizotypy (Platek & Gallup, 2002).

Many designs have explored the separate subtypes of schizotypy with relation to affect processing, rather than considering it only as a unitary construct. While this allows for a deeper understanding of how the different dimensions and variables of schizotypy relate to perception of differently valent stimuli, the results have been inconsistent. It has been found that positive schizotypy is associated with greater attention towards emotions but less clarity of emotions than controls (Kerns, 2005). Williams, Henry and Green (2007) found that negative aspects of schizotypy, but not positive or disorganised dimensions, were related to reduced ability to identify negative facial emotions. Similarly, the interpersonal dimension of schizotypy (especially relating to social anxiety) but not the disorganised or cognitive-perceptual dimension, was associated with poorer facial affect recognition (Abbott & Green, 2013). It is of no surprise then, that affect processing has been recognised as a specific characteristic of disorders in the schizotypal spectrum, more than simply a part of the generalised cognitive deficits (Bryson, Bell & Lysaker, 1997; Penn et al., 2000). Understanding the mechanisms relating to deficits in affect-related processes thus has both clinical and theoretical significance in elucidating the aetiology of schizophrenia.

### 1.6. Learning, Schizophrenia and Schizotypy

While the studies mentioned previously have examined emotion recognition, identification or expression with relation to schizophrenia and schizotypy, associative learning about emotional outcomes (such as facial affect) remains yet unexplored. Learning associations between events and objects in the environment is a fundamental cognitive skill and when learning about emotions are included in the picture, it is also an essential social skill. Among other things, learning to associate neutral cues with facial expressions allows us to predict and adapt to emotional reactions from others and is therefore an integral part of social

communication. Associative learning can be observed in numerous species and has been identified as having a major role in the evolutionary history of humans, being a key factor in driving the Cambrian explosion (Ginsburg & Jablonka, 2010). It follows then, that difficulties with associative learning is related to psychopathologies such as schizophrenia (Farkas et al., 2008; Wadehra, Pruitt, Murphy & Diwadkar, 2013), anxiety (Grillon, 2002) and higher levels of schizotypy (Le Pelley, Schmidt-Hansen, Harris, Lunter & Morris, 2010). Conversely, genetic vulnerabilities for the psychotic symptoms of schizophrenia can be traced to abnormalities in associative learning (Hall et al., 2009).

Associative learning is often measured using classical conditioning paradigms, where an initially neutral conditioned stimulus (CS) acquires the motivational properties of an unconditioned stimulus (US; a biologically significant event such as receiving food) following repeated pairings. It can also be demonstrated with human causal learning tasks, where participants learn to form associations between neutral stimuli. Research using both these paradigms has shown that humans and other animals often do not learn all possible associations between stimuli, but rather, only non-redundant ones. For example, in Kamin's blocking design (1968), where learning about a cue (A) previously paired with the outcome is compared to that of a second cue (B) which is also paired with the outcome, but only in conjunction with the first cue. That is, for an appetitive outcome: A+ is presented first, followed by AB+, where + represents an outcome such as a US. In this paradigm, learning about B is 'blocked' by learning about A; there is weaker responding to B compared to if A was not presented or if A had signaled the absence of the outcome. Thus, people (and other species) tend to ignore or learn less about the redundant cue B, which does not signal the outcome above and beyond A. This and other

effects have led researchers to conclude that learning can be selective (Rescorla & Wagner, 1972).

It has been found that higher levels of schizotypy, particularly in the negative or interpersonal dimensions is associated with increased learning about B in the blocking paradigm, indicating a reduced blocking effect and hence impaired selective learning (Haselgrove & Evans, 2010; Moran, Owen, Crookes, Al-Uzri & Reveley, 2007). Others, however, have found that positive schizotypy (including cognitive disorganisation and unusual experiences) is associated with reduced blocking (Moran, Al-Uzri, Watson, & Reveley, 2003), and other selective learning effects (Gray, Fernandez, Williams, Ruddle & Snowden, 2002). It is therefore still unclear which schizotypy dimension is associated with reduced selective learning. Furthermore, learning about affective stimuli in relation to the dimensions of schizotypy has not yet been investigated.

### 1.7. The Current Study

Taking into account the lack of research on affect-related outcomes and these conflicting associations with schizotypy subtypes, the present study has two broad exploratory aims: examining overall (affective) learning and selective learning about facial affect stimuli (both positively and negatively valanced), and whether individual differences in overall and selective learning are related to specific schizotypy dimensions.

Instead of the aforementioned selective learning paradigm (blocking), the current study investigates the Relative Validity Effect (RVE) (Baetu, Baker, Darredeau & Murphy, 2005; Wagner, Logan, Haberlandt, & Price, 1968). In this design, we assess learning about the target cue (X) in a true discrimination (TD) condition compared to a pseudo-discrimination (PD) condition. In the TD condition, when the target (X) is paired with a cue (A), the outcome occurs 100% of the time (AX – 100% outcome) and when it is paired with another cue (B), the outcome

does not occur (i.e. 0% occurrence; BX – 0% outcome). Here, the participants learn to pay greater attention to A or B to predict the occurrence of the outcome or its absence and learn the irrelevance of the target cue (X) which does not help in solving the discrimination between AX and BX trials. In the PD conditions however, cues AX or BX both result in a 50% chance of the outcome occurring (AX – 50% outcome and BX – 50% outcome) and thus, greater attention would be paid to the target cue (X) in the PD condition compared to the TD condition since neither of the cues is a better predictor of the outcome. In this design, the competing cues are exposed to the participants at the same frequency, simultaneously and thus, this design overcomes confounds such as differences in familiarity of cue or simple memory effects that are present in the blocking paradigm (Baetu et al, 2005).

# CHAPTER 2

# METHOD

## 2.1. Participants

Participants (N = 62) were aged between 18-51 years (M = 24, SD = 7.64) and 44% were female. They were either recruited from the first-year psychology participant pool via the online Research Participation System SONA (n = 22) or were personal contacts of the researchers (n = 42). The results of two participants were excluded due to incomplete data.

This study was approved by the Human Research Ethics Committee of the University of Adelaide. The inclusion criteria for participation were: aged between 18 to 60 years, no history of brain injury or neurological disorder, not suffering from drug and alcohol dependency, not smoking more than 5 cigarettes per day and not using medication that affects neurological function (e.g. antidepressants).

## 2.2. Materials

This study was programmed using the software Xojo (Xojo, Inc., Austin, Texas) and the task was conducted on standard 21-inch Apple iMac computers with a corded HP keyboard and mouse. The information sheets were provided (see Appendix C), and consent forms (see Appendix D) were signed before commencing participation.

**2.2.1. Schizotypy Measure.** The SPQ-BRU (Davidson et al., 2016) is a 32-item questionnaire that reliably measures levels of schizotypy in the non-clinical population. The original SPQ questionnaire (Raine, 1991) was developed to detect the nine features of Schizotypal Personality Disorder (SPD) as defined by the DSM-III-R. Subsequent research found that three higher-order factors can be used to describe dimensions of schizotypy: interpersonal, disorganised and cognitive-perceptual (Raine et al., 1994). Reliability has been

improved by using a 5-point likert scale rather than a binomial yes/no answer to the items and the three-factor higher-order model had an acceptable goodness-of-fit index (Davidson et al., 2016).

## 2.3. Procedure

The current experiment forms part of a combined battery of tasks that requires around 90 minutes to complete in a single testing session. The task is entirely computerised, and all instructions are provided on-screen. Both components of this study – the training and testing stage, are self-paced. Participants completed the computerised version of the SPQ-BRU questionnaire at the conclusion of the study, after all other tasks.

**2.3.1. Training stage.** In the training stage, participants are asked to predict what outcome (the unconditioned stimuli i.e. US) will follow certain pairs of stimuli (the conditioned stimuli i.e. CS); either the image of a happy face (positively valent outcome), a fearful face (negatively valent outcome) or no face at all (non-valent outcome). As seen in Figure 2.1, the participants make a choice between two buttons containing two possible outcomes (counterbalanced) on either side of a grey rectangle within which the correct outcome is later displayed. Feedback is given by green or red outlines on the buttons as well as the words "Correct!" or "Incorrect!" at the bottom of the screen, followed by a display of the correct outcome for 3 seconds in the central grey rectangle. Following a 1.5 second inter-trial interval, the subsequent trial is displayed. Appendix A contains instructions that were provided to participants in the training phase.

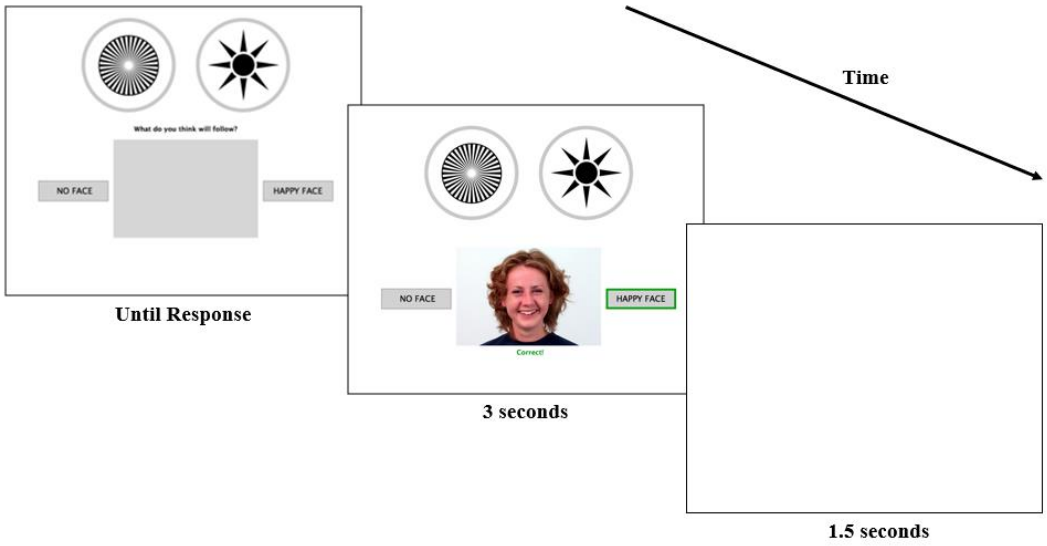



*Figure 2.1.* An example training trial. Participants are first presented with two conditioned stimuli and asked to predict whether a happy face (or fearful face on different trials) will follow by clicking on either of the buttons on the side of the central grey rectangle. Then, feedback is provided – in this case, feedback shows that the choice of a happy face is correct. Then the correct outcome is displayed for 3 seconds, followed by a 1.5 second inter-trial interval.

The conditioned stimuli (CS) are a total of 12 abstract black-and-white geometrical shapes that are randomly allocated within the 8 different conditions as described in Table 1. The outcomes or unconditioned stimuli (US) are images of happy or fearful faces derived from Olszanowski et al., (2015) that have been used to reliably induce positive or negative valence with human participants. In true discrimination (TD) trials, two pairs of stimuli with one common element are presented (e.g. AW and BW, where W is the common stimulus). The paired stimuli reliably predict either a happy face, a fearful face or no face with a probability of 1.0 (i.e. 100% of the time). More specifically, in the positive valence condition, AW always signals a happy face and BW always signals no face. In the negative valence condition, EY always signals

a fearful face and FY signals no face. In the pseudo-discrimination trials (PD), there is a 0.5 probability (i.e. 50% of the time) of the paired stimuli being followed by a happy or fearful face and a 0.5 probability of the paired stimuli being followed by no face. Each trial type is presented 8 times, thus there is a total of 64 trials in the training stage. The trials were randomly intermixed.

Table 1.

*The Relative Validity Effect Paradigm*

| | | |
|---|---|---|
| | Positive Valence Conditions | |
| TD | A + W → 1.0 😊 | B + W → 1.0 ☐ |
| PD | C + X → 0.5 😊, 0.5 ☐ | D + X → 0.5 😊, 0.5 ☐ |
| | Negative Valence Conditions | |
| TD | E + Y → 1.0 😐 | F + Y → 1.0 ☐ |
| PD | G + Z → 0.5 😐, 0.5 ☐ | H + Z → 0.5 😐, 0.5 ☐ |

*Note.* During the training stage, pairs of stimuli predict the outcomes (happy face, fearful face or no face) with differing probabilities. In true discrimination (TD) conditions, the pairs of stimuli always predict the same outcome- whether it is a happy face, fearful face or no face. In the pseudo-discrimination conditions (PD), there is equal chance of the outcome being a happy/fearful face or no face. In the positive valence conditions, the outcome can either be a happy face or no face whereas in the negative valence conditions, the outcome can either be a fearful face or no face. TD = True discrimination. PD = Pseudo-discrimination. The numerical values displayed beside the outcomes indicate the probability of their occurrence.

**2.3.2. Testing Stage.** In the testing stage, participants are presented with each CS separately and are asked to assess the likelihood of it being followed by a relevant US with the question "Do you think that this will follow?". In the positive valence conditions, the CS (A, B, C, D, W, X) are tested with each possible US i.e. happy face or no face. Similarly, in the negative valence conditions, the CS (E, F, G, H, Y, Z) are tested with each possible US i.e. fearful face or no face. The positive and negative valence conditions are randomly intermixed. Participants are asked to rate how likely it is that the US shown will follow that particular CS on a rating scale ranging from 0 ("No, it's very unlikely") to 100 ("Yes, it's very likely"), although the numerical values are not visible to participants. Ratings closer to 100 indicates participants' belief that there is a high probability of that particular outcome to follow the stimulus in question, and ratings closer to 0 indicates belief that there is a low probability of that outcome to follow the stimulus. Ratings that are mid-range indicates uncertainty whereas ratings in the extremities indicates greater confidence in their decision. In each trial, only one of the relevant outcomes is presented along with a single stimulus and participants are not given feedback. For example, as seen in Figure 2.2, the high rating indicates that the participant believes there is a high probability of that stimulus being followed by a happy face. Following their rating, participants then click the "Next" button to proceed to the subsequent trial following a 1.5 second inter-trial interval. Appendix B contains instructions that were provided to participants in the testing phase.

There are 12 stimuli in total and each stimulus can predict two possible outcomes (e.g. happy face or no face), and each stimulus-outcome pairing (e.g. A + happy face) is repeated twice, resulting in a total of 48 trials in the testing stage.

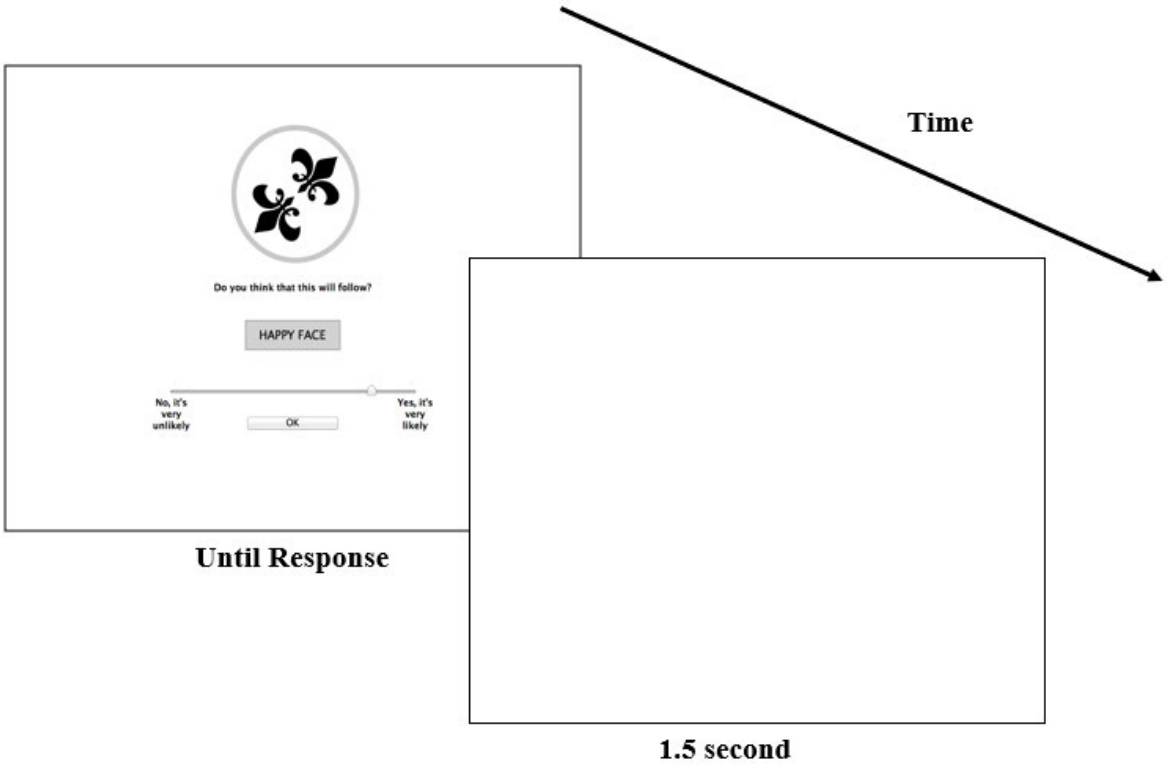



*Figure 2.2.* An example testing trial. Participants are asked to rate the likelihood of a stimulus being followed by an outcome on a scale ranging from 0 ("No, it's very unlikely") to 100 ("Yes, it's very likely") and then click the 'OK' button. Trials are separated by a 1.5 second inter-trial interval and feedback is not provided.

### 2.4. Computation of learning scores

Learning scores for each CS is calculated by subtracting the ratings for a no face outcome from a valent face outcome during the testing stage (e.g. learning score for A = average happy face outcome rating for A – average no face outcome rating for A).

Selective learning is calculated by comparing scores for the target CS on TD trials (W or Y) with scores for the target CS on PD trials (X or Z). For the positive valence conditions, this is measured by subtracting the average rating for W from the average rating for X (i.e. X – W).

Similarly, for the negative valence condition, it is measured by subtracting the average rating for Y from the average rating for Z (i.e. Z – Y). A positive score for either of these variables (i.e. 'X – W' or 'Z – Y') indicates that greater learning has occurred for the target in the PD trials compared to the TD trials, indicating the occurrence of selective learning via the relative validity effect (RVE).

Overall (affect-related) learning is measured both during test and training. Since discriminative learning between the target and non-target CS was not expected to occur in the PD conditions, only the TD conditions were examined. Overall learning as measured by test scores is calculated by comparing predictions for face and no face trials (e.g., participants should be able to report that A would be followed by a happy face and that B would be followed by no face). In the positive valence condition, this was calculated by subtracting the average rating for B from the average rating for A (i.e. A – B) and similarly in the negative valence condition, this was calculated by subtracting the average rating for F from the average rating for E (i.e. E – F). This allows to estimate how much learning has occurred for each type of outcome (whether positive or negative). Additionally, learning during training was calculated by assigning a binomial value (correct = 1 or incorrect = 0) to the final four trials of the TD condition and averaging these scores to calculate a 'training accuracy' measure of learning. Again, learning about positive outcomes (AW, BW) was assessed separately from learning about negative outcomes (EY, FY).

# CHAPTER 3

# RESULTS

## 3.1. Descriptive Statistics

**3.1.1. Schizotypy.** Table 2 presents descriptive statistics for scores on the SPQ-BRU including scores on the specific dimensions: cognitive perceptual, disorganised and interpersonal – all of which were significantly inter-correlated ($p < 0.001$).

Table 2

*Descriptive statistics for scores on the Schizotypy Personality Questionnaire Brief Revised Updated (SPQ-BRU)*

| | Mean | Standard Deviation | Min | Max | Skew | Kurtosis |
|---|---|---|---|---|---|---|
| SPQ Total | 88.05 | 18.31 | 53 | 129 | 0.1 | -0.62 |
| CP | 34.52 | 9 | 16 | 51 | -0.12 | -0.94 |
| DO | 25.73 | 5.64 | 14 | 38 | 0.39 | -0.52 |
| IP | 27.81 | 8.19 | 12 | 47 | 0.31 | -0.57 |

*Note.* SPQ Total = aggregate scores on the Schizotypy Personality Questionnaire Brief Revised Updated (SPQ-BRU) ranging from 0-160. CP = scores on the Cognitive Perceptual dimension of the SPQ-BRU, ranging from 0-70. DO = scores on the Disorganised dimension of the SPQ-BRU, ranging from 0-40. IP = scores on the Interpersonal dimension of the SPQ-BRU, ranging from 0-50.

**3.1.2. Learning Measures.** Descriptive statistics for overall learning scores (as measured by test and training learning scores) and selective learning scores (as measured by the relative validity paradigm) are displayed in Table 3. It was found that on average, there has been above-chance performance in training accuracy and test discrimination, indicating overall learning has

occurred about both positively and negatively valent outcomes. Selective learning scores showed possible evidence for the RVE in negatively valent outcome conditions and, unexpectedly, a reversal of the RVE in positively valent outcome conditions.

Table 3

*Descriptive Statistics for Overall Learning Scores and Selective Learning Scores*

| | Mean | Standard Deviation | Min | Max | Skew | Kurtosis |
|---|---|---|---|---|---|---|
| Training Accuracy | | | | | | |
| Positive Valence | 0.72 | 0.26 | 0.12 | 1 | -0.45 | -1.08 |
| Negative Valence | 0.68 | 0.23 | 0.25 | 1 | -0.10 | -1.36 |
| Test Discrimination | | | | | | |
| Positive Valence | 78.98 | 72.61 | -51 | 200 | -0.01 | -1.07 |
| Negative Valence | 56.92 | 56.92 | -200 | 200 | -0.60 | 0.52 |
| Selective Learning | | | | | | |
| Positive Valence | -17.13 | 58.88 | -161 | 114 | -0.17 | 0.22 |
| Negative Valence | 7.23 | 54.22 | -154 | 100.5 | -0.58 | 0.51 |

*Note*. Training accuracy was calculated by averaging binomial scores (1 = correct, 0 = incorrect) on the last four training trials. Thus, they range from 0 to 1, with 0.5 indicating random performance. Test discrimination scores were calculated by comparing the average ratings for non-target CS in TD trials; that is, 'A – B' for the positive valence condition and 'E – F' in the negative valence condition. The test discrimination scores range from -200 to 200, with 0

indicating random performance. The selective learning scores were calculated by comparing the average ratings for the target CS on TD trials with the ratings for the target CS on PD trials; that is, 'W – X' for the positive valence condition and 'Y – Z' in the negative valence condition. Positive values for these measurements indicate the RVE and a score of 0 indicates no RVE.

### 3.2. Overall (Affect-Related) Learning

**3.2.1. T-tests.** One-sample t-tests revealed that significant learning had occurred about positively valent faces during training ($t$ (61) = 6.64, $p < 0.01$, CI95 [0.65, 0.79]) as well as at test ($t$ (61) = 8.57, $p < 0.01$, CI95 [60.5, 97.4]). Similarly, significant learning had also been observed for negatively valent faces, as seen in both training ($t$ (61) = 6.36, $p < 0.01$, CI95 [0.63, 0.74]) and test scores ($t$ (61) = 5.87, $p < 0.01$, CI95 [38.5, 76.3]). Paired sample t-tests comparing learning about positive versus negative outcomes, however, were non-significant during training ($t$ (61) = 0.92, $p = 0.36$, CI95 [-0.04, 0.11]) and test ($t$ (61) = 1.95, $p = 0.06$, CI95 [-0.55, 44.66]). This indicates that although more learning had occurred for positively valent faces (evidenced by higher means, as seen in Table 3), it was not significantly greater than learning about negatively valent faces.

**3.2.2. Principal Components Analysis.** The test discrimination scores, and the training accuracy scores, both of which were used to assess overall learning, were highly correlated in both positive (0.69) and negative (0.53) valence conditions. Thus, these measures were subjected to a principal components analysis (PCA) to collapse both into a single 'overall learning' measure. The PCA reveals that the first unrotated factor accounts for 84% of the variance in learning scores in the positive valence condition, and 76% of variance in learning scores in the negative valence condition. Training and test scores had equal loading in the PCA for both positively and negatively valent conditions; 0.92 and 0.87 respectively.

### 3.2.3. Correlations between Schizotypy Dimensions and Overall Learning.

Correlations between schizotypy, it's dimensions, and overall learning (as measured by values derived from the PCA) are presented in Table 4. Total schizotypy was significantly correlated with learning about both positively valent and negatively valent outcomes ($p = 0.01$ for both).

The CP and DO dimensions of schizotypy were significantly correlated with learning about positively valent outcomes but not significantly correlated with learning about negatively valent outcomes. On the other hand, the IP dimension of schizotypy was significantly correlated with learning about negatively valent outcomes but not significantly correlated with learning about positively valent outcomes.

Table 4.

*Correlations Between Schizotypy Dimensions and Overall Learning Scores*

| | CP | DO | IP | Total Schizotypy |
|---|---|---|---|---|
| Positive Valence | 0.33* | 0.46** | 0.10 | 0.35** |
| Negative Valence | 0.24 | 0.24 | 0.33* | 0.34** |

*Note:* The dimensions of schizotypy: CP = cognitive perceptual, DO = disorganised and IP = interpersonal. Overall learning was measured by values derived from the principle components analysis (PCA). * = $p < 0.05$. ** = $p \leq 0.01$.

Figure 3.1 illustrates that the CP and DO dimensions are most highly correlated with learning about positively valent outcomes as seen by the greater positive slopes, then followed by the IP dimension. The IP dimension was most highly correlated with learning about negatively valent outcomes as seen by the greatest positive slope in Figure 3.2, followed by the DO and CP dimensions.

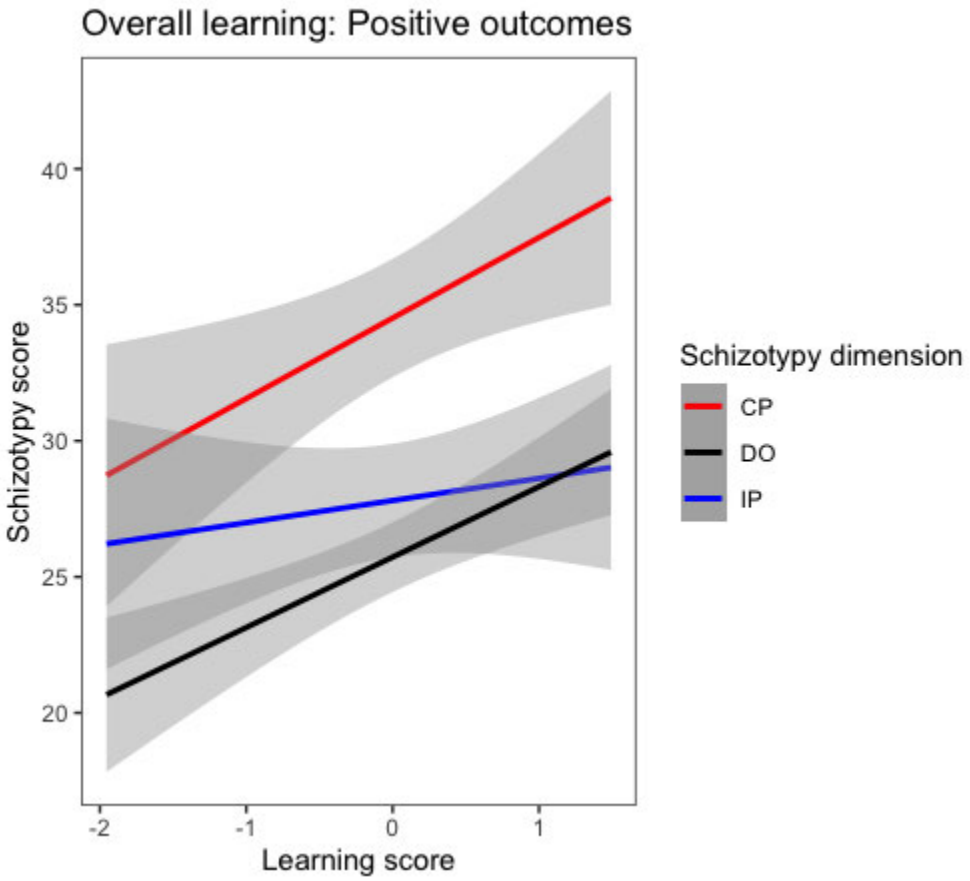



*Figure 3.1.* Linear regression slopes showing the relationship between the dimensions of schizotypy and learning about positively valent outcomes.

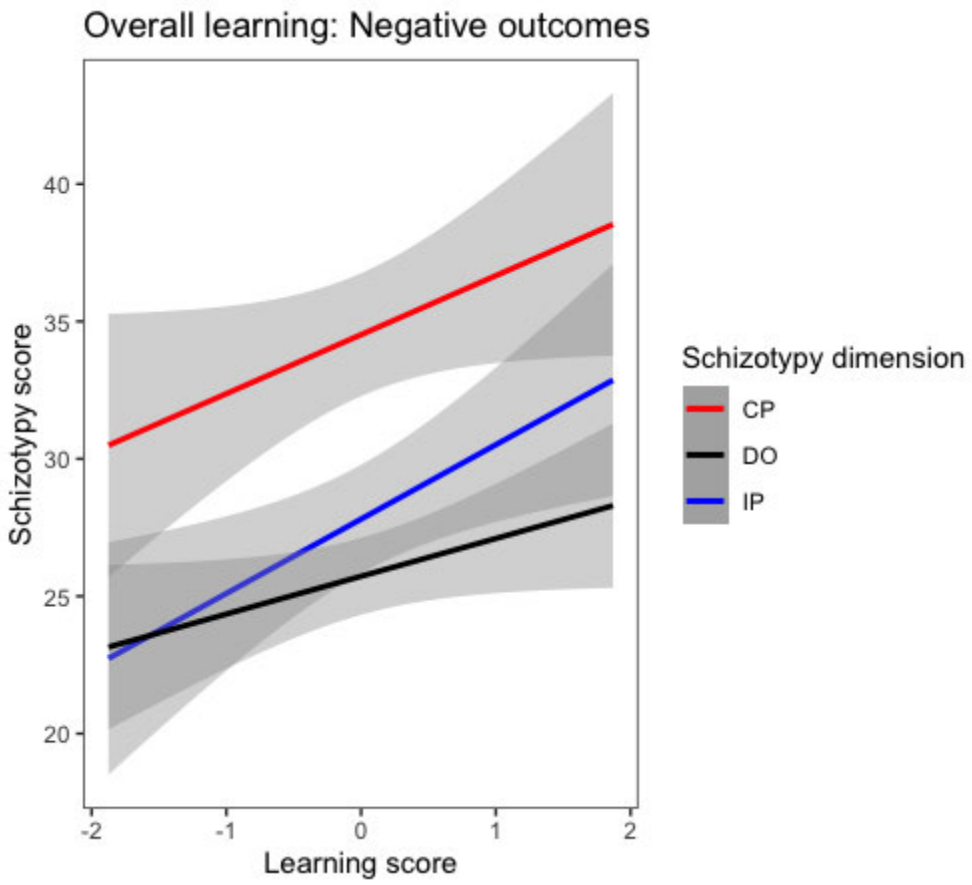



*Figure 3.2.* Linear regression slopes showing the relationship between the dimensions of schizotypy and learning about negatively valent outcomes.

**3.2.4. Linear Regression Models.** First, the overall learning scores were regressed on schizotypy (as a single construct) as well as age and gender to control for their potentially confounding effects (Bora & Baysan Arabaci, 2009). It was found that schizotypy indeed explains a significant portion of the variance in overall learning scores about positively valent outcomes ($R^2 = 0.18$, $F$ (3, 58) = 4.15, $p < 0.01$; and coefficient for total schizotypy = *0.02, p = 0.02* ) and a large portion of the variance for overall learning about negatively valent outcomes ($R^2 = 0.22$, $F$ (3, 58) = 5.57, $p < 0.01$; and coefficient for total schizotypy *B = 0.01, p = 0.06*), after controlling for age and gender.

To further investigate the contribution of each schizotypy dimension, the overall learning scores were regressed on all three dimensions of schizotypy as well as age and gender (see Table 5). Both the positive valence model ($R^2 = 0.34$, $F$ (5, 56) = 5.66, $p < 0.01$) and the negative valence model ($R^2 = 0.24$, $F$ (5, 56) = 3.54, $p < 0.01$) were significant (Table 5).

Table 5

*Regression Models Predicting Overall Learning*

| | $R^2$ | *B* | *SE B* | *p* |
|---|---|---|---|---|
| *Positive Valence* | 0.34*** | | | |
| Age | | -0.03 | 0.02 | 0.090 |
| Gender | | -0.20 | 0.23 | 0.371 |
| CP | | 0.03 | 0.01 | 0.082 |
| DO | | 0.08 | 0.02 | <0.001 |
| IP | | -0.03 | 0.02 | 0.051 |
| *Negative Valence* | 0.24** | | | |
| Age | | -0.04 | 0.02 | 0.023 |
| Gender | | -0.36 | 0.24 | 0.147 |
| CP | | -0.00 | 0.02 | 0.883 |
| DO | | 0.03 | 0.02 | 0.149 |
| IP | | 0.02 | 0.02 | 0.432 |

*Note*. $R^2$ = explained variance. B = regression coefficient. SE B = standard error of regression coefficient. The dimensions of schizotypy: CP = cognitive perceptual, DO = disorganised and IP = interpersonal. * = p < 0.05. ** = p < 0.01. *** = p < 0.001.

To analyse the contributions of each predictor within each model in explaining the variance in affect-related learning, a relative importance regression (Groemping, 2006) was conducted. Figure 3.3 visually represents these contributions as well as the direction of effect on the model (indicated by a '+' = positive effect and '–' = negative effect). While all the factors (i.e. age, gender and schizotypy dimensions) explain 34% of the variance in positively valent learning scores, the DO dimension explains most of this variance, followed by the CP dimension. For negatively valent learning scores, the combined factors explain 24% of the variance and age is the greatest contributor (i.e. membership to the female gender compared to the male gender is more highly correlated with learning about negatively valent outcomes), followed by the IP dimension of schizotypy.

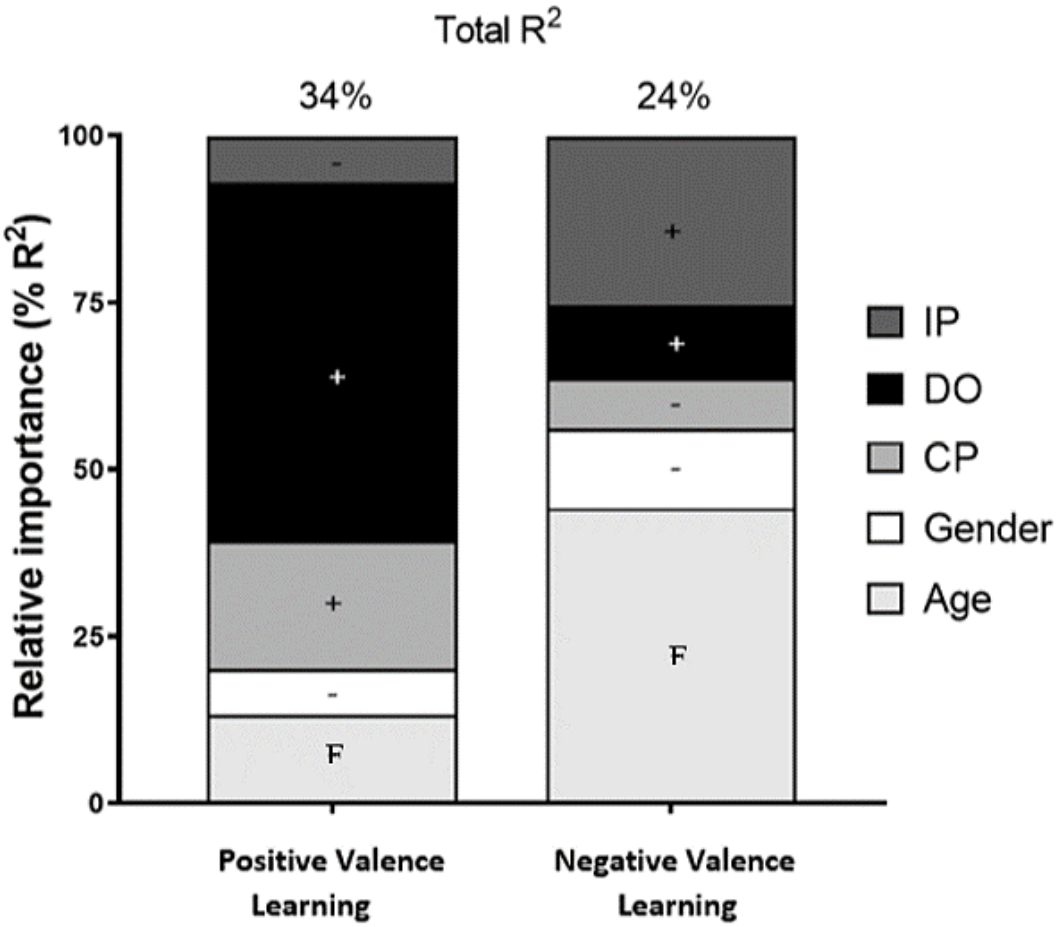


*Figure 3.3.* Relative importance metrics for learning about positively and negatively valent outcomes derived from relative importance regression models. Values over the bar denote the total amount of variance in learning that is explained by the combination of factors: age, gender and the dimensions of schizotypy: CP = cognitive perceptual, DO = disorganised and IP = interpersonal. Direction of the effect of the on each model is represented by: '+' = positive effect and '–' = negative effect. F = female gender bias

## 3.3. Selective Learning

**3.3.1. One-Sample T-tests.** The relative validity effect was not found in this experiment; i.e. learning about the target CS in the PD condition was not significantly greater than learning about the target CS in the TD condition. In fact, a significant reverse RVE was found in the positive valence condition (i.e. the test scores were greater for W than for X; $t$ (61) = 2.29, $p$ = 0.03, CI95 [2.2, 32.1]). There was no significant difference between learning about the target CS in TD or PD trials in the negative valence condition (i.e. there was no significant difference between the test scores for Y and Z; $t$ (61) = 1.05, $p$ = 0.30, CI95 [-6.5, 21.0]).

**3.3.2. Correlations Between Schizotypy and Selective Learning.** Table 6 shows the correlations between the selective learning scores (i.e. the relative validity scores) for positively (X – W) and negatively (Z – Y) valent stimuli, with schizotypy and its dimensions, none which were significant.

Table 6.

*Correlations for Dimensions of Schizotypy and Selective Learning Scores*

| | CP | DO | IP | Total Schizotypy |
|---|---|---|---|---|
| Positive Valence | -0.18 | 0.14 | -0.05 | -0.07 |
| Negative Valence | 0.04 | 0.06 | 0.19 | 0.12 |

*Note.* The dimensions of schizotypy: CP = cognitive perceptual, DO = disorganised and IP = interpersonal.

**3.3.4. Linear Regression.** A regression model examining overall schizotypy (i.e. without considering the separate dimensions) while controlling for the possibly confounding effects of age and gender failed to significantly explain the variance in selective learning scores for positively valent ($R^2 = 0.05$, $F$ (3, 58) = 0.98, $p = 0.4$ and coefficient for schizotypy = 0.05, p = 0.91) and negatively valent ($R^2 = 0.05$, $F$ (3, 58) = 0.97, $p = 0.41$ and coefficient for schizotypy = 0.19, p = 0.64) outcomes. Linear regression models in which the selective learning scores were regressed on all three dimensions of schizotypy as well as age and gender were also non-significant for both the positive valence condition ($R^2 = 0.11$, $F$ (5, 56) = 1.40, $p = 0.24$) and the negative valence condition ($R^2 = 0.06$, $F$ (5, 56) = 0.74, $p = 0.60$) (see Table 7).

Table 7

*Regression Models Predicting Selective Learning*

| | $R^2$ | *B* | *SE B* | *p* |
|---|---|---|---|---|
| *Positive Valence* | 0.11 | | | |
| Age | | 1.48 | 1.06 | 0.168 |
| Gender | | -4.44 | 15.39 | 0.774 |
| CP | | -1.40 | 1.01 | 0.171 |
| DO | | 2.43 | 1.50 | 0.110 |
| IP | | 0.20 | 1.19 | 0.867 |
| *Negative Valence* | 0.06 | | | |
| Age | | -0.96 | 1.00 | 0.345 |
| Gender | | 11.75 | 14.55 | 0.423 |
| CP | | -0.51 | 0.95 | 0.595 |
| DO | | 0.01 | 1.42 | 0.992 |
| IP | | 1.08 | 1.12 | 0.338 |

*Note*. $R^2$ = explained variance. B = regression coefficient. SE B = standard error of regression coefficient. The dimensions of schizotypy: CP = cognitive perceptual, DO = disorganised and IP = interpersonal.

# CHAPTER 4

# DISCUSSION

## 4.1. Overview

While previous studies indicate that schizophrenia and higher levels of schizotypy is related to deficits in facial affect processing (Kohler et al., 2009; Poreh et al., 1994), the implications of this upon learning about affective outcomes had not yet been explored. Since associative learning, particularly relating to facial affect, is essential to social functioning (Morrison et al, 1988), it is important to explore impairments in facial affect-related learning as it can explain the origins of many psychopathologies with symptoms of interpersonal dysfunction. By understanding the relationships between the dimensions of schizotypy and affect-related learning, we can better understand the aetiology and improve the clinical models of psychopathologies such as schizophrenia and related disorders (Vollema et al., 2002), anxiety and depression (Lewandowski et al., 2006) and even autism spectrum disorders (Dinsdale et al., 2013).

The current study, to the best of our knowledge, is one of the first investigating the relationship between schizotypy and affect-related learning; by measuring both overall learning about affective faces as well as selective learning via the relative validity paradigm. There was particular interest in exploring the associations between specific dimensions of schizotypy as defined by the three-factor model (Raine et al., 1994) and differential learning about positively valent outcomes (i.e. happy faces) and negatively valent outcomes (i.e. fearful faces).

## 4.2. The Current Study

Results of the current study demonstrates that significant learning has occurred about both positively and negatively valent outcomes, indicating that our design reliably captures

associative learning between neutral cues and affective outcomes. While there was more learning about positively valent outcomes compared to negatively valent outcomes, this difference was non-significant, suggesting that participants learnt almost equally well about both positive and negative outcomes.

Crucially, a strong relationship between schizotypy and affect-related learning was found. Taken as a unitary construct, schizotypy significantly explained much of the variance in learning; that is, higher levels of schizotypy was associated with greater overall learning about both positively and negatively valent outcomes. This was evident from simple correlations as well as linear regression models that control for possibly confounding factors such as age and gender (Bora & Baysan Arabaci, 2009). Furthermore, overall learning about the affective outcomes was modulated disparately by the dimensions of schizotypy. The CP and DO dimensions of schizotypy were found to be strongly associated with learning about positively valent outcomes, whereas the IP dimension was found to be strongly associated with learning about negatively valent outcomes. The relative importance regression reinforced these patterns of results when explaining the variance in the regression models, and also revealed that females demonstrated better learning than males, particularly about negatively valent outcomes. Finally, this study did not find evidence of selective learning as measured by the relative validity paradigm, nor any relationships between selective learning and schizotypy.

### 4.3. Implications of Overall Affect-Related Learning

Previous studies have shown that interpersonal deficits in individuals with high levels of schizotypy and those in the clinical range (e.g. with schizophrenia or schizotypal personality disorder) can be traced to cognitive dysfunction (Walker et al., 2004); particularly relating to the processing of emotional faces (Bediou et al., 2005). There is evidence that neurological

abnormalities exist in facial affect processing areas such as the right prefrontal cortex (Blonder et al., 1991) and general affect processing areas like the amygdala (Kosaka et al., 2002) in people with schizophrenia. Individuals with high schizotypy levels display inferior performance on facial affect identification and recognition tasks (Poreh et al., 1994). However, the implications of this upon learning mechanisms had not yet been explored in sufficient depth. Associative learning about cues in the environment, particularly affective cues, are crucial in social interactions (Morrison et al, 1988) and misunderstandings of facial affect cues have been linked to antisocial behaviour (Marsh & Blair, 2008). Thus, it is important to investigate associative learning about facial affect with relation to schizotypy as this may be a causal mechanism for some of the interpersonal symptoms seen in schizotypy and schizophrenia.

The present study found that higher levels of schizotypy was related to enhanced affect-related learning. While this may initially appear contradictory to previous findings of learning deficits, the poor performance on selective learning tasks such as blocking is actually due to greater learning, albeit about irrelevant cues (Haselgrove & Evans, 2010). It has been found that individuals with high schizotypy (Moran, Al-Uzri, Watson & Reveley, 2003) and schizophrenia (Jones, Gray & Hemsley, 1992) struggle to accurately direct attention and attribute salience to the correct environmental cues. Directing attention away from irrelevant cues in selective learning paradigms (Gray & Snowden, 2005) aims to reduce cognitive load (Kalyuga & Singh, 2016) and failure in this mechanism results in the distorted performance on selective learning paradigms that is observed in people with schizophrenia or high levels of schizotypy. However, there are mixed findings when relating this to the specific dimensions of schizotypy and the symptoms of schizophrenia.

One account proposes that failure to regulate learning is associated with positive symptoms such as hallucinations and delusions – which are ascribed to be a consequence of hyperactive dopaminergic systems (Howes & Kapur, 2009; Kapur, Mizrahi & Li, 2005). Winton-Brown, Fusar-Poli, Ungless & Howes (2014) found that dysregulation in dopamine pathways leads to incorrect salience attributions, leading to psychotic symptoms. This is why poor performance in selective learning paradigms such as blocking is observed in acute, but not chronic patients with schizophrenia; since the acute phase involves heightened dopamine levels whereas chronic patients are likely medicated with neuroleptics which dampen dopamine activity in the brain.

Dopamine plays an important role in learning about both appetitive and aversive events (Salamone, 1994) and promotes associative learning though two mechanisms. Firstly, it signals prediction error – that is, the release of dopamine corresponds to new learning of associative links between unexpected environmental stimuli (Kapur et al., 2005). Since the firing of dopamine neurons is a motivational occurrence (Arias-Carrión & Pöppel, 2007), it is unsurprising then, that disorders such as schizophrenia, which are characterised by heightened dopamine activity in the midbrain, is related to greater associative learning. Secondly, dopamine is involved in assigning motivational salience upon reward-associated stimuli, causing it to accrue greater attention and thus result in improved long-term, goal-directed learning about such stimuli (Kapur et al., 2005). Moreover, dopamine agonists such as amphetamine have been found to worsen performance on selective learning tasks, indicating similar enhancements in learning as in people with schizophrenia (Gray & Snowden, 2005). Taken together, it is evident that dopamine (which plays a causal role in the positive symptoms of schizophrenia) functions as a

promoter to associative learning, particularly when learning about biologically relevant, rewarding outcomes.

The present study has found that the CP and DO dimensions of schizotypy which encompass positive symptoms (such as paranoid thoughts and eccentric behaviour) is correlated with enhanced learning about happy face outcomes. Positively valent outcomes such as happy faces are appetitive, and they elicit strong effects on the amygdala since they are biologically relevant (Santos, Mier, Kirsch & Meyer-Lindenberg, 2011). Thus, with an ecologically valid design, the present study demonstrates that the CP and DO dimensions of schizotypy is indeed related to dopamine-enhanced learning about positively valent (appetitive) outcomes.

This has a number of clinical implications, such as when examining the high co-morbidity of schizophrenia with addiction behaviours (Hambrecht & Häfner, 1996). Cleghorn et al. (1991) found that substance abuse in schizophrenia is initially related to the occurrence of positive symptoms and in later stages can cause social withdrawal which leads to the development of negative symptoms. Thus, a bias in associative learning about rewarding stimuli such as drugs (and related cues) due to dopamine can be a key causal factor in eliciting both positive and negative symptoms of schizophrenia. This aligns with the longitudinal study by Kwapil et al. (2013) which found a temporal association between the positive symptoms of schizotypy and elevated rates of substance abuse. It also highlights a possible reason for the high rates of medication non-compliance in patients with schizophrenia; the dulling of dopamine activity due to neuroleptics is an aversive outcome since dopamine is inherently rewarding and medication is thus avoided to prevent these effects (Fenton, Blyler & Heinssen, 1997).

On the other hand, it was found that the IP dimension of schizotypy, which includes symptoms directly related to interpersonal dysfunction such as anxiety and blunted affect, was

related to improved learning about negative outcomes. Anxiety disorders have been known to cause attentional bias towards aversive stimuli, especially negatively valent faces (Lee, Lim, Lee, Kim & Choi, 2009). This has been recognised as the basis for the persistence of anxiety through reinforcement of negative thoughts and perceptions. Haddad, Lissek, Pine & Lau (2011) note that anxiety disorders develop from augmented fear learning due to greater attention towards social threat cues. This can result in avoidance of social interactions, further perpetuating the issues with social interaction (American Psychiatric Association., 2013). Thus, from this study, it can be seen that the IP dimension of schizotypy modulates attention and improves learning about aversive outcomes (such as negatively valent faces) in much the same way as in anxiety disorders, resulting in similar symptoms of interpersonal dysfunction. Furthermore, the greater learning about negatively valent outcomes demonstrated by female participants aligns with the greater prevalence of anxiety disorders and depression in the female population, attributed to their stronger interpersonal orientation than males (Hale III, Raaijmakers, Muris, & Meeus, 2008).

Considering these divergent relationships between the dimensions of schizotypy and affect-related learning, it is clear that schizotypy cannot be considered only as a unitary construct; the heterogeneity of symptom dimensions that comprise the personality measure must be acknowledged and handled appropriately in research designs. In the same way, there are distinct patterns of learning about positively valent and negatively valent outcomes. This indicates that the affective nature of the outcome plays an important role in associative learning – in alignment with the vast literature comparing appetitive and aversive conditioning which have differently valent outcomes resulting in different patterns of learning due to the involvement of different neural circuits (Barberini, Morrison, Saez, Lau & Salzman, 2012).

### 4.4. Failure to Detect the Relative Validity Effect

In the current experiment, participants failed to elicit the relative validity effect i.e. they did not learn more about the target cue in the PD trials compared to the TD trials. This indicates that participants did not learn the irrelevance of the target cue (X) in TD trials where there were better predictors of the outcome (A and B). Moreover, in the positive valence condition, there was found to be a significant reversal of the relative validity effect – more learning has occurred for the target cue in TD trials than PD trials. While it may be that the RVE is a small effect that we aimed to detect and a larger sample size may overcome this issue with the provision of greater power, there are alternative perspectives which explain a failure in detecting the occurrence of selective learning.

Contemporary models of associative learning as presented by Soto (2018) can illuminate a possible reason for this finding. Selective learning paradigms such as blocking and the RVE design acknowledge cue competition effects that can influence the learning of associations between a CS and US. That is, learning about an outcome (i.e. US) is dependent upon the relative salience of the cues (i.e. CS) that are presented, which can 'compete' with each other for predictive value (e.g., CSs that convey more information about the occurrence of a US may reduce learning about other, redundant, CSs that do not convey any additional information). However, associative learning also occurs between the CSs themselves i.e. learning about each CS (e.g. A and X) are not independent; especially if they are always presented in conjunction with each-other, as in the current study. Thus, a strong association between one CS (e.g. A) and the US (e.g. happy face) can also be passed onto a paired CS (e.g. X) such that it also acquires association with the US, a phenomenon known as second-order conditioning (Rizley & Rescorla, 1972). This effect can be further enhanced by greater similarity between the CS such as when

they are both of the same modality and could hence result in a failure to detect the relative validity effect.

### 4.5. Strengths of the Current Study

A primary strength of the present study is the use of a non-clinical population to assess the personality measure of schizotypy. This has overcome a number of confounds relating to medication and hospitalisation that are associated with studies of clinical participants (Barrantes-Vidal et al, 2015) and it has allowed for broader generalisations to pathological disorders beyond the schizophrenia-spectrum (such as anxiety and depression; Lewandowski et al., 2006).

Secondly, the utilisation of regression models such as linear regression and relative importance regression allow for control of correlations between dimensions of schizotypy as well as age and gender which may have potentially confounding effects (Bora & Baysan Arabaci, 2009). It also provides a more comprehensive understanding of the contributions of these factors in explaining the variance in affect-related learning. Regression methods such as these avoid arbitrary distinctions in the data (such as with a median split into high/low schizotypy categories) and allows for examination of individual differences across the observed range of schizotypy.

In this study, associative learning occurred between a neutral CS and an affective US (positively or negatively valanced face) or non-affective US (no face, grey square). Since each training trial required participants to make a choice between either an affective outcome or a non-affective outcome, there was no direct discrimination required between the different affective outcomes themselves i.e. happy face and fearful face. Thus, learning about a happy face or fearful face occurred independently of one-another. Furthermore, during the testing phase, the CS was presented only with relevant outcomes that were presented during training. For example, if training involved learning that AX results in a happy face 100% of the time and no face 0% of

the time, testing of A and X would only consist of rating the possibility of a happy face or no face outcome – participants were not asked to rate the possibility of a fearful face outcome in this scenario. Thus, comparative associative learning between happy and fearful faces (i.e. positively and negatively valent outcomes) was not assessed, but rather the study focused on associations between the neutral CS and affective US. This is an important strength in the design of this study when taking into consideration that higher levels of schizotypy is associated with disturbances in facial affect identification and recognition (Poreh et al., 1994). Since learning about happy faces and fearful faces was assessed separately, this study overcomes the need to discriminate between facial affects or identify the exact facial affect. Therefore, the individual differences in learning that were observed were not confounded by an inability to discriminate between faces with different expressions, since each discrimination involved only one face outcome and a no-face outcome.

### 4.6. Limitations and Future Directions

In examining the relationship between existing levels of schizotypy and affect-related learning, only correlational analyses were performed and neither schizotypy levels nor ability to learn about affect-related outcomes were directly manipulated. Thus, it can be difficult to discern direction of causality from this study design i.e. whether schizotypy levels influence affect-related learning or whether dysfunctions in affect-related learning are a cause of schizotypal traits. Hence, a longitudinal study can be of use to answer this question as it can examine temporal patterns in the development of both schizotypy and patterns of learning.

The regression analyses of the present study revealed that females had greater learning than males, particularly about negatively valent outcomes. Future studies with a larger sample size may allow for exploration of the possible influence of gender on affect processing (as

suggested by Scholten, Aleman, Montagne & Kahn, 2005) and may have greater power to detect a selective learning effect, if such an effect exists.

While utilising biologically relevant cues such as emotional faces have allowed the present study to validly measure affect-related learning, incorporating other stimulus modalities for the CS (such as auditory or olfactory cues) may expand upon the current findings. However, the biological significance of affective faces can overshadow cue-competition effects (Oberling, Bristol, Matute & Miller, 2000), hence this must be accounted for if designs may use such stimuli as a CS.

It has been shown that dopamine activity moderates associative learning, and affect processing is related to amygdala function. Future research could explore these mechanisms further by utilising techniques such as fMRI (such as in Mitchell, Elliott & Woodruff, 2001), electrodermal measures, pharmacological manipulations (e.g. with a multi-factorial experimental design) or genetic examinations of individual differences. Moreover, it has been shown that even brief training can improve recognition of facial affect in patients with chronic schizophrenia (Silver, Goodman, Knoll & Isakov, 2004). The right prefrontal cortex has been shown to be important for facial affect processing and deficits in this area have been observed in schizophrenia (Blonder, Bowers & Heilman, 1991; Daskalakis et al., 2002). Thus, stimulation with transcranial magnetic stimulation, which has been previously used to attenuate symptoms such as hallucinations in schizophrenia (Hoffman et al., 2000) in conjunction with other training methods may allow for improvements in facial affect processing and hence, a possible avenue for treatment.

Finally, deficits in affect processing have been linked to a broader dysfunction in schizophrenia – a problem with comprehending the mental states of themselves and others (Frith,

2014; Frith, & Corcoran, 1996). Brüne (2005) describes the deviant social behaviours in schizophrenia and deficits in facial emotion recognition as arising from a poor understanding of the 'theory of mind' and notes that the brain systems that represent these functions are overlapping. Exploring affect-related learning with this understanding of theory of mind difficulties can allow for a more comprehensive understanding of the heterogenous syndrome of schizophrenia and pave the way to better address the observed social impairment.

### 4.7. Conclusions

The current study is, to the best of our knowledge, first in investigating schizotypy in conjunction with affect-related learning. An exploration of overall learning as well as selective learning explicates the disparate effects of the dimensions of schizotypy, as defined by the three-factor model, upon learning about affective faces. Individuals with higher scores on the DO and CP dimensions demonstrated greater overall learning about positively valanced outcomes, indicating a greater propensity for appetitive learning whereas those who scored higher on the IP dimension demonstrated greater overall learning about negatively valanced outcomes, indicating a greater propensity for aversive learning. These divergent results have widespread theoretical and clinical implications for schizophrenia-spectrum disorders, substance abuse and anxiety disorders.

## References


Abbott, G. R., & Green, M. J. (2013). Facial affect recognition and schizotypal personality characteristics. *Early intervention in psychiatry*, *7*(1), 58-63.

Addington, J., & Addington, D. (1998). Facial affect recognition and information processing in schizophrenia and bipolar disorder. *Schizophrenia research*, *32*(3), 171-181.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.

Andreasen, N. C. (1997). The evolving concept of schizophrenia: from Kraepelin to the present and future. *Schizophrenia Research*, *28*(2-3), 105-109.

Andreasen, N. C., & Olsen, S. (1982). Negative v positive schizophrenia: Definition and validation. *Archives of general psychiatry*, *39*(7), 789-794.

Andreasen, N. C., Hoffman, R. E., & Grove, W. M. (1985). Mapping abnormalities in language and cognition.

Arias-Carrión, Ó., & Pöppel, E. (2007). Dopamine, learning, and reward-seeking behavior. *Acta neurobiologiae experimentalis*.

Arieti, S. (1955). Interpretation of schizophrenia. Oxford, England: Robert Brunner.

Arieti, S. Interpretation of Schizophrenia (Basic Books, 1974).

Baetu, I., Baker, A. G., Darredeau, C., & Murphy, R. A. (2005). A comparative approach to cue competition with one and two strong predictors. *Learning & behavior*, *33*(2), 160-171.

Barberini, C. L., Morrison, S. E., Saez, A., Lau, B., & Salzman, C. D. (2012). Complexity and competition in appetitive and aversive neural circuits. *Frontiers in neuroscience*, *6*, 170.

Barrantes-Vidal, N., Grant, P., & Kwapil, T. R. (2015). The role of schizotypy in the study of the etiology of schizophrenia spectrum disorders. *Schizophrenia Bulletin*, *41*(suppl_2), S408-S416.

Bediou, B., Franck, N., Saoud, M., Baudouin, J. Y., Tiberghien, G., Daléry, J., & d'Amato, T. (2005). Effects of emotion and identity on facial affect processing in schizophrenia. *Psychiatry research*, *133*(2-3), 149-157.

Bhugra, D. (2005). The global prevalence of schizophrenia. *PLoS medicine*, *2*(5), e151.

Blanchard, J. J., & Neale, J. M. (1992). Medication effects: Conceptual and methodological issues in schizophrenia research. *Clinical Psychology Review*, *12*(3), 345-361.

Bleuler, E. (1911) Dementia Praecox oder Gruppe der Schizophrenien. Leipzig: F. Deuticke.

Bleuler, E. (1950) Dementia praecox or the group of schizophrenias (Transl. Zenkin, J.). New York International University Press.

Blonder, L. X., Bowers, D., & Heilman, K. M. (1991). The role of the right hemisphere in emotional communication. *Brain*, *114*(3), 1115-1127.

Bora, E., & Baysan Arabaci, L. (2009). Effect of age and gender on schizotypal personality traits in the normal population. *Psychiatry and clinical neurosciences*, *63*(5), 663-669.

Bowman, C. H., & Turnbull, O. H. (2009). Schizotypy and flexible learning: A pre-requisite of creativity. *Philoctetes*, *2*(2), 5-30.

Braus, D. F., Weber-Fahr, W., Tost, H., Ruf, M., & Henn, F. A. (2002). Sensory information processing in neuroleptic-naive first-episode schizophrenic patients: a functional magnetic resonance imaging study. *Archives of General Psychiatry*, *59*(8), 696-701.

Brüne, M. (2005). Emotion recognition, 'theory of mind,' and social behavior in schizophrenia. *Psychiatry research*, *133*(2-3), 135-147.

Bryson, G., Bell, M., & Lysaker, P. (1997). Affect recognition in schizophrenia: a function of global impairment or a specific cognitive deficit. *Psychiatry Research*, *71*(2), 105-113.

Cannon, T. D., van Erp, T. G., & Glahn, D. C. (2002). Elucidating continuities and discontinuities between schizotypy and schizophrenia in the nervous system. *Schizophrenia Research*, *54*(1-2), 151-156.

Claridge, G., & Beech, T. (1995). Fully and quasi-dimensional constructions of schizotypy. *Schizotypal personality*, *29*, 192-216.

Claridge, G., & Broks, P. (1984). Schizotypy and hemisphere function—I: Theoretical considerations and the measurement of schizotypy. *Personality and Individual Differences*, *5*(6), 633-648.

Cleghorn, J. M., Kaplan, R. D., Szechtman, B., Szechtman, H., Brown, G. M., & Franco, S. (1991). Substance abuse and schizophrenia: effect on symptoms but not on neurocognitive function. *The Journal of clinical psychiatry*.

Cochrane, M., Petch, I., & Pickering, A. D. (2012). Aspects of cognitive functioning in schizotypy and schizophrenia: evidence for a continuum model. *Psychiatry research*, *196*(2-3), 230-234.

Coyle, J. T. (2006). Glutamate and schizophrenia: beyond the dopamine hypothesis. *Cellular and molecular neurobiology*, *26*(4-6), 363-382.

Daskalakis, Z. J., Christensen, B. K., Chen, R., Fitzgerald, P. B., Zipursky, R. B., & Kapur, S. (2002). Evidence for impaired cortical inhibition in schizophrenia using transcranial magnetic stimulation. *Archives of General Psychiatry*, *59*(4), 347-354.

Davidson, C, Hoffman, L., & Spaulding, W. (2016). Schizotypal personality questionnaire–brief revised (updated): An update of norms, factor structure, and item content in a large non-clinical young adult sample. *Psychiatry research*, *238*, 345-355.

Dickey, C. C., Panych, L. P., Voglmaier, M. M., Niznikiewicz, M. A., Terry, D. P., Murphy, C., Zacks, R., Shenton, M. E. & McCarley, R. W. (2011). Facial emotion recognition and facial affect display in schizotypal personality disorder. *Schizophrenia research*, *131*(1-3), 242-249.

Dinsdale, N. L., Hurd, P. L., Wakabayashi, A., Elliot, M., & Crespi, B. J. (2013). How are autism and schizotypy related? Evidence from a non-clinical population. *PloS one*, *8*(5), e63316.

Diwadkar, V. A., Flaugher, B., Jones, T., Zalányi, L., Ujfalussy, B., Keshavan, M. S., & Erdi, P. (2008). Impaired associative learning in schizophrenia: behavioral and computational studies. *Cognitive Neurodynamics*, *2*(3), 207.

Ekman, P., & Oster, H. (1979). Facial expressions of emotion. *Annual review of psychology*, *30*(1), 527-554.

Evans, L. H., Gray, N. S., & Snowden, R. J. (2007). A new continuous within-participants latent inhibition task: Examining associations with schizotypy dimensions, smoking status and gender. *Biological Psychology*, *74*(3), 365-373.

Farkas, M., Polgár, P., Kelemen, O., Réthelyi, J., Bitter, I., Myers, C. E., ... & Kéri, S. (2008). Associative learning in deficit and nondeficit schizophrenia. *Neuroreport*, *19*(1), 55-58.

Feinberg, T. E., Rifkin, A., Schaffer, C., & Walker, E. (1986). Facial discrimination and emotional recognition in schizophrenia and affective disorders. *Archives of general psychiatry*, *43*(3), 276-279.

Fenton, W. S., Blyler, C. R., & Heinssen, R. K. (1997). Determinants of medication compliance in schizophrenia: empirical and clinical findings. *Schizophrenia bulletin*, *23*(4), 637-651.

Fossati, A., Raine, A., Carretta, I., Leonardi, B., & Maffei, C. (2003). The three-factor model of schizotypal personality: Invariance across age and gender. *Personality and individual differences*, *35*(5), 1007-1019.

Frith, C. D. (2014). *The cognitive neuropsychology of schizophrenia*. Psychology press.

Frith, C. D., & Corcoran, R. (1996). Exploring 'theory of mind' in people with schizophrenia. *Psychological medicine*, *26*(3), 521-530.

Ginsburg, S., & Jablonka, E. (2010). The evolution of associative learning: A factor in the Cambrian explosion. *Journal of theoretical biology*, *266*(1), 11-20.

Goldfarb, W. (1961). Childhood schizophrenia.

Goldfarb, W. (1961). The mutual impact of mother and child in childhood schizophrenia. *American Journal of Orthopsychiatry, 31*(4), 738-747.

Gray, N. S., & Snowden, R. J. (2005). The relevance of irrelevance to schizophrenia. *Neuroscience & Biobehavioral Reviews*, *29*(6), 989-999.

Gray, N. S., Fernandez, M., Williams, J., Ruddle, R. A., & Snowden, R. J. (2002). Which schizotypal dimensions abolish latent inhibition?. *British Journal of Clinical Psychology*, *41*(3), 271-284.

Grillon, C. (2002). Associative learning deficits increase symptoms of anxiety in humans. *Biological psychiatry*, *51*(11), 851-858.

Groemping, U. (2006). *Relative importance for linear regression in R: The Package relaimpo 2006*. 17(1), 27.

Haddad, A. D., Lissek, S., Pine, D. S., & Lau, J. Y. (2011). How do social fears in adolescence develop? Fear conditioning shapes attention orienting to social threat cues. *Cognition & emotion*, *25*(6), 1139-1147.

Hale III, W. W., Raaijmakers, Q., Muris, P., & Meeus, W. (2008). Developmental trajectories of adolescent anxiety disorder symptoms: A 5-year prospective community study. *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(5), 556-564.

Hall, J., Romaniuk, L., McIntosh, A. M., Steele, J. D., Johnstone, E. C., & Lawrie, S. M. (2009). Associative learning and the genetics of schizophrenia. *Trends in neurosciences*, *32*(6), 359-365.

Hambrecht, M., & Häfner, H. (1996). Substance abuse and the onset of schizophrenia. *Biological psychiatry*, *40*(11), 1155-1163.

Haselgrove, M., & Evans, L. H. (2010). Variations in selective and nonselective prediction error with the negative dimension of schizotypy. *The Quarterly Journal of Experimental Psychology*, *63*(6), 1127-1149.

Hofer, E., Doby, D., Anderer, P., & Dantendorfer, K. (2001). Impaired conditional discrimination learning in schizophrenia. *Schizophrenia Research*, *51*(2-3), 127-136.

Horan, W. P., Blanchard, J. J., Clark, L. A., & Green, M. F. (2008). Affective traits in schizophrenia and schizotypy. *Schizophrenia bulletin*, *34*(5), 856-874.

Howes, O. D., & Kapur, S. (2009). The dopamine hypothesis of schizophrenia: version III—the final common pathway. *Schizophrenia bulletin*, *35*(3), 549-562.Walker, E., Kestler, L., Bollini, A., & Hochman, K. M. (2004). Schizophrenia: etiology and course. *Annu. Rev. Psychol.*, *55*, 401-430.

Insel, T. R. (2010). Rethinking schizophrenia. *Nature*, *468*(7321), 187.

Jentsch, J. D. (2003). Pre-clinical models of cognitive dysfunction in schizophrenia: new avenues to addressing unmet needs. *Clinical Neuroscience Research*, *3*(4-5), 303-313.

Jones, S. H., Gray, J. A., & Hemsley, D. R. (1992). Loss of the Kamin blocking effect in acute but not chronic schizophrenics. *Biological psychiatry*, *32*(9), 739-755.

Kamin, L. J. "Attention-like" processes in classical conditioning. In M. R. Jones (Ed.), Miami symposium on the prediction of behavior, 1967: aversive stimulation. Coral Gables, Fla.: University of Miami Press, 1968. Pp. 9-31.

Kapur, S., Mizrahi, R., & Li, M. (2005). From dopamine to salience to psychosis—linking biology, pharmacology and phenomenology of psychosis. *Schizophrenia research*, *79*(1), 59-68.

Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophrenia bulletin*, *13*(2), 261-276.

Kerns, J. G. (2005). Positive schizotypy and emotion processing. *Journal of Abnormal Psychology*, *114*(3), 392.

Kerr, S. L., & Neale, J. M. (1993). Emotion perception in schizophrenia: specific deficit or further evidence of generalized poor performance?. *Journal of abnormal psychology*, *102*(2), 312.

Kim, H. S., Shin, N. Y., Jang, J. H., Kim, E., Shim, G., Park, H. Y., Hong, K. S. & Kwon, J. S. (2011). Social cognition and neurocognition as predictors of conversion to psychosis in individuals at ultra-high risk. *Schizophrenia research*, *130*(1-3), 170-175.

Kohler, C. G., Walker, J. B., Martin, E. A., Healey, K. M., & Moberg, P. J. (2009). Facial emotion perception in schizophrenia: a meta-analytic review. *Schizophrenia bulletin*, *36*(5), 1009-1019.

Kosaka, H., Omori, M., Murata, T., Iidaka, T., Yamada, H., Okada, T., ... & Wada, Y. (2002). Differential amygdala response during facial recognition in patients with schizophrenia: an fMRI study. *Schizophrenia research*, *57*(1), 87-95.

Kraepelin, E. (1896). Dementia praecox and paraphrenia, trans. RM Barclay.

Kuperberg, G., & Heckers, S. (2000). Schizophrenia and cognitive function. *Current opinion in Neurobiology*, *10*(2), 205-210.

Kwapil, T. R., Barrantes-Vidal, N., & Silvia, P. J. (2007). The dimensional structure of the Wisconsin schizotypy scales: Factor identification and construct validity. *Schizophrenia Bulletin*, *34*(3), 444-457.

Kwapil, T. R., Brown, L. H., Silvia, P. J., Myin-Germeys, I., & Barrantes-Vidal, N. (2012). The expression of positive and negative schizotypy in daily life: an experience sampling study. *Psychological Medicine*, *42*(12), 2555-2566.

Kwapil, T. R., Gross, G. M., Silvia, P. J., & Barrantes-Vidal, N. (2013). Prediction of psychopathology and functional impairment by positive and negative schizotypy in the Chapmans' ten-year longitudinal study. *Journal of Abnormal Psychology*, *122*(3), 807.

Laing, P. A., Burns, N., & Baetu, I. (2019). Individual differences in anxiety and fear learning: The role of working memory capacity. *Acta psychologica*, *193*, 42-54.

Lang, F. U., Walther, S., Stegmayer, K., Anderson-Schmidt, H., Schulze, T. G., Becker, T., & Jäger, M. (2015). Subtyping schizophrenia: A comparison of positive/negative and system-specific approaches. *Comprehensive psychiatry*, *61*, 115-121.

Le Pelley, M. E., Schmidt-Hansen, M., Harris, N. J., Lunter, C. M., & Morris, C. S. (2010). Disentangling the attentional deficit in schizophrenia: Pointers from schizotypy. *Psychiatry Research*, *176*(2-3), 143-149.

Lecomte, T., & Théroux, L. (2017). 153. Facial Emotion Recognition in Individuals With Schizophrenia With or Without Social Anxiety. *Schizophrenia bulletin*, *43*(Suppl 1), S79.

Lee, T. H., Lim, S. L., Lee, K., Kim, H. T., & Choi, J. S. (2009). Conditioning-induced attentional bias for face stimuli measured with the emotional Stroop task. *Emotion*, *9*(1), 134.

Lenzenweger, M. F. (2006). Schizotypy: an organizing framework for schizophrenia research. *Current Directions in Psychological Science*, *15*(4), 162-166.

Lewandowski, K. E., Barrantes-Vidal, N., Nelson-Gray, R. O., Clancy, C., Kepley, H. O., & Kwapil, T. R. (2006). Anxiety and depression symptoms in psychometrically identified schizotypy. *Schizophrenia research*, *83*(2-3), 225-235.

Lewis, S. F., & Garver, D. L. (1995). Treatment and diagnostic subtype in facial affect recognition in schizophrenia. *Journal of Psychiatric Research*, *29*(1), 5-11.

Madras, B. K. (2013). History of the discovery of the antipsychotic dopamine D2 receptor: a basis for the dopamine hypothesis of schizophrenia. *Journal of the History of the Neurosciences*, *22*(1), 62-78.

Marsh, A. A., & Blair, R. J. R. (2008). Deficits in facial affect recognition among antisocial populations: a meta-analysis. *Neuroscience & Biobehavioral Reviews*, *32*(3), 454-465.

Mason, O., Claridge, G., & Jackson, M. (1995). New scales for the assessment of schizotypy. *Personality and Individual differences*, *18*(1), 7-13.

McGrath, J., Saha, S., Chant, D., & Welham, J. (2008). Schizophrenia: a concise overview of incidence, prevalence, and mortality. *Epidemiologic reviews*, *30*(1), 67-76.

McReynolds, P. (1960). Anxiety, Perception, and Schizophrenia. In D. D. Jackson (Ed.), *The etiology of schizophrenia* (pp. 248-292). Oxford, England: Basic Books.

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, *17*(12), 827.

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

Mitchell, R. L., Elliott, R., & Woodruff, P. W. (2001). fMRI and cognitive dysfunction in schizophrenia. *Trends in cognitive sciences*, *5*(2), 71-81.

Montague, E. (2016). Defining Dimensions In Schizotypy: Factor Structure Replication And External Validation Of The Schizotypal Personality Questionnaire–Brief Revised Updated (SPQ-BRU).

Moran, P. M., Al-Uzri, M. M., Watson, J., & Reveley, M. A. (2003). Reduced Kamin blocking in non paranoid schizophrenia: associations with schizotypy. *Journal of Psychiatric Research*, *37*(2), 155-163.

Moran, P. M., Owen, L., Crookes, A. E., Al-Uzri, M. M., & Reveley, M. A. (2007). Abnormal prediction error is associated with negative and depressive symptoms in schizophrenia. Progress in Neuropsychopharmacology & Biological Psychiatry, 32, 172–181

Morrison, R. L., Bellack, A. S., & Mueser, K. T. (1988). Deficits in facial-affect recognition and schizophrenia. *Schizophrenia Bulletin*, *14*(1), 67-83.

Mueser, K. T., Penn, D. L., Blanchard, J. J., & Bellack, A. S. (1997). Affect recognition in schizophrenia: a synthesis of findings across three studies. *Psychiatry*, *60*(4), 301-308.

Murphy, D., & Cutting, J. (1990). Prosodic comprehension and expression in schizophrenia. *Journal of Neurology, Neurosurgery & Psychiatry*, *53*(9), 727-730.

Nelson, M. T., Seal, M. L., Pantelis, C., & Phillips, L. J. (2013). Evidence of a dimensional relationship between schizotypy and schizophrenia: a systematic review. *Neuroscience & Biobehavioral Reviews*, *37*(3), 317-327.

Oberling, P., Bristol, A. S., Matute, H., & Miller, R. R. (2000). Biological significance attenuates overshadowing, relative validity, and degraded contingency effects. *Animal Learning & Behavior*, *28*(2), 172-186.

Olszanowski, M., Pochwatko, G., Kuklinski, K., Scibor-Rylski, M., Lewinski, P., & Ohme, R. K. (2015). Warsaw set of emotional facial expression pictures: a validation study of facial display photographs. *Frontiers in psychology*, *5*, 1516.

Parnas, J., & Handest, P. (2003). Phenomenology of anomalous self-experience in early schizophrenia. *Comprehensive psychiatry*, *44*(2), 121-134.

Penn, D. L., Combs, D. R., Ritchie, M., Francis, J., Cassisi, J., Morris, S., & Townsend, M. (2000). Emotion recognition in schizophrenia: Further investigation of generalized versus specific deficit models. *Journal of Abnormal Psychology*, *109*(3), 512.

Platek, S. M., & Gallup Jr, G. G. (2002). Self-face recognition is affected by schizotypal personality traits. *Schizophrenia Research*, *57*(1), 81-85.

Poreh, A. M., Whitman, R. D., Weber, M., & Ross, T. (1994). Facial recognition in hypothetically schizotypic college students: the role of generalized poor performance. *Journal of Nervous and Mental Disease*.

Quintana, J., Davidson, T., Kovalik, E., Marder, S. R., & Mazziotta, J. C. (2001). A compensatory mirror cortical mechanism for facial affect processing in schizophrenia. *Neuropsychopharmacology*, *25*(6), 915.

Raine, A. (1991). The SPQ: a scale for the assessment of schizotypal personality based on DSM-III-R criteria. *Schizophrenia bulletin*, *17*(4), 555-564.

Raine, A., & Lencz, T. (1995). Conceptual and theoretical issues in schizotypal personality research. *Schizotypal personality*, 3-15.

Raine, A., Lencz, T., & Mednick, S. A. (Eds.). (1995). *Schizotypal personality*. Cambridge University Press.

Raine, A., Reynolds, C., Lencz, T., Scerbo, A., Triphon, N., & Kim, D. (1994). Cognitive-perceptual, interpersonal, and disorganized features of schizotypal personality. *Schizophrenia bulletin*, *20*(1), 191-201.

Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. *Classical conditioning II: Current research and theory*, *2*, 64-99.

Rizley, R. C., & Rescorla, R. A. (1972). Associations in second-order conditioning and sensory preconditioning. *Journal of comparative and physiological psychology*, *81*(1), 1.

Rosell, D. R., Futterman, S. E., McMaster, A., & Siever, L. J. (2014). Schizotypal personality disorder: a current review. *Current psychiatry reports*, *16*(7), 452.

Rossi, A., & Daneluzzo, E. (2002). Schizotypal dimensions in normals and schizophrenic patients: a comparison with other clinical samples. *Schizophrenia Research*, *54*(1-2), 67-75.

Saha, S., Chant, D., Welham, J., & McGrath, J. (2005). A systematic review of the prevalence of schizophrenia. *PLoS medicine*, *2*(5), e141.

Salamone, J. D. (1994). The involvement of nucleus accumbens dopamine in appetitive and aversive motivation. *Behavioural brain research*, *61*(2), 117-133.

Santos, A., Mier, D., Kirsch, P., & Meyer-Lindenberg, A. (2011). Evidence for a general face salience signal in human amygdala. *Neuroimage*, *54*(4), 3111-3116.

Scholten, M. R., Aleman, A., Montagne, B., & Kahn, R. S. (2005). Schizophrenia and processing of facial emotions: sex matters. *Schizophrenia Research*, *78*(1), 61-67.

Searles, H. F. (1961). Sexual processes in schizophrenia. *Psychiatry*, *24*(sup2), 87-95.

Serra, A. M., Jones, S. H., Toone, B., & Gray, J. A. (2001). Impaired associative learning in chronic schizophrenics and their first-degree relatives: a study of latent inhibition and the Kamin blocking effect. *Schizophrenia Research*, *48*(2-3), 273-289.

Shean, G., Bell, E., & Cameron, C. D. (2007). Recognition of nonverbal affect and schizotypy. *The Journal of Psychology*, *141*(3), 281-292.

Silver, H., Goodman, C., Knoll, G., & Isakov, V. (2004). Brief emotion training improves recognition of facial emotions in chronic schizophrenia. A pilot study. *Psychiatry Research*, *128*(2), 147-154.

Skosnik, P. D., Park, S., Dobbs, L., & Gardner, W. L. (2008). Affect processing and positive syndrome schizotypy in cannabis users. *Psychiatry Research*, *157*(1-3), 279-282.

Soto, F. A. (2018). Contemporary associative learning theory predicts failures to obtain blocking: Comment on Maes et al.(2016).

Stanfield, A. C., Moorhead, T. W. J., Harris, J. M., Owens, D. G., Lawrie, S. M., & Johnstone, E. C. (2008). Increased right prefrontal cortical folding in adolescents at risk of schizophrenia for cognitive reasons. *Biological Psychiatry*, *63*(1), 80-85.

Streit, M., Wölwer, W., & Gaebel, W. (1997). Facial-affect recognition and visual scanning behaviour in the course of schizophrenia. *Schizophrenia research*, *24*(3), 311-317.

Strik, W., Wopfner, A., Horn, H., Koschorke, P., Razavi, N., Walther, S., & Wirtz, G. (2010). The Bern psychopathology scale for the assessment of system-specific psychotic symptoms. *Neuropsychobiology*, *61*(4), 197-209.

Tandon, R., Gaebel, W., Barch, D. M., Bustillo, J., Gur, R. E., Heckers, S., ... & Van Os, J. (2013). Definition and description of schizophrenia in the DSM-5. *Schizophrenia research*, *150*(1), 3-10.

Upadhyay, R. K. (1987). *Hospitalised Schizophrenics: Their Socio-sexual Life and Rehabilitation*. Mittal Publications.

Vollema, M. G., Sitskoorn, M. M., Appels, M. C. M., & Kahn, R. S. (2002). Does the Schizotypal Personality Questionnaire reflect the biological–genetic vulnerability to schizophrenia?. *Schizophrenia research*, *54*(1-2), 39-45.

Wadehra, S., Pruitt, P., Murphy, E. R., & Diwadkar, V. A. (2013). Network dysfunction during associative learning in schizophrenia: Increased activation, but decreased connectivity: An fMRI study. Schizophrenia Research, 148, 38-49.

Wagner, A. R., Logan, F. A., & Haberlandt, K. (1968). Stimulus selection in animal discrimination learning. *Journal of experimental psychology*, *76*(2p1), 171.

Waldeck, T. L., & Miller, L. S. (2000). Social skills deficits in schizotypal personality disorder. *Psychiatry Research*, *93*(3), 237-246.

Walker, E., Kestler, L., Bollini, A., & Hochman, K. M. (2004). Schizophrenia: etiology and course. *Annu. Rev. Psychol.*, *55*, 401-430.

Weickert, T. W., Goldberg, T. E., Marenco, S., Bigelow, L. B., Egan, M. F., & Weinberger, D. R. (2003). Comparison of cognitive performances during a placebo period and an atypical antipsychotic treatment period in schizophrenia: critical examination of confounds. *Neuropsychopharmacology*, *28*(8), 1491.

Williams, B. T., Henry, J. D., & Green, M. J. (2007). Facial affect recognition and schizotypy. *Early Intervention in Psychiatry*, *1*(2), 177-182.

Winton-Brown, T. T., Fusar-Poli, P., Ungless, M. A., & Howes, O. D. (2014). Dopaminergic basis of salience dysregulation in psychosis. *Trends in Neurosciences*, *37*(2), 85-94.

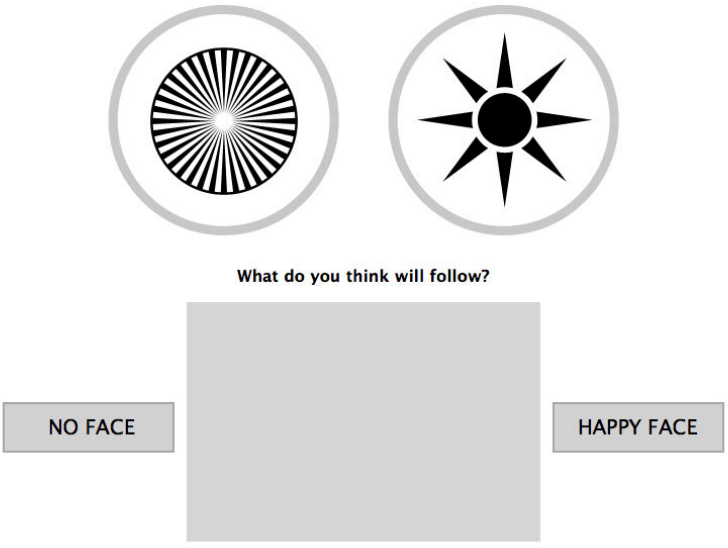
Wölwer, W., Streit, M., Gaebel, W., & Polzer, U. (1996). Facial affect recognition in the course of schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience*, *246*(3), 165-170.

## APPENDIX A

### Training Stage Instructions

In this part of the experiment, you will observe a series of pictures. Some of these pictures will be followed by a happy face, whereas others will be followed by a fearful face, and some will not be followed by any face. Your task is to learn about what will follow each picture – whether it will be a happy face, a fearful face or no face.

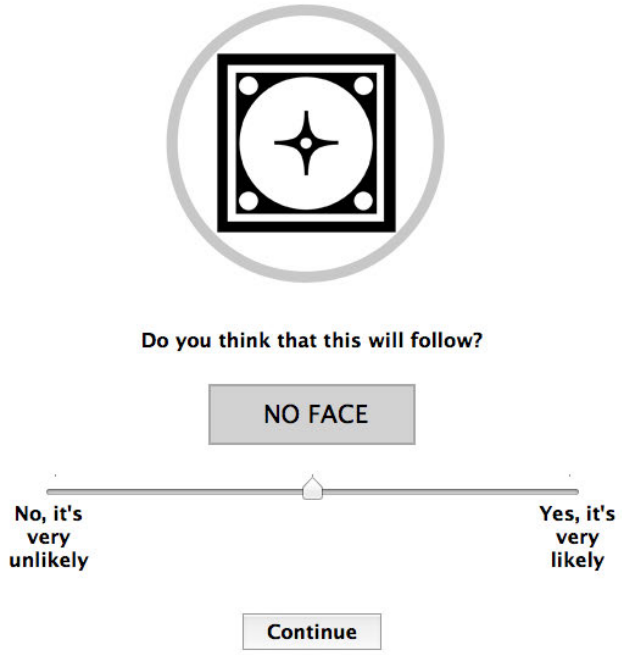
You will be shown two pictures at a time and then asked to predict what kind of face will be shown in the middle rectangle by clicking on one of two buttons on the sides. Following your prediction, you will be shown a happy face, a fearful face, or a blank rectangle (no face) and then you will be informed whether your prediction was correct. This feedback will help you make more accurate predictions later. Pay close attention to the pictures and try to make accurate predictions, as your memory will be tested later on. Have a go at the practice question below!



## APPENDIX B

### Testing Stage Instructions

It's time to test what you have learned. You will be shown some of the pictures you have seen before and asked whether they are likely to be followed by a happy face, a fearful face, or no face. You will be asked to make your prediction using a scale as in the example below. Unlike the previous learning phase, you will no longer receive feedback after your predictions.



THE UNIVERSITY of ADELAIDE

SCHOOL OF PSYCHOLOGY
FACULTY OF HEALTH SCIENCES

THE UNIVERSITY OF ADELAIDE
SA 5005
AUSTRALIA

# APPENDIX C

## Information Sheet

Study Title

Relationship between individual differences in personality and working memory on learning ([redacted])

Investigators

Siri Damathmari

Bartosz Kowalik

Andy Nguyen

Dr. Irina Baetu

Purpose of the Study

This project investigates how people learn to associate stimuli based on individual differences. Associative learning is fundamental as it enables people to predict future events and ignore irrelevant stimuli. Although everyone is capable of such learning, there are known differences in the way people learn to associate stimuli. Working memory, defined as the ability to maintain information in an active state, is one particular factor that is known to influence the formation and strength of associations, and this is an individual difference that is often highly variable between people. By studying the role of working memory and personality in associative learning, a more holistic understanding can be developed of how people are able to learn. Specifically, prior conceptions of associative learning tend to only briefly consider the role of working memory, thus results from this study may provide preliminary evidence that future research of associative learning will require more focus on the role of working memory.

What Happens During the Study

To investigate the learning process, participants are asked to complete computerised tasks that will involve responding to and making predictions about various types of stimuli. One task will involve quickly responding to a sequence of randomised letters, while other tasks will involve making predictions about a series of visual stimuli.

Additionally, the study will also explore the relationship between personality and learning. Measures of personality will be captured through a number of self-reported questionnaires. Finally, participants will be asked to perform several tests that assess working memory. Because learning ability might depend on working memory, we will test whether these cognitive functions mediate the relationship between associative learning and personality.

Location

The study takes place in the Hughes building room 240, School of Psychology, University of Adelaide, North Terrace Campus.

Who Can Participate

Volunteers will be eligible for inclusion in this study only if all of the following apply:

- Aged 18-60 years
- Not suffering from a neurological disorder and no history of brain injury
- Not suffering from a drug or alcohol dependency, either a current or previous condition
- Not smoking more than 5 cigarettes per day
- Not using medication that affects neurological function (e.g., antidepressants, sedatives, antipsychotics)
- Not suffering from an uncorrected visual disorder

Safety and Ethical Issues

The Human Ethics Subcommittee of The University of Adelaide has approved this study (ethics approval number [redacted]). All potential participants will provide their written informed consent before commencing the study. The risks of this study are considered minimal. Every effort will be made to ensure that the discomfort levels are kept to a minimum.

Leaving the Study

You are free to withdraw from the study at any time and for any reason. You are not required to explain your reasons to the study staff. You may also decide to withdraw any collected data. In this case, none of your data will be used for research purposes. Withdrawal from the study will not affect your involvement in any future research programs that you may wish to participate in.

Duration

The study lasts approximately 2 hours.

Confidentiality

All information collected about you from the study is completely confidential. Your results in this experiment will not be associated with your personal information at any point in time (e.g., in publications or presentations). Number codes rather than names will be used to assign identification.

Contact Information

If you have any questions about the study please feel free to contact [redacted]. Please see the attached independent complaints form if you have any concerns regarding the ethics of this research, or would like to speak to someone independent of the project.

**The University of Adelaide**

**Human Research Ethics Committee (HREC)**

*This document is for people who are participants in a research project.*

**CONTACTS FOR INFORMATION ON PROJECT AND INDEPENDENT COMPLAINTS PROCEDURE**

The following study has been reviewed and approved by the University of Adelaide Human Research Ethics Committee:

| | |
|---|---|
| **Project Title:** | **Relationship between individual differences in personality and working memory on learning** |

| | |
|---|---|
| **Approval Number:** | [redacted] |

The Human Research Ethics Committee monitors all the research projects which it has approved. The committee considers it important that people participating in approved projects have an independent and confidential reporting mechanism which they can use if they have any worries or complaints about that research.

This research project will be conducted according to the NHMRC National Statement on Ethical Conduct in Human Research (see http://www.nhmrc.gov.au/publications/synopses/e72syn.htm)

1. If you have questions or problems associated with the practical aspects of your participation in the project, or wish to raise a concern or complaint about the project, then you should consult the project co-ordinator:

| | |
|---|---|
| **Name:** | **Dr Irina Baetu** |
| **Phone:** | [redacted] |

2. If you wish to discuss with an independent person matters related to:

- making a complaint, or
- raising concerns on the conduct of the project, or
- the University policy on research involving human participants, or
- your rights as a participant,

contact Dr. Diana Dorstyn, Deputy Convenor, Human Research Ethics Subcommittee,

[redacted]

## APPENDIX D

SCHOOL OF PSYCHOLOGY
FACULTY OF HEALTH SCIENCES

THE UNIVERSITY OF ADELAIDE
SA 5005
AUSTRALIA

## CONSENT FORM

1. I have read the attached Information Sheet and agree to take part in the following research project:

| | |
|---|---|
| **Title:** | Individual differences in learning, schizotypy and impulsivity |
| **Ethics Approval Number:** | [redacted] |

2. I have had the project, so far as it affects me, and the potential risks and burdens fully explained to my satisfaction by the research worker. I have had the opportunity to ask any questions I may have about the project and my participation. My consent is given freely.
3. Although I understand the purpose of the research project, it has also been explained that my involvement may not be of any benefit to me.
4. I agree to participate in the activities outlined in the participant information sheet.
5. I understand that I am free to withdraw from the project at any time and that this will not affect my study at the University, now or in the future.
6. I have been informed that the information gained in the project may be published in a journal article, thesis or in conference presentations.
7. I have been informed that in the published materials I will not be identified and my personal results will not be divulged.
8. I understand my information will only be disclosed according to the consent provided, except where disclosure is required by law.

9. I am aware that I should keep a copy of this Consent Form, when completed, and the attached Information Sheet.

**Participant to complete:**

Name: _____ Signature: _____ Date: _____

**Researcher to complete:**

I have described the nature of the research to _____

*(print name of participant)*

and in my opinion she/he understood the explanation.

Signature: _____ Position: _____ Date: _____