

**The impact of psychopathy traits on facial expression processing  
among individuals with a psychotic disorder: associations with  
symptomatology, emotion regulation and cognitive functioning**

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## **Declaration**

### STATEMENT OF ORIGINALITY

I hereby certify that the work embodied in this thesis is my own work, conducted under normal supervision. The thesis contains no material which has been accepted, or is being examined, for the award of any other degree or diploma in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. I give consent to the final version of my thesis being made available worldwide when deposited in the University's Digital Repository, subject to the provisions of the Copyright Act 1968 and any approved embargo.

Ketrina Anne Sly

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## **Abstract**

**Scope:** Individuals with psychosis exhibit marked deficits in facial expression perception and processing. Similar impairments are observed among people with a history of psychopathy. While both groups display atypical scanning patterns and associated poorer social functioning, the relative contribution of psychosis and psychopathy traits to these deficits remains unclear.

**Purpose:** This thesis aims to extend previous facial emotion processing research in psychosis by utilising visual scanning eye-tracking tasks to examine the impact of coexisting psychopathy traits, as well as considering the contribution of symptomatology, emotion regulation and cognitive functioning.

**Methodology:** Sixty-three participants were recruited, including 37 diagnosed with psychosis (Schizophrenia or Schizoaffective disorder) and 24 healthy controls. The Psychopathy Checklist: Screening-Version (PCL: SV) was used to assess psychopathy traits, and to divide psychosis participants into low (N=18) and high psychopathy (N=19) groups. Three visual-cognitive eye-tracking tasks were utilised to examine emotion recognition, emotion induction, and face recognition and working memory.

**Results:** Among the psychosis group, relative to the control group, emotion recognition accuracy (*Study 1*) was significantly poorer, and while some atypical visual scan-paths were apparent, visual scanning strategies were not associated with recognition accuracy. Emotion induction (*Study 2*) was not impaired, although recall accuracy for faces (*Study 3*) was reduced and significant neuropsychological deficits apparent, although, again no accompanying visual scanning deficits were observed. Overall performance accuracy was associated with better immediate memory and higher premorbid IQ. Coexisting elevated psychopathy traits in psychosis were not associated

with increased socio-cognitive performance deficits in either emotion or face recognition or mood induction.

**Conclusions:** An aggregate index based on key task performance indices revealed a trend level difference for psychosis status, reflective of poorer social-cognition performance across tasks. These findings indicate combined treatments targeting both social cognitive and neurocognitive impairments may provide optimal clinical benefit in improving emotion-processing strategies. Diagnostic complexity in psychosis, involving elevated psychopathy traits should not be a barrier to remediation aimed at improving social-cognition, given the observed lack of association between psychopathy traits and impaired visual-cognitive strategies across the three facial emotion-processing tasks.

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## **Chapter 1 – Introduction: Facial Expression Processing in Psychosis and Psychopathy**

### **1.1 Introduction**

People are social beings, generally striving for a life connected in fulfilling and meaningful social interactions with others. In part, this involves recognising emotions, actions and the intentions of others (Fusar-Poli et al., 2009), and being able to process affective content accurately to infer mental and emotional state (Said, Haxby, & Todorov, 2011). The face is one of the richest sources of information for making such inferences (Said et al., 2011), with much of this information gleaned from facial expressions (Fusar-Poli et al., 2009). Faces provide us with socially relevant information, and correctly, rapidly and continuously identifying or discriminating emotional expressions is essential in everyday interpersonal interactions (De Sanctis et al., 2013; Lee, Gosslin, Wynn, & Green, 2010). A failure to perceive or experience the range and depth of emotions expressed by others has serious implications for interpersonal communication (Decety, Skelly, Yoder, & Kiehl, 2014), particularly if individuals are unable to modify their behaviour according to the social environment (Blair, 2003). Essentially, these non-verbal affective cues are vital for normal social functioning and establishing and maintaining stable interpersonal relationships (Schönenberg et al., 2014). Our everyday social interactions also have broader implications in terms of our general health and wellbeing, with social connectedness a strong predictor of subsequent mental health (Saeri, Cruwys, Barlow, Stronge, & Sibley, 2017).

Individuals with schizophrenia are known to exhibit marked deficits in facial expression perception (Hooker & Park, 2002; Kee, Green, Mintz, & Brekke, 2003;

Kohler, Bilker, Hagendoorn, Gur, & Gur, 2000; Kohler & Brennan, 2004; Kohler, Walker, Martin, Healey, & Moberg, 2010). These deficits are thought to underlie some of the difficulties observed for these individuals in social and interpersonal communication (Schneider et al., 2006). Psychosocial and occupational functioning, independent living ability, subjective wellbeing and quality of life have all been significantly associated with emotion perception deficits (Green et al., 2012; Hooker & Park, 2002; Horan et al., 2012; Kee et al., 2003; Kohler et al., 2010). People with psychopathy exhibit similar emotion perception difficulties, with evidence supporting deficits particularly in the recognition of negatively valenced emotions, such as fear or sadness (Dawel, O'Kearney, McKone, & Palermo, 2012; Fullam & Dolan, 2006b; Marsh & Blair, 2008). However, the relative contribution of psychopathy traits to emotion processing deficits in psychosis remains unclear.

Additional research to increase our understanding of emotion processing deficits is required to assist in driving the translation of research findings towards evidence-based clinical practice change, with the potential aim of improved social outcomes for this population. Psychotic disorders can be chronic and debilitating, and while psychotic symptoms may abate with antipsychotic medication, marked social cognitive impairments often remain. Findings from the second Australian national survey of psychosis (Survey of High Impact Psychosis – SHIP) revealed that, following illness onset, 90.4% of people with a psychotic disorder report deterioration in functioning; with 63.2% having an obvious to severe dysfunction in their capacity to socialise (Morgan et al., 2012). In contrast, prior to illness, normal levels of functioning were reported by 68.7% for work and study, and by 63.9% for social functioning (Morgan et al., 2012).

The last decade has seen developments in both broad based and targeted social cognitive interventions, with most demonstrating positive gains (see systematic review: Tan, Lee, & Lee, 2018). For example, promising results have been found in social-cognitive remediation studies aimed at drawing attention to salient features or improving sensitivity to emotional expressions among both schizophrenia (Combs, Chapman, Waguspack, Basso, & Penn, 2011; Hooker et al., 2013; Marsh, Lockett, Russell, Coltheart, & Green, 2012) and offender populations with high psychopathy traits (Schönenberg et al., 2014). More recently, advances in online social cognition interventions including “e-Motivational Training” for schizophrenia have been successful in improving emotion recognition ability (Maroño Souto et al., 2018). However, additional research is still required examining the underlying mechanisms and the translatability of findings, particularly in relation to comorbidity or diagnostic complexity in psychosis.

### *1.1.1 Summary of objectives*

The main objective of this thesis is to extend previous research on emotion processing deficits in psychosis:

- *Firstly*, to gain a greater understanding of emotion processing deficits in psychosis, by assessing the contribution of coexisting psychopathy traits with respect to their potential impacts on face processing deficits, utilising visual scanning techniques; and
- *Secondly*, to examine inter-relationships with symptomatology, emotion regulation, general social-cognitive and overall neurocognitive functioning.

Facial emotion processing will be assessed (utilising visual scanning tasks) in terms of emotion recognition, emotion induction, face recognition and working memory, amongst a group of outpatients with a psychotic disorder presenting to mental health services (with and without a history of offending behaviour), as well as healthy controls. Initial assessments with all participants will also include an examination of the degree of coexisting psychopathy traits. The primary objective is to explore the link between psychopathy traits and psychosis with respect to face and emotion processing impairments, using eye-tracking as an objective psychophysiological assessment method.

There is a vast amount of research literature on facial emotion processing, and associated visual scanning among individuals with psychosis. A discrete body of work has been undertaken in psychopathy largely examining deficits in facial emotion recognition, however there is a paucity of research on the associated visual processing, particularly in relation to visual scan-path performance with relatively little known about the differential elements. This introductory chapter provides a critical review of the relevant literature in relation to the spectrum and varied presentations of the disorders of interest, as well as the existing research undertaken in face perception and facial expression processing, in psychosis and psychopathy. A brief introductory precis, and the main objectives of undertaking this research have been outlined in this Section. The following Section 1.2 provides background information on face and emotion perception, theoretical models, and neuropsychological aspects of face and emotion processing, as well as visual processing during face and emotion perception among non-clinical populations. Section 1.3 provides background information on psychosis, specifically related to symptoms and functioning, face and emotion perception and

visual processing during face and emotion perception. Similarly for psychopathy, in Section 1.4 background on symptoms and functioning, face and emotion perception and the limited literature on visual processing during face and emotion perception is covered. Finally, Section 1.5 provides a summary and thesis overview. This chapter seeks to provide an empirical background to the investigations reported in subsequent chapters (Chapters 2 to 4), and for the synthesis and interpretations of the overall findings in the final chapter (Chapter 5).

## **1.2 Face and emotion perception, and visual processing in non-clinical populations**

Irrespective of psychiatric illness or psychopathy traits, there are a number of important aspects of emotion processing that warrant exploration. These include, current theories around facial emotion processing; innate and environmental (nature versus nurture) aspects of how individuals process displays of emotion; and the importance of faces and aspects of facial features, which may have graded importance in social cognition. In addition, there are potential differences across processing categories (i.e., happy, sad, fear, etc.) or depending on the intensity of emotion, as well as complexities of the visual system (i.e., visual scanning parameters) in processing temporal and spatial information. Although an extensive in-depth review of each of these fields clearly falls outside the scope of this dissertation, given the primary focus of this research is on facial emotion processing deficits in psychosis, and the potential impact of coexisting psychopathy traits, important theoretical underpinnings around face perception and models of emotion processing will be discussed.

### *1.2.1 Face and emotion perception*

Faces represent a special kind of stimuli. From birth an orienting bias towards faces is apparent, and faces are generally accepted as a close physical representation of a person (Haxby & Gobbini, 2007). Face perception, focusing on detecting or discriminating faces in the context of other stimuli and on the recognition of uniqueness or identity, occurs very rapidly, often within less than a few hundred milliseconds, however, this accounts for only a small proportion of the actual time people spend looking at faces during interpersonal interactions (Haxby & Gobbini, 2007). Face perception is a highly developed visual skill, involving rapidly accessing information essential in interacting effectively, and the parallel processes involved are numerous (Haxby & Gobbini, 2007). With respect to brain regions, in healthy individuals face viewing has been associated with robust activity in a collection of cortical and subcortical regions including: the fusiform gyrus, occipital cortex, and posterior superior temporal sulcus (pSTS) as the core face processing network; and inferior frontal gyrus (IFG), amygdala, orbitofrontal cortex (OFC), and anterior insula (aINS), as supporting regions (Decety et al., 2014; Haxby, Hoffman, & Gobbini, 2000).

The fusiform face area (FFA) has been identified by Haxby and Gobbini (2007) as a key node in neural systems for face perception. However, this region does not account for key processes such as the recognition of expression, emotional resonance, shared attention, spontaneous activation of a person's knowledge, and trait inferences (Haxby & Gobbini, 2007). For example, visual representations rely on extrastriate visual regions like the pSTS, and include nonvisual neural systems like the inferior parietal lobule and frontal operculum for mirroring actions, the amygdala and insula in the representation of emotion, the intraparietal sulcus and frontal eye fields in spatial

attention, and temporal parietal occipital junction (TPJ), anterior temporal cortex, and medial prefrontal cortex in theory of mind (Haxby & Gobbini, 2007). Thus, face processing represents a complex and dynamic integrative process within and between brain regions and limbic system and brain substrates that occur in a seemingly seamless fashion.

During social interactions the face provides a complex source of information (Yankouskaya, Humphreys, & Rotshtein, 2014b). A person's face conveys information not only related to identity but expresses emotion, and can often signal different blends of emotion at the same time (Adolphs, Tranel, Damasio, & Damasio, 1994). Facial expressions are crucial in emotional and social behaviour, and considered to represent innate and automatic behaviour patterns (Darwin, 1872: cited in Blair (2003)). Subtleties in the recognition of emotional expressions may also be partially determined by culture (Derntl et al., 2012). The capacity to recognise facial expressions is one of the most important abilities in social interaction, allowing inferences about another's state of mind to be made, and it facilitates communication (Nakamura, Maess, Knosche, & Friederici, 2014).

Research focusing on emotion perception and utilising social cues from faces (seen broadly as the field of social neuroscience) has gained worldwide momentum. Studies have determined that people can reliably discriminate at least six distinct expressions of emotion including happiness, surprise, fear, sadness, anger and disgust, relatively easily (Ekman, 1993; Posamentier & Abdi, 2003). Existing evidence suggests that information on identity and emotion are largely processed separately; however, the underlying brain mechanisms are complex and still not completely understood

(Nakamura et al., 2014). Research continues to focus on how a range of facial information is processed by the perceptual system, with studies independently examining each type or manipulating both in order to elucidate if identity and expression are indeed processed independently or interactively (see review: Yankouskaya et al., 2014b).

### *1.2.2 Theoretical models of face and emotion perception*

Initial theoretical models have endeavoured to explain the mechanisms underlying facial identity and expression perception. To date, proposed models have been described as falling broadly into three areas: 1) “*independent or separate and parallel processing*” of facial identity and emotional expression; 2) “*asymmetric processing*”, which posits that the processing of facial emotion depends on facial identity but not the reverse; and 3) “*integrated processing*” of facial identity and emotion (Yankouskaya et al., 2014b). Bruce and Young’s (1986) cognitive model of face processing provided a framework to explore face perception, comprising a set of interrelated yet independent modules, as an integrated system for processing different kinds of facial information. Seen as the first type of “*separate and parallel processing*” (Yankouskaya et al., 2014b), the model proposed distinct module-based processing pathways for facial identification, emotional expression and speech-related facial movements (Bruce & Young, 1986). Evidence supporting the claim that information on identity and emotion are largely processed separately included studies examining: patients with prosopagnosia (impairment in face recognition), whose facial expression recognition was intact (Ectoff, 1984; Tranel, Damasio, & Damasio, 1988); people with impaired facial expression recognition, whose facial identification ability was preserved (Adolphs et al., 1994; Bowers, Bauer, Coslett, & Heilman, 1985;

DeKosky, Heilman, Bowers, & Valenstein, 1980); and behavioural tasks, which reveal processing differences for facial identity and expression (Ellis, Young, & Flude, 1990; Young, McWeeny, Hay, & Ellis, 1986).

Founded on Bruce and Young's (1986) model, Haxby and colleagues (2000) later proposed a neural model with two major pathways, one related to processing invariant aspects of facial identification and the other related to changeable aspects of faces such as eye gaze, expression and lip movements (Haxby et al., 2000; Haxby, Hoffman, & Gobbini, 2002). Haxby et al. (2002) outlined a hierarchical organisation system of face recognition, with a core system consisting of occipitotemporal regions in extrastriate visual cortex (FFA) that mediate the visual analysis of faces, and an extended system of regions from neural systems for other cognitive functions that act with the core system to extract meaning from faces. In the extended system the amygdala plays a central role in processing socially relevant information from faces, such as affect (particularly when the information may signal a potential threat) (Haxby et al., 2002).

Haxby et al. (2000) argued that invariant aspects of faces influence how changeable aspects are processed, although information on changeable aspects do not influence invariant face properties; which can be seen as consistent with "*asymmetric processing*", where emotion processing is dependent of facial identity coding (Yankouskaya et al., 2014b). Research seen as supporting asymmetric dependency included studies revealing that it is possible to ignore emotion and speech while attending and responding to identity, but not visa-versa (Schweinberger, Burton, & Kelly, 1999; Schweinberger & Soukup, 1998). Functional neuroimaging studies

confirmed that the ventral occipitotemporal region (FFA) plays an essential role in the recognition of invariant aspects of faces (Haxby et al., 2002); however findings from studies examining recognition of facial expressions implicate several brain regions as having important functions, revealing a more complex picture (Nakamura et al., 2014).

Models of “*integrated processing*” represent an alternate view to dissociated (separate) parallel processing, and support a single multidimensional mechanism for processing identity and expression (Calder & Young, 2005; Yankouskaya et al., 2014b). Evidence suggesting mechanisms for processing identity and emotions are interconnected include: studies finding performance differences for familiar faces, whereby expressions are more easily derived (Ganel, Goshen-Gottstein, & Ganel, 2004); redundancy related gains in processing capacity (Yankouskaya, Booth, & Humphreys, 2012; Yankouskaya, Rotshtein, & Humphreys, 2014a); and studies providing support for dynamic face processing, whereby the processing of facial identity and emotion interact (are not independent) and are shaped by task and experience (Yankouskaya et al., 2014a; Yankouskaya et al., 2014b). Mixed results have been reported in the literature examining unfamiliar faces, however, a recent study found facial expressiveness variability can lead to identify confusion in the recognition of unfamiliar faces, supporting the notion that expressions are not disregarded when processing identity, as well as theoretical approaches in which emotion are readily incorporated when processing facial identity (Redfern & Benton, 2017). While the exact mechanism remains unclear about whether identity and emotion is processed independently or in an integrated manner, additional computational, neuropsychological and neuroimaging research continues.

### *1.2.3 Neuropsychological aspects of face and emotion perception*

Processing facial emotional expressions is known to activate several distinct brain regions. The role of the amygdala in facial expression recognition has been well documented, and appears to be a necessary component of neural systems, particularly its role in the perception of fear, in both clinical (Adolphs et al., 1994; Calder, 1996) and neuroimaging studies (Breiter et al., 1996; Morris et al., 1998; Morris et al., 1996). The amygdala has also been implicated in neural processing of sad facial expressions (Blair, Morris, Frith, Perrett, & Dolan, 1999). Enhanced activity in the orbitofrontal cortex (OFC) has been associated with processing angry facial expressions, and both sad and angry expression activate the anterior cingulate and right temporal pole (Blair et al., 1999). Activation of the insula and basal ganglia in response to the expression of disgust has repeatedly been found (Phillips et al., 1997; Sprengelmeyer, Rausch, Eysel, & Przuntek, 1998).

Lesion studies have revealed involvement of the right primary and secondary somatosensory cortices in judging facial expression of emotion (Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000; Adolphs, Damasio, Tranel, & Damasio, 1996). The right inferior frontal (Leslie, Johnson-Frey, & Grafton, 2004; Nakamura et al., 1999) and OFC (Hornak, Rolls, & Wade, 1996; Vuilleumier, Armony, Driver, & Dolan, 2001) have been reported to contribute to the processing of facial displays of emotion. The superior temporal sulcus (STS) is also considered important, with neurons in this region tuned to respond to social signals (or socially relevant stimuli as part of the social perception system) including facial expression, gestures, eye gaze, or movements of the eyes, mouth, lips, hands and body (Allison, Puce, & McCarthy, 2000; Hoffman & Haxby, 2000).

It has been suggested that early perceptual processing of faces draws on cortices in the occipital and temporal lobes to construct detailed representations from the configuration of facial features (Adolphs, 2002). Recognition requires a set of structures, including the amygdala and OFC that link the perceptual representation of the face to the generation of knowledge about the emotion signalled (Adolphs, 2002). A meta-analysis of functional Magnetic Resonance Imaging (fMRI) studies concluded that the processing of emotion from facial expressions draws on diverse psychological processes implemented in a large array of neural structures, while the exact functional interplay between these areas is unclear (Fusar-Poli et al., 2009). Emotional gesture perception, reactions to the face, eye gestures and social attention have also been linked to the TPJ (Wible, 2012). Due to the number of brain regions implicated as having important roles, a concise picture of the underlying brain mechanisms involved in the recognition of facial expression remains elusive (Nakamura et al., 2014).

In addition, differential neural responses to emotions have been detected in the limbic system, with happy and fearful faces activating the amygdala bilaterally, sad faces show laterality, and angry and disgusted faces have no effect on this region. However, conflicting theories have been proposed around hemispheric lateralisation in emotion processing (Nakamura et al., 2014). Studies supporting the right hemisphere hypothesis propose a right predominance, regardless of emotional valence (i.e., positive or negative) (Adolphs et al., 2000; Nakamura et al., 1999). On the other hand, studies supporting the valence hypothesis suggest that hemispheric role is dependent on emotion type, with the left hemisphere dominant for positive and the right hemisphere dominant for negative emotions (Canli, Desmond, Zhao, Glover, & Gabrieli, 1998; Graham & Cabeza, 2001). Recent electrophysiological findings also suggest that the

right and left hemispheres may play different roles in the recognition of facial expression, depending on the cognitive context (Nakamura et al., 2014). Specifically that reading facial expressions activates the parieto-frontal network in the right hemisphere, while the emotional value of the facial expression (e.g., happy) mainly causes activity in the inferior and medial temporal regions of the left hemisphere (Nakamura et al., 2014).

Research among healthy adults has revealed some performance variations on behavioural tasks; for example, age (Isaacowitz et al., 2007; Orgeta, 2010), gender (Donges, Kersting, & Suslow, 2012), and IQ (Andric et al., 2015) have all been found to correlate significantly with facial emotion recognition performance. Healthy individuals with higher degrees of neuroticism have also been found to exhibit poorer recognition of happy facial expressions (Andric et al., 2015). Happiness is generally one of the easiest emotions to recognise even at low intensity levels, although a higher threshold for identifying happy facial expressions has been reported among patients with depression in both acute and remitted symptom phases (Kohler, Turner, Gur, & Gur, 2004). As can be ascertained from these findings, the identification of sub-group differences may have important implications for targeted early interventions and staging models among clinical groups (Andric et al., 2015).

#### *1.2.4 Visual processing during face and emotion perception*

While the face provides non-verbal cues about emotional expressions, some parts of the face are subject to a more detailed analysis during emotion processing (Hoffmann, Traue, Limbrecht-Ecklundt, Walter, & Kessler, 2013). Emotion processing refers to all aspects involved in the perception, understanding, regulation, and use of

emotions in social functioning (Bertone, Diaz-Granados, Vallejos, & Muniello, 2017). Emotion-specific differences (for basic emotions) have been found related to the importance of specific facial areas, for example, observing wide-open eyes in the recognition of surprise, or a wrinkled nose and lifted upper lip in the recognition of disgust (Hoffmann et al., 2013). Essentially, when categorising expressions of emotion, visual attention will typically be drawn towards salient facial features, such as the eyes and mouth (Eisenbarth, Alpers, Segre, Calogero, & Angrilli, 2008). Visual scanning technologies allow for an objective examination of the accompanying behavioural processing of emotional information. Likewise, visual scan-path performance provides one experimental approach for exploring social cognition during emotion processing tasks.

Visual scan-path measurements provide a marker of directed attention, basically a map tracking the direction and extent of a person's gaze, comprised of '*fixations*' which are consecutive gaze positions or points of attention, and '*saccades*' which are voluntary eye movements between fixations (Phillips, Senior, & David, 2000). Eye tracking technologies provide a continuous record of processing and an effective tool for collecting relevant information for an individual, such as patterns of visual exploration of the face during emotion recognition tasks (Bortolon, Capdevielle, Salesse, & Raffard, 2016). Eye-tracking technologies have also been used to explore the underlying neurocognitive mechanisms during socio-emotional perception and processing in order to gain a greater understanding of any impairments among clinical populations (Elbogen, Dennis, & Johnson, 2016).

The scan-path strategies of healthy individuals have been observed in response to a large range of visual stimuli, generally in comparison to clinical populations (i.e. schizophrenia) across a variety of processing tasks (see review: Beedie, Benson, & St Clair, 2011), including a number of studies exploring face or emotion processing (Loughland, Williams, & Gordon, 2002b; Loughland, Williams, & Harris, 2004a; Phillips & David, 1994; Williams, Senior, David, Loughland, & Gordon, 2001). Visual scanning strategies of healthy individuals have generally been described as more extensive or holistic, with attention being appropriately paid to perceptual and semantically relevant areas (Beedie et al., 2011). For example, in the perception of facial expressions as illustrated by Loughland et al. (2002b, Fig.2), visual eye-movement patterns characterised by a triangular scan-path strategy, indicating directional attention between the eyes and mouth, with increased fixations to salient features (i.e., eyes, mouth). In addition, the processing of faces and associated affect among healthy individuals has been suggested as being undertaken in a relatively automatic manner, with accuracy and scan-paths remaining independent (Loughland et al., 2002b).

The principal focus of this dissertation is on facial emotion processing in psychosis, and the potential impact of psychopathy traits. Consequently, some background information about these disorders is provided below (e.g., prevalence, aetiology, symptomatology, comorbidity, functioning), followed by consideration of relevant facial emotion processing and visual scanning research associated with these disorders. Throughout this dissertation, the term psychosis has been used interchangeably in referring collectively to psychotic disorders, such as schizophrenia

and schizoaffective disorders, encompassing their similarities whilst acknowledging apparent clinical differences.

### **1.3 Face and emotion perception, and visual processing in psychosis**

#### *1.3.1 Psychosis: background, symptoms and functioning*

##### *1.3.1.1 Prevalence and aetiology*

Schizophrenia is a low prevalence disorder, with worldwide estimates of around 4.6 per 1,000 population and 4.0 per 1,000 for lifetime prevalence (See systematic review: Saha, Chant, Welham, & McGrath, 2005). The estimated median incidence per year is 15.2 per 100,000 persons (with a 5.6-fold variance across regions) worldwide diagnosed with the illness, with increased rates among males, urban sites and migrants reported (See reviews: McGrath, Saha, Chant, & Welham, 2008; McGrath et al., 2004). Similar estimates for Australia reveal a treated prevalence of psychotic disorders in public mental health services of 3.1 per 1,000 population using 1-month data, and 4.5 per 1000 using 12-month data (Morgan et al., 2012). In addition, a median lifetime morbidity risk of 7.2 per 1,000 persons has previously been reported, together with a two- to threefold increased risk of dying compared to a median standardised mortality ratio of 2.6 for all-causes (McGrath et al., 2008). A recent meta-analysis estimated that the relative risk of dying for all-causes was up to 2.5 times higher for those with a psychotic disorder (Walker, McGee, & Druss, 2015), confirming a persistent and widening gap in life expectancy for individuals with psychosis compared to the general population (Chesney, Goodwin, & Fazel, 2014; Lawrence, Hancock, & Kisely, 2013). Indeed, around a 10-20 year life expectancy gap has been found in a number of countries, including: Australia (Lawrence et al., 2013); the UK (Chang et al., 2011); US (Druss, Zhao, Von Esenwein, Morrato, & Marcus, 2011); Denmark, Finland, and

Sweden (Laursen et al., 2013). Although suicide is a cause of some excess deaths, increased mortality is mainly due to physical health conditions such as cardiovascular disease, respiratory disease and cancer (Lawrence et al., 2013).

While individuals vary in terms of symptoms exhibited, those diagnosed with a psychotic disorder typically suffer debilitating positive symptoms, such as delusions, hallucinations, disorganization of thought and behaviour, as well as negative symptoms, such as emotional withdrawal and an inability to focus on day-to-day tasks. Early descriptions of the disorder first emerged in the late 1800s; with the German physician Emile Kraepelin in 1887 initially using the term "dementia praecox" (a particular form of early onset dementia) to describe psychotic symptoms of disordered thought and intellectual decline, which we now associate with schizophrenia (Kraepelin, 1919). The Swiss psychiatrist Eugen Bleuler in 1911 later introduced the term "schizophrenia" and was the first to describe "positive" and "negative" symptoms (Bleuler, 1911/1950), Bleuler's characterisation differed in that schizophrenia did not always commence in adolescence or progress to dementia in the classic sense of memory distortion and loss (McGlashan, 2011). Both Bleuler (1950) and Kraepelin (1919) observed deficits in attention, memory, associative thinking, reasoning and language (Seidman & Mirsky, 2017) and described categories based on prominent symptoms to classify types of schizophrenia. Current diagnostic systems, such as the Diagnostic and Statistical Manual of Mental Disorders, Fifth edition (DSM-5) (American Psychiatric Association, 2013) classify five types: disorganized, catatonic, paranoid, residual, and undifferentiated; the first three of which were originally proposed by Kraepelin (1919). Bleuler's characterisation included four A's: "Association" with a focus on disordered language; "Affectivity" or emotional deterioration; "Ambivalence"; and "Autism", all

indicative of different aspects of social functioning and relevant to the continued study of cognitive and social-communication dysfunction today (McGlashan, 2011; Niznikiewicz, Kubicki, Mulert, & Condray, 2013).

Since that time, and over the last 50 years, there have been substantial gains in knowledge around the varied clinical presentation, structural and functional brain region differences, genetic and environmental risk factors, gene-environment interactions or epigenetics, as well as treatment advances in antipsychotic medications, and adjunctive psychosocial and biomedical treatments (See reviews: Brown, 2011; Matheson, Shepherd, & Carr, 2014; Seidman & Mirsky, 2017). Although, for many individuals diagnosed with the illness, lifelong impairments in day-to-day cognitive, social and occupational functioning remain (Shenton, Whitford, & Kubicki, 2010; van Os & Kapur, 2009). Altered brain development has been identified, shared partly with other developmental and affective disorders (van Os & Kapur, 2009). Poor cognitive functioning (also to a lesser degree in first-degree relatives), subtle but diverse structural brain changes, altered electrophysiological measures (P50, P300, N400 & mismatch-negativity), neurological soft signs (also in first-degree relatives) and sensory changes (reduced olfactory and pain sensitivity) have all been confirmed (Matheson et al., 2014). Epidemiological studies provide evidence for developmental factors, with large effect sizes found for pregnancy and birth complications, exposure to infections, developmental motor delays, lower premorbid IQ, childhood viral infections, as well as adversity, migrant, urbanity and premorbid cannabis use as risk factors (Matheson et al., 2014).

Accumulated epidemiological evidence suggests that environmental factors play a significant role in both cause and prevention of schizophrenia, with a diversity of factors likely to interact along with genetic factors in complex ways, and with other mechanisms (Brown, 2011; Matheson et al., 2014). Exposure to environmental risk factors as early as peri-conception, pregnancy, childhood, and as late as adolescence and adulthood have been identified as playing an important role in susceptibility, and rather than an inherited schizophrenia illness, a gene–environment interaction is likely (Brown, 2011). Overall epidemiological findings suggest that the onset and course of the disorder is shaped by a combination of intrinsic (genetic) and extrinsic (environmental) factors, somewhere between conception and adolescence (Matheson et al., 2014). The disorder involves widespread neural dysfunction, with altered inflammatory and or immunological processes suggested as having a causal role, likely infective in origin (Matheson et al., 2014).

In broad general terms, a “*stress-vulnerability model*” proposes that some individuals have a greater predisposition, due to genes, trauma or environmental factors, with psychosis triggered by a stressful event, usually in late adolescence or early adulthood. Such long-standing aetiological explanations (proposing, an interaction between external stressors and pre-existing vulnerabilities) have been, coupled with recent genetic, epidemiological and imaging studies positing mechanisms involving the immune system, in which microglial cells may play an important role in altered synaptic pruning (Howes & McCutcheon, 2017). For example, the “*inflammation and neural diathesis-stress*” model proposes that environmental risk factors impact on the immune system, where over activation of microglial cells may result in gray matter loss in prefrontal cortex and hippocampus regions, leading to negative and cognitive

symptoms, and dopaminergic dysregulation of subcortical structures (Howes & McCutcheon, 2017). Neuroimaging research has confirmed, as speculated by Kraepelin in his early descriptions of “*dementia praecox*” as a result of cerebral cortex damage, that schizophrenia is a disorder of brain function, with some progress towards understanding the mechanisms involved (Barch & Ceaser, 2012; Niznikiewicz et al., 2013), and neurocognitive dysfunction identified at all phases of the illness (including, prodromal, high risk, and first episode psychosis) (Seidman & Mirsky, 2017).

Antipsychotic medications targeting changes in dopamine neurotransmission have been found to be effective treatments for delusions and hallucinations, but less so for cognitive and motivational impairments (with limited evidence for reversibility), with memory, executive functioning, affect and social communication all altered in schizophrenia, (Seidman & Mirsky, 2017; Shenton et al., 2010). Recent research also supports illness categories of treatment-resistant and treatment-responsive schizophrenia, as distinct illness subtypes, with clozapine found to be a more effective for treatment-resistant schizophrenia (Gillespie, Samanaite, Mill, Egerton, & MacCabe, 2017a). The importance of assessing neurocognition has been identified due to the strong association between neurocognitive deficits and social and role functioning (Seidman & Mirsky, 2017). Promising results from cognitive remediation studies also highlight the importance of focusing on cognitive interventions and potential early intervention for at risk populations (Hooker et al., 2014; Seidman & Mirsky, 2017).

Although a comprehensive theory of schizophrenia remains elusive, psychosis research continues to make progress, but there are no definitive answers. Both neurodevelopmental and neurodegenerative factors have been supported in the aetiology

of schizophrenia (Buoli, Serati, Caldiroli, Cremaschi, & Altamura, 2017).

*“Neurodevelopmental models”* propose that a disruption of brain development in early life is responsible for the later onset of symptoms, while *“Neurodegenerative models”* highlight the negative effects of illness (Altamura, Buoli, & Pozzoli, 2014; Buoli et al., 2017). Current research focuses on both exploring how specific factors contribute to altered or impaired development and on neurodegeneration or physiological aging processes (Buoli et al., 2017). Given the current evidence that neurobiological phenotypes in schizophrenia overlap with other neuropsychiatric disorders including bipolar disorder, research has tended to move towards environmental determinants of structural and functional brain phenotypes (Brown, 2011; Buoli et al., 2017).

Despite increasing knowledge, the debate over the nature of the disorder continues, with the main perspectives being neurobiological and psychological (Perez-Alvarez, Garcia-Montes, Vallina-Fernandez, & Perona-Garcelan, 2016). Schizophrenia has been conceptualised by some as a developmental neurocognitive disorder, with Seidman and Mirsky (2017) describing it as a *“dynamically evolving developmental neuropsychiatric disorder best understood from a psychobiological perspective”*, where *“gene-environment interaction, stress, and risk and protective factors all play a role in the onset, maintenance and recovery from illness”*. An alternate view sees schizophrenia as a disorder of the person rather than the brain. For example, Perez-Alvarez et al. (2016) conceptualise alteration of the self, its modern origin, juvenile onset, improved prognosis in developing countries, high incidence among immigrants, epigenetic findings, and recovery of one's sense of self through psychotherapy, as all supportive of the notion that schizophrenia is a disorder of the person. Importantly, psychosis research continues on multiple fronts, with the aim of obtaining a better understanding of not

only aetiology, risk factors, prognosis, illness management, and associated neurodegeneration, but potential early interventions aimed at optimising recovery and rehabilitation of persons affected by psychotic illnesses.

### *1.3.1.2 Symptomatology*

In terms of current diagnostic profiles, psychotic disorders fall under the category of schizophrenia spectrum and other psychotic disorders in DSM-5 (American Psychiatric Association, 2013) and within schizophrenia, schizotypal and delusional disorders in the 10<sup>th</sup> edition of the International Statistical Classification of Diseases and related health problems (ICD-10) (World Health Organization, 2011). In DSM-5, schizophrenia is defined by abnormalities in one or more of five domains: delusions, hallucinations, disorganised thinking (speech), grossly disorganised or abnormal motor behaviour, and negative symptoms (American Psychiatric Association, 2013). . The definition of schizophrenia disorders in ICD-10 identifies fundamental distortions of thinking and perception, and inappropriate or blunted affect. Psychopathological phenomena include: thought echo, insertion, withdrawal or broadcasting, delusional perception and delusions of control, influence or passivity, hallucinatory voices commenting or discussing in the third person, thought disorders and negative symptoms (World Health Organization, 2011).

Delusions: defined in DSM-5 as fixed beliefs that are resistant to change, these can fall into a number of themes such as persecutory, referential, somatic, religious or grandiose. Persecutory delusions are among the most common, involving the fixed belief that a particular individual, organisation, or group will cause harm or harass. Referential delusions are also quite common, involving beliefs around gestures,

comments or environmental cues, directed towards oneself. Grandiose delusions refer to an individual holding the belief that they have exceptional abilities, wealth or fame. Erotomaniac delusions involve a false belief that another person is in love with them. Nihilistic delusions involve the belief that a major catastrophe will take place, while somatic delusions relate to a preoccupation with health or organ function. Delusions are deemed bizarre if they are clearly implausible, such as the belief that one's thoughts have been removed by an outside force (thought withdrawal), or that alien thoughts have been put into one's mind (thought insertion), or that one's body or actions are manipulated by an outside force (delusions of control) (American Psychiatric Association, 2013).

*Hallucinations:* as defined in DSM-5, these are perceptual experiences occurring in any sensory modality without external stimulus, with the full impact of normal perceptions, that are vivid and clear and not under voluntary control. Auditory hallucinations are the most common in schizophrenia and related disorders, experienced as familiar or unfamiliar voices, that are distinct from the individual's own thoughts. (American Psychiatric Association, 2013).

*Disorganised thoughts and behaviour:* Disorganized thinking or formal thought disorder, usually inferred from speech, may involve switching from topic to topic (derailment) or answers to questions being completely unrelated (tangentiality). Speech may be incomprehensible but is rarely severely disorganised (i.e., receptive aphasia), while mild disorganised speech is common, impairing effective communication. Less severe disorganised thinking and speech can occur during prodromal and residual periods. Grossly disorganised or abnormal motor behaviour can range from silliness to

unpredictable agitation, including catatonia, leading to difficulties in day-to-day activities. Catatonic behaviour can range from negativism (involving resisting instructions) to rigid, inappropriate or bizarre posture; or mutism or stupor involving a complete lack of verbal and motor responses. Purposeless excessive motor activity (catatonic excitement) and other features can include repeated stereotyped movements, staring, grimacing, mutism and echoing of speech (American Psychiatric Association, 2013).

Negative symptoms: prominent and enduring in schizophrenia, these are often associated with substantial morbidity. They are represented by a deficit or loss in functioning in two main areas: *diminished emotional expression* of flattened affect and alogia (i.e., facial expression, eye contact, intonation and poverty of speech); and *avolition*, including anhedonia and asociality (i.e., inability to initiate/persist in goal directed activity, diminished capacity to experience pleasure and social withdrawal) (American Psychiatric Association, 2013). A meta-analysis examining treatments for negative symptoms revealed that while symptom reduction is apparent (i.e., using scales such as PANSS, SANS and BPRS), most treatments do not provide clinically meaningful improvement (Fusar-Poli et al., 2014). This suggests that currently there are no effective treatments for negative symptoms, which together with cognitive impairment represent one of the most disabling features of schizophrenia, and a major unmet clinical need warranting new and targeted treatments (Fusar-Poli et al., 2014).

In diagnosing psychotic disorders, conditions are also considered that don't meet full criteria, are limited to one domain (i.e., delusional disorder or catatonia), time-limited (i.e., brief psychotic disorder-less than 1 month, or schizophreniform disorder-

less than 6 months) or induced by another condition that may give rise to psychosis (i.e., substance/medication or medical condition). In diagnosing schizophrenia symptoms are required to last for at least 6 months, including 1 month of active-phase symptoms. In schizoaffective disorder, a mood episode and active phase symptoms co-occur, preceded or followed by at least 2 weeks of delusions or hallucinations without prominent mood symptoms. Psychotic disorder may also be substance or medication induced, with psychotic symptoms ceasing after the agent is removed. Schizotypal personality disorder (PD) is also considered within the schizophrenia spectrum of disorders, although it fits within PDs. It is characterised by a pervasive pattern of social and interpersonal deficits, including a reduced capacity for close relationships, cognitive and perceptual distortions, and eccentricities of behaviour. Usually apparent in early adulthood, but in some cases in childhood and adolescence, abnormal beliefs, thinking and perception are below the threshold for a psychotic disorder (American Psychiatric Association, 2013). The diagnostic profiles outlined here highlight not only the complex and varied clinical presentations but also inextricably the potential for associated trauma, stigmatisation and victimisation among individuals diagnosed with a psychotic illness.

### *1.3.1.3 Comorbidity*

Adding to the diagnostic and treatment complexity, comorbid conditions commonly occur with psychotic disorders. In Australia, increasingly high rates of comorbid substance use have been reported, including a lifetime history of alcohol abuse or dependence (50.4%), and lifetime history of illicit drug abuse or dependence (54.5%) (Morgan et al., 2012). Poor physical health is often an added burden; for example, the SHIP study reported elevated rates for chronic pain (31.8%),

heart/circulatory conditions (26.8%), diabetes (20.5%), asthma (30.1%), arthritis (20.8%), respiratory problems (18.0%), hepatitis (11.2%), eating disorders (8.0%), and epilepsy (7.3%) (Morgan et al., 2012). A range of metabolic and cardiovascular risk factors related to lifestyle were also reported, including high rates of smoking (66.1%), obesity (45.1%), a lack of physical activity (33.5%) and poor nutrition (71.1%), antipsychotic medication use associated weight gain (37.5%), and familial risk factors for diabetes, cardiovascular disease and related conditions (i.e., 38.8% hypertension; 35.0% diabetes; 34.9% heart disease) (Morgan et al., 2012).

Within the US prison system, a high prevalence of serious mental illness has been reported, particularly psychotic, bipolar and major depressive disorders with psychotic features (Torrey et al., 2014). It is estimated that US jails hold as high as 10 times the number of people with mental illness as state hospitals, with around 356,000 of the 2.1 million inmates having a serious mental illness compared to 35,000 people in state psychiatric hospitals (McCarthy, 2014; Torrey et al., 2014). In addition, those who remain in psychiatric hospitals under court order are often later “trans-institutionalized” from hospital to prison, often without appropriate treatment, at risk of physical and sexual abuse and subject to harsh detrimental conditions. This report concluded that functional mental health treatment systems are needed, so that mentally ill persons do not end up in the prison system, including: eliminating legal barriers preventing treatment prior to acts being committing that lead to arrest; diversion programs to obtain treatment; and appropriate treatments in prison and after release back into the community (McCarthy, 2014; Torrey et al., 2014).

A recent study, exploring challenges faced in treating offenders with schizophrenia, noted that the majority have additional problems compared to non-offender groups with psychosis, such as neurocognitive impairment, medication side effects, persistent positive symptoms, a history of substance abuse, conduct disorder, comorbid antisocial personality disorder, psychopathy, and stressful life experiences or adversity (Lau, 2017). Furthermore, people with mental health issues are more often victims than offenders (Teasdale, Daigle, & Ballard, 2014). Consequently, those with offending behaviours potentially represent a sub-group requiring special attention in terms of treatment and research. For example, interviews conducted for Australia's second national psychosis survey (SHIP), revealed that only 11.3% of people with a psychotic disorder reported having been arrested or charged with an offence, while 38.6% reported victimisation (i.e., break in, assault), with 16.4% being actual victims of violence (i.e., assault); although this sample did not include people with psychosis in the prison system (Morgan et al., 2012).

#### *1.3.1.4 Cognitive and social functioning*

Cognition involves the acquisition, processing, storage and use of information, encompassing a variety of abilities. There has been substantial research in the area of cognition in schizophrenia, with core deficits identified, including learning and memory impairments (Rajji, Ismail, & Mulsant, 2009; Salavati et al., 2015). Severity has previously been related to age of illness onset, with more severe cognitive deficits associated with youth-onset, in comparison to relatively preserved functioning seen in late-onset schizophrenia (Rajji et al., 2009). Cognitive functioning has been found to be a key determinant of quality of life, more so than severity of symptoms (i.e., hallucination or delusions), with a clear relationship between neurocognitive

functioning and patient outcomes (i.e., recovery of everyday and work functioning) in schizophrenia (Nuechterlein et al., 2011; Seidman & Mirsky, 2017). Cognitive factors broadly fall into two areas, non-social “*neurocognition*” and “*social cognition*”. Neurocognitive factors predictive of work outcome include working memory; attention and early perceptual processing; verbal memory and processing speed (Nuechterlein et al., 2011). Longer-term follow-up studies indicate these neurocognitive impairments are stable rather than progressive (Bonner-Jackson, Grossman, Harrow, & Rosen, 2010; Rund et al., 2016).

Social cognition on the other hand, refers to the mental operations or processes involved in social interactions, such as perceiving, interpreting, and generating responses (Green et al., 2008). Social cognition in psychosis is described as a multidimensional construct, distinct from neurocognition and social skills but also overlapping (Mancuso, Horan, Kern, & Green, 2011). Impairments in emotion processing, theory of mind and social relationship perception are prominent. They also exhibit longitudinal stability with no evidence of improvement or progression across illness phases (prodromal risk, first episode and chronic psychosis), and they are predictive of clinical and functional outcomes (Green et al., 2012; Horan et al., 2012). The ability to process emotion has long been established as a key indicator of social and occupational functioning, and independent living ability in schizophrenia (Hooker & Park, 2002; Kee et al., 2003; Penn et al., 2000a; Weiss et al., 2006), and more recently as a predictor of functional outcomes in first episode psychosis (Horan et al., 2012).

In schizophrenia, significant deficits are exhibited across a wide range of cognitive domains (Harvey & Rosenthal, 2018), which adds to the complexity in

understanding the mechanisms involved (Barch & Ceaser, 2012). Common mechanisms are believed to underlie some deficits, such as an impaired proactive control, which is thought to impact multiple domains (Barch & Ceaser, 2012). The term “*cold cognition*” has been used to describe dysfunction in attention, memory systems, language and perceptual mechanisms (Barch & Ceaser, 2012; Niznikiewicz et al., 2013), while “*hot cognition*” includes abnormalities in emotion and affect processing (Chung & Barch, 2011; Mathews & Barch, 2010; Niznikiewicz et al., 2013). Social cognition draws on both processes, being the ability to express attitudes and intentions and to predict and interpret those in others, involving: recognising social cues, such as emotions from a person’s face, tone of voice or posture; theory of mind; being able to express empathy; and making decisions in social situations (Niznikiewicz et al., 2013). In broad terms, schizophrenia related impairments in social cognition are negatively associated with patient outcomes (Hoertnagl & Hofer, 2014), including poorer community functioning (McCleery et al., 2016).

Given the focus of this dissertation, emphasis here will predominately be on aspects of neurocognitive and social cognitive functioning related to processing facial displays of emotion and face perception more broadly. The majority of individuals with schizophrenia exhibit neurocognitive impairments, ranging from mild to substantial deficits that are apparent across all illness phases, including prodromal and first episode psychosis (Seidman & Mirsky, 2017). Individuals with improved aspects of neurocognition, such as in executive function, working memory, attention, and verbal learning, are more likely to obtain better vocational and quality of life outcomes (Green & Mandal, 2002; Shayegan & Stahl, 2005). Correlations between impaired face processing and attention, working memory, memory-learning, abstraction-flexibility

and language abilities have previously been reported (Addington & Addington, 1998; Chen, Norton, McBain, Ongur, & Heckers, 2009; Kohler et al., 2000; Sachs, Steger-Wuchse, Kryspin-Exner, Gur, & Katschnig, 2004; Schneider, Gur, Gur, & Shtasel, 1995).

Impairments in at least four aspects of social cognition (including emotion processing, theory of mind, social relationship perception, and attributional style/bias) remain relatively stable across illness phase, representing a useful vulnerability indicator that is potentially predictive of associated functional outcomes, but may also provide a viable early intervention target (Comparelli et al., 2013; Green et al., 2012; Horan et al., 2012; Pinkham et al., 2014). However, findings around the stability of social cognitive impairments are mixed, with some studies suggesting a progressive impairment. Increased impairment in social and emotion perception has been associated with acute inpatient status, while greater impairment in emotion processing has been linked to illness duration (See meta-analysis: Savla, Vella, Armstrong, Penn, & Twamley, 2013). On the other hand, a meta-analysis of facial emotion and vocal prosody identification, and social perception found illness duration did not moderate task performance (Kohler et al., 2010).

A recent meta-analysis focusing on individuals at ultra-high risk for psychosis, further highlights the existence of consistent impairments in social cognition, with specific impairments in facial affect recognition and theory of mind predictive of transition to psychosis (van Donkersgoed, Wunderink, Nieboer, Aleman, & Pijnenborg, 2015). Given the observed marked impairments in social cognition (which are present in prodromal, first episode and chronic psychosis populations), and the potential as a

vulnerability marker in schizophrenia, recent research has sought to confirm whether these deficits are indeed stable or worsen as a function of illness chronicity (McCleery et al., 2016). In their 5-year follow up study of social perception and emotion processing, McCleery et al. (2016) found support for a trait-like stability in these selected areas of social cognition in schizophrenia. This confirmed previous findings over 12 months (Green et al., 2012; Horan et al., 2012), with a moderately large association detected between social cognition and community functioning. However, baseline social cognition was not predictive of community functioning at the 5-year follow-up, leading the authors to suggest short-term implications for functional outcomes rather than longer-term consequences (McCleery et al., 2016).

#### *1.3.1.5 Theoretical Models of Social Cognition.*

Taking into account the nature and extent of cognitive dysfunction, some researchers have conceptualised schizophrenia as a disorder of social communication, since the ability to effectively use language and deploy communicative devices to achieve successful social functioning is known to be poor (Niznikiewicz et al., 2013; Wible, 2012). Meaningful social communication is described as involving a range of sensory and cognitive processes and complex behaviours, both formal language (rules of phonology, grammar, syntax and semantics) and conveying emotional state and attitudes, as well as successfully interpreting these behaviours in others, dependent on effective perceptual and intact higher order processes (i.e., working memory, attention, and inhibition and response selection) (Niznikiewicz et al., 2013). Due to the complexity of behaviours involved, research has tended to focus on the discrete areas of language, executive function and perception within the framework of “*cold cognition*”, or the study of emotion, theory of mind, and agency within “*hot cognition*”

(Niznikiewicz et al., 2013). Although it has been acknowledged that research focusing on either language or social cognition in isolation may not capture the full extent of communicative difficulties experienced in schizophrenia (Niznikiewicz et al., 2013).

Within cognition research, a number of theoretical models have been proposed that examine the underlying mechanisms of successful social interactions. One such theory focuses on “*abnormal control mechanisms*”, suggesting a complex system of symbolic and multilayered semantics and syntax, affected by an impaired capacity to manipulate different elements, where abnormal language function is central (Arcuri et al., 2012; Boudewyn, Carter, & Swaab, 2012; Ketteler, Theodoridou, Ketteler, & Matthias, 2012). For example, impaired language comprehension in schizophrenia, particularly a difficulty processing meaningful discourse, has been suggested to result from deficits in pre-frontally mediated cognitive control mechanisms, involving integrating multiple levels of meaning and maintaining context (i.e., in high-level/demanding language processing) (Boudewyn et al., 2012).

Research conceptualising schizophrenia as a disorder of social cognition has instead focused on impaired social cognition processes, such as affect processing and negative facial affectivity bias (Fatouros-Bergman, Spang, Merten, Preisler, & Werbart, 2012; Pinheiro, McCarley, Thompson, Gonçalves, & Niznikiewicz, 2012). It is suggested that conversation involves more than just words and meanings, but relies on correctly interpreting facial expressions (emotion processing), tone of voice, social salience, agency (who is doing the talking), theory of mind (ability to anticipate another person’s actions and represent another point of view), and intention (Wible, 2012). The theory of “*abnormal social communication*” provides an alternate perspective on how

formal language may interface with social communicative devices (gestures, emotional facial expressions, tone of voice (prosody)) and faculties (theory of mind, sense of agency and intention), in which communication disorder is seen as the core clinical deficit (Niznikiewicz et al., 2013; Wible, 2012). Under this model, social communication abnormality is central, and language is considered within the context of the body-based gesture system, with greater communicative capabilities possible than within the language system alone (Wible, 2012).

The theory of “*abnormal control mechanisms*” suggests the importance of prefrontal brain regions; while the theory of “*abnormal social communication*” suggests the involvement of the TPJ (including the pSTS and surrounding regions) in the perception of dynamic social, emotional and attentional gestures (body, face and eyes) for self and others, as well as other social processes (speech and prosody) (Wible, 2012). It has been suggested that over-activation of this core system (TPJ, pSTS and surrounding regions), for moment-to moment social communication, may produce the symptoms and cognitive deficits seen in schizophrenia (Wible, 2012).

In terms of symptomatology, the multimodal representation of dynamic gestures, auditory, visual and tactile, match the predominant hallucinatory categories in schizophrenia, while many negative symptoms are characterised by deficits in responding in these domains (i.e., abnormal social responding and lack of social expression) (Wible, 2012). Hallucinations and delusions are described as involving abnormalities in cognitive control (Frith, Friston, Liddle, & Frackowiak, 1992), in perceptual and attentional processes (Hugdahl, Loberg, & Nygard, 2009), as well as abnormalities in the sense of agency (Startup & Startup, 2005). Wible (2012) points out that the TPJ supports most of

these functions (with projections to inferior frontal regions, hippocampus, and insular brain regions) and, given the strong evidence of TPJ involvement in psychosis, suggests further studies to systematically examine the role of the TPJ are required. Several properties of this system that map onto the schizophrenia syndrome have been discovered through single neuron recording, brain stimulation, neuroimaging and neurological impairment studies; while research under this model continues to build links between brain dysfunction and the syndrome of symptoms seen in schizophrenia (Wible, 2012).

Irrespective of the underlying mechanisms, what we do know from the second Australian national psychosis survey (SHIP) is that around 90.4% of people with a psychotic disorder report an overall deterioration in functioning (Morgan et al., 2012). In terms of social participation, 63.2% experienced an obvious to severe dysfunction in their capacity to socialise, although prior to illness onset 68.7% had reported functioning normally in their work or study roles, and 63.9% had reported normal social functioning (Morgan et al., 2012). In addition, around a third (32.3%) experienced severe impairment in their ability to care for themselves, with 85.0% receiving a government pension as their main source of income, and only 21.5% were currently in paid employment (Morgan et al., 2012). The majority (61.2%) of SHIP participants were single, having never married or been in a de facto relationship, with a large number (69.3%) revealing their illness made it hard to maintain close relationships (Morgan et al., 2012). The top challenges people with psychosis faced included financial problems (42.7%), loneliness and social isolation (37.2%), and lack of employment opportunities (35.1%) (Morgan et al., 2012). Moreover, these key challenges (which included financial, social isolation, unemployment, as well as poor

physical and mental health) were found among both younger and older age groups, and males and females (Morgan et al., 2016).

### *1.3.2 Face and emotion perception in psychosis*

Given the known impacts on social and functional outcomes (Green et al., 2012; Hooker & Park, 2002; Horan et al., 2012; Irani, Seligman, Kamath, Kohler, & Gur, 2012; Kee et al., 2003; Kohler et al., 2010), and their presence even in early prodromal phases (Amminger et al., 2012a; Amminger et al., 2012b; Barkl, Lah, Harris, & Williams, 2014; Roddy et al., 2012), facial emotion processing abnormalities seen in psychotic disorders continue to be an ongoing focus of research. Many aspects have been explored including: emotion perception (Kerr & Neale, 1993); emotion experience (Cohen & Minor, 2010; Kring, Kerr, Smith, & Neale, 1993); the expression of emotion (Gur et al., 2006); effects on cognitive processes (Bozikas, Kosmidis, Anezoulaki, Giannakou, & Karavatos, 2004; Combs & Gouvier, 2004; Huang & Hsiao, 2017); as well as variations due to affective properties of the emotional stimuli, such as valence or intensity (Pineiro et al., 2012).

Difficulties are exhibited across a variety of face tasks, including face detection (Bauser et al., 2012; Darke, Peterman, Park, Sundram, & Carter, 2013), facial emotion perception and associated eye-movement abnormalities (Loughland, Williams, & Gordon, 2002a), as well as through comparisons with other stimulus categories (Kronbichler et al., 2018). While facial emotion processing has been explored extensively, aspects of general face processing have often been less of a focus, and recently a shift in the literature has occurred towards addressing whether non-emotional aspects of facial processing are intact in psychosis (Bortolon, Capdevielle, & Raffard,

2015). It has also previously been suggested, that at a behavioural level these visual perception deficits might not be specific to faces, and that impairment may increase with cognitive and perceptual demands (Bortolon et al., 2015; Darke et al., 2013).

Facial emotion perception impairments have been reported to be independent of acute psychotic symptoms, illness severity, age of illness onset and duration, and treatment with antipsychotic medication (Albus et al., 2002; Comparelli et al., 2013; Edwards, Pattison, Jackson, & Wales, 2001; Kohler et al., 2010; Song et al., 2015; Streit, Wolwer, & Gaebel, 1997). They are also present in clinically high-risk and first episode psychosis (Amminger et al., 2012a; Amminger et al., 2012b; Barkl et al., 2014; Romero-Ferreiro et al., 2016). Leading researchers have also suggested that facial emotion recognition deficits may be a potential trait marker or endophenotype of schizophrenia (Song et al., 2015). Some studies have reported relationships between the severity of negative symptoms and facial emotion processing performance (Mandal, Jain, Haque-Nizamie, Weiss, & Schneider, 1999; Martin, Baudouin, Tiberghien, & Franck, 2005), and between cognitive disorganized symptoms and facial emotion recognition, and to a lesser with extent face recognition performance (Barkhof, de Sonnevile, Meijer, & de Haan, 2015; Comparelli et al., 2014). Age and gender specific error patterns have also been observed, with a decline in facial emotion recognition performance associated with age (Weiss et al., 2007), and an increased accuracy among women (Campellone & Kring, 2013; Scholten, Aleman, Montagne, & Kahn, 2005).

Initially, there was some debate around whether impaired facial affect recognition reflected a specific deficit or a generalized difficulty with face perception (Hooker & Park, 2002), involving low level sensory processes underpinning more basic

perception (Kring & Campellone, 2012). In early studies, individuals with schizophrenia were found to perform poorly on both face recognition and facial emotion recognition tasks, suggestive of a more generalised impairment (Addington & Addington, 1998; Gessler, Cutting, Frith, & Weinman, 1989). Other studies report a particular difficulty identifying negative facial emotions such as fear, sadness or anger, suggestive of a negative emotion specific deficit (Addington & Addington, 1998; Heimberg, Gur, Erwin, Shtasel, & Gur, 1992; Schneider et al., 1995; Song et al., 2015). However, other research suggests that deficits in recognising negatively valenced emotions may be associated largely with increased task difficulty (Gur et al., 2002; Johnston, Katsikitis, & Carr, 2001; Johnston, Devir, & Karyanidis, 2006).

Due to the diversity of tasks, small sample sizes, and varied clinical/demographic characteristics, the generalisability of findings from earlier studies was limited with respect to determining whether a differential or generalised impairment in face processing existed (Johnston et al., 2001; Penn et al., 2000b). More recent, methodologically consistent studies have confirmed that individuals with schizophrenia have difficulty across most categories of facial emotion expression perception (Kohler et al., 2010), including studies using degraded stimuli (Johnston et al., 2006), supporting earlier studies disregarding emotion specific deficits (Kerr & Neale, 1993). Furthermore, with respect to general face perception deficits, recent studies examining non-emotional aspects of face processing suggest a more generalised visual perceptual difficulty may exist (Darke et al., 2013; Watson, 2013), that is not specific to faces (Bortolon et al., 2015).

Tasks traditionally used to examine facial expression perception include: ‘*identification tasks*’, asking a participant to identify the emotion displayed; or ‘*discrimination tasks*’, displaying two pictures side by side and asking a participant to decide if the emotion is the same or different (Kring & Campellone, 2012). The Kohler et al. (2010) meta-analysis included 86 studies, confirming difficulties both identifying and discriminating facial expressions of emotion. Historically, emotion processing research focused on the perception of static faces (e.g., stimulus sets including: Ekman and Friesen (1976); Tottenham, Borscheid, Ellersten, Marcus, and Nelson (2002); (Tottenham et al., 2009)), but more recently the importance of context has been examined, and its role in helping to construct the perception of emotion in schizophrenia (Kring & Campellone, 2012). A considerable body of research demonstrates a difficulty perceiving emotion, while another body of research demonstrates context processing deficits exist, suggesting contextual information in the perception of emotion is not fully utilised by individuals with schizophrenia (Kring & Campellone, 2012).

In day-to-day situations, emotion cues can be more ambiguous and the integration of contextual information can assist or hinder perception, which is particularly relevant in identifying areas of difficulty in schizophrenia (Kring & Campellone, 2012; Phillips, Drevets, Rauch, & Lane, 2003). Efforts to understand how emotion processing is influenced in real life situations is an important area of current research, by exploring cognitive processes and relationships between contextual processing and social and emotional functioning (Kring & Campellone, 2012). Individuals with psychosis have been found to display less impaired processing of static stimuli compared to dynamic emotional stimuli (Song et al., 2015), providing further

evidence that variations in the recognition of facial expressions of emotions depend on the nature of the stimuli (Johnston et al., 2010).

Despite well documented evidence of impaired emotion identification, some recent research suggests that individuals with schizophrenia may actually retain the ability to implicitly perceive facial affect, in that the initial perception of salient facial features remains intact, with deficits arising during subsequent stages of contextual processing, such as integration and appraisal (Shasteen et al., 2016). It is proposed that examining the stream of face processing has important implications for mechanistic models of social cognitive impairment, as well as treatment strategies aimed at improving functional outcomes in schizophrenia (Shasteen et al., 2016). Face perception problems (i.e., categorisation, discrimination and identification of facial stimuli) have previously been associated with neurocognitive deficits in working memory, attention and executive function (Addington & Addington, 1998; Chen et al., 2009; Heinrichs & Zakzanis, 1998) and linked to prefrontal cortex dysfunction in schizophrenia (Hodgson et al., 2002; Zihl & Hebel, 1997). One such study, among individuals in the remitted phase of schizophrenia, revealed that emotion recognition performance was influenced by a combination of cognitive flexibility and memory encoding ability, with overall neurocognition predicting 39% of the variance in performance (Mehta, Bhagyavathi, Thirthalli, Kumar, & Gangadhar, 2014). Similarly, cognitive training programs have shown improved functioning in specific neurocognitive abilities to be predictive of social cognition outcomes (Tan et al., 2018).

Facial emotion recognition performance has previously been shown to be associated with both clinical symptoms and basic neurocognition (Gur et al., 2006),

with a likely interdependence between facial emotion recognition and some neurocognitive domains (Bozikas et al., 2004; Combs & Gouvier, 2004). Various subdomains of social functioning have been associated with general intelligence, neurocognition (attention, working memory, executive functioning), clinical symptoms (positive and negative) and facial affect recognition (Huang & Hsiao, 2017). Current research continues to focus on attempting to disentangle the complex interrelationships between clinical symptoms, neurocognition and emotion recognition, in order to better understand the underlying mechanisms and brain related aspects of these impairments in social functioning (Huang & Hsiao, 2017).

Prior neurophysiological research implies facial emotion recognition could be an independent neural event, via activation in brain regions related to autonomic processing of emotions, such as the limbic structures (Gur et al., 2002); although, interconnected execution related medial-frontal areas are also reported in studies of emotion recognition deficits in schizophrenia (Hempel, Hempel, Schönknecht, Stippich, & Schröder, 2003). Neuroanatomical abnormalities in frontal and temporal regions, have been consistently implicated in facial emotion recognition (Nakamura et al., 2014), with impaired recognition linked to anomalous activation in these areas, such as hypo activation in response to negatively valenced emotions in schizophrenia (Ji et al., 2015; Phillips et al., 1999). Emerging neural and neurophysiological evidence suggests impaired early visual processing in psychosis, possibly deficits in the interaction of magnocellular and parvocellular pathways, impacts on processing prior to illness onset, which may not be specific to face perceptions (Bortolon et al., 2015).

Experimental studies have demonstrated abnormalities in early visual encoding of facial features (Turetsky et al., 2007), and cognitive influences on down-stream information processing, including attention and executive functioning (Bozikas et al., 2004; Combs & Gouvier, 2004). Emerging research, considering the significance of neurocognition and facial affect recognition on social functioning in schizophrenia, implies both have crucial roles in predicting social outcomes (Huang & Hsiao, 2017). This research also provides confirmation facial emotion recognition correlates with many domains of social functioning, including interpersonal communication, independence (competence and performance), and employment outcomes (Huang & Hsiao, 2017).

Encouraging cognitive remediation results have also emerged (Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007; Hodge et al., 2008; Kurtz, 2003) among studies using strategies aimed at improving facial affect recognition (Penn & Combs, 2000; Russell et al., 2006). Impaired early stage visual processing in emotion recognition deficits has generally been the target of remediation (Butler et al., 2009), such as utilising eye-movement recordings to re-direct attention to relevant facial features; which has been shown to be successful in normalising prior extended view strategies in schizophrenia (Russell et al., 2006). Campellone and Kring (2013), using an emotion perception task that included context, found that women with and without schizophrenia showed similar performance, while men with schizophrenia exhibited deficits, being most likely to benefit from cognitive remediation training involving processing emotions in context. Given that recognition accuracy was related to functional capacity and social skills, training may not only help to improve task performance but more importantly social functioning (Campellone & Kring, 2013).

### *1.3.3 Visual processing during face and emotion perception in psychosis*

Abnormalities in visual scan-paths have been consistently reported for schizophrenia and are suggested as a possible trait or neurophysiological biomarker for the disorder (Beedie et al., 2011). Recent research utilising scan-path indices to produce an integrated eye-movement score (i.e., scan-path length, horizontal position gain, and duration of fixations) has been able to successfully discriminate with 82% accuracy between schizophrenia patients and healthy controls (Morita et al., 2017). Eye-tracking dysfunction in schizophrenia is a behavioural deficit that has been replicated in multiple studies, described as involving a decline in saccade control and smooth pursuit eye-movement (Levy, Sereno, Gooding, & O'Driscoll, 2010). Visual scanning deficits involving eye-movement abnormalities, such as a shorter mean scan-path length and longer duration of fixations to face stimuli, have been widely researched, and shown to remain stable over time, occurring during both acute and remitted illness phases (Streit et al., 1997). Many studies have confirmed a relationship between impaired visual processing and emotion recognition deficits, involving atypical scan-path strategies characterised by a differential pattern of fixations and saccades (de Wilde, Dingemans, Linszen, Bour, & Boeree, 2007; Kojima et al., 1992; Minassian, Granholm, Verney, & Perry, 2005; Phillips & David, 1997; Turetsky et al., 2007).

Visual scanning disturbances in processing facial displays of emotion in schizophrenia have been characterised by a restricted scan-path strategy, involving fewer fixations of longer duration, reduced scan-path length and distance between fixations, as well as fewer fixations on salient facial features (like the eyes and mouth) likely to assist in discriminating affect (Loughland et al., 2002a, 2002b; Williams et al., 2001). These visual-cognitive and perception deficits have also been observed among

individuals at risk for schizophrenia, such as: first-degree relatives (Loughland et al., 2004a; Toomey, Seidman, Lyons, Farone, & Tsuang, 1999); those with prodromal symptoms (Walker & al., 1993); schizotypal traits (Mikhailova, Vladimirova, Iznak, Tsusulkovskaya, & Sushko, 1996; Statucka & Walder, 2017; Waldeck & Miller, 2000), including children and adolescents (Habel, Krasenbrink, Bowi, Ott, & Schneider, 2006); as well as first episode psychosis (Benson, Leonards, Lothian, Clair, & Merlo, 2007; Edwards et al., 2001; Lee et al., 2015a). It is likely that these deficits are present before illness onset (Habel et al., 2006), consistent with a trait based deficit (Beedie et al., 2011; Edwards et al., 2001; Streit et al., 1997).

Familial studies also provide some evidence that components of visual scan-path dysfunction may represent a vulnerability marker in the familial transmission of schizophrenia (Loughland et al., 2004a); although one study found shorter scan-paths among individuals with schizophrenia but not their healthy siblings (de Wilde et al., 2007). However, task differences can make direct study comparisons difficult. For example, the de Wilde et al., (2007) study utilised extended stimuli exposures (20 sec), potentially in excess of that required to process content (Thematic Apperception Test - an operating room or a crying female followed by storytelling), whereas the study by Loughland et al. (2004) used brief static facial stimuli (during an emotion recognition task). Different scan-path strategies may apply when integrating aspects of more complex stimuli. However, suggestions that some scan-path abnormalities may be specific to schizophrenia (Addington & Addington, 1998; Loughland et al., 2002a) are supported by recent studies distinguishing schizophrenia patients from healthy controls utilising integrated eye-movement scores, which have revealed that some scan-path indices are more discriminating or of pathologic relevance (e.g., decreased scan-path

length, decreased gain during smooth pursuit, and a reduced duration of fixations) (Benson et al., 2012; Miura et al., 2014; Morita et al., 2017).

The impact of specific psychotic symptoms on visual scanning performance, in terms of abnormal information processing, has also previously been examined. For example, persecutory delusions have been associated with directing gaze to less threatening areas when viewing stimuli depicting ambiguous scenes (Phillips et al., 2000). Biased processing of contextual information in ambiguous settings has been suggested as perhaps leading to inappropriately perceiving threat (Phillips et al., 2000). In addition, visual scanning abnormalities have been observed among other clinical groups, including individuals with an affective disorder (Loughland et al., 2002a) and psychopathy traits (Gillespie, Rotshtein, Beech, & Mitchell, 2017b; Gillespie, Rotshtein, Wells, Beech, & Mitchell, 2015c), raising concerns about the potential impacts of coexisting comorbid conditions. Impaired facial affect recognition has been frequently linked to aggressive behaviour, and posited as a possible contributor to increased aggression in psychosis (Malone, Carroll, & Murphy, 2012). In examining relationships between facial emotion recognition, psychosis and aggression, several potential confounding variables have been suggested as possible contributors, including positive symptoms, psychopathic personality traits, childhood trauma, and substance use (Malone et al., 2012). Psychopathic personality traits have also been robustly linked to increased aggression (Loney, Frick, Clements, Ellis, & Kerlin, 2003) as well as to emotion processing impairments (Dawel et al., 2012).

## **1.4 Face and emotion perception and visual processing in psychopathy**

### *1.4.1 Psychopathy: background, symptoms and functioning*

#### *1.4.1.1 Prevalence and aetiology*

Prevalence rates for psychopathy are low, around 1% (0.6-1.2%) of the general community possess enough traits to be considered psychopathic (Coid, Yang, Ullrich, Roberts, & Hare, 2009; Hare, 1996; Neumann & Hare, 2008). However, higher estimates have been reported for specific populations, including offenders and psychiatric patients, with around 15-25% of male and 7.5% of female prisoners, 10% of male forensic psychiatric patients, and 1-2% of inpatient psychiatric patients meeting criteria for psychopathy (Douglas, Ogloff, Nicholls, & Grant, 1999; Hare, 1996; Hart, N., & Hare, 2003). Epidemiological studies amongst community samples are relatively scarce. Coid et al. (2009) utilising the Psychopathy Checklist: Screening-Version (PCL:SV) (Hare et al., 1995), found a prevalence rate of 0.6% at a cut off score of 13, but using a more liberal cut score of 11 reported a prevalence rate of 2.3%, with 3.7% for men and 0.9% for women in the UK. A half normal distribution of psychopathy was reported, with the majority of the general population (70.8%) having no traits, and a severe subgroup with multiple traits (Coid et al., 2009). Psychopathy in the community was associated with: younger age; being male; violent behaviour; suicide attempts; homelessness; imprisonment; drug dependence; histrionic, borderline and antisocial personality disorders; and panic and obsessive-compulsive disorders (Coid et al., 2009). Similarly, an Australian community sample reported correlates of self-reported psychopathy as being male, having high levels of callous affect, pronounced empathy deficits, alcohol misuse, and pro-violence sentiments (Watt & Brooks, 2012).

With the highest prevalence estimates reported among prison populations (15% - 25%), a disproportionate amount of violence, criminal behaviour and recidivism has been associated with the disorder (Hare, 1996); and, at a societal level psychopathy, is seen as dangerous and costly (Burley, Gray, & Snowden, 2017). On the other hand, many psychopaths in the community are suggested to be equally as callous, manipulative, and egocentric as their criminal counterparts; however, due to factors such as intelligence, social skills, family background or other environmental circumstances, they reside in the community (Hare, 1999). Not unlike psychosis, psychopathy is a complex disorder of unknown aetiology. Biological, psychological, social and environmental factors have been explored and, while no definitive answers have been obtained, a complex interaction between biological predisposition and social forces seem likely (Hiatt & Newman, 2006).

In terms of the development of psychopathy, a review of studies on childhood psychopathy revealed that it presents in a very similar way to psychopathy in adulthood, and is characterized by the same traits (Lynam & Gudonis, 2005). Psychopathy appears to be quite stable across adolescence, characterised by serious and stable patterns of offending. Affected children are prone to externalizing disorders, with extremely low levels of agreeableness and conscientiousness, and they exhibit similar processing deficits to adults (Lynam & Gudonis, 2005).

Several theories have been proposed, offering accounts for the origins of psychopathy, but the empirical base supporting these theories is limited (Lynam & Gudonis, 2005). Up until recently, research on correlates and aetiology were heavily reliant on forensic and psychiatric samples, with a lack of epidemiological evidence due

largely to the low prevalence amongst community samples (Coid et al., 2009). However, more recently research among non-forensic samples has important implications, given that psychopathy traits are believed to be continuously distributed, among forensic, clinical and community samples (Neumann & Hare, 2008).

It is generally accepted that genetics and environment play a role in the aetiology of psychopathy, particularly in the development of callous unemotional traits. Both genetic and prenatal factors are thought to contribute to the abnormal development of particular neural systems, with social and environmental factors thought to influence the probability that antisocial behaviour will be subsequently displayed (Blair, 2015). Neuroimaging studies have implicated amygdala dysfunction in the aetiology of psychopathic traits (Marsh & Cardinale, 2014), with structural brain differences also identified (Gregory et al., 2012). Environmental factors, including enrichment, diet, paternal deprivation, and maternal substance abuse during pregnancy, have all been implicated in the development of psychopathy traits, largely due to impacts on neural systems like the amygdala, caudate and ventromedial prefrontal cortex (vmPFC) (Barker, Oliver, Viding, Salekin, & Maughan, 2011; Blair, 2015; Seidel, Poeggel, Holetschka, Helmeke, & Braun, 2011; Workman, Fonken, Gusfa, Kassouf, & Nelson, 2011). While specific details around the molecular genetics are unknown, research looking at callous unemotional traits proposes a genetically driven system-wide alteration in serotonin function (Moul, Dobson-Stone, Brennan, Hawes, & Dadds, 2015). In this regard, there may be two pathways to callous unemotional traits that involve methylation of the serotonin 1B receptor gene: one driven by a genotypic risk; and another associated with risk for generally increased levels of methylation (Moul et

al., 2015). Overall, a biopsychosocial perspective on aetiology is currently implied (Vasconcellos et al., 2017).

#### 1.4.1.2 Symptomatology

Psychopathy, which is traditionally characterised as a disorder of personality, affective deficits and to a lesser extent behaviour, was one of the first personality disorders recognised. Early historical texts detail characteristics of individuals later referred to as psychopathy. In the early 1800s, the French physician Philippe Pinel used the label '*manie sans delire*' (mania without confusion of the mind) to describe individuals whose affective faculties were disordered but were otherwise capable and displayed good judgement (Pinel, 1809; cited in Ogloff (2006)). Based on Pinel's earlier work, Prichard used the term '*moral insanity*' (insanity without delusion) which became an accepted diagnosis in the 19th century, to account for morally objectionable behaviour where the personality of the individual was distorted but intellectual faculties were unimpaired (Pritchard 1835 cited in Blair (2006)).

In 1904, Kraepelin introduced the term '*psychopathic personalities*' in characterising individuals who were neither neurotic or psychotic (cited in:Moreira, Almeida, Pinto, & Fávero, 2014). Schneider, a German psychiatrist, later used the term '*psychopathy*' to refer to a variety of psychopathic personalities as extreme variants of normal personality (Schneider (1923); cited in Hildebrand and de Ruiter (2004)). Across the course of history, a number of labels for the condition now known as psychopathy were utilised, starting with: '*manie sans delire*'; '*moral insanity*'; '*moral imbecility*'; '*degenerate constitution*'; '*congenital delinquency*'; '*constitutional inferiority*'; '*psychopathic inferiority*'; '*psychopathic taint*'; '*character deficiency*'; '*manipulative*

*personality*'; *psychopathic personality*'; *sociopathic personality disorder*'; *psychopathy*' and more recently *'Antisocial'* and *'Dissocial Personality Disorder (PD)'* (Hare, 1991; Ogloff, 2006).

Current conceptualisations of psychopathy are based on the work of American psychiatrist Hervey Cleckley. His book *'The mask of sanity'* first published in 1941 described individuals who seemed sane, intelligent and competent but who were clearly disturbed, or in Cleckley's terms wore "*masks of sanity*" (Cleckley, 1988; Lynam & Gudonis, 2005). Cleckley (1988) outlined the first formal classification criteria, which included: 1) superficial charm and good intelligence; 2) absence of delusions and other signs of irrational thinking; 3) absence of 'nervousness' or psychoneurotic manifestations; 4) unreliability; 5) untruthfulness and insincerity; 6) lack of remorse or shame; 7) inadequately motivated antisocial behaviour; 8) poor judgement and failure to learn from experience; 9) pathological egocentricity and incapacity for love; 10) general poverty in major affective reactions; 11) specific loss of insight; 12) unresponsiveness in general interpersonal reactions; 13) fantastic and uninviting behaviour, with drink and sometimes without; 14) suicide rarely carried out; 15) sex life impersonal, trivial and poorly integrated; and 16) failure to follow any life plan.

Cleckley (1941) described the core features of psychopathy as intrapersonal, interpersonal and behavioural characteristics. "*Intrapersonally*" referred to deficits in the experience of nervousness and major emotions, loss of insight regarding attitudes and behaviour, a limited ability to learn from experience, and poor judgement in interpersonal relationships (Cleckley, 1988). "*Interpersonally*", the psychopath was pathologically egocentric, with superficial interactions, callousness towards others, and

insincere and shallow interactions (Cleckley, 1988). “*Behaviourally*”, the psychopath was likely to engage in inadequately motivated antisocial behaviour, lack long term goals, remorse and guilt (Cleckley, 1988). Cleckley focused on affective and interpersonal characteristics, emphasising an emotional deficit influencing the ability to have emotional experiences that were necessary for the development of a conscience. Since Cleckley’s description, definitions of psychopathy among other clinicians and researchers, such as Hart et al. (2003), have remained relatively similar (Lynam & Gudonis, 2005). However, in Cleckley’s model, criminal behaviour was not considered a defining feature; which essentially asserts that these traits are not only found among criminals but in the community, including, for example, successful individuals of high social status, whose façade of normality could lead to superficial material and social success (Coid et al., 2009). This is in contrast to Hare’s model with a focus on criminal and violent behaviour (Ogloff, 2006).

Furthering Cleckley’s work, Hare’s empirically-driven model intended to quantify the criteria, conceptualising psychopathy as comprising two factors “*interpersonal/affective*” and “*social deviance*”, with four facets: 1) interpersonal; 2) affective; 3) impulsive and irresponsible lifestyle and 4) antisocial behaviour (Hart et al., 2003; Ogloff, 2006). Hare developed the Psychopathy Checklist (PCL) in 1980, followed by the revised version (PCL-R) in 1991 (2nd edition, 2003), as a clinical assessment tool, which has been extensively used clinically and in research for over 30 years. The “*interpersonal facet*” includes superficial charm, grandiosity, pathological lying and manipulation (Hart et al., 2003). The “*affective facet*” is characterised by callousness towards others, lack of remorse or guilt, shallow affect and failure to take responsibility for their actions (Hart et al., 2003). Impulsivity and irresponsibility,

demonstrated by a lack of long term goals, a parasitic lifestyle and an unusual proneness to boredom or need for stimulation, represent the third “*lifestyle facet*” (Hart et al., 2003). The final “*antisocial facet*” focuses on criminal behaviour commencing in adolescence, versatility of criminal behaviour, and failure to comply with legal conditions (Hart et al., 2003). A screening version of the PCL: SV, with the same four facet model, has been developed for non-forensic samples, which is suitable for research use (Hart, Cox, & Hare, 1995) and which has been found to be highly correlated with the corresponding PCL-R constructs (Cooke, Michie, Hart, & Hare, 1999; Guy & Douglas, 2006).

With respect to DSM and ICD diagnostic profiles, while psychopathy is not specifically included, there are traits associated with other personality disorders that are remarkably similar: in DSM - antisocial; paranoid; histrionic; narcissistic; borderline; and passive aggressive traits; and in ICD - impulsive; dissocial; paranoid; histrionic; and borderline dimensions (Coid et al., 2009). However, as Ogloff (2006) points out, whilst often used inter-changeably, the diagnostic constructs of psychopathy, and antisocial or dissocial PD are in fact distinct. Hare (1991) defines psychopathy as a dysfunction of interpersonal and affective traits, as well as impulsive, irresponsible and antisocial behavioural traits, leading to a failure to abide by social norms, obligations and responsibilities.

*DSM Antisocial PD:* DSM-5 defines antisocial PD (criteria A) as a pervasive pattern of disregard for and violation of the rights of others, occurring since the age of 15 years, beginning in childhood or early adolescence and continuing in to adulthood. This is characterised by (in brief, three or more of the following: 1) persistent violations

of social norms; 2) deceitfulness, lying, stealing; 3) impulsivity; 4) irritability and aggressiveness; 5) reckless disregard for safety of self or others; 6) irresponsibility, inconsistent work behaviour; and 7) lack of remorse (American Psychiatric Association, 2013). Additional criteria (B, C & D) include being at least 18 years; evidence of conduct disorder before the age of 15 years, indicating that the PD is of long duration; and not exclusively during the course of schizophrenia or bipolar disorder (APA, 2013). DSM-5 states this pattern has been referred to as psychopathy, sociopathy or dyssocial PD (APA, 2013).

While antisocial PD shares characteristics with psychopathy, it is seen as being much broader, with many more behavioural (social deviance) than personality (interpersonal or affective) traits, leading to a tendency to over identify individuals (Ogloff, 2006). This is particularly evident among offender populations, explaining the higher estimates of 50% to 80% of prisoners meeting criteria for antisocial PD, with only a small percentage of around 15% expected to meet criteria for psychopathy (Hart et al., 2003). Importantly, whilst the majority of psychopaths meet criteria for antisocial PD, not all individuals diagnosed with antisocial PD will meet criteria for psychopathy (Hare, 1996; Hildebrand & de Ruiter, 2004).

ICD-10 Dissocial PD: dissocial PD, as defined in ICD-10, is characterised by: a disregard for social obligations and callous unconcern for the feelings of others; a gross disparity between behaviour and social norms; behaviour that is not readily modifiable; low tolerance to frustration and threshold for aggression, including violence; and a tendency to blame others or offer plausible rationalizations for behaviour (World Health Organization, 2011). The criteria for dissocial PD, while conceptually closer to

psychopathy than antisocial PD, have been suggested to place greater emphasis on affective deficits, and do not comprehensively cover the broad personality and behavioural components (Ogloff, 2006). On the other hand, the PCL-R has been designed to measure the extent to which an individual possesses characteristics consistent with psychopathy, using comprehensive criteria to assesses two main factors: interpersonal and affective facets, tapping personality; and social deviance in lifestyle and antisocial facets, in terms of behavioural deficits (Hart et al., 2003).

More importantly, given these definitional differences, caution in drawing clinical implications from the existing psychopathy literature is recommended, as not all findings will be directly relevant across populations (Ogloff, 2006). In addition, cut-off scores utilised on the PCL-R may vary, typically, a score of 30/40 or more is utilised (Hare, 1991), although studies in some European countries (Scotland, England and Sweden) report lower cut off scores of 25 or 26 as more useful (Bo, Abu-Akel, Kongerslev, Haahr, & Simonsen, 2011). Similarly, cut off scores using the PCL: SV may vary; typically, a score of 13/24 (Hart et al., 1995) is used, but lower more conservative cut-offs among community samples have been considered, such as 11/24 (Coid et al., 2009).

#### *1.4.1.3 Comorbidity*

Seen as a socially devastating disorder, there is a strong relationship between psychopathy, as a central personality characteristic, and offending and violent behaviour (Hare, 1996; Watt & Brooks, 2012). As expected, psychopathy among forensic psychiatric patients is highly associated with antisocial PD, with rates as high as 81% reported (Hildebrand & de Ruiter, 2004). Psychopathy has also been found to co-occur

with schizophrenia, although inconsistent rates among forensic samples have been reported ranging from 4-8% (Hart & Hare, 1989; Rice & Harris, 1995) to as high as 22-25% (Rasmussen & Levander, 1996; Tengstroem, Grann, Langstroem, & Kullgre, 2000). Research examining psychopathy amongst individuals diagnosed with schizophrenia has tended to occur in forensic settings, with a focus on symptomatology and associations with risk of violence or aggression (Abushua'leh & Abu-Akel, 2006; Fullam & Dolan, 2006a; Weiss et al., 2006). Of particular interest, there is research showing that both disorders are characterised by similar impairments in emotion perception (Dolan & Fullam, 2009; Fullam & Dolan, 2006b).

Research examining relationships between psychopathy and other diagnostic criteria have considered facets of psychopathy (using measures such as the PCL: R). A positive association between intelligence and the interpersonal facet, and a negative association between intelligence and the affective facet has previously been reported (Neumann & Hare, 2008; Vitacco, Neumann, & Jackson, 2005). Among offender populations, the lifestyle and antisocial facets have consistently been shown to be positively correlated with substance use disorders (Walsh, Allen, & Kosson, 2007). Psychopathy traits appear to be continuously distributed, which has led researchers to speculate on the presence of these traits in the general population (Neumann & Hare, 2008). In general community samples, psychopathy factors (measured using the PCL: SV) have been found to be associated with violent behaviour, alcohol use, and intelligence, in much the same way (Neumann & Hare, 2008) as among offender and psychiatric samples (Hart et al., 2003; Vitacco et al., 2005). Similarly, in an Australian community sample using a Self-Report Psychopathy Scale (SRP-III), psychopathy was associated with low levels of empathy (higher levels of callous-affect), high alcohol use,

pro-violence thoughts, as well as elevated depression, anxiety and stress (Watt & Brooks, 2012). Inconsistently with previous findings, a negative correlation was found between anxiety and stress and the affective/interpersonal aspects of psychopathy (i.e., associations with callous affect were not found using a self-report measure (Watt & Brooks, 2012).

#### *1.4.1.4 Cognitive and social functioning*

Two main cognitive impairments, “*empathic dysfunction*” and “*impaired decision-making*”, have been associated with the callous-unemotional and impulsive-antisocial components of psychopathy (Blair, 2013). The first, reduced empathic responding to emotional and verbal displays of affect in others, particularly responsiveness to distress cues (including fear, sadness, pain), has been found to be associated with reduced amygdala functioning (Blair, 2013). Empathy is characterised as the process of understanding another by putting ourselves into the other person’s place for a “shared experience” (Gallese, 2003 cited in Montgomery, Seeherman, and Haxby (2009). However, cognitive empathy, in terms of theory of mind, appears to be intact in adults (Dolan & Fullam, 2004; Richell et al., 2003), and adolescents with psychopathic traits (Jones, Happe, Gilbert, Burnett, & Viding, 2010). The empathic deficit seen in psychopathy also differs to that seen in autistic spectrum disorder (ASD). While both groups may appear uncaring, in psychopathy the deficit is in affective empathy, involving a difficulty in affective information processing, specifically resonating with others distress, where as in ASD the difficulty relates to cognitive perspective taking, or not knowing what people think (Jones et al., 2010).

In terms of social functioning, empathy relates to the interpersonal and affective components of psychopathy (Hart et al., 2003), with the lack of empathic concern shown by individuals with psychopathy a particular area of interest (Watt & Brooks, 2012). Empathy is also seen as a protective factor for aggression and violent behaviour, with Blair (1993) initially proposing an “*impaired violence inhibition mechanism*” model among psychopaths. For example, lacking remorse or guilt and having callous-unemotional traits may predispose them to early onset aggression, with little or no remorse for victims (Blair, Jones, Clark, & Smith, 1997). Proposed as a model of the development of morality, the violence inhibition mechanism is considered a cognitive mechanism which, when activated by the non-verbal communication of distress (i.e., sad facial expression), initiates a withdrawal response (Blair, 1995). It is argued that such plans or schema which otherwise predispose an individual to withdraw from the attack, do not occur with psychopathy (Blair, 1995).

The second cognitive impairment associated with psychopathy influences aspects of decision-making, specifically reinforcement learning, which relates more to the impulsive-antisocial component. This functional impairment has been associated with dysfunction in the vmPFC and striatum (Blair, 2013). Imaging (fMRI) studies reporting inferior parietal lobe and frontal operculum activation during observation and execution of actions, thus forming a putative human mirror neuron system (hMNS) (Montgomery et al., 2009). Impairments in executive functioning have also been implicated in reduced behaviour control, and impulsivity, representing a risk factor for violence and criminal behaviour (Slotboom et al., 2017).

One critical element for cognitive control and goal directed behaviour is visual attention, which has been extensively examined among offenders with psychopathy (Slotboom et al., 2017). Interpersonal and affective traits (i.e., deceitful interpersonal style, callousness, emotional superficiality, and lack of empathy), have been related to improved or superior selective attention, while impulsive and antisocial lifestyle has been related to poorer attentional performance (Baskin-Sommers, Curtin, Li, & Newman, 2012; Baskin-Sommers, Curtin, & Newman, 2011). A specific set of attentional abnormalities, in top-down (e.g. goal directed) attention and selection history, but not in bottom-up (e.g. stimulus driven) attention have been reported in psychopathic offender and community samples (Hoppenbrouwers, Van der Stigchel, Sergiou, & Theeuwes, 2016b; Hoppenbrouwers, Van der Stigchel, Slotboom, Dalmaijer, & Theeuwes, 2015).

While characterisations of psychopathy have tended to emphasize emotion-processing deficits (i.e., poor fear conditioning and reduced startle potentiation), information-processing deficits also provide critical insight into the disorder (Hiatt & Newman, 2006). An earlier review by Hiatt and Newman (2006) outlining broad cognitive processing deficits, such as dual-task attention, and behavioural inhibition (among homogenous samples using the PCL or PCL-R), suggest a primary challenge for researchers is to obtain a detailed understanding of the relationships between cognitive and emotional deficits to reach a unified understanding of the disorder (Hiatt & Newman, 2006). Given the characteristics and impairments outlined above, the psychopath is seen to pose harm to and exact costs from society (Lynam & Gudonis, 2005). For example, among criminal populations, psychopathic offenders have been

found to have more versatile offence patterns, committing significantly more violent and non-violent offences (Kosson, Smith, & Newman, 1990).

#### 1.4.2 *Face and emotion perception in psychopathy*

Psychopaths lack empathy and remorse, and are infamous for using charm, manipulation, threats and violence to control others to satisfy their own selfish needs (Hart et al., 2003). They often use aggression in a planned way, lacking any fear of punishment, and have difficulty regulating emotion (Hart et al., 2003). Psychopathy has previously been related to an innate deficiency in fearfulness (Lykken, 1995), supported by research evidence of: poor fear conditioning, and passive avoidance learning (Lykken, 1995); weak electro-dermal response to anticipation of aversive events (Hare, 1999); lack of startle response to unpleasant stimuli (Patrick, Bradley, & Lang, 1993); reduced amygdala activation during aversive conditioning (Birbaumer et al., 2005), and in making moral judgements about causing fear or distress (Marsh & Cardinale, 2014). Atypical amygdala response to fear in psychopathy has been reported for several kinds of stimuli (Marsh & Cardinale, 2014), including a number of studies reporting face recognition deficits for fear among psychopathic and antisocial populations (see meta-analyses by Dawel et al., 2012; Marsh & Blair, 2008; Wilson, Juodis, & Porter, 2011). Given the role facial expressions play in social interaction, emotion perception deficits have been extensively explored (Decety et al., 2014), still remain a current area of focus, and appear to extend beyond processing fear (Dawel et al., 2012), although findings have differed in relation to the specificity of the impairment.

While definitional differences exist between studies, there is general agreement that antisocial individuals have problems recognising emotions. Impaired facial affect

recognition for negatively valenced emotions has been linked to antisocial behaviour among violent offender populations (Schönenberg et al., 2014). A meta-analysis of studies including antisocial populations, has confirmed deficits in facial affect recognition, specifically in identifying fearful and sad expressions (Marsh & Blair, 2008). However, another meta-analysis, examining both facial and vocal stimuli specifically in psychopathy, found evidence suggestive of a more pervasive emotion recognition deficit, not only for fear and sadness but both positive and negative emotions, including fear, happiness, sadness and surprise (Dawel et al. (2012). Similarly, a meta-analysis of facial affect recognition in psychopathy supports a general impairment in emotion recognition, with larger effect sizes for fear and sadness but deficits overall (Wilson et al., 2011). Consequently, some uncertainty remains, due to differing samples and inconsistent findings, around whether emotion recognition deficits among individuals who meet the criteria for psychopathy are only for particular emotions, or whether the impairment is more pervasive.

Notwithstanding the diverse results, impairments in facial emotion processing among individuals with psychopathy exist, although the underlying mechanisms are unclear. Deficits in affective functioning are suggested to reflect core psychopathy, while cognitive factors such as attention are also seen as important. There are currently two major aetiological theories proposing different mechanisms as to how facial expressions are processed by individuals with elevated psychopathic traits (Munneke et al., 2018). The “*Integrated Emotions System theory*” proposes that a fundamental amygdala dysfunction precludes adequate responsiveness to the distress of others, while the “*Response Modulation Hypothesis*” suggests that emotional deficits are a

consequence of attentional deployment, and likely to be situation-specific (Blair, 2006; Munneke et al., 2018; Newman, Curtin, Bertsch, & Baskin-Sommers, 2010).

The first of these approaches, Blair's (2006) neurobiological *Integrated Emotions Systems* model, focuses on the emergence of psychopathy. It proposes that specific deficits in experiencing fear and sadness contribute to the development of psychopathy, involving a basic deficit in reactivity to aversive stimuli, indicative of amygdala dysfunction (Blair, 2006; Blair et al., 2004). The amygdala is seen as the primary area of dysfunction underlying these emotion processing deficits, with atypical neural functioning in the OFC and striatum also linked to elevated psychopathic traits (Blair, 2005; Blair et al., 2004; Gordon, Baird, & End, 2004; Marsh & Blair, 2008). Amygdala deficits are hypothesised to cause an impairment in the formation of aversive stimulus-reinforcement associations in psychopathic individuals, with aversive stimuli extending to emotional expressions such as fear and anger (Birbaumer et al., 2005; Blair, 2005). These emotion processing deficits have been implicated in impulsive aggressive behaviour, whereby normal instrumental conditioning may not occur, particularly with fear stimuli, preventing the inhibition of aggression and violence, and the development of pro-social behaviour (Blair, 2006; Blair et al., 2004). Essentially, it is proposed that as a consequence of not effectively processing emotions signalling distress in others, psychopathic individuals exhibit diminished social behaviour. Amygdala dysfunction among general community populations has been associated with impaired fear recognition (Adolphs et al., 1994; Calder, 1996), supporting an amygdala-mediated deficit in the impaired recognition of fear and sad facial expressions observed in psychopathy.

An alternate view to an “*amygdala-mediated deficit*”, proposed by Newman et al. (2010), is the *response modulation theory*, which suggests deficits in fear conditioning, as well as other behavioural traits and emotion deficits displayed in psychopathy, are the result of a failure to process information in the periphery (Newman et al., 2010). Rather than reduced sensitivity to punishment cues, there is some evidence to support the view that higher-order cognitive processes may moderate deficits of diminished reactivity to fear stimuli and emotion-related cues generally (Newman et al., 2010). Psychopathic behavioural traits are suggested to originate from deficits in the ability to rapidly switch from goal-directed behaviour to attending to task-irrelevant information, when processing this irrelevant information could actually lead to beneficial behaviour or improved social interaction (Patterson & Newman, 1993). That is, there may be an attention-related deficit limiting the processing of peripheral information, providing crucial context for interpretation, decision-making, and interpersonal interactions (Newman et al., 2010). Consequently, self-regulation may be lacking, which could account for chronic disinhibition and an insensitive interpersonal style in psychopathy, and possibly provide a target for early clinical intervention (Newman et al., 2010). This malfunction in information-processing ability is seen as an important contributor to psychopathic behaviour, whereby individuals use a rigid, inflexible mechanism of top-down attentional controls (Hoppenbrouwers et al., 2016b; Hoppenbrouwers et al., 2015; Munneke et al., 2018; Newman & Wallace, 1993).

Some evidence to support the *response modulation theory* comes from studies that have identified attentional abnormalities in psychopathy. Lorenz and Newman (2002) suggested that deficient response modulation may underlie the patterns of emotional and cognitive deficits detected among psychopaths during a lexical decision

task. While individuals were found to be capable of normal emotional responses (i.e., normal appraisal) they had difficulty processing affective information (i.e., impaired use of emotional cues) peripheral to primary attentional focus. Similarly, Dadds et al. (2006), based on early developmental psychopathy work with children, posited that a dysfunction in attentional mechanisms, specifically abnormal attention to socially relevant cues, may cause the observed emotion recognition deficits. In line with Newman et al. (2010) and Dadds et al. (2006), Decety et al. (2014) suggested that the mechanisms for instrumental learning may be intact but the abnormal intake of affective information drives learned behaviour, and that if distress cues are processed in an atypical manner, functional empathy may not develop. Recently, Munneke et al. (2018) found evidence for top down attention to emotional faces among individuals with elevated psychopathic traits; however, there were different response patterns for happy and fearful faces, suggesting that top-down attention may not determine the processing of all types of emotional facial expressions in psychopathy.

A substantial number of neurobiological studies also provide evidence that psychopathic traits are associated with a difficulty processing others emotional displays of distress (such as fear, sadness and pain), which is related to the lack of empathy displayed (Blair, 2015). This reduced empathic responding has been linked to decreased amygdala responsiveness to distress cues, while deficits in decision making and reinforcement learning have been associated with dysfunction in the vmPFC and striatum (Blair, 2013). Stimulus reinforcement learning (i.e., aversive conditioning) studies suggest that if distress cues serve as aversive social reinforcers, then an individual who finds actions that cause others distress to be less aversive would be more likely to engage in antisocial behaviour, such as aggression (Blair, 2013). The amygdala

has been shown to be critical for stimulus-reinforcement learning (threat/fear, learning and memory) (Sears, Schiff, & LeDoux, 2014), and responds to distress cues, particularly fear (Murphy, Nimmo-Smith, & Lawrence, 2003), but also sadness (Blair et al., 1999) and pain in others (Lamm, Decety, & Singer, 2011).

In psychopathy studies, both the amygdala and vmPFC have been found to be involved in making moral judgements about actions that could harm others (Blair, 2013). Children, adolescents and adults with psychopathy traits (particularly callous unemotional traits) have also been found to display reduced amygdala responsiveness to distress cues (Decety, Skelly, & Kiehl, 2013; Marsh et al., 2013; Marsh et al., 2008); as well as during aversive conditioning (stimulus-reinforcement learning) (Birbaumer et al., 2005). Consequently, it has been suggested that these findings are consistent with the idea that dysfunctional learning results in individuals who socialise poorly (Blair, 2015). In addition, poor fear conditioning in young children has been found to be predictive of the development of antisocial behaviour (Gao, Raine, Venables, Dawson, & Mednick, 2010).

Extensive evidence supports atypical emotion processing in psychopathy. Including studies of: executive function, revealing deficits for tests with affective components (Lapierre, Braun, & Hodgins, 1995); poor performance on cognitive empathy tasks (Brook & Kosson, 2013); deficits in emotion recognition (Dawel et al., 2012; Marsh & Blair, 2008; Wilson et al., 2011); a lack of automatic avoidance behaviour to socially threatening cues, such as angry faces (Louise von Borries et al., 2012); and reduced amygdala activity and medial frontal cortex to fearful faces among individuals with callous unemotional traits (Birbaumer et al., 2005; Deeley et al., 2006;

Gordon et al., 2004; Han, Alders, Greening, Neufeld, & Mitchell, 2012; Marsh et al., 2008). However, the emotion recognition findings have been mixed due to methodological and sampling limitations, with one study finding no difference in facial affect recognition performance between psychopathic offenders and healthy controls (Glass & Newman, 2006). Overall, impairments in emotion recognition have been found among children, adolescents and adults with psychopathic tendencies (Blair, Colledge, Murray, & Mitchell, 2001a; Dadds, El Masry, Wimalaweera, & Guastella, 2008; Dadds et al., 2006), as well as a decline in recognition accuracy related to increased psychopathy traits (Dolan & Fullam, 2006).

A range of criticisms of existing meta-analyses of emotion recognition have been expressed, including the limited numbers of studies for some categories of emotion (resulting in low reliability), and the collapsing of child, adolescent and adult samples based on psychopathy measures, when they may indeed differ in cognitive and affective capabilities (Hoppenbrouwers, Bulten, & Brazil, 2016a). More recently, neuroimaging researchers have attempted to address some issues related to inconsistent findings. An fMRI study using dynamic facial emotional stimuli to examine neural processing found that individuals with high psychopathy traits (on the PCL-R) displayed a reduction in neuro-hemodynamic response in the face processing (inferior occipital gyrus, fusiform gyrus, and STS) and extended networks (IFG, OFC, and vmPFC)) compared to controls across four categories of emotion, including fear, sadness, happiness and pain (Decety et al., 2014). These findings are consistent with a more pervasive deficit across emotions, as proposed by Dawel et al. (2012), in contrast to a fear specific deficit or one limited to expressions of distress (i.e., fear, sadness and pain), as proposed by Marsh and Blair (2008). However, Blair (2015) points out that the processing of anger and

disgust tend to be relatively preserved in psychopathy, on the basis of findings from the Dawel et al. (2012) meta-analysis.

Findings from structural Magnetic Resonance Imaging (sMRI) studies among individuals with antisocial PD, who also met criteria for psychopathy, have led to suggestions that psychopaths brains may differ structurally, with gray matter volume reductions in frontopolar and orbitofrontal regions, anterior temporal cortex, the STS and insula (Müller et al., 2008). Once again, evidence varies, and methodological differences make direct study comparisons difficult. For example, as noted by Gregory et al. (2012) there have been differences in techniques (i.e., whole brain versus manual tracing); assessment methods (i.e., PCL-R cut off scores ranging from 23 to 30); sample sizes; and mixed diagnostic groupings (i.e., antisocial or borderline PD, schizophrenia spectrum and comorbid substance use disorders). All of which could independently be associated with differences in the brain regions of interest (Gregory et al., 2012). However, the structural imaging (sMRI) study by Gregory et al. (2012), among 44 violent male offenders diagnosed with antisocial PD, suggests that psychopathy may represent a distinct neurodevelopmental sub-group or phenotype. This suggestion was based on differences identified in key brain regions important in understanding emotions, including: significantly reduced gray matter volume bilaterally; in the anterior rostral prefrontal cortex (Brodmann area 10); and temporal poles (Brodmann area 20/38) (Gregory et al., 2012). MRI scans identified structural brain abnormalities only among offenders with antisocial PD who also met criteria for psychopathy, when compared to offenders with antisocial PD who did not meet criteria for psychopathy, and healthy non-offenders (Gregory et al., 2012). These researchers propose that the reduced gray matter volume in areas implicated in empathic processing, moral reasoning and

pro-social emotion (i.e., guilt and embarrassment) may contribute to the social behaviour abnormalities observed in psychopathy (Gregory et al., 2012).

Of particular relevance to this dissertation, only a small number of researchers have examined the impact of coexisting psychopathy on facial affect recognition among offenders with schizophrenia. Fullam and Dolan (2006b) using a forensic group of patients with schizophrenia, were one of the first to explore the relative contributions of psychopathic traits to disturbed facial affect recognition. In their study, faces were morphed to create variable expression intensity. Patients with schizophrenia who had a higher number of psychopathic traits showed greater impairment in the recognition of sadness at the lowest intensity (Fullam & Dolan, 2006b). Recognition accuracy for disgust was also negatively related to severity of cognitive symptoms (Fullam & Dolan, 2006b). A subsequent functional imaging (fMRI) study among violent patients with schizophrenia revealed that individuals with high levels of psychopathic traits displayed blunted amygdala response to fearful faces (Dolan & Fullam, 2009). In addition, for sub-facets of psychopathy, specifically antisocial groups, a differential relationship to functioning in amygdala-prefrontal circuitry was found (Dolan & Fullam, 2009).

Whilst psychopathy was not assessed, another study explored the relationship between violent criminal offending, schizophrenia and emotion recognition, with criminal behaviour being associated with poorer emotion recognition, particularly for facial expressions of fear and anger (Weiss et al., 2006). A misinterpretation of social cues (i.e., angry and fearful expression) is believed to lead to a failure in socially adaptive behaviour, with aggressive and impulsive behaviour (typically associated with criminal/antisocial behaviour) thought to be a product of emotion dysregulation (Weiss

et al., 2006). Emotion dysregulation has also previously been implicated in violent behaviour in both schizophrenia and antisocial PD (Kumari et al., 2009). In addition, it has been suggested that an impaired ability to perceive and avoid dangerous situations or actions that cause harm to others, may leave individuals unaware that their behaviour unintentionally generates fear in others (Weiss et al., 2006). However, as can be ascertained from the literature reviewed here, diagnostic differences among study populations can hamper direct comparisons.

#### *1.4.3 Visual processing during face and emotion perception in psychopathy*

Eye-tracking technologies are seen as a way to look beyond mental illness, to examine the neurocognitive mechanisms underlying socio-emotional perception and processing (Elbogen et al., 2016), but they also allow an examination of visual scan-paths abnormalities and associations with characteristics such as the level of psychopathy traits. Given that psychopathy traits are linked with impaired facial expression recognition (Dawel et al., 2012; Marsh & Blair, 2008; Wilson et al., 2011), utilising eye-tracking tasks to help determine what might affect functional processes could assist in further understanding these deficits. Indeed, some of the observed deficits and dysfunction may be due, in part, to reduced attention to the eyes (Dadds et al., 2014). For example, impaired fear recognition among children with psychopathic traits has been associated with impaired eye contact (Dadds et al., 2008; Dadds et al., 2006). As is the case in patients with amygdala damage (Adolphs et al., 2005), the observed deficits have also been shown to be temporarily corrected by directing a child's focus to the eyes (Dadds et al., 2006).

Eye-tracking technology has been utilised to examine the visual strategies used by adolescent males high in callous unemotional traits during emotion processing tasks. This has revealed that high psychopathy traits predict poorer recognition of fear, with a significantly lower number, and duration of eye fixations, and fewer first foci in the eye region (Dadds et al., 2008). An impairment in fear recognition, associated with reduced attention to other people's eyes, provided some support for models of psychopathy emphasising *amygdala dysfunction* (Dadds et al., 2008; Dadds et al., 2006). In addition, visual processing in children with callous unemotional traits, characterised by a failure to attend to the eyes of attachment figures, has been found to be independent of maternal behaviour (affection and level of eye contact) and associated with psychopathic traits in fathers (including fearlessness) (Dadds et al., 2014; Dadds et al., 2012).

Dadds et al. (2012) proposed that psychopathic disorder may begin as a failure to attend to the eyes of attachment figures, with the ability to attend to others emotions important in the development of empathy. Furthermore, these impairments could have functional significance, and be potentially amenable to change, making them an important focus for longitudinal and treatment studies (Dadds et al., 2014). An impairment in fear recognition has been replicated in studies among adults high in callous unemotional traits, seen as the emotion dysfunction factor of psychopathy (Dawel et al., 2012; Marsh & Blair, 2008). Interestingly, when children with callous unemotional traits are directed to attend to the eyes, a normal level of fear recognition has been found to occur (Dadds et al 2008, 2006). Similarly, in adults with psychopathy, when attention is directed to fear stimuli normal startle response has been shown to occur (Newman et al., 2010). Newman et al. (2010) would suggest that

reduced reactivity to fear, and emotion related cues more generally, is reflected by idiosyncrasies in attention that limit processing of peripheral information, in support of the *response modulation theory*.

A study among adult males, using visual scanning technology to examine the relationship between psychopathy traits and emotion recognition, found primary psychopathy traits (selfish, uncaring) were associated with reduced attention to the eyes, which was characterised by: a reduced number of fixations; and overall dwell time on the eyes, relative to the mouth, across six expressions (Gillespie et al., 2015c). However, no relationship was apparent between psychopathy traits and recognition accuracy (Gillespie et al., 2015c). Moreover, the relationship between primary psychopathic traits and attention to the eyes of angry and fearful faces was influenced by the gender and intensity of the expression; in this instance, a greater number of fixations on the eyes (relative to the mouth) was associated with increased recognition accuracy for these emotions.

Similarly, in another eye-tracking study by the same research group among violent male offenders, reduced attention toward salient aspects of the face (i.e., the eyes) was reported to be associated with boldness psychopathic traits (Gillespie et al., 2017b). This involved a reduced number of fixations and shorter dwell times, as well as slower first fixation to the eyes (compared with the mouth); variations by emotion and with the intensity of the facial expression were also found (Gillespie et al., 2017b). These studies suggest that psychopathic traits are associated with reduced attention to the eyes of emotional faces, and support amygdala based accounts of psychopathy (Gillespie et al., 2015c). As acknowledged by the authors, the study of non-offenders

(Gillespie et al., 2015c) utilised a self-report psychopathy scale and included only males with mild levels of psychopathy, while the study among offenders (Gillespie et al., 2017b) included males with a narrow range of offences and did not assess comorbid clinical disorders.

## **1.5 Summary and thesis overview**

Research exploring facial emotion processing and visual scanning among different phenotypes has important implications, particularly when diagnostic complexities are paramount. Psychotic disorders are indeed complex, with schizophrenia often referred to as a spectrum of disorders due to the heterogeneous presentation of symptoms and treatment outcomes. For example, patients with first episode psychosis and cannabis-induced psychosis have been shown to exhibit the same visual scanning impairments, comprising fewer saccades and fixations of longer duration; however, the spatial distribution of fixations was found to be more clustered, and there was less diversity in the number of features fixated upon among individuals with cannabis-induced psychosis (Benson et al., 2007). In terms of other comorbidities, such as the impact on psychopathy traits, no studies to date have focused on visual scanning parameters among individuals with psychosis and coexisting psychopathy traits.

It remains unclear whether the specific facial emotion recognition alterations that are exhibited relate only to psychosis, or are influenced by psychopathy. Similarly, studies investigating facial expression perception in psychopathy have predominately been undertaken at the higher end of the psychopathy spectrum, among offender populations, with a focus on aggressive and violent behaviour, and they have not

explicitly examined the contribution of comorbid psychosis. Additional research among individuals with psychotic disorders, encompassing the full range of psychopathy traits, will assist in further improving our understanding of these social impairments.

Identifying active or inhibited cognitive strategies also potentially increases remediation opportunities. These could include targeted emotion recognition training, aimed at improving social cognition in psychotic disorders, particularly when diagnostic complexities are an issue and poorer social-cognitive rehabilitation outcomes are anticipated.

The following chapters examine discrete but inter-related research questions in the area of social cognition, specifically facial expression perception and processing. Chapter 2 details findings from the initial study (Study 1) aimed at extending research on *facial emotion recognition* performance in psychosis. Visual scanning techniques are utilised to explore face processing among individuals with and without a history of offending behaviour, assessed with respect to their degree of coexisting psychopathy. From the current literature, we know that individuals diagnosed with psychosis exhibit marked impairments in emotion recognition, characterised by a pattern of restricted visual scanning. There is evidence to suggest individuals who meet diagnostic criteria for psychopathy may exhibit similar impairments in emotion recognition, particular for negatively valenced emotions, although details around the emotion processing strategies employed or in indeed whether atypical patterns of visual scanning for differing emotions exist is as yet unknown.

Findings from Study 2 are detailed in chapter 3, which aimed to extend previous research on emotion processing in psychosis by examining visual scan-path

performance during a novel *emotion induction task*. The ability to evoke particular emotions is examined in terms of emotion responsivity, by comparing the same groups in Study 1 using a different task; that is, individuals with a psychotic disorder, and low to high levels of psychopathy traits, will be compared with those from a healthy comparison group. Change in mood state is assessed and visual scanning parameters, in order to explore potential differential strategies when asked to evoke or experience versus recognise emotions. In addition to the contribution of coexisting psychopathy traits, the impact of difficulties in emotion regulation are investigated (emotional clarity; emotional awareness; impulse control; emotional non-acceptance; difficulty engaging in goal-directed behaviour; limited access to emotion regulation strategies).

Chapter 4 details findings from the final *face recognition* study (Study 3), which aimed to extend research findings further by examining whether any impairment in *working memory for faces* (immediate or delayed) was apparent among individuals with a psychotic disorder, with low and high levels of psychopathy traits, when compared to a healthy comparison group. The visual scanning strategies utilised, the contribution of coexisting psychopathy traits, and neurocognitive functioning for specific domains (and overall) will be explored (including immediate and delayed memory; visuospatial/constructional abilities; language; and attention).

The final chapter (Chapter 5) provides a detailed synthesis of the key findings, as well as discussing the broader clinical relevance and implications of this research.

## **Chapter 2 – Study 1: Facial Emotion Recognition**

### **2.1 Introduction**

Individuals with mental illnesses including psychosis are over represented among offending populations, within the prison system (Prins, 2011). Evidence indicates significant comorbidity between disorders such as psychosis and psychopathy exist (Lau, 2017). However, the relative contribution with respect to aspects of facial emotion processing deficits to these disorders remains unclear. The impact that psychopathy traits may have with respect to aspects of facial emotion processing in psychosis is unclear. This first study sought to clarify research findings related specifically to facial emotion recognition deficits observed amongst individuals with psychosis, considering coexisting psychopathy traits. Currently, based on existing evidence we know both groups exhibit emotion recognition impairments, atypical visual scanning patterns, and that these emotion-processing deficits have been associated with poorer social functioning outcomes. A more focused exploration of the evidence on emotion recognition ability and scan-path performance, amongst individuals with psychosis and psychopathy will be presented.

### **2.2 Emotion recognition deficits in psychosis.**

#### *2.2.1 Emotion recognition performance*

Individuals with psychosis exhibit marked impairments in the ability to perceive and interpret facial displays of emotion (Chan, Li, Cheung, & Gong, 2010; Hooker & Park, 2002; Kee et al., 2003; Kohler et al., 2000; Kohler & Brennan, 2004; Kohler et al., 2010). Essentially, these impairments in facial emotion perception involve a difficulty identifying or differentiating between emotions (contributing to misattribution errors) (Kohler et al., 2010; Schneider et al., 2006). Meta-analyses of facial emotion perception

in schizophrenia have confirmed large effect sizes, in the order of 0.91, ranging from 0.71 to 0.89 for identification, and 1.01 to 1.09 for discrimination (Kohler et al., 2010; Kurtz & Richardson, 2012). Neutral emotional stimuli, for example, have previously been observed to be misinterpreted as expressing emotion (Kohler 2003), more specifically expressing negatively valenced emotions, suggestive of a negativity bias, both in schizophrenia (Edwards et al., 2001), and in at risk groups (i.e. high schizotypal traits: Statucka & Walder, 2017). For example, one study reported a tendency for first episode psychosis patients to recognise happy faces as expressing negative emotions (Catalan et al., 2016). However, findings are mixed, other investigators reporting impaired categorical perception of ambiguous stimuli from one emotion category to another, but no negativity bias *per se* (Kee, Horan, Wynn, Mintz, & Green, 2006).

Studies examining emotion recognition performance among both at risk, and first episode psychosis, indicate deficits exist prior to onset of active psychotic symptoms (see meta-analysis: Barkl et al., 2014), with one study reporting deficits linked to perceptual aberration largely independent of broad neurocognitive deficits (Lee et al., 2015b). A study examining affect recognition performance in both early and late stages of the illness, comparing first episode psychosis and multi-episode schizophrenia patients, indicating a differential pattern of deficits may exist (Romero-Ferreiro et al., 2016). First episode patients only demonstrated impaired fear recognition, while those with multi-episode schizophrenia exhibited a more generalised deficit, with impaired recognition of angry, sad and fearful faces, as well as misattribution of neutral faces as expressing emotion (Romero-Ferreiro et al., 2016). More broadly, increased social-cognitive impairment in later illness stages is consistent with the evidence from longitudinal psychosis studies, which have found men with

longer illness duration exhibit increased disability both socially and globally (Hanlon et al., 2017).

Facial emotion perception is crucial in social interactions, with impairments suggested as a specific cognitive marker for schizophrenia (She et al., 2017). These deficits are strongly associated with poorer social and functional outcomes, including psychosocial and occupational functioning, independent living ability, subjective wellbeing and quality of life (Green et al., 2012; Hooker & Park, 2002; Horan et al., 2012; Huang & Hsiao, 2017; Kee et al., 2003; Kohler et al., 2010). Attribution or categorisation errors are thought to contribute to impaired socialisation, due to the misinterpretation of subtle ambiguous cues (Song et al., 2015). Of clinical significance, it has been suggested that this tendency to misinterpret emotions may assist in understanding the mechanisms by which paranoid symptoms arise and persist, and along with misjudging language and behaviour, may potentially contribute to the development of persecutory delusions (Song et al., 2015).

Severity of positive symptoms and mania have been found to correlate with facial affect processing performance, with reduced accuracy in affect matching (Rossell, Van Rheenen, Joshua, O'Regan, & Gogos, 2014). While schizophrenia and bipolar disorder represent separate diagnostic constructs, associated behavioural, cognitive, and brain abnormalities overlap, including aspects of facial emotion recognition deficits (Goghari & Sponheim, 2013; Rossell et al., 2014). Studies comparing emotion recognition performance, report more subtle deficits in bipolar disorder, in comparison to more pronounced deficits in schizophrenia (Rossell et al., 2014). Specifically in recognising angry expressions compared to bipolar patients and healthy controls, while

both patient groups are more likely to mislabel anger as fear (Goghari & Sponheim, 2013). Similarly, another study demonstrated impaired recognition of angry, disgusted, sad and happy facial expressions among schizophrenia patients, while bipolar disorder patients only exhibited impaired recognition of disgusted and happy expressions in comparison to healthy controls (Yalcin-Siedentopf et al., 2014). In comparing patient groups with remitted symptoms, those with bipolar disorder outperformed those with schizophrenia, specifically in recognising angry expressions (Yalcin-Siedentopf et al., 2014). Another study of schizophrenia and bipolar patients during acute psychosis and seven weeks after antipsychotic treatment, found similar to those with schizophrenia, bipolar patients exhibited deficits in recognising both happy and sad expressions, which did not resolve with treatment (Daros, Ruocco, Reilly, Harris, & Sweeney, 2014). While overall poorer performance following antipsychotic treatment was related to the ongoing severity of negative symptoms among otherwise clinically stable schizophrenia patients (Daros et al., 2014). These findings suggest, during periods of symptomatic remission, patients with bipolar disorder exhibit less impaired facial emotion recognition than those with schizophrenia (Yalcin-Siedentopf et al., 2014).

Research demonstrating emotion-processing deficits among other clinical groups indicates a need to consider diagnostic complexity, as individuals will not necessarily meet diagnostic specificity for only one condition. Psychotic disorders such as schizophrenia are complex, and are characterised by several independent symptom domains, specifically positive (e.g., delusions, and hallucinations), disorganised (e.g., disorganised speech) and negative (e.g., flat affect, social withdrawal) symptom clusters (American Psychiatric Association, 2013), which can restrict individuals in meaningful social engagement (Shayegan & Stahl, 2005). Most individuals with schizophrenia

present a mixed syndrome. Individuals experiencing paranoid (positive) symptoms exhibit impaired judgement and reasoning (Iqbal, Birchwood, Chadwick, & Trower, 2000), believed to contribute to emotion perception problems (Combs & Gouvier, 2004). Impaired emotional functioning is a prominent feature, with negative symptoms, including flat affect, contributing to debilitation and treatment resistance (Gur et al., 2006). Severity of negative symptoms being strongly correlated with facial emotion processing, with improved performance associated with less severe symptoms (Daros et al., 2014; Heimberg et al., 1992; Nieman, Bour, Linszen, Gersons, & Ongeboer de Visser, 2005; Sachs et al., 2004; Turetsky et al., 2007).

Evidence supports emotion processing deficits as both state and trait dependent, with facial affect recognition deficits associated with symptomatic remission (i.e., trait dependent), indicating these deficits are an integral part of the disorder, but they are also impacted during active phases (i.e., state dependent) of psychosis (Daros et al., 2014; Maat et al., 2015). In addition, a meta-analysis of facial emotion identification studies among early-onset and first-episode psychosis confirmed trait-like generalised deficits as being present at illness onset, prior to diagnosis, with a substantial effect size for facial emotion identification of -0.88; indicative of significantly poorer emotion recognition accuracy compared to healthy controls (Barkl et al., 2014). Research evidence provides support, that some emotions are harder to identify, with among early onset and first-episode patients, the largest magnitude of impairments reported for disgust, fear and surprise, followed by medium impairments for sad and happy facial expressions, while no differences for anger and neutral expressions were reported (Barkl et al., 2014).

Studies addressing heritability provide additional support that emotion recognition deficits may be an endophenotype for schizophrenia, revealing facial emotion recognition deficits among psychosis patients (including first-episode) as well as their first degree relatives (Allott et al., 2015; Li, Chan, Zhao, Hong, & Gong, 2010), although findings are inconsistent with some studies reporting no deficits among first degree relatives (Toomey et al., 1999). Impaired performance for anger, disgust and fear among first-episode schizophrenia, and poorer recognition of fear among their first degree relatives, compared to health controls was found in one study, supporting deficits specifically in recognising fear as potentially a heritable characteristic of schizophrenia (Allott et al., 2015). Similarly, a longitudinal study of ultra-high risk individuals examining whether emotion recognition performance predicts transition to psychosis, found while overall facial emotion recognition accuracy was not predictive, recognition accuracy specifically for neutral and fearful emotions predicted transition to psychosis (Allott et al., 2014). Reduced accuracy identifying neutral emotion, and increased accuracy for fear, were both predictive of transition to a psychotic disorder within 12 months (Allott et al., 2014). While a recent meta-analysis among individuals at ultra-high risk for psychosis, highlighting impairments in facial affect recognition, as well as theory of mind as being predictive of transition to psychosis (van Donkersgoed, Wunderink, Nieboer, Aleman, & Pijnenborg, 2015).

Furthermore, a study examining facial affect recognition across illness phases, which examined PANSS symptom factors has shown: in first episode psychosis, the depressed factor was positively correlated with fear recognition accuracy; while among later stage multi-episode schizophrenia, the negative factor positively correlated with fear recognition, and the recognition of sad and neutral expressions inversely associated

with the disorganised factor (Romero-Ferreiro et al., 2016). In addition, the misattribution of neutral expressions with increased attribution of emotional salience was characteristic of social cognition at later illness stages (Romero-Ferreiro et al., 2016). The relationship between emotion recognition and negative symptoms have been confirmed across a number of studies that utilised both facial affect detection (identification) and matching tasks (Martin et al., 2005; Norton, McBain, Holt, Ongur, & Chen, 2009). Likewise, associations have been detected between facial affect search performance and severity of negative symptoms in schizophrenia (She et al., 2017). Indicating that the relationship between facial affect processing deficits and negative symptoms is both reliable and generalizable across different tasks, and reinforcing emotion recognition deficits as a candidate cognitive marker for schizophrenia (She et al., 2017).

Importantly, emotion recognition ability is crucial in predicting social and occupational functioning, in addition to the contribution of clinical symptoms and non-social neurocognition in schizophrenia (Gur et al., 2006; Huang & Hsiao, 2017). Emotion recognition deficits have been related to a variety of social competence and functioning outcomes, that remain relatively stable over time (Yalcin-Siedentopf et al., 2014). Emotion recognition task performance has been suggested to be indicative of early phase perception difficulties being related to down-stream processes of judging emotional implications, though to lead to dysfunctional problem solving in social settings, impacting community functioning (Abdi & Sharma, 2004; Huang & Hsiao, 2017). While imaging research, including a recent study of facial emotion processing in first episode schizophrenia, has found that hypo-metabolism in brain regions is implicated in emotion processing, and a failure to optimally recruit brain circuitry during

emotion processing, irrespective of confounding neuroleptic effects and illness duration (Choudhary et al., 2015).

Essentially, impairments in recognising facial expressions of emotion are a widely accepted, prominent and stable feature of schizophrenia (Chan et al., 2010; Kohler et al., 2010), although the underlying mechanisms remain unclear (Lee et al., 2010). Some studies have reported slower facial affect recognition, among those at risk of psychosis (Eack et al., 2010), and in schizophrenia (Li et al., 2010), although only slightly but not significantly slower in some studies (Gur et al., 2002). The core presence of generalised emotion recognition deficits, prior to (Allott et al., 2014) and early in illness onset (Barkl et al., 2014), and across illness phases (Daros et al., 2014; Maat et al., 2015), highlighting the need for treatments addressing these emotion processing deficits, with the potential to improve functional outcomes in psychosis.

### *2.2.2 Visual scanning: eye-tracking abnormalities*

Established evidence has shown that visual processing is not the same for all basic emotions (Calder, Young, Keane, & Dean, 2000; Hoffmann et al., 2013), and it is not the entire face but specific key components that provide information as the basis for classification, with the relevance of each dependent on the basic emotion expressed (Hoffmann et al., 2013). Investigations of how patients with schizophrenia use specific aspects of visual information to decode facial displays of emotion, revealed that accurate expression identification required exposure of more facial areas (Lee et al., 2010). Such studies have provided evidence of atypical strategies for using visual information in emotion recognition (Lee et al., 2010). Another study utilising a bubbles technique to determine what facial information was critical in correct identification

(Gosselin & Schyns, 2001), found that patients identification of happy expressions relied more on mouth and eye regions, and less on bilateral eye regions in identifying fearful faces.

A number of eye-tracking studies reveal that atypical visual scanning during emotion recognition processing in psychosis is characterised by a restricted scan-path strategy. This includes fewer fixations of longer duration, reduced scan-path length, and distance between fixations, as well as fewer fixations on salient facial features like the eyes and mouth, that are likely to assist in discriminating affect (Loughland et al., 2002a, 2002b; Williams et al., 2001). These eye-tracking abnormalities have been confirmed as being present across illness phases, including in at risk groups (Benson et al., 2007; Edwards et al., 2001; Habel et al., 2006; Lee et al., 2015a; Loughland et al., 2004a; Mikhailova et al., 1996; Toomey et al., 1999; Waldeck & Miller, 2000; Walker & al., 1993). Consequently, it is likely that these visual scan-path deficits were present before illness onset (Habel et al., 2006), consistent with a trait based deficit (Beedie et al., 2011; Edwards et al., 2001) and potentially representing a vulnerability marker in the familial transmission of schizophrenia (Loughland et al., 2004a). These findings are supported by recent studies utilising scan-path indices, to produce integrated eye-movement scores, which have been able to distinguish individuals with schizophrenia from healthy controls (Benson et al., 2012; Miura et al., 2014; Morita et al., 2017).

Interventions that target improving emotion recognition have the potential to improve social functioning in schizophrenia (Goghari & Sponheim, 2013). Of clinical utility, some successful remediation outcomes have already been achieved. This includes a local pilot study of bottom-up biofeedback remediation training targeting low

level visual processing during emotion recognition, which demonstrated normalising of some visual scan-path parameters, including a significant increase in scan-path length along with improved emotion recognition performance (McCabe, Loughland, Hunter, Lewin, & Carr, 2010). More recently, advances towards readily available online social cognition interventions such as, “e-Motivational Training” for schizophrenia (Maroño Souto et al., 2018), provide promising new clinical tools for potentially improving emotion recognition ability in complex and difficult to engage populations.

## **2.3 Emotion recognition deficits in psychopathy**

Psychopathy is characterised by interpersonal (i.e., callousness, manipulative, grandiose), emotional (i.e., lack of remorse & empathy, blunted emotional experience) and behavioural traits (i.e., impulsivity, irresponsibility) (Burley et al., 2017). The disorder now more viewed as a dimensional construct, has allowed researchers to investigate psychopathy traits within community samples (Burley et al., 2017). A number of studies have reported face recognition deficits, particularly for fear, among both psychopathic and antisocial populations (see meta-analyses by Dawel et al., 2012; Marsh & Blair, 2008; Wilson et al., 2011). Although deficits appear to extend beyond processing fear or distress (Dawel et al., 2012), as findings have differed in relation to the specificity of the impairment.

### *2.3.1 Emotion recognition performance*

Impairments in emotion recognition have been associated with psychopathy traits among child, adolescent and adult samples (Blair et al., 2001a; Dadds et al., 2008; Dadds et al., 2006). Studies finding deficits specifically in recognising fear and sad facial expressions among both children with psychopathic tendencies (Blair, Budhani,

Colledge, & Scott, 2005; Stevens, Charman, & Blair, 2001) and adults with psychopathy (Blair et al., 2004), coupled with imaging studies reporting reduced amygdala activity during affect processing (Birbaumer et al., 2005; Gordon et al., 2004) have been suggested to provide support for an amygdala model of psychopathy (i.e., *Integrated Emotion Systems model*) (Blair, 2006; Blair et al., 2005). However, not all studies support facial affect recognition deficits as being associated with psychopathy traits. For example, Glass and Newman (2006) found no evidence of facial affect recognition deficits for anger, fear, happy or sad expressions during an emotion matching task among psychopathic offenders, with performance comparable to controls. Similarly, Kosson, Suchy, Mayer, and Libby (2002) reported finding deficits in the recognition of disgust but no psychopathy related deficits in the ability to recognise anger, fear, happy or sad expressions among criminal psychopaths. While Habel, Kuhn, Salloum, Devos, and Schneider (2002) reported poorer performance during an emotion discrimination task in psychopathy, but improved performance was related to higher scores for the emotional detachment factor on the PCL.

Evidence from existing meta-analyses has differed, with variation in the studies included (i.e., adult, child and adolescent samples; including antisocial, psychopathy traits, and psychopathic offender populations; as well as variations in tasks). A meta-analysis by Wilson et al. (2011) reported psychopathy was associated with recognition deficits across multiple emotions (although effect sizes were small  $r = 0.06 - 0.12$ ), with larger effect sizes for fear and sadness. Similarly, Dawel et al. (2012) identified deficits for fear, sad, happy, and surprised facial expressions, not only fear and sadness (or distress) as previously reported by Marsh and Blair (2008) among antisocial populations. A recent neuroimaging study also identified reduced neuro-hemodynamic

responses in face processing (inferior occipital gyrus, fusiform gyrus, and STS) and extended networks (IFG, OFC, and vmPFC), among individuals with high psychopathy (based on PCL-R scores), while viewing dynamic face stimuli across four categories of emotion (fear, sadness, happiness and pain) (Decety et al., 2014). Similarly, another recent study, reported psychopathy related emotion recognition deficits (for sad, happy, and fear), were associated changes in brain morphology, involving gray matter volume reductions in the prefrontal cortex, somatosensory cortex, anterior insula, cingulate cortex and posterior lobe of the cerebellum (Pera-Guardiola et al., 2016). Adding further support to a more pervasive deficit in facial emotion recognition in psychopathy.

In line with a more generalised deficit, an alternate view to an amygdala-mediated dysfunction associated with responsiveness to distress, is that emotion recognition deficits may occur as a consequence of attentional deployment (i.e., *Response Modulation Hypothesis*) (Newman et al., 2010). Whereby, high-order cognitive processes may moderate deficits, related not only to diminished reactivity to fear stimuli but emotion cues more generally (Newman et al., 2010). A recent study by Munneke et al. (2018), using a visual search task found top-down attention influences the way emotional faces attract attention among individuals with high psychopathy traits. Although, different response patterns for happy and fear suggest top down attention may not determine the processing of all types of facial expressions of emotion in psychopathy (Munneke et al., 2018).

A decline in emotion recognition accuracy has also previously been reported as being associated with an increased number of psychopathy traits (Dolan & Fullam, 2006). A recent study by Cigna, Guay, and Renaud (2017) among undergraduate male

students, examining the degree of psychopathic traits and emotion recognition using dynamic emotion stimuli, reported higher levels of antisocial behaviour was associated with poorer performance, and a specific impairment in the recognition of sadness. Callous affect, was also positively correlated with identifying sad expressions predicting heightened sensitivity to facial affect (Cigna et al., 2017). This study raises an important point, in that the ability to recognise signs of vulnerability may be an adaptive strategy among some psychopathic individuals, useful in manipulating others, and highlights that distinct facets of psychopathy in facial emotion recognition may have important implications (Cigna et al., 2017).

Forensic studies with an interest in examining associations between psychopathy, violence and criminality, have included a few psychiatric samples. One such emotion recognition study using static images during an identification task (black and white photographs displayed for 170ms) among males from a psychiatric hospital, found psychopathy was associated with deficits in recognising both fear and surprise, regardless of criminality (Stanković, Nešić, Obrenović, Stojanović, & Milošević, 2015); however, no details on psychiatric diagnoses were provided. Another study by Wolfkuhler et al. (2012), among forensic and non-forensic patients with schizophrenia, found impaired emotion recognition across all emotion tasks, in comparison to controls, as well as superior recognition for disgust among the forensic group; however, psychopathy traits were not assessed.

Of particular interest here, one study examining the impact of psychopathy traits on emotion recognition in schizophrenia found that patients with a high number of psychopathic traits exhibited increased impairment in the recognition of sadness at the

lowest intensity, while recognition accuracy for disgust was negatively related to severity of cognitive symptoms (Fullam & Dolan, 2006b). Another neural processing study, among violent patients with schizophrenia, also revealed that individuals with high levels of psychopathic traits display blunted amygdala response to fearful faces (Dolan & Fullam, 2009). In addition, within that study, for sub-groups with antisocial characteristics, a differential relationship was found with respect to functioning in amygdala-prefrontal circuitry (Dolan & Fullam, 2009).

Whilst coexisting psychopathy traits were not examined, findings from an event related potentials (ERP) study also have relevance here, as they investigated whether emotion perception deficits, leading to misinterpreting neutral stimuli as threatening, increased the risk for violence (De Sanctis et al., 2013). They found that sensory-perceptual processing dysfunction at P35 and P100 for negatively valenced stimuli was pronounced (N100) among individuals with schizophrenia and a history of violence (De Sanctis et al., 2013). While no difference in task accuracy was found, responsivity differences were evident, with around a 10ms faster mean reaction time among patients with a history of violence compared to healthy controls and the psychosis group with no history of violence (De Sanctis et al., 2013). Abnormal processing of negatively valenced stimuli was more pronounced among violent individuals, with this study being one of the first to report electrophysiological measures that distinguish patients depending on history of violence, linking processing impairments for affective inputs to violent behaviour in schizophrenia (De Sanctis et al., 2013). Of significance, these findings imply more severe emotion processing deficits may be apparent among schizophrenia patients with a history of violence, although, as already noted, psychopathy traits were not assessed.

### 2.3.2 *Visual scanning: eye-tracking abnormalities*

A study among adult males used visual scanning technology, to examine the relationship between psychopathy traits and emotion recognition (Gillespie et al., 2015c). They found that primary psychopathy traits (selfish, uncaring) were associated with reduced attention to the eyes, characterised by a reduced number of fixations, and overall dwell time on the eyes relative to the mouth, across six expressions (Gillespie et al., 2015c). However, this study found no relationship between psychopathy traits, either primary or secondary (impulsive, antisocial) and recognition accuracy (Gillespie et al., 2015c). The relationship between primary psychopathic traits with attention to the eyes of angry and fearful faces was influenced by the gender and intensity of the expression, with a greater number of fixations on the eyes (relative to the mouth) being associated with increased recognition accuracy for these emotions (Gillespie et al., 2015c).

Another study by the same research group, examining eye movements among violent male offenders, also reported reduced attention toward salient aspects of the face (i.e., the eyes). Psychopathic traits of boldness (on the Triarchic Psychopathy self-report measure) were associated with a reduced number of fixations, and dwell time, as well as slower first fixation to the eyes (compared with the mouth). Variations with emotion and intensity of the facial expression were also found, implying visual eye-tracking scan-paths are sensitive to stimulus driven effects (Gillespie et al., 2017b). These studies suggest that psychopathic traits are associated with reduced attention to the eyes in emotional faces, and support amygdala based accounts of psychopathy (Gillespie et al., 2017b; Gillespie et al., 2015c).

Similarly, studies among children and adolescents with callous and unemotional psychopathy traits, have found impaired fear recognition as being associated with impaired eye contact (Dadds et al., 2008; Dadds et al., 2006); including a significantly lower number of, and duration of eye fixations, as well as fewer first foci in the eye region (Dadds et al., 2008). These fear recognition deficits have been shown to improve by directing a child's focus to the eyes (Dadds et al., 2006), suggesting they may be amenable to change, which has important implications for potential early interventions (Dadds et al., 2014). As noted earlier, further research by this same group has also found visual processing among children with callous and unemotional traits is characterised by a failure to attend to the eyes of attachment figures, and is associated with the presence of paternal psychopathy traits (Dadds et al., 2014; Dadds et al., 2012).

Findings from another recent eye tracking study among healthy young adults examining facets of psychopathy (using the Psychopathic Personality Inventory), found fearless dominance (interpersonal-emotional facet), described as equivalent to boldness in the Gillespie et al. (2017b) study, and cold heartedness was associated with reduced scanning behaviour for emotional faces, consistent with lower emotional reactivity (Boll & Gamer, 2016). While participants high in social deviance (self-centred impulsivity), equivalent to disinhibition in the Gillespie et al. (2017b) study, showed a reduced bias to shift attention towards the eyes (Boll & Gamer, 2016). Furthermore, these studies provide some support to notion that distinct facets of psychopathy may modulate face processing, and that attentional mechanisms might be responsible for the social perception and behaviour impairments observed (Boll & Gamer, 2016; Gillespie et al., 2017b). However, due to differing study samples, measures (i.e., similar and differing

constructs of psychopathy) as well as emotion processing tasks (i.e., stimulus presentation features, and display times), direct study comparisons can be challenging.

## **2.4 Aims and hypotheses**

This initial study aimed to extend existing research findings on emotion recognition deficits in psychosis utilising visual scanning techniques by examining the impact of coexisting psychopathy traits on facial emotion processing performance. What we know from the extensive existing literature is that individuals diagnosed with a psychotic disorder exhibit impairments in emotion recognition (Kohler et al., 2010; Kurtz & Richardson, 2012), with a pattern of restricted visual scanning (Loughland et al., 2002a, 2002b; Williams et al., 2001). There is evidence that individuals who meet criteria for psychopathy also exhibit impairments in emotion recognition (Dawel et al., 2012; Wilson et al., 2011), particularly a difficulty with negatively valenced emotions, such as fear. However, deficits may be more pervasive across emotions, not unlike those seen in psychosis. Visual scanning abnormalities among individuals with high psychopathy traits have been found among general community (Gillespie et al., 2015c), and violent offender populations (Gillespie et al., 2017b). However, among individuals with psychosis, details around the emotion processing strategies used by those with high levels of psychopathy, or indeed, whether there is a restricted pattern in visual scanning, are unknown.

The primary hypotheses for this first study are: 1) Compared to a healthy control group, participants who meet criteria for a diagnosis of a psychotic disorder will exhibit poorer overall emotion recognition accuracy and an atypical restricted pattern of visual scanning; and 2) The level of impairment exhibited will increase in relation to the

number of psychopathy traits, whereby participants with a high number of traits will perform more poorly, exhibiting greater deficits in affect recognition and more restricted visual scanning, particularly for negatively valenced expressions. While current symptoms and overall cognitive function are likely to be associated with facial emotion perception performance, it is anticipated that these effects will be less marked than the hypothesised diagnosis and trait-related group differences, especially given the recruitment of community-based participants with a history of psychosis who were not acutely unwell.

## **2.5 Method**

### *2.5.1 Participants*

Sixty-three participants aged between 18 - 65 years, with no significant visual or hearing impairment, were referred to the study from Community Mental Health Services (MHS) within the Hunter New England Local Health District (HNE-LHD), or recruited as healthy controls via the broader local community of Newcastle, Australia. Thirty-nine were clinical participants recruited from Community MHS via ongoing service contact, which included in-service presentations and meetings with key members of staff. Participants were referred from a number of services, which included Newcastle, Lake Macquarie and Hunter Valley Community Mental Health (CMH), the Newcastle Mental Health Court Liaison (NMFCL) service (primary place of community referrals for offenders with a mental illness in Newcastle), Mental Health Substance Use Service (MHSUS) and Psychiatric Rehabilitation Services (PRS). In addition, HNE-LHD Community MHS clients who had previously consented to being contacted about future research projects (as part of the SHIP study; (Morgan et al., 2012)), were approached to ask if they may be interested in taking part in an emotion

processing study; however, no data from the SHIP project were utilised in the current study. Two clinical participants recruited into the study were later excluded from the psychosis group (N = 37) as there was no apparent evidence of a psychotic disorder, although they met criteria for another major mental illness (DSM-IV - Axis I Mood disorder).

The choice to participate in the study was voluntary. Participants were contacted by the research team once they expressed an interest in being involved following the receipt of an information statement (see [Appendix A](#)) about the study (in the case of the clinical participants, from Community MHS staff). Those provided with the information statement had as much time as they liked to consider participating. Clinical participants with a history of offending were only provided information about taking part in the study after any pending court matters had been resolved to ensure there could be no perceived coercion to take part. Participants who agreed to participate were contacted by phone, followed by an appointment letter sent in the mail inviting them to attend an interview at the Visual-Cognition (Eye Tracking) Laboratory at the Centre for Brain and Mental Health Research (CBMHR). Fully informed written consent was obtained prior to study commencement (see [Appendix B](#)). A study flyer (see [Appendix C](#)) and associated recruitment video was also developed (see additional details, [Appendix D](#)) and placed on a MHS collaborative space website for clinicians, and in the public domain on the University of Newcastle's CBMHR (Mental Health Hub) website. All components of the study protocol were approved by the Hunter New England Human Research Ethics Committee (HNEHEC: 07/02/21/5.05; see [Appendix E](#), for approval certification); approval was also registered with the University of Newcastle Human Research Ethics Committee (HREC: H-425-047; see [Appendix F](#)).

A staggered recruitment method was used for the comparison group of healthy control participants (N = 24) based on a general matching strategy. They were recruited via the local community, using websites (mentioned above) advertising that recruitment was underway, as well as printed flyers on notice boards. Where possible, healthy control participants were selected if they matched the demographic profile of the psychosis group participants, on the basis of age and gender (within a 5 year range; see [Appendix G](#) for a breakdown of the sample by age, gender and group). Healthy control participants were excluded from the study if they reported a family history of a psychotic disorder or a personal history of psychosis; however, no potential participants were actually excluded for this reason. Individuals with a history of severe head injury (resulting in loss of consciousness for 20 minutes or longer), a severe intellectual disability, a diagnosis of bipolar disorder, or a purely substance-induced psychosis were not included in the study. All participants were reimbursed for time and travel undertaken to take part in the study, receiving either a monetary payment of \$60 or gift card voucher of equal value.

### *2.5.2 Measures*

All recruited participants completed a structured interview that comprised a standardized set of assessments, which included a diagnostic interview to collect demographic and clinical information, a neuropsychological evaluation, behavioural and self-report measures. The complete set of assessment measures is summarised in [Table 2.1](#), together with approximate administration times. In addition, a mapping of the measures to the primary research questions addressed in this thesis is provided in [Table 2.2](#). The behavioural measures, and specifically details about the face stimuli,

experimental apparatus and visual-cognitive emotion recognition task utilised in this initial study, are provided in sections 2.5.3, 2.5.4 and 2.5.5.

#### *2.5.2.1 Clinical assessments*

Demographic information collected included age, gender, marital status, education and employment history, psychopathology, hospitalisations, medication and substance use, current social functioning and history of head injury. The Structured Clinical Interview for DSM-IV (SCID) (First, Spitzer, Gibbon, & Williams, 2001) was used to collect demographic information and confirm current or lifetime DSM-IV Axis-I disorders, Psychosis, Mood or Substance Use diagnoses. This included administration of the SCID-I overview and the following modules by a trained psychologist - A: Mood Episodes; B: Psychotic and Associated Symptoms; C: Differential Diagnosis of Psychotic disorder, D: Mood differential; and E: Substance Use Disorders. Current psychiatric symptoms were assessed using the 24-item version of the Brief Psychiatric Rating Scale (BPRS) (Ventura et al., 1993), which records the presence and severity of symptoms rated on a Likert scale ranging from 1 (Not present) to 7 (Extremely severe), with a possible range of scores from 24 to 168 (whereby lower scores indicate less severe psychopathology). In addition, clinician rated assessment of current level of functioning was undertaken using two established measures. General functioning was assessed using the DSM-IV, Axis V: Global Assessment of Functioning (GAF), measuring psychological, social and occupational functioning on a scale of 0-100 points (American Psychiatric Association, 2000). The Social Occupational Functioning Scale (SOFAS) was also utilised to assess social and occupational functioning on a scale of 0-100 points, independently of severity of psychological symptoms (American Psychiatric Association, 2000). Detailed assessment of positive and negative symptoms and

disorganisation (e.g., utilising measures such as the SAPS and SANS) were not undertaken, as it was anticipated current psychosis symptoms levels would be low among clinical participants.

The Hare Psychopathy Checklist: Screening Version (PCL: SV) was used to assess the presence of psychopathy traits (Hart et al., 1995). Derived from the PCL: R, the brief 12-item PCL: SV is an effective short form for use in non-forensic settings. Ratings of interpersonal, behavioural and affective symptoms on the PCL: SV are made on a 3-point scale, according to the degree to which a characteristic is present (0 = does not apply, 1 = applies to a certain extent, 2 = applies), and summed to obtain a total score (ranging from 0 to 24). This total score is effectively a dimensional measure of the degree to which a given individual matches the prototypical psychopath. In addition, measuring all four PCL: SV facets produces two factors (6-items each), with factor 1 assessing interpersonal and affective traits (superficial, grandiose, deceitful, lacks remorse, lacks empathy, doesn't accept responsibility), and factor 2 assessing lifestyle and antisocial features (impulsive, lacks goals, irresponsible, poor behavioural controls, adolescent antisocial behaviour, adult antisocial behaviour). The PCL: SV has been found to have sound psychometric properties, good reliability and validity, and to be strongly related to the full PCL-R, both conceptually and empirically (Cooke et al., 1999; Guy & Douglas, 2006; Hart et al., 2003).

#### *2.5.2.2 Neuropsychological assessments*

The Wechsler Test of Adult Reading (WTAR) was used to obtain an estimate of a person's reading level, and in the case of the clinical participants to derive an estimate of premorbid intellectual functioning (Wechsler, 2001). The Repeatable Battery for

Assessment of Neuropsychological Status (RBANS) was used to measure neurocognitive performance (Randolph, Tierney, Mohr, & Chase, 1998). The RBANS total scale score provides information on global level of cognitive functioning, with RBANS index scores providing information on cognitive functioning in the domains of Immediate Memory; Visuospatial/Constructional abilities; Language; Attention; and Delayed Memory.

#### *2.5.2.3 Self-report measures*

Participants completed an abbreviated version of the Profile of Mood States (POMS), which identifies and assesses mood state across 6 dimensions including, Tension/Anxiety; Depression/Dejection; Anger/Hostility; Vigour/Activity; Fatigue/Inertia; and Confusion/Bewilderment, providing a total mood disturbance score (McNair, Lorr, & Droppleman, 2005). Ratings are provided in relation to how a person is feeling “Right Now” on a Likert scale ranging from 0 to 4 for each item (0= Not at all, 1 = A little, 2= Moderately, 3 = Quite a bit, 4 = Extremely). An abbreviated version was used to monitor change in mood state (see [Appendix H](#)), which consisted of 12-items selected from the 65-item version (McNair et al., 2005), whereby two adjectives were chosen to most closely represent the labels given to each of the 6 domains. The Difficulties in Emotion Regulation Scale (DERS) (Gratz & Roemer, 2004) was also used, consisting of 36-items rated with respect to how often each statement applies, using a Likert scale ranging from 1 to 5 (1= Almost Never, 2 = Sometimes, 3 = About half of the time, 4 = Most of the time, 5 =Almost always). The DERS identifies and assesses six facets of emotion regulation including: difficulties with emotional clarity; emotional awareness; impulse control; emotional non-acceptance; difficulty engaging in goal-directed behaviour; limited access to emotion regulation strategies; as well as providing a total emotion dysregulation score. The scale has been reported to have sound

psychometric properties, high internal consistency, good test-retest reliability, and construct and predictive validity (Gratz & Roemer, 2004).

A modified 20-item version of the Childhood Adversity Questionnaire (CAQ) (Rosenman & Rodgers, 2004) was used to obtain additional information on experiences of adversity. The CAQ is a composite measure of items adapted from the Parental Bonding Instrument (Parker, 1979), the British National Survey of Health and Development (Parker, Tupling, & Brown, 1979), the US National Comorbidity Survey (Riso, Miyatake, & Thase, 2002), and the Australian cross sectional study (Henderson et al., 1998). The first six-items assess parental affection (0 = A little, somewhat or very affectionate; 1 = Not at all affectionate, 2 = No father figure); nervous, emotional problems or depression; and alcohol or substance use problems (0= No problems, 1 = Had problems). The next two items record household conflict and tension (0 = No conflict, 1 = Some, 2 = A lot) and parental divorce or separation (0= No parents together throughout childhood, 1 = Separation/divorce). A further nine items identify parental physical, psychological or sexual abuse, neglect, authoritarian upbringing, poverty or financial hardship, excessive physical punishment or witnessing physical or sexual abuse of others in the family (0 = No, 1 = Yes); with a further three items assessing positive aspects of upbringing, including having a happy childhood, normal upbringing, or parents doing their best (1 = No, 0 = Yes) (Rosenman & Rodgers, 2004). Additional items assessing loss due to the death of a parent, sibling or significant other (0= No, 1 = Yes), were also included, consistent with CAQ administration among an Australian psychosis and community sample (McCabe, Maloney, Stain, Loughland, & Carr, 2012).

A modified version of the Assessment of Quality of Life instrument (AQoL-6D) (Richardson, Peacock, Lezzi, Atherton-Day, & Hawthorne, 2007; Richardson et al., 2012) was used to provide a multidimensional evaluation of health related impairment in quality of life. The AQoL-6D contains 20 items, rated using multi-tier descriptive response options ranging from 4-6 levels. Higher AQoL-6D scores indicate quality of life impairment, which can be summarised into six dimensions: 1 – Independent living (4 items); 2 – Relationships (3 items); 3 – Mental Health (4 items); 4 – Coping (3 items); 5 – Pain (3 items); and 6 – Senses (3 items). Australian normative data are available among community samples (Allen, Inder, Lewin, Attia, & Kelly, 2013), and the measure has sound psychometric properties, including construct, concurrent and convergent validity (Allen et al., 2013; Richardson et al., 2012).

The 59-item screening version of the International Personality Disorder Examination Questionnaire (IPDEQ) (Loranger, Janca, & Sartorius, 1997) was used to assess for possible Axis II personality disorders. The IPDEQ is a self-report screening version developed by the World Health Organization based on the original IPDE semi-structured clinician administered interview (Loranger et al., 1997). The IPDEQ items assess the nine ICD-10 personality disorders: including Cluster A – paranoid and schizoid; Cluster B – dissocial, impulsive, borderline and histrionic; and Cluster C – anankastic, anxious and dependent. The measure is relatively brief and found to have satisfactory psychometric properties (Lewin, Slade, Andrews, Carr, & Hornabrook, 2005).

### 2.5.3 Facial expression stimuli

Facial expression stimuli were utilised to assess emotion processing across three separate visual-cognitive eye-tracking tasks. Details on the face stimuli used in the first emotion recognition task are provided here (stimuli utilised in the remaining tasks are detailed in subsequent chapters). The stimuli consisted of 28 colour images, equal numbers of male and female actors depicting one of seven facial expressions, selected for high interrater agreement for categorisation of each emotion (Mean validation score = 0.81, SD = 0.16) from the standardised NimStim set of facial expressions (Tottenham et al., 2002; Tottenham et al., 2009; <http://www.macbrain.org/resources.htm>). All 7 categories of stimuli were selected based on validity data that indicated a moderate-high mean proportion correct for the chosen facial expressions: happy (M = 0.93, SD = 0.03), sad (M = 0.87, SD = 0.08), surprise (M = 0.83, SD = 0.09), anger (M = 0.91, SD = 0.13), fear (M = 0.58, SD = 0.85), disgust (M = 0.94, SD = 0.21), and neutral (M = 0.61, SD = 0.07). A neutral expression was included in order to assess performance when not viewing emotional faces. All stimuli had a resolution of 506 × 650 pixels, displayed within equivalent parameters on a computer screen, in order to maintain a similar set location of facial features, such as eyes and mouth across all stimuli presentations.

Table 2.1: *Key clinical and behavioural assessment measures*

Assessment Measure	Administration time	Key Variables
<i>Structured Clinical Interview</i>		
1. Demographics	5-10 min	Age, gender, education, employment history, past psychopathology, hospitalisations, medication and illicit substance use, and head injury
2. SCID – Psychosis, Mood and Substance use disorder modules	30-45 min	DSM-IV Axis I diagnoses: Current & Lifetime Psychosis, Mood and Substance Use Disorders
3. PCL: SV - Part I: Interview Schedule:	30-45 min	School adjustment, work history, health problems, goals, family history, sexual relationships, child/adolescent impulsive and antisocial behaviour, adult impulsive and antisocial behaviour, and general questions (to ascertain level of Psychopathy traits)
4. BPRS	15-20 min	Psychiatric Symptomatology ratings
5. GAF	2-3 min	Single rating of Global Assessment of (psychological, social and occupational) Functioning
6. SOFAS	2-3 min	Single rating of Social and Occupational functioning
<i>Neuropsychological Assessment</i>		
7. WTAR	5 min	Adult Reading/Pre-morbid IQ
8. RBANS	30 min	Cognitive functioning domains: Language; Immediate and Delayed Memory; Attentional; and Visuospatial/Constructional abilities
<i>Collateral File Information</i>		
9. PCL: SV - Part II: Collateral Information Schedule	60-180 min	Demographics, family, educational, employment, marital, medical, antisocial behaviour, offending and substance use history (MHS Medical Record file review: to ascertain level of Psychopathy traits)
<i>Visual-Cognitive Tasks</i>		
10. Task A: Emotion recognition	3-5 min	Affect recognition, scan-path performance
11. Task B: Emotion induction	10 min	Emotion induction scan-path performance
12. Task C: Face recognition / working memory	5 min	Recognition/working memory, immediate and delayed recall, scan-path performance
<i>Self-Report</i>		
13. POMS (abbreviated)	5-10min	Change in mood state (during Task B)
14. DERS	10-15 min	Emotion regulation
15. CAQ	3-5 min	Childhood Adversity
16. AQoL-6D	5-10 min	Quality of life
17. IPDEQ	10-15 min	Personality Disorder Screener

*Note: SCID-Structured Clinical Interview for DSM-IV; PCL: SV-Psychopathy Checklist: Screening Version; BPRS-Brief Psychiatric Rating Scale; GAF-Global Assessment of Functioning; SOFAS-Social Occupational Functioning Scale; WTAR-Wechsler Test of Adult Reading; RBANS-Repeatable Battery for Assessment of Neuropsychological Status; POMS-Profile of Mood States; DERS-Difficulties in Emotion Regulation Scale; CAQ-Childhood Adversity Questionnaire; AQoL6D-Assessment of Quality of Life; and IPDEQ-International Personality Disorder Examination Questionnaire.*

#### 2.5.4 Eye tracking apparatus

Visual scanning technology (Eyelink: 1000; SR Research Ltd, Ontario, Canada) was utilised to record eye movements while participants viewed face stimuli. A View Point eye tracker (1000 Hz sampling frame) recorded individual visual scan-paths, detailing the facial emotion processing strategies used. The optical assembly consisted of an infrared low light camera that recorded retinal and corneal reflections from the participant's right eye (while viewing was binocular, only right eye movements were recorded) to obtain the point of fixation. A procedure for calibration of eye fixation position conducted prior to each recording, using a 9-point matrix, ensured a point of fixation error rate of less than 0.5 degrees. As shown in [Figure 2.1](#), one computer managed the recording of eye movements, while face stimuli presented on a 19" colour monitor using the SR-Research Experiment builder software was running on a separate computer. The SR Eye Tracking software ensured that the face stimuli appeared only when a participant had maintained fixation on a centrally presented point for 1000ms, thereby controlling for initial direction of retinal attention.

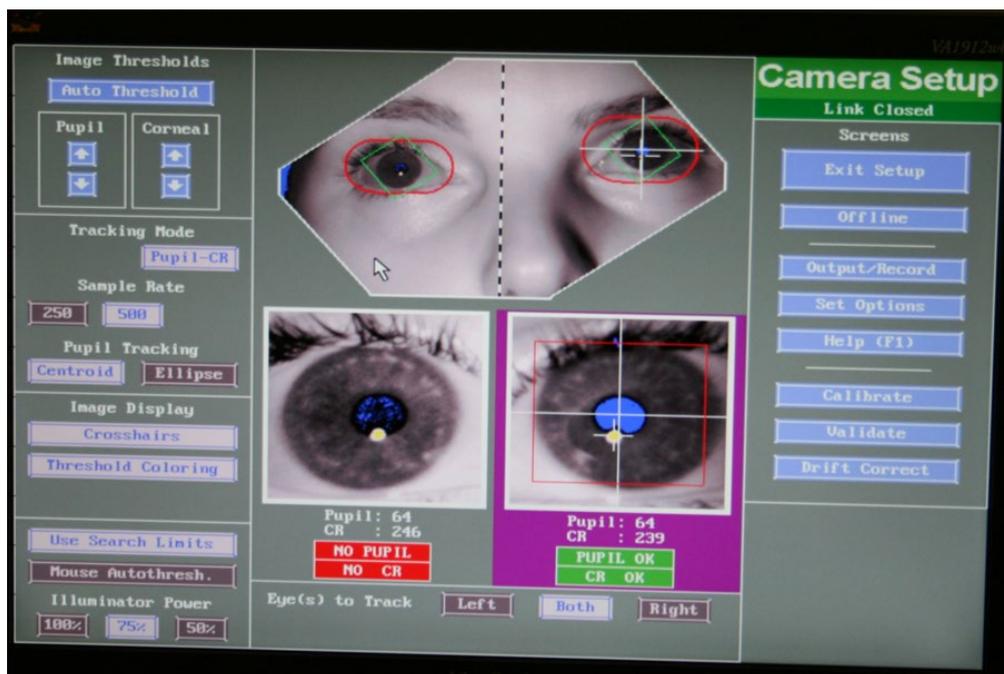


Figure 2.1 Illustration of Eyelink SR software recording from View Point eye-tracker.

### *2.5.5 Procedures*

Where possible, all study components were completed during a single structured interview at the CBMHR visual-cognitive laboratory. Although, if necessary due to current level of functioning and/or symptomology, clinical participants were provided with the option of undertaking clinical and neuropsychological assessments during an initial appointment, with the remaining behavioural eye tracking and self-report measures completed at a subsequent appointment, the following week. All clinical and neuropsychological measures were administered by a registered psychologist (PhD Candidate/author of dissertation), fully trained in the administration procedures for each instrument (SCID, BPRS, GAF, SOFAS, WTAR & RBANS by Associate Professor Carmel Loughland; and PCL: SV accredited by the Darkstone Research Group).

The PCL: SV assessment was undertaken during clinical interview (Part I: Interview Schedule) and finalised following the integration of collateral information (Part II: Collateral Information schedule) obtained from a file review of paper and electronic medical records held by MHS in the HNE-LHD (in adherence with recommended completion procedures), for all clinical participants. The collateral information schedule was utilised to confirm demographic, family, education, employment, relationship, medical, antisocial behaviour, offending, substance use and diagnostic history. In the case of the healthy control participants, clinical inferences were based on interview data alone. The same registered psychologist trained in the administration, scoring and interpretation of the PCL: SV conducted all assessments.

While the PCL: SV measures a dimensional construct, researchers using the scale have previously adopted convenient cut scores. A score of 18 or greater for

“probable psychopathy”, scores between 13 and 17 as an indication of “possible psychopathy”, and scores of 12 or lower as “non-psychopathic” (Hart et al., 2003). As assessments based primarily on interview data indicate scores may be lower than those that include additional collateral information (Hart et al., 2003), and consistent with research previously undertaken using the PCL: SV amongst a community sample, a conservative cut score (i.e., 11/24) was utilised (Coid et al., 2009). For descriptive purposes, in the current study clinical participants scoring 11 or above were assigned to the high-psychopathy group (N = 19; with scores ranging from 11 to 18). The low-psychopathy group comprised clinical participants scoring below 11 (N = 18; with scores ranging from 0 to 10). In practice, continuous scores on the PCL: SV were used in the major statistical analyses ([see Section 2.5.7](#)). All healthy control participants (N= 24; with scores ranging from 0 to 2) exhibited low-psychopathy. An examination of individual PCL:SV items, and scoring using a four-factor model, have also been reported as having some clinical utility, particularly in individual clinical practice (Vitacco et al., 2005), allowing subcomponents of psychopathy to be further investigated, or to address issues of possible differing aetiologies (Coid et al., 2009).

Offending details obtained during the PCL: SV interview for all participants, and/or supplemented by collateral MHS file review for clinical participants, were coded into the 16 broad divisions (including subdivisions) of criminal offences used by the Australian Bureau of Statistics, Catalogue No. 1234.0 - Australian and New Zealand Standard Offence Classification (ANZSOC) framework (A.B.S., 2011). This classification was used as it provides a standardised clinical framework of criminal offences, overcoming any differences in legal offence definitions across states and territories (A.B.S., 2011). For reporting purposes, these 16 broad overall divisions were

further collapsed into three categories of offence types: 1) offences against people, 2) offences against property, and 3) offences against public order. Offences against people included: homicide; acts intended to cause injury (i.e., assault); sexual assault; dangerous or negligent acts endangering persons; abduction, harassment and other offences against the person; robbery, extortion and related offences (i.e., blackmail). Offences against property included: unlawful entry with intent/burglary, break and enter; theft; fraud, deception and related; property damage and environmental pollution. Offences against public order included: illicit drug offences; prohibited and regulated weapons and explosives offences; public order offences; traffic and regulatory offences; offences against justice procedures; government security and government operations; and miscellaneous offences.

Prior to commencement of the visual-cognitive eye tracking tasks, evidence of any ocular pathology was checked, with details on both visual acuity and ocular-motor dominance recorded. Visual acuity was assessed using a half size Snellen chart at the recommended distance of 3 meters, whereby participants were required to exhibit normal vision at a 20/20 level of acuity by accurately reciting the line second from the bottom of the chart (82% without glasses; while 18% of the sample required glasses). A check of ocular-motor dominance by recording the participant's response to set questions around any shift in visual field following the opening and closing of each eye was also undertaken, with 63.9% of the sample reporting right-eye, and 36.1% left-eye dominance. Participants were then oriented to the laboratory facilities specifically designed for undertaking eye movement recordings (using a non-invasive technique), ensuring participant comfort and safety (with a duress system in place in case of psychiatric emergency).

All participants took part in three visual-cognitive eye movement recording tasks examining different aspects of facial emotion processing: A) facial emotion recognition, B) emotion induction, and C) recognition and working memory (procedural details for Tasks B and C are provided in subsequent chapters, 3 and 4). As noted earlier, during the first emotion recognition task (Task A) 28 face stimuli were presented in four gender balanced (14 Males/14 females) pre-randomised order blocks, consisting of 7 images for each category of emotion (happy, sad, surprise, anger, fear, disgust, and neutral; see [Figure 2.2](#)). Recognition accuracy and visual scan-path performance was recorded concurrently. Participants were seated at a desk approximately 60 cm away from a computer display screen running the experimental task, linked to a second computer, which monitored and recorded the visual scan-path performance (as seen earlier, in [Figure 2.1](#)). The participants chair allowed for height and distance positioning and, as shown in [Figure 2.3](#), a soft head and chin rest was used to both position the participants head and minimise movement during the eye movement recordings.



Figure 2.2 *Example of facial expression stimuli utilised from the NimStim set (Tottenham et al., 2009 – stimuli available for illustrative use include happy, sad, surprise, anger, fear, disgust, and neutral expressions).*

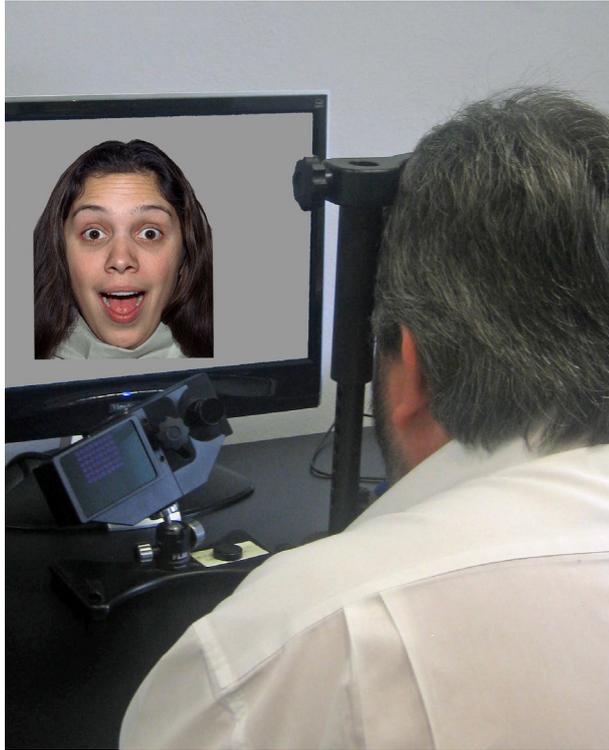


Figure 2.3 *Illustration of a participant taking part in the visual cognitive eye-tracking emotion recognition task (not an actual research participant).*

Prior to commencing the task, a standard SR EyeLink calibration and validation procedure was undertaken, using a 9-point matrix, consisting of a series of fixation points presented randomly in one of nine locations on the screen, to ensure a point of fixation error rate of less than 0.5 degrees. At the start of each trial, an image was cued when a participant fixated on a centrally located dot (3 cm diameter) presented on the computer screen for more than 1000ms. The facial expression stimuli were presented for 6000ms, followed by a list of response options (afraid, happy, no emotion, sad, surprised, disgusted, angry), with all seven categories of emotion presented in the same order, printed horizontally across the screen after each image presentation. Participants were given as much time as they required to provide a verbal response about their emotion selection (see [Appendix I](#), for task instructions). The experimenter recorded their response manually, with recognition accuracy measured by the number of emotions correctly identified.

Participants completed additional self-report measures subsequent to, and in combination with, the three visual-cognitive eye-tracking tasks. Following Task A, the abbreviated version of the POMS questionnaire was presented on a computer screen, in conjunction with the emotion induction task (Task B). The POMS provided a baseline measure of mood state prior to the task, and change in mood state after exposure to each block of face stimuli (as detailed in Chapter 3: Section 3.5.5). Participants were then asked to complete paper and pencil versions of the remaining self-report questionnaires following the immediate recall component of the final emotion processing task (Task C), which examined recognition and working memory (as detailed in Chapter 4: Section 4.2.4).

Whilst the remaining questionnaires were self-completed, administration was supervised by the interviewer, who provided general guidance if required. The DERS, assessing difficulties in emotion regulation, was scored using the original 6-factor solution developed from a student sample by Gratz and Roemer (2004), which has also been used in clinical populations, including an Australian psychosis sample (Ayre, 2013). Likewise, the CAQ has been utilised in a number of clinical and general community samples to detail the prevalence of adversities, including an Australian psychosis sample (McCabe et al., 2012). The CAQ was scored using the 5-factor model detailed by McCabe et al. (2012), which included Factor 1 – Abusive Parenting; Factor 2 – Loss, Poverty and Sexual Abuse; Factor – 3 Neglectful Parenting; Factor 4 – Dysfunctional parenting; and Factor 5 - Sibling Loss. All items were scored to be dichotomous (0 = 0, and 1/2 = 1) and a factor was considered present if at least one of the associated items was endorsed.

The AQoL-6D, which assesses quality of life impairment, was scored to produce two higher order factors as described by Allen et al. (2013), providing an indication of impairment in the physical and psychological components of quality of life, as well as overall. The IPDEQ screener for personality disorder traits was scored to provide Cluster A, B, and C subscale scores, and a total score, using the dimensional scoring method as detailed by Lewin et al. (2005), which was also utilised by McCarter et al. (2016) among an Australian clinical sample. Following completion of all of the self-report measures, the delayed recall component of the final emotion-processing task was administered (Task C - examining recognition and working memory). The entire research protocol took approximately 3 hours per participant, including all clinical, neuropsychological, behavioural visual-cognitive eye-tracking tasks and self-report measures. For completeness, outlined in [Table 2.2](#) is a mapping of the measures to the primary research questions addressed throughout this dissertation.

#### *2.5.6 Eye movement parameters*

Visual scan-paths were utilised as a potential guide to the accompanying brain processes, with a total of 6-scan-path parameters extracted for analysis. These included two temporal indices, the mean fixation duration (time/ms), and mean number of fixations (count); and two spatial indices, the mean distance between fixations (mm), and overall mean scan-path length (mm) (see [Figure 2.4](#) for further illustration). In addition, two attentional measures were used for examining regions of interest (i.e., salient feature areas such as eyes, nose, and mouth) versus non-feature areas (i.e., other areas of the face), measured by mean number and duration of fixations within the region of interest (see [Figure 2.5](#) for an illustration of the type of template utilised).

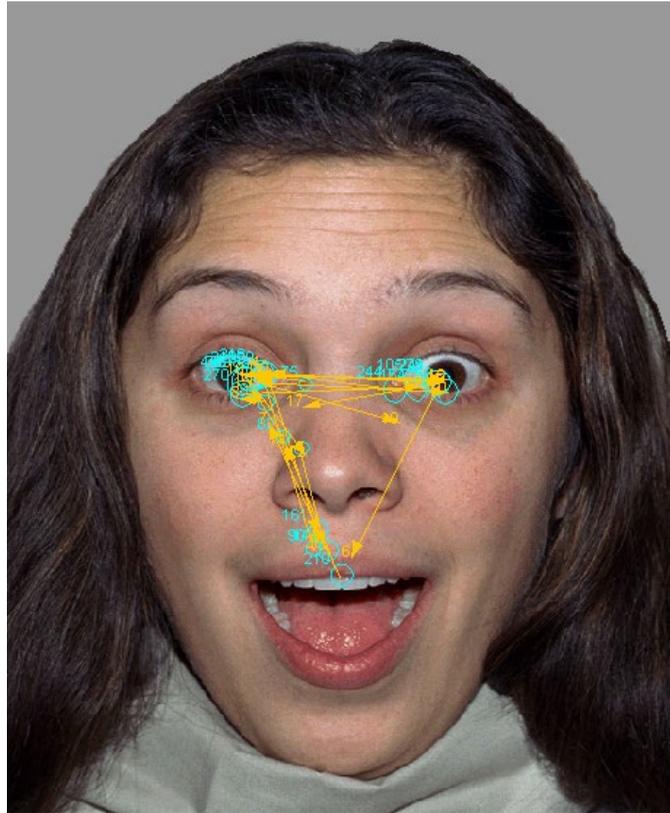


Figure 2.4 *Illustration of the visual scan-path parameters extracted, the number and duration of fixations are shown in blue circles, while distance between fixations and scan-path length and direction is depicted by the yellow lines with directional arrows.*

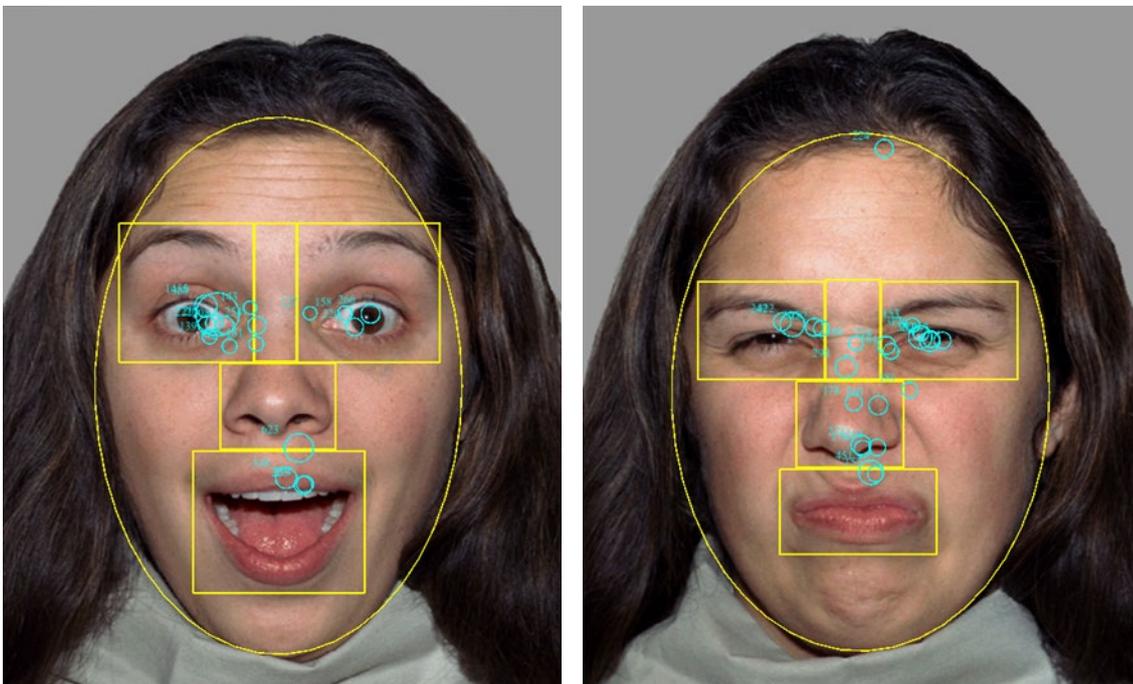


Figure 2.5 *Illustration of the region of interest templates utilised.*

Table 2.2: *Mapping of measures to primary research questions*

Assessment Measure	Characterising the sample	Primary research questions		
		Chapter 2: Emotion Recognition	Chapter 3: Emotion induction	Chapter 4: Face recognition and memory
2. SCID	*	*	*	*
	(Group assignment Psychosis)			
3. PCL: SV	*	*	*	*
	(Group assignment Psychopathy)			
4. BPRS	*			
5. GAF	*			
6. SOFAS	*			
7. WTAR	*			*
8. RBANS	*			*
10. Task A:		*		
11. Task B:			*	
12. Task C:				*
13. POMS			*	
14. DERS	*		*	
15. CAQ	*			
16. AQoL-6D	*			
17. IPDEQ	*			

### 2.5.7 Data analysis

Data coding and analyses were undertaken using SPSS Statistical Software (Version 24.0; SPSS, Armonk, NY, USA). Chi-square tests were utilised to assess simple associations between categorical variables, such as source of recruitment and offending profiles. To explore the relative contributions of the individual PCL: SV items to the psychopathy categorisation, univariate analysis of variance (ANOVA) techniques were utilised solely to estimate the variance ( $\eta^2$ ) associated with differences between the low and high psychopathy groups. Chi-square analyses and planned contrasts (helmet style orthogonal contrasts) within one-way ANOVAs were utilised initially to assess group differences on sociodemographic, neurocognitive functioning and symptom characteristics (i.e., simple planned orthogonal contrasts comparing psychosis versus not, and low versus high psychopathy within the psychosis groups).

Given that no potentially useful sociodemographic covariates were apparent (e.g., an absence of group differences in age, gender, education or IQ), thereafter a continuous psychopathy contrast was used, which provided additional statistical power over a somewhat arbitrary division of psychopathy into categorical subgroups (i.e., low and high). Likewise, for analyses examining general functioning, quality of life, childhood adversity and personality disorder traits, a multiple regression-based approach was utilised which incorporated a binary contrast (Psychosis vs. Not) and a continuous contrast based on dimensional scores for psychopathy. It should also be noted that, throughout this dissertation, where such multiple regression-based analyses were conducted (e.g., Tables 2.7, 3.2 to 3.6, 4.3 and 4.4), and as an aid to interpretation and discussion, means are reported for the Low vs. High Psychopathy comparison (as opposed to regression weights for the various contrasts). Although the associated statistical test were based on the continuous version of the psychopathy contrast.

Misattribution patterns during the emotion recognition task were examined by calculating for each emotion category a 99% confidence bound for a random distribution of misattribution errors. As Task A involved recognition of seven categories of facial expression, analyses of accuracy and the visual cognitive eye-tracking scan-path indices, were undertaken using a Generalised Linear Model (GLM) incorporating a series of planned orthogonal contrasts. As detailed in [Table 2.3](#), twenty planned orthogonal contrasts were defined in order to reduce the number of significance tests, and control for variations between groups, and within emotion categories. The planned contrasts comprised two between-group contrasts (GC1 and GC2), six within-group emotion contrasts (EC1 to EC6), and twelve interaction or product contrasts (GC1 x EC1 to EC6, and GC2 x EC1 to EC6). Contrast coefficients were standardised (i.e.,

weighted mean of zero, and standard deviation of 1.000). The relationships between scan-path indices were examined using Pearson product-moment correlations, with two-tailed significance. As a partial control for the number of statistical tests conducted, the threshold for statistical significance was set at  $p < 0.01$  for all analyses, although statistical trends ( $p < 0.05$ ) are also noted.

Table 2.3: *Planned orthogonal contrasts – Standardised coefficients*

<i>Group Contrasts (Between-groups)</i>	Healthy Control (N = 24)		Psychosis Groups				
			Low Psychopathy (N = 18)	High Psychopathy (N = 19)			
GC1: Psychosis Vs. Not	1.240		-0.804	-0.804			
GC2: Low Vs. High Psychopathy (Continuous Contrast)	0.000		-0.099 to -2.085	0.144 to 2.154			
<i>Emotion Contrasts (Within-groups)</i>							
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral
EC1: Negative Vs. Positive or Neutral	1.153	-0.865	1.153	-0.865	-0.865	-0.865	1.153
EC2: Neutral Vs. Happy or Surprise	1.079	0.000	1.079	0.000	0.000	0.000	-2.158
EC3: Surprise Vs. Happy	1.869	0.000	-1.869	0.000	0.000	0.000	0.000
EC4: Other Negative Vs. Sad	0.000	2.289	0.000	-0.763	-0.763	-0.763	0.000
EC5: Fear or Disgust Vs. Anger	0.000	0.000	0.000	2.158	-1.079	-1.079	0.000
EC6: Disgust Vs. Fear	0.000	0.000	0.000	0.000	1.869	-1.869	0.000
<i>Interaction Contrasts</i>							
GC1 x EC1 to EC6 (Six Contrasts)							
GC2 x EC1 to EC6 (Six Contrasts)							

*Note. Twenty planned orthogonal contrasts were defined, comprising two between-group contrasts (GC1 and GC2), six within-group emotion contrasts (EC1 to EC6) and twelve interaction or product contrasts (GC1 x EC1 to EC6, and GC2 x EC1 to EC6). Contrast coefficients have been standardised (i.e., weighted mean of zero, and standard deviation of 1.000).*

## 2.6 Results

### 2.6.1 Sample characteristics

#### 2.6.1.1 Diagnostic and offending profiles

Diagnostic and offending profiles [presented as number and percentage for categorical variables, and mean (SD) for continuous variables] are displayed in [Table 2.4](#) for the study sample of 61 participants, including 24 healthy control participants and 37 community mental health outpatients with a psychotic disorder. All healthy control participants self-referred from the community, while the psychosis group participants were referred to the study from either Community Mental Health or Psychiatric Rehabilitation Services. Utilising the Psychopathy Checklist: Screening Version (PCL: SV), the psychosis group participants were allocated to either the low or high psychopathy group. A cut point based on a PCL: SV total score of 11 or more was used, coinciding with prior research utilising the psychopathy screener in community settings (Coid et al., 2009), where a score of less than 11 is likely to be “*non-psychopathic*” (Hart et al., 2003). The psychosis group categorised as having low psychopathy traits included 18 participants, while a further 19 had high levels of psychopathy traits as measured on the PCL: SV. The majority (94.4%) of the low psychopathy group, compared to 57.9% of the high psychopathy group, were referred from Community Mental Health Services (living independently), with the remainder living in supported accommodation recruited from Psychiatric Rehabilitation Services ( $\chi^2 = 6.71, p < 0.01$ ).

The psychosis group participants had either a primary lifetime diagnosis or schizophrenia or a schizoaffective disorder (based on the SCID; and confirmed via file review). There were fewer individuals with a diagnosis of schizophrenia among the low psychopathy group (with 55.6%) compared to the high psychopathy group (84.2%).

Table 2.4: Diagnostic and offending profiles (SCID, PCL: SV and Collateral file)

Characteristic	Healthy Control	Psychosis Groups	
	N=24	Low Psychopathy N=18	High Psychopathy N= 19
<b>Primary Lifetime Diagnoses - SCID</b>			
<i>Schizophrenia N (%)</i>		10 (55.6%)	16 (84.2%)
<i>Schizoaffective disorder N (%)</i>		8 (44.4%)	3 (15.8%)
<b>Psychosis Onset and Illness Duration -SCID</b>			
<i>Mean Age of onset in years (SD)</i>		27.39 (7.83)	23.84 (5.99)
<i>Mean Duration in years (SD)</i>		19.06 (10.37)	16.79 (9.54)
<b>Current Illness Severity - SCID</b>			
<i>Mild to Moderate symptoms N (%)</i>		5 (27.8%)	6 (31.6%)
<i>In Remission N (%)</i>		13 (72.2%)	13 (68.4%)
<b>Number of Hospital Admissions - SCID</b>			
<i>Mean Psychiatric admissions (SD)</i>		4.89 (3.77)	5.68 (4.51)
<i>Mean General admissions (SD)</i>	2.29 (1.99)	1.28 (1.23)	1.21 (1.51)
<b>Current Medications</b>			
<i>Antipsychotic N (%)</i>		17 (94.4%)	19 (100%)
<i>Antidepressant/Mood stabiliser N (%)</i>	1 (4.2%)	4 (22.2%)	6 (31.6%)
<i>Anxiolytic (%)</i>		3 (16.7%)	1 (5.3%)
<i>Anti-craving N (%)</i>		1 (5.6%)	1 (5.3%)
<i>Anticonvulsant N (%)</i>		1 (5.6%)	4 (21.1%)
<b>Comorbid Diagnoses - SCID</b>			
<i>Lifetime Mood disorder N (%)</i>		4 (22.2%)	6 (31.6%)
<i>Lifetime alcohol abuse/dependence N (%)</i>	3 (12.5%)	13 (72.2%)	14 (73.7%)
<i>Lifetime substance abuse/dependence N (%)</i>	1 (4.2%)	11 (61.1%)	17 (89.5%)
<b>Collateral History – Diagnostic Categories <sup>a</sup></b>			
<i>Psychosis N (%)</i>		18 (100.0%)	19 (100%)
<i>Mood Disorder N (%)</i>		5 (27.8%)	4 (21.1%)
<i>Substance misuse N (%)</i>		7 (38.9%)	14 (73.7%)
<i>Personality disorder N (%)</i>		1 (5.6%)	4 (21.1%)
<i>Other MH problems N (%)</i>			1 (5.3%)
<i>Other non-MH problems N (%)</i>			2 (10.5%)
<b>Psychopathy Traits - PCL:SV</b>			
<i>Mean Part 1 Score (SD)</i>	0.21 (0.42)	1.33 (1.46)	6.05 (2.01)
<i>Mean Part 2 Score (SD)</i>	0.29 (0.46)	3.50 (2.83)	8.16 (1.39)
<i>Mean Total Score (SD)</i>	0.50 (0.59)	4.83 (3.45)	14.21 (2.39)
<b>Offending Profiles (Arrested or Charged)</b>			
<i>Any Offending N (%)</i>	1 (4.2%)	14 (77.8%)	19 (100%)
<i>Mean per offender (SD)</i>	1	2.43 (1.16)	3.42 (1.74)
<i>Against People N (%)</i>		11 (61.1%)	18 (94.7%)
<i>Mean per offender (SD)</i>		1.09 (0.30)	1.44 (0.70)
<i>Against Property N (%)</i>		4 (22.2%)	13 (68.4%)
<i>Mean per offender (SD)</i>		1 (0.00)	1.46 (0.66)
<i>Against Public Order N (%)</i>	1 (4.2%)	11 (61.1%)	13 (68.4%)
<i>Mean per offender (SD)</i>	1	1.64 (0.67)	1.54 (0.78)

Note. Low Psychopathy < 11 on PCL: SV; <sup>a</sup> CHIME - Broad diagnostic categories extracted from medical records of formal diagnoses and presenting problems.

The two psychosis groups were relatively similar in terms of illness onset, duration, current level of severity, and number of hospital admissions. The average age of illness onset was 25.6 years, with a mean illness duration of 17.9 years (SD = 9.88). The majority were currently in remission of psychotic symptoms (70.3%), having had a median number of four psychiatric and two general hospital admissions. At the time of interview, all of the psychosis participants were currently taking antipsychotic medication as prescribed, with the exception of one person in the low psychopathy group who recently ceased taking their antipsychotic medication. The two psychosis groups had similar levels of antidepressant or mood stabiliser, anxiolytic, anticonvulsant, and anti-craving medication use, as well as medication use for comorbid physical conditions (i.e., diabetes, high cholesterol or blood pressure; in 22.2% of the low vs. 26.3% of the high psychopathy group). In addition, one healthy control participant had been prescribed antidepressant medication by their GP to assist with generalised anxiety, and another healthy control was taking medication for comorbid physical conditions (i.e., high blood pressure/thyroid problem).

Similar levels of comorbidity were apparent within the low and high psychopathy groups. Overall 27.0% of the psychosis group participants met criteria for a lifetime mood disorder, 73.0% lifetime alcohol abuse (48.6% dependence), and 75.7% lifetime substance abuse (67.9% dependence) on the SCID. While lifetime levels of substance misuse were slightly higher among the high psychopathy group, current levels of misuse were low overall, 2.7% for current alcohol abuse/dependence (one low psychopathy group participant), and 5.4% for substance abuse/dependence (two high psychopathy group participants). In addition, while 12.5% of the healthy controls reported a lifetime history of alcohol abuse (8.3% dependence), current levels were 0%

for alcohol and substance abuse/dependence. A collateral file review of diagnostic and presenting problems recorded in community mental health service records confirmed primary lifetime psychosis diagnoses, and similar levels of comorbidity. Although documented rates of substance misuse appeared to be lower in the patient medical records, particularly among the low psychopathy group.

Patterns of offending revealed a higher rate and number of offences among the high psychopathy group, with a mean of 2.43 (SD = 1.16) offences per offender in the low compared to 3.42 (SD = 1.74) offences per offender in the high psychopathy group. In terms of categories of offending, there were proportionately more offences involving crimes against people (i.e., attempted murder/assault),  $\chi^2 = 8.46$ ,  $p = 0.04$ , as well as property crimes (i.e., break, enter & steal),  $\chi^2 = 9.51$ ,  $p = 0.02$ , among the high compared to the low psychopathy group. Similar rates of public order offences (i.e., illicit drug use & against justice procedures) were apparent amongst both the low and high psychopathy groups. In addition, one healthy control participant had a public order offence recorded.

On the PCL: SV, Part 1 psychopathy scores (assessing interpersonal and affective traits) were slightly lower than Part 2 scores (assessing lifestyle and antisocial features). Total PCL: SV scores were well below the clinical range for the healthy controls (Mean = 0.50, SD = 0.59) and the low psychopathy group (Mean = 4.83, SD = 3.45) participants. A mean PCL: SV total score of 14.21 (SD = 2.39) was obtained for the high psychopathy group (median of 15; with scores ranging from 11 to 18), which is within the spectrum of psychopathy traits worthy of further clinical examination (scores of 13-17 representing possible; and  $\geq 18$ , providing a strong indication of psychopathy).

As shown in [Table 2.5](#), individual item profiles for the PCL: SV were also examined with respect to the amount of variance associated with psychopathy group differences (low vs. high psychopathy traits). Although all 12 items were useful, the two items making the largest contribution were “lacks remorse” and “doesn’t accept responsibility”, while the items contributing least were “lacks goals” and “adolescent antisocial behaviour”. Consequently, PCL: SV items from Part 1 contributed more to the overall psychopathy score and group differences ( $\eta^2 = 0.654$ ), compared with Part 2 items ( $\eta^2 = 0.540$ ).

#### *2.6.1.2 Socio-demographic, neurocognitive functioning and symptoms*

Analyses of sample characteristics by group, as shown in [Table 2.6](#), revealed no significant differences for age, gender, education level, or premorbid IQ based on performance on the Wechsler Test of Adult Reading (WTAR). Overall, 78.4% of psychosis group participants were male, with an average age of 43.5 years (SD = 10.13). Similarly, 75.0% of the healthy control participants were males, with an average aged of 43.6 years (SD = 10.46) (see [Appendix G](#): Supplementary Table S1 for additional details on the recruited sample by age, gender and group). A significantly higher proportion of the healthy control participants were currently married or in a de facto relationship (91.7% vs. only 8.1% of the psychosis group participants,  $\chi^2 = 42.11$ ,  $p < 0.001$ ). In addition, all of the healthy controls were living with family or friends, compared to only 37.8% of the psychosis group participants, with a large number living alone 35.1% ( $\chi^2 = 23.95$ ,  $p < 0.001$ ). Within the psychosis groups, a higher proportion of the high psychopathy group were residing in supported accommodation (42.1% vs. only 5.6% of the low psychopathy group,  $\chi^2 = 9.07$ ,  $p = 0.028$ ), as noted previously in relation to recruitment source.

Table 2.5: *Psychopathy Check List: Screening Version (PCL: SV) item profiles for the psychosis group*

PCL: SV item	Low Psychopathy N=18			High Psychopathy N= 19			Variance associated with group differences ( $\eta^2$ )
	Maybe (%)	Yes (%)	Mean (SD)	Maybe (%)	Yes (%)	Mean (SD)	
<u>Part 1:</u>			1.33 (1.46)			6.05 (2.01)	0.654
<i>Superficial</i>	0.0	0.0	0.00 (0.00)	42.1	0.0	0.42 (0.51)	0.261
<i>Grandiose</i>	11.1	0.0	0.11 (3.23)	31.6	21.1	0.74 (0.81)	0.212
<i>Deceitful</i>	27.8	0.0	0.28 (0.46)	36.8	31.6	1.00 (0.82)	0.236
<i>Lacks Remorse</i>	16.7	0.0	0.17 (0.38)	42.1	42.1	1.26 (0.73)	0.477
<i>Lacks Empathy</i>	22.2	11.1	0.44 (0.71)	52.6	36.8	1.26 (0.65)	0.278
<i>Doesn't Accept Responsibility</i>	33.3	0.0	0.33 (0.49)	63.2	36.8	1.37 (0.50)	0.540
<u>Part 2:</u>			3.5 (2.83)			8.16 (1.39)	0.540
<i>Impulsive</i>	27.8	5.6	0.39 (0.61)	42.1	47.4	1.37 (0.68)	0.376
<i>Poor Behavioural Controls</i>	55.6	27.8	1.11 (0.67)	15.8	78.9	1.74 (0.56)	0.212
<i>Lacks Goals</i>	33.3	11.1	0.56 (0.71)	26.3	42.1	1.11 (0.88)	0.112
<i>Irresponsible</i>	33.3	11.1	0.56 (0.71)	47.4	52.6	1.53 (0.51)	0.398
<i>Adolescent Antisocial Behaviour</i>	16.7	5.6	0.28 (0.58)	42.1	21.1	0.84 (0.77)	0.154
<i>Adult Antisocial Behaviour</i>	50.0	5.6	0.61 (0.61)	42.1	57.9	1.58 (0.51)	0.442
<u>Overall</u>			4.83 (3.45)			14.21 (2.39)	0.727

Note. Low Psychopathy < 11 on PCL: SV. To illustrate the relative contributions of the PCL: SV items to the psychopathy categorisation, the last column reports the variance associated with differences between the two groups.

Employment rates were significantly different, with the majority (91.9%) of the psychosis group currently unemployed, compared to only 12.5% of the healthy controls ( $\chi^2 = 38.45$ ,  $p < 0.001$ ), with a substantial proportion of the psychosis group (86.5%) receiving a disability support pension. In terms of current neurocognitive functioning and symptoms, neuropsychological functioning as measured using the Repeatable Battery for Assessment of Neuropsychological functioning (RBANS) was significantly lower among the psychosis group ( $t_{(58)} = 6.27$ ,  $p < 0.001$ ), with a mean total scale score of 82.11 (SD = 12.17) compared to 101.21 (SD = 10.95) for the healthy controls. The WTAR and RBANS total scores were also significantly correlated ( $r = 0.44$ ,  $p < 0.001$ ). As expected, significantly higher levels of current psychiatric symptoms were apparent on the Brief Psychiatric Rating Scale (BPRS) among the psychosis groups ( $t_{(58)} = -6.29$ ,  $p < 0.001$ ). However, the psychosis participants were not highly symptomatic at the time of interview, with mean total score = 37.86 (SD = 10.26), predominately in the mild range.

### *2.6.1.3 General functioning, quality of life, childhood adversity, and personality traits*

As shown in [Table 2.7](#), in subsequent analyses (within which psychopathy scores were examined as a continuous contrast within multiple regression analyses) significant functional impairments were detected among the psychosis participants, for both global ( $t_{(58)} = 18.15$ ,  $p < 0.001$ ) and social and occupational functioning ( $t_{(58)} = 17.26$ ,  $p < 0.001$ ). The global assessment of functioning (GAF) measure had a mean of 53.64 (SD = 10.14) for psychosis participants compared to 89.67 (SD = 3.74) for healthy controls, while on the social and occupational functioning scale (SOFAS) the corresponding means were 53.95 (SD = 11.14) and 90.83 (SD = 4.07). In addition, functioning was differentially influenced by the number of psychopathy traits, with

Table 2.6: Sample characteristics by group: socio-demographic, neurocognitive functioning and symptoms

Characteristic	Healthy Control	Psychosis Groups		Planned Comparisons <sup>a</sup>	
	N=24 Mean (SD)	Low Psychopathy N=18 Mean (SD)	High Psychopathy N= 19 Mean (SD)	Psychosis Vs. Not	Low Vs. High Psychopathy
Age	43.58 (10.46)	46.44 (10.22)	40.63 (9.45)	$t_{(58)}=0.02, p=0.986$	$t_{(58)}=-1.75, p=0.085$
Gender					
Male N (%)	18 (75.0%)	12 (66.7%)	17 (89.5%)	$\chi^2=0.09, p=0.759$	$\chi^2=2.84, p=0.092$
Female N (%)	6 (25.0%)	6 (33.3%)	2 (10.5%)		
Marital Status					
Married/de facto N (%)	22 (91.7%)	2 (11.2%)	1 (5.3%)	$\chi^2=42.11, p<0.001^{**}$	$\chi^2=0.47, p=0.789$
Divorced/separated/widowed N (%)	1 (4.2%)	5 (27.8%)	5 (26.3%)		
Never married N (%)	1 (4.2%)	11 (61.1%)	13 (68.4%)		
Education					
Never completed High School N (%)	3 (12.55)	5 (27.8%)	8 (42.1%)	$\chi^2=4.50, p=0.105$	$\chi^2=3.42, p=0.180$
Completed High School N (%)	4(16.7%)	2 (11.1%)	5 (26.3%)		
Further formal study N (%)	17 (70.8%)	11 (61.1%)	6 (31.6%)		
Accommodation					
Family / Friend(s) N (%)	24 (100%)	10 (55.6%)	4 (21.1%)	$\chi^2=23.95, p<0.001^{**}$	$\chi^2=9.07, p=0.028^{\#}$
Alone N (%)		6 (33.3%)	7 (36.8%)		
Supported Accommodation N (%)		1 (5.6%)	8 (42.1%)		
Homeless/No-fixed address N (%)		1 (5.6%)			
Employment Status					
Working N (%)	21 (87.5%)	2 (11.1%)	1 (5.3%)	$\chi^2=38.45, p<0.001^{**}$	$\chi^2=0.42, p=0.515$
Not working N (%)	3 (12.5%)	16 (88.9%)	18 (94.7%)		
Wechsler Test of Adult Reading (WTAR)					
Mean standardised Score	101.29 (11.08)	97.44 (12.39)	92.32 (17.70)	$t_{(58)}=1.77, p=0.082$	$t_{(58)}=1.13, p=0.264$
Repeatable Battery for Assessment of Neuropsychological Status (RBANS)					
Mean total scaled score	101.21 (10.95)	85.11 (11.90)	79.26 (12.03)	$t_{(58)}=6.27, p<0.001^{**}$	$t_{(58)}=1.54, p=0.130$
Brief Psychiatric Rating Scale (BPRS)					
Mean total score	24.67 (0.76)	36.11 (8.96)	39.53 (11.34)	$t_{(58)}=-6.29, p<0.001^{**}$	$t_{(58)}=-1.30, p=0.198$

Note. Low Psychopathy < 11 on PCL: SV; <sup>a</sup> Chi-square analyses and planned contrasts within one-way ANOVAs; # trend ( $p<0.05$ ); \*  $p < 0.01$ ; \*\*  $p < 0.001$ ;

psychosis participants with higher psychopathy traits displaying increased impairment in global (Means: 50.26 vs. 57.22;  $t_{(58)} = -3.48, p = 0.001$ ) as well as social and occupational functioning (Means: 48.89 vs. 58.22;  $t_{(58)} = -3.86, p < 0.001$ ). Consistent with recruitment source, and as noted earlier (Section 2.6.1.1.), a significantly greater proportion of the high psychopathy group were recruited from specialised psychiatric rehabilitation services, living in supported outpatient accommodation, indicative of an increased level of need or service dependence.

In keeping with the clinician rated findings, and as shown in [Table 2.7](#), self-assessment of quality of life impairment (AQoL-6D) revealed that the psychosis group participants were currently experiencing significantly increased impairment in both psychological ( $t_{(58)} = -2.74, p < 0.01$ ) and physical ( $t_{(58)} = -4.28, p < 0.001$ ) domains, as well as overall ( $t_{(58)} = -3.64, p = 0.001$ ). Although there were no associations with psychopathy traits among the psychosis groups, the mean total AQoL-6D impairment score was higher for the psychosis group, at 2.00 (SD = 0.54) compared to 1.54 (SD = 0.37) for healthy control participants. The psychosis group also reported experiencing increased childhood adversity, particularly in the areas of abusive or dysfunctional parenting, loss, poverty or sexual abuse, and sibling loss, as measured on the Childhood Adversity Scale (CAQ). The mean total CAQ score for the psychosis groups was 6.22 (SD = 4.33) compared with 3.25 (SD = 3.29) for healthy control participants ( $t_{(58)} = -2.86, p < 0.01$ ). As expected, the self-report International Personality Disorder Examination Questionnaire (IPDEQ) demonstrated significant clinical group differences for the psychosis compared to healthy control participants on all three Cluster scores [Cluster A ( $t_{(58)} = -4.89, p < 0.001$ ); B ( $t_{(58)} = -7.76, p < 0.001$ ); and C ( $t_{(58)} = -4.84, p < 0.001$ )]. As well as on the overall IPDEQ dimensional total score ( $t_{(58)} = -6.76, p <$

Table 2.7: Additional characteristics: global, social & occupational functioning, quality of life, childhood adversity, and personality traits

Characteristic	Healthy Control	Psychosis Groups		R <sup>2</sup>	Comparisons	
	N=24 Mean (SD)	Low Psychopathy N=18 Mean (SD)	High Psychopathy N= 19 Mean (SD)		Psychosis Vs. Not	Low Vs. High Psychopathy <sup>a</sup>
Global Assessment of Functioning (GAF)	89.67 (3.74)	57.22 (11.27)	50.26 (7.79)	0.86	t <sub>(58)</sub> =18.15,p<0.001**	t <sub>(58)</sub> =-3.48,p=0.001**
Social & Occupational Functioning Scale (SOFAS)	90.83 (4.07)	58.22 (12.17)	49.89 (8.51)	0.84	t <sub>(58)</sub> =17.26,p<0.001**	t <sub>(58)</sub> =-3.86,p<0.001**
Quality of Life Impairment (AQoL-6D)						
<i>Psychological</i>	1.82 (0.67)	2.32 (0.58)	2.31 (0.80)	0.12	t <sub>(58)</sub> =-2.74,p<0.01*	t <sub>(58)</sub> =-0.84,p=0.404
<i>Physical</i>	1.25 (0.15)	1.71 (0.43)	1.66 (0.53)	0.24	t <sub>(58)</sub> =-4.28,p<0.001**	t <sub>(58)</sub> =0.54,p=0.594
<i>Total</i>	1.54 (0.37)	2.02 (0.46)	1.99 (0.63)	0.19	t <sub>(58)</sub> =-3.64,p=0.001**	t <sub>(58)</sub> =0.81,p=0.422
Childhood Adversity Scale (CAQ)						
<i>Factor 1: Abusive Parenting</i>	0.75 (1.33)	1.44 (1.29)	2.47 (2.52)	0.14	t <sub>(58)</sub> =-2.61,p=0.012*	t <sub>(58)</sub> =1.53,p=0.132
<i>Percent with any</i>	[41.7%]	[77.8%]	[63.2%]			
<i>Factor 2: Loss Poverty &amp; Sexual Abuse</i>	0.75 (0.74)	1.22 (1.00)	1.21 (0.85)	0.07	t <sub>(58)</sub> =-2.07,p=0.043 <sup>#</sup>	t <sub>(58)</sub> =-0.13,p=0.899
<i>Percent with any</i>	[62.5%]	[77.8%]	[78.9%]			
<i>Factor 3: Neglectful Parenting</i>	0.79 (1.21)	0.89 (1.49)	1.58 (1.77)	0.04	t <sub>(58)</sub> =-1.15,p=0.225	t <sub>(58)</sub> =1.01,p=0.319
<i>Percent with any</i>	[45.8%]	[38.9%]	[57.9%]			
<i>Factor 4: Dysfunctional Parenting</i>	0.96 (0.10)	1.72 (1.18)	1.53 (0.90)	0.10	t <sub>(58)</sub> =-2.46,p=0.017 <sup>#</sup>	t <sub>(58)</sub> =-0.35,p=0.731
<i>Percent with any</i>	[62.5%]	[77.8%]	[84.2%]			
<i>Factor 5: Sibling Loss</i>	0.00 (0.00)	0.17 (0.38)	0.16 (0.37)	0.07	t <sub>(58)</sub> =-2.10,p=0.040 <sup>#</sup>	t <sub>(58)</sub> =0.16,p=0.868
<i>Percent with any</i>	[0.00%]	[16.7%]	[15.8%]			
<i>Total Score</i>	3.25 (3.29)	5.44 (3.40)	6.95 (5.05)	0.14	t <sub>(58)</sub> =-2.86,p<0.01*	t <sub>(58)</sub> =0.96,p=0.338
Personality Disorder Screener (IPDEQ)						
<i>Cluster A</i>	0.17 (0.11)	0.34 (0.13)	0.37 (0.19)	0.29	t <sub>(58)</sub> =-4.89,p<0.001**	t <sub>(58)</sub> =0.55,p=0.061
<i>Cluster B</i>	0.07 (0.06)	0.31 (0.11)	0.33 (0.18)	0.51	t <sub>(58)</sub> =-7.76,p<0.001**	t <sub>(58)</sub> =1.06,p=0.293
<i>Cluster C</i>	0.17 (0.15)	0.44 (0.19)	0.39 (0.25)	0.30	t <sub>(58)</sub> =-4.84,p<0.001**	t <sub>(58)</sub> =-1.36,p=0.180
<i>Total Score</i>	0.13 (0.08)	0.37 (0.11)	0.36 (0.19)	0.44	t <sub>(58)</sub> =-6.76,p<0.001**	t <sub>(58)</sub> =-1.30,p=0.897

Note. Low Psychopathy < 11 on PCL: SV; <sup>a</sup> tested as a continuous contrast within a multiple regression analysis; <sup>#</sup> trend (p<0.05); \* p < 0.01; \*\* p < 0.001

0.001). However, there were no significant self-reported differences for low versus high psychopathy.

## 2.6.2 Emotion recognition performance – Task A

### 2.6.2.1 Accuracy and misattribution patterns

Overall emotion recognition accuracy and misattribution patterns for the seven categories of facial expression in Task A are presented in [Table 2.8](#), based on responses obtained for all 61 participants. Response patterns regardless of group were examined initially, to reduce the likelihood of biased selection of misattribution segments for subsequent analysis of group differences. In terms of recognition performance, expressions of happy (96.7%), followed by anger (93.4%) were the most likely to be identified correctly, while fear (35.2%) was most likely to be misidentified. There was an overall mean accuracy of 80.5% (SD =10.69) of emotions correctly categorised during the emotion recognition eye-tracking Task A.

Table 2.8: *Emotion recognition accuracy and misattribution patterns (Task A) – Mean percent correct across seven categories of facial expression*

Emotion Category	Response						
	Happy %	Sad %	Surprise %	Anger %	Fear %	Disgust %	Neutral %
Happy	96.7	0.8	1.6	0	0	0.4	0.4
Sad	1.6	79.5	2.9	1.2	1.6	<b>7.8</b>	5.3
Surprise	<b>6.6</b>	0	90.6	0.4	2.5	0	0
Anger	0	0.4	0.4	93.4	2.5	2.5	0.8
Fear	<b>0</b>	<b>1.2</b>	<b>50.4</b>	<b>2.9</b>	35.2	8.6	<b>1.6</b>
Disgust	2.0	1.6	5.7	5.3	2.5	81.1	1.6
Neutral	0.4	3.7	2.9	2.9	1.2	2.0	86.9

Note: Row percentages based on 244 stimulus presentations (N = 61 participants by 4-stimuli per emotion category). Bolded percentages fall outside the 99% confidence bounds for a random distribution of misattribution errors.

In order to examine patterns of misattributions for each emotion category, 99% confidence intervals around a chance level distribution of errors were constructed. For example, if the 20.5% error rate for sad was distributed evenly across the other response categories, the expected error rate per emotion would be 3.42% (99% CI: 0.43%, 6.41%), with only a misattribution for disgust (7.8%) falling outside the chance level range. In categorising happy facial expressions, there was a high level of accuracy with no significant misattributions. Similarly, for anger, disgust or neutral facial expressions there were no misattribution rates outside of the chance range. For surprise, an overall error rate of 9.4% was observed, with misattribution to happy falling significantly outside the chosen confidence bound (99% CI: 0.00%, 3.61%).

A large error rate of 64.8% for fear was found, with misattributions (99% CI: 5.69%, 15.91%) occurring largely to surprise (50.4%), followed by disgust (8.6%). The fear stimuli utilised in the stimulus set for Task A had lower mean validation ratings ( $M = 0.58$ ,  $SD = 0.85$ ) for interrater agreement on expression categorisation, in comparison to the stimuli for the other emotion categories (see Section 2.5.3), which could explain some of the recognition difficulty observed. In order to explore this further, when all fear stimuli identified as surprise were regarded as correct, the overall error rate dropped to 14.4%, leaving only misattributions for disgust outside the 99% confidence bound (CI: 0.00%, 4.92%) for a random distribution of misattributions.

As detailed in [Table 2.9](#), recognition accuracy analyses undertaken by emotion and group revealed an overall group difference between the psychosis and healthy control participants ( $GC1: W^2 = 8.11$ ,  $p = 0.004$ ). Healthy control participants (Mean = 84.82) exhibited significantly higher emotion recognition accuracy relative to the

psychosis groups (Mean = 77.70), in terms of the percent of emotions correctly identified. There were also significant main effects for emotion (i.e., irrespective of group), with all six contrasts statistically significant. On average, accuracy was reduced in the recognition of negative compared to positive facial expressions of emotion (EC1: Means = 72.34 vs. 91.39). Likewise, there was reduced accuracy when comparing neutral to happy or surprise (EC2: Means = 86.89 vs. 93.65); surprise to happy (EC3: Means = 90.57 vs. 96.72); other negative emotions to sad (EC4: Means = 69.95 vs. 79.51); fear or disgust to anger (EC5: Means = 58.20 vs. 93.44); and fear compared to disgust (EC6: Means = 35.25 vs. 81.15). However, there were no significant group by emotion interactions for psychosis status, or low versus high psychopathy traits.

Further analyses of misattribution patterns by group were also undertaken examining the misidentification of “sad as disgust”, “surprise as happy” and “fear as disgust. In the analysis of “sad as disgust” misattributions, there was a statistical tendency for the high psychopathy group to make fewer misattributions of that type compared to the psychosis group with low psychopathy traits (3.95% vs. 9.72;  $W^2 = 5.88$ ,  $p = 0.015$ ). The psychosis participants with high psychopathy actually tended to misattribute sad expressions as neutral (11.84%), which did not happen in the low psychopathy group (2.78%) or the healthy controls (2.08%). There were no other significant differences between groups in terms of misattribution errors when recognising facial expressions of surprise or fear.

Table 2.9: Emotion recognition accuracy (Task A) – Mean (SD) percent correct by emotion and group, and associated analyses

Group	Emotion Recognition Accuracy – Mean (SD) percent correct							
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall
Healthy Control (N = 24)	97.92 (7.06)	84.38 (21.88)	94.79 (12.72)	95.83 (9.52)	40.63 (29.32)	87.50 (22.12)	92.71 (13.75)	84.82 (8.89)
Psychosis - Low Psychopathy (N = 18)	94.44 (13.71)	79.17 (19.65)	86.11 (19.60)	97.22 (8.08)	33.33 (25.73)	83.33 (22.69)	81.94 (20.66)	79.37 (11.59)
Psychosis - High Psychopathy (N = 19)	97.37 (7.88)	73.68 (24.26)	89.47 (17.31)	86.84 (25.51)	30.26 (27.10)	71.05 (23.95)	84.21 (17.10)	76.13 (10.31)
<i>Group contrasts (Between-groups)</i>	<i>Emotion Contrasts (Within-groups)</i>		<i>Emotion main effect contrasts</i>		<i>GC1 by EC1 to EC6 interaction contrasts</i>		<i>GC2 by EC1 to EC6 interaction contrasts</i>	
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not	EC1: Negative Vs. Positive or Neutral		9.44	103.58 (<0.001)**	-0.40	0.19 (0.665)	1.53	2.04 (0.153)
Beta	EC2: Neutral Vs. Happy or Surprise		2.09	8.51 (0.004)*	-0.78	1.45 (0.229)	-0.41	0.30 (0.585)
3.48	EC3: Surprise Vs. Happy		1.65	7.87 (0.005)*	-0.65	1.35 (0.246)	0.34	0.27 (0.602)
8.11 (0.004)*	EC4: Other Negative Vs. Sad		3.13	10.68 (0.001)**	0.04	0.00 (0.966)	0.29	0.13 (0.719)
GC2: Low Vs. High Psychopathy (Continuous Contrast)	EC5: Fear or Disgust Vs. Anger		10.89	152.69 (<0.001)**	-0.87	1.11 (0.293)	-0.74	0.43 (0.514)
Beta	EC6: Disgust Vs. Fear		-12.28	153.20 (<0.001)**	-0.21	0.04 (0.835)	0.56	0.31 (0.578)
-1.01								
	W <sup>2</sup> (p)							
	0.41 (0.520)							

Note. Grand Mean (SD) [N = 61] = 80.50 (10.69). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Recognition Accuracy (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

### 2.6.2.2 Scan-path performance for temporal indices: number and duration of fixations

Scan-path performance for the two temporal parameters extracted during the emotion recognition task, which comprised the number and duration of fixations are detailed in [Table 2.10](#) and [Table 2.11](#), respectively. No significant overall group differences were obtained for either the number or duration of fixations. Statistically significant scan-path differences for the number of fixations across categories of emotion were apparent (Table 2.10), irrespective of group. There were a greater number of fixations for neutral expressions compared to happy or surprise (EC2: Means = 18.92 vs. 17.84); fear or disgust compared to anger (EC5: Means = 18.55 vs. 17.93); and for fear compared to disgust (EC6: Means = 19.02 vs. 18.07); although no group by emotion interactions were detected. Significant scan-path differences across categories of emotion for the duration of fixations were also apparent (Table 2.11), with on average shorter duration of fixations for neutral expressions compared to happy or surprise (EC2: Means = 281.39ms vs. 300.82ms), as well a trend for a shorter duration of fixations for fear compared to disgust (EC6: Means = 286.37ms vs. 301.59ms).

In addition, there were two group by emotion interactions (Table 2.11), although both were only trends. The psychosis group (Means = 292.24ms vs. 301.80ms) exhibited a shorter fixation duration for positive or neutral expressions compared to negative emotions (GC1 by EC1), while the healthy controls (Means = 297.58ms vs. 293.45ms) displayed similar fixation durations for either category. Psychosis participants with high psychopathy traits (Means = 297.57ms vs. 314.32ms) also exhibited shorter fixation durations for fear compared to disgust (GC2 by EC6), while those with low psychopathy traits (Means = 304.40ms vs. 305.29ms) had fixation durations that were roughly the same.

Table 2.10: Fixation counts (Task A) – Mean (SD) number of fixations by emotion and group, and associated analyses

Group	Fixation counts – Mean (SD) number of fixations							
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall
Healthy Control (N = 24)	17.88 (3.47)	18.26 (3.41)	17.78 (3.42)	17.83 (3.59)	19.13 (3.04)	18.33 (3.55)	18.76 (3.11)	18.28 (3.12)
Psychosis - Low Psychopathy (N = 18)	17.57 (5.07)	17.90 (5.42)	18.36 (4.28)	17.14 (5.34)	18.06 (5.85)	18.04 (5.42)	18.78 (4.70)	18.09 (4.98)
Psychosis - High Psychopathy (N = 19)	17.38 (3.84)	18.68 (3.60)	18.11 (2.99)	18.79 (3.52)	19.11 (3.77)	17.78 (3.53)	19.26 (3.63)	18.44 (3.32)
<i>Group contrasts (Between-groups)</i>	<i>Emotion Contrasts (Within-groups)</i>		<i>Emotion main effect contrasts</i>		<i>GCI by EC1 to EC6 interaction contrasts</i>		<i>GC2 by EC1 to EC6 interaction contrasts</i>	
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not	EC1: Negative Vs. Positive or Neutral		-0.06	0.65 (0.419)	-0.05	0.49 (0.482)	-0.01	0.01 (0.918)
Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise	-0.33	21.10 (<0.001)**	0.04	0.30 (0.584)	-0.05	0.40 (0.526)
0.01	0.00 (0.990)	EC3: Surprise Vs. Happy	-0.11	3.24 (0.072)	0.11	2.86 (0.091)	0.03	0.29 (0.591)
GC2: Low Vs. High Psychopathy (Continuous Contrast)	EC4: Other Negative Vs. Sad		-0.02	0.07 (0.788)	-0.03	0.19 (0.662)	0.03	0.31 (0.578)
Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger	-0.19	7.01 (0.008)*	-0.07	0.86 (0.355)	0.11	2.67 (0.102)
-0.21	0.11 (0.743)	EC6: Disgust Vs. Fear	0.25	18.97 (<0.001)**	-0.03	0.33 (0.565)	0.08	2.34 (0.126)

Note. Grand Mean (SD) [N = 61] = 18.27 (3.75). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixation Counts (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 2.11: Fixation duration (Task A) – Mean (SD) fixation duration (ms) by emotion and group, and associated analyses

Group	Fixation duration – Mean (SD) fixation duration (ms)								
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall	
Healthy Control (N = 24)	304.49 (77.44)	291.68 (68.32)	304.12 (126.39)	315.15 (162.59)	278.23 (71.04)	288.74 (69.75)	284.12 (63.16)	295.21 (81.80)	
Psychosis - Low Psychopathy (N = 18)	309.10 (132.39)	315.62 (137.19)	294.82 (113.79)	326.93 (140.15)	304.40 (142.38)	305.29 (128.81)	282.22 (100.76)	305.48 (125.51)	
Psychosis - High Psychopathy (N = 19)	301.78 (102.52)	279.38 (61.04)	288.88 (60.01)	291.29 (113.53)	279.57 (76.05)	314.32 (94.43)	277.16 (59.69)	290.34 (68.54)	
<u>Group contrasts</u> (Between-groups)			<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>		
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	
GC1: Psychosis Vs. Not			EC1: Negative Vs. Positive or Neutral	-2.07	1.55 (0.213)	3.32	4.93 (0.026)#	1.41	0.34 (0.559)
	Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise	6.00	14.45 (<0.001)**	0.19	0.01 (0.906)	-0.17	0.01 (0.993)
	-1.22	0.01 (0.914)	EC3: Surprise Vs. Happy	2.24	0.82 (0.365)	-1.73	0.37 (0.545)	-0.88	0.44 (0.508)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad	-1.57	0.88 (0.349)	0.65	0.14 (0.713)	-2.70	2.25 (0.134)
	Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger	5.32	1.79 (0.181)	3.60	0.70 (0.402)	0.31	0.00 (0.948)
	-2.98	0.04 (0.852)	EC6: Disgust Vs. Fear	-4.07	5.73 (0.017)#	1.02	0.35 (0.553)	-3.78	4.91 (0.027)#

Note. Grand Mean (SD) [N = 61] = 296.72 (92.06). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixation Duration (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

### 2.6.2.3 Scan-path performance for spatial indices: distance between fixations and overall scan-path length

Scan-path performance on the two spatial parameters extracted during the emotion recognition eye-tracking task, which comprised the mean distance between fixations and total scan-path length, are detailed in [Table 2.12](#) and [Table 2.13](#), respectively. There were no overall group differences or interaction effects for either the mean distance between fixations or the total scan-path length. Although irrespective of group, the distance between fixations was significantly shorter for negative expressions compared to positive or neutral emotions (EC1: Means = 45.27mm vs. 48.61mm). Similarly, as shown in [Table 2.13](#), the total scan-path length was significantly shorter for negative expressions compared to positive or neutral emotions (EC1: Means = 707.95mm vs. 741.74mm), and also significantly longer for fear compared to disgust (EC6: Means = 745.38mm vs. 644.51mm). In addition, the average total scan-path length tended to be longer for neutral compared to happy or surprise (EC2: Means = 776.67mm vs. 724.28mm); and for sad in comparison to the other negative emotions (EC4: Means = 732.32mm vs. 699.83mm), both at the trend level of significance.

### 2.6.2.4 Scan-path performance for regions of interest: feature versus non-feature

As illustrated in [Figure 2.6](#), the proportion of fixations significantly differed for all three salient feature regions during the emotion recognition task [ $F_{(2, 59)} = 43.30$ ,  $p < 0.001$ ], with the largest percentage of fixations to the eyes, followed by the nose and mouth (Means = 38.57, 23.70, 13.42). There was also a significant overall difference in the average duration of fixations between regions ( $N = 59$ ;  $F_{(3, 56)} = 29.95$ ,  $p < 0.001$ ), with fixations to the eyes and mouth having a similar duration (Means = 297.96ms, 301.50ms) followed by the nose (272.69ms) and other facial regions (233.42ms).

Table 2.12: Distance between fixations (Task A) – Mean (SD) distance (mm) by emotion and group, and associated analyses

Group	Distance between fixations – Mean (SD) distance (mm)									
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall		
Healthy Control (N = 24)	39.67 (10.59)	42.26 (12.21)	54.67 (50.71)	41.77 (12.97)	42.06 (12.30)	39.04 (19.75)	43.75 (13.55)	43.30 (17.12)		
Psychosis - Low Psychopathy (N = 18)	53.19 (43.78)	48.19 (25.05)	60.17 (64.94)	63.22 (77.77)	50.64 (31.45)	49.82 (43.59)	55.99 (42.92)	54.46 (43.16)		
Psychosis - High Psychopathy (N = 19)	45.54 (26.48)	42.39 (15.43)	44.50 (25.55)	47.48 (36.40)	43.24 (14.69)	38.98 (16.05)	43.25 (15.36)	43.62 (20.00)		
<u>Group contrasts</u> (Between-groups)			<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>			
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)		
GC1: Psychosis Vs. Not			EC1: Negative Vs. Positive or Neutral		1.65	10.76 (0.001)**	0.57	1.05 (0.305)	-0.72	3.69 (0.055)
	Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise		0.65	1.48 (0.223)	0.33	0.32 (0.569)	-0.27	0.69 (0.406)
	-2.73	0.80 (0.371)	EC3: Surprise Vs. Happy		-2.04	2.03 (0.155)	-1.59	1.11 (0.292)	0.08	0.00 (0.951)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad		-0.53	0.29 (0.593)	0.78	0.86 (0.354)	0.70	0.42 (0.517)
	Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger		1.945	3.14 (0.076)	-1.265	1.99 (0.159)	-0.75	0.45 (0.504)
	-6.48	2.45 (0.118)	EC6: Disgust Vs. Fear		0.74	2.52 (0.112)	0.06	0.02 (0.892)	0.15	0.08 (0.771)

Note. Grand Mean (SD) [N = 61] = 46.69 (28.03). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Distance between Fixations (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 2.13: Scan-path length (Task A) – Mean (SD) total scan-path length (mm) by emotion and group, and associated analyses

Group	Scan-path length – Mean (SD) total scan-path length (mm)									
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall		
Healthy Control (N = 24)	641.73 (184.95)	710.35 (269.82)	741.88 (206.82)	666.73 (202.31)	729.42 (196.02)	624.70 (249.22)	750.90 (272.28)	694.78 (206.46)		
Psychosis - Low Psychopathy (N = 18)	761.44 (612.57)	791.77 (494.72)	816.21 (431.95)	708.36 (464.01)	777.89 (528.39)	708.43 (477.89)	869.48 (552.02)	776.09 (487.84)		
Psychosis - High Psychopathy (N = 19)	695.76 (395.07)	703.76 (324.72)	712.51 (394.82)	764.93 (598.72)	734.76 (311.37)	608.98 (314.62)	721.29 (296.32)	706.00 (355.64)		
<u>Group contrasts</u> (Between-groups)			<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>			
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)		
GC1: Psychosis Vs. Not			EC1: Negative Vs. Positive or Neutral		16.74	7.40 (0.007)*	-2.03	0.14 (0.709)	-9.20	2.56 (0.109)
	Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise		-16.19	4.70 (0.030)#	-1.67	0.05 (0.817)	2.67	0.16 (0.693)
	-22.04	0.33 (0.563)	EC3: Surprise Vs. Happy		-16.26	3.70 (0.054)	-8.49	1.42 (0.233)	-2.56	0.04 (0.839)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad		10.65	4.67 (0.031)#	1.12	0.05 (0.817)	-3.71	1.00 (0.318)
	Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger		4.53	0.27 (0.603)	-6.22	0.63 (0.428)	7.91	2.77 (0.096)
	-77.74	1.63 (0.202)	EC6: Disgust Vs. Fear		26.99	22.86 (<0.001)**	0.83	0.02 (0.879)	4.65	0.65 (0.422)

Note. Grand Mean (SD) [N = 61] = 722.27 (350.67). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Scan-path Length (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Scan-path performance for fixations within the eye region, by group and emotion, in this instance characterised as the percent and duration of fixations, are detailed in [Table 2.14](#) and [Table 2.15](#), respectively. No significant overall group differences for either the percent or duration of fixations within the eye region were found. Irrespective of group, a significantly greater percentage of fixations within the eye region were found for other negative emotions compared to sad (EC4: Means = 38.99 vs. 35.47); and for fear compared to disgust (EC6: Means = 41.60 vs. 36.91). A group by emotion interaction effect for percent of fixations was also found (GC1 by EC3), at a trend level of significance (Table 2.14). Healthy control participants (Means = 43.94 vs. 38.53) paid proportionately more attention to the eyes when viewing happy expressions compared to surprise, while the psychosis group (Means = 37.36 vs. 37.27) exhibited around 6.6% less fixations to the eyes for happy, and a similar percentage of fixations for surprise.

In addition, for low versus high psychopathy traits a significant group by emotion interaction for fixation duration within the eye region was found (Table 2.15, GC2 by EC6:  $W^2 = 9.09$ ,  $p = 0.003$ ). Psychosis participants with high psychopathy traits (Means = 284.82ms vs. 304.88ms) spent less time per fixation within the eye region when viewing the fear compared to disgust stimuli, relative to those with low psychopathy traits who tended to have higher fixation durations for fear and lower fixation durations for disgust (Means = 302.05ms vs. 291.87ms).

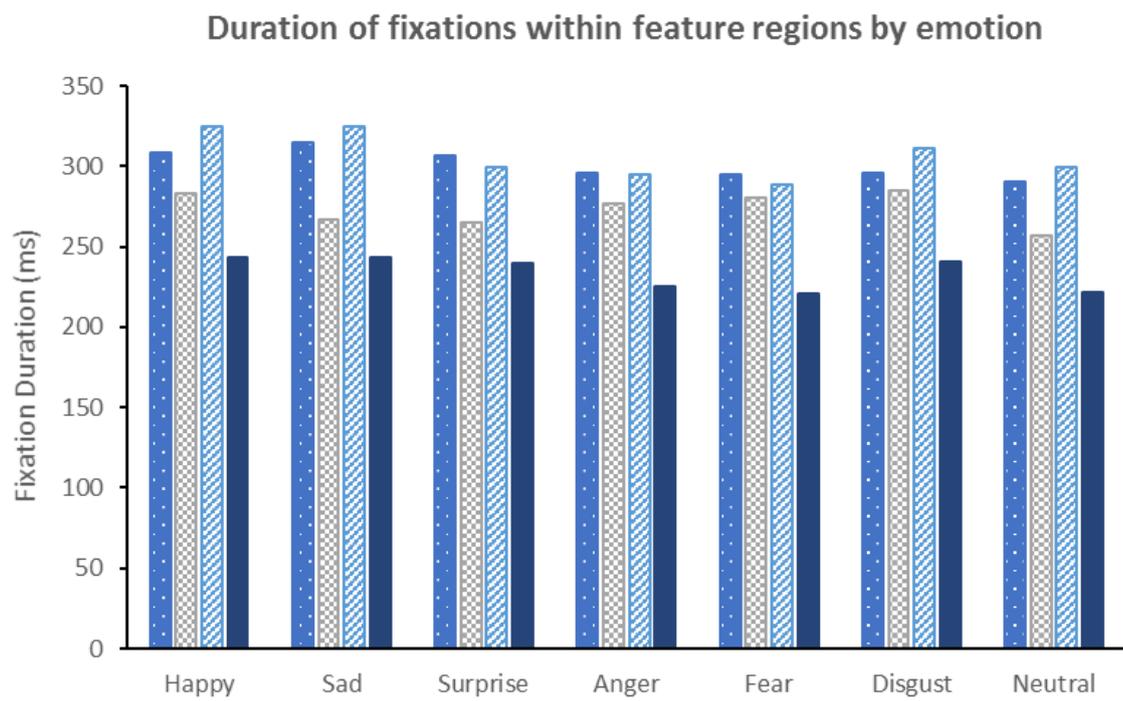
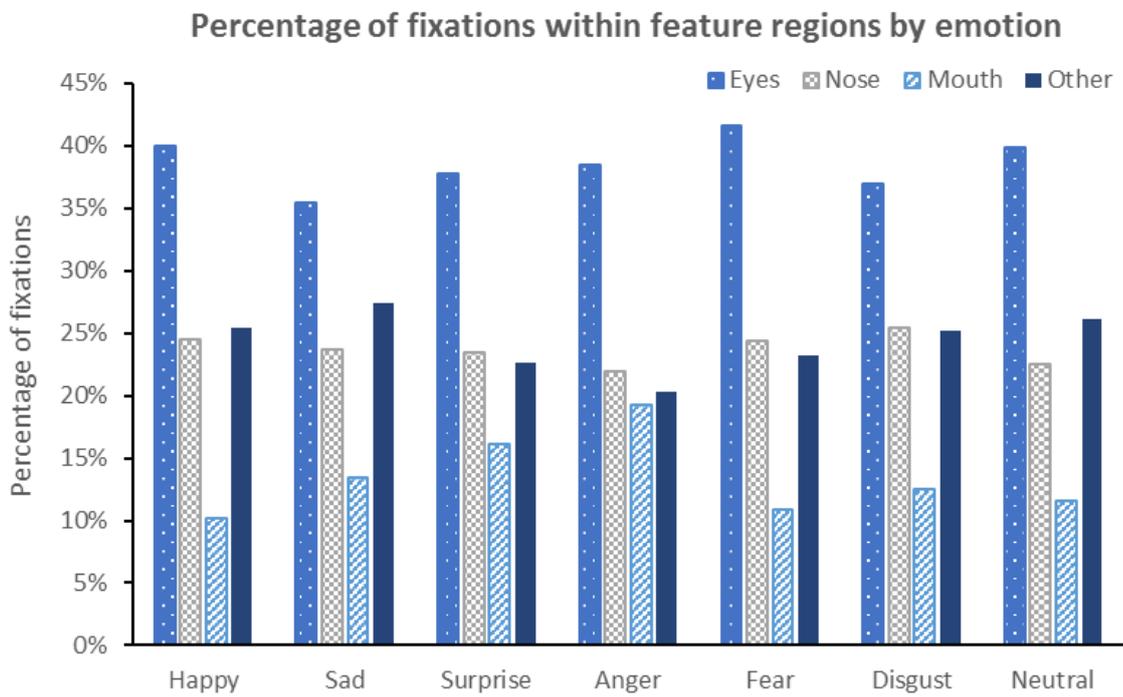


Figure 2.6 Percentage and duration of fixations within feature regions by emotion

Table 2.14: Fixations within eye region (Task A) – Mean (SD) percent within region by emotion and group, and associated analyses

Group	Fixations within eye region – Mean (SD) percent							
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall
Healthy Control (N = 24)	43.94 (20.17)	38.55 (18.27)	38.53 (17.83)	41.58 (18.61)	42.66 (18.72)	40.50 (19.13)	42.42 (19.43)	41.18 (17.98)
Psychosis - Low Psychopathy (N = 18)	39.67 (21.35)	35.02 (18.87)	38.76 (19.43)	40.60 (20.74)	46.33 (18.85)	37.56 (17.82)	40.14 (18.48)	39.71 (18.30)
Psychosis - High Psychopathy (N = 19)	35.17 (17.39)	32.01 (18.60)	35.85 (20.63)	32.48 (17.27)	35.79 (18.73)	31.76 (18.57)	36.30 (19.62)	34.19 (17.28)
<i>Group contrasts (Between-groups)</i>	<i>Emotion Contrasts (Within-groups)</i>		<i>Emotion main effect contrasts</i>		<i>GCI by EC1 to EC6 interaction contrasts</i>		<i>GC2 by EC1 to EC6 interaction contrasts</i>	
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not	EC1: Negative Vs. Positive or Neutral		0.53	3.13 (0.077)	-0.11	0.12 (0.729)	0.42	2.18 (0.140)
Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise	-0.31	0.71 (0.401)	-0.05	0.02 (0.881)	-0.05	0.01 (0.914)
2.10	0.88 (0.348)	EC3: Surprise Vs. Happy	0.59	2.76 (0.096)	0.70	3.90 (0.048)#	0.02	0.00 (0.951)
GC2: Low Vs. High Psychopathy (Continuous Contrast)	EC4: Other Negative Vs. Sad		-1.15	11.20 (0.001)**	0.13	0.15 (0.700)	0.35	1.11 (0.293)
Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger	-0.25	1.01 (0.315)	0.20	0.72 (0.395)	0.02	0.01 (0.937)
-2.78	1.36 (0.243)	EC6: Disgust Vs. Fear	1.26	11.92 (0.001)**	-0.55	2.71 (0.100)	-0.28	0.58 (0.445)

Note. Grand Mean (SD) [N = 61] = 38.57 (17.82). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixations within Eye Region (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 2.15: Fixation durations within eye region (Task A) – Mean (SD) duration (ms) by emotion and group, and associated analyses

Group	Fixation durations within eye region – Mean (SD) duration (ms)								
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall	
Healthy Control (N = 24)	306.13 (71.76)	318.20 (103.66)	332.61 (173.97)	290.71 (99.58)	297.61 (86.67)	293.23 (77.51)	278.37 (62.93)	296.56 (56.36)	
Psychosis - Low Psychopathy (N = 18)	310.23 (97.11)	325.31 (176.59)	297.91 (96.18)	320.52 (108.43)	302.05 (109.81)	291.87 (103.36)	296.77 (60.77)	300.08 (93.50)	
Psychosis - High Psychopathy (N = 19)	311.42 (151.36)	299.33 (54.09)	280.52 (55.70)	278.40 (76.79)	284.82 (56.50)	304.88 (65.81)	301.69 (72.60)	294.04 (56.69)	
<u>Group contrasts</u> (Between-groups)			<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>		
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	
GC1: Psychosis Vs. Not			EC1: Negative Vs. Positive or Neutral	0.86	0.08 (0.781)	1.61	0.29 (0.588)	3.00	0.65 (0.420)
	Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise	5.12	2.18 (0.139)	6.09	2.75 (0.097)	0.66	0.04 (0.834)
	1.03	0.01 (0.905)	EC3: Surprise Vs. Happy	0.77	0.02 (0.877)	-6.33	1.34 (0.248)	2.17	0.19 (0.665)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad	6.09	2.80 (0.094)	1.53	0.20 (0.655)	-3.73	0.46 (0.496)
	Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger	-0.41	0.00 (0.994)	-1.19	0.10 (0.747)	-3.09	0.84 (0.360)
	-3.40	0.08 (0.784)	EC6: Disgust Vs. Fear	1.61	0.03 (0.864)	1.28	0.20 (0.652)	-3.25	9.09 (0.003)*

Note. Grand Mean (SD) [N = 61] = 296.86 (68.47). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixation Durations within Eye Region (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Scan-path performance for fixations within the nose region, by group and emotion, for the percent and duration of fixations respectively, are detailed in [Table 2.16](#) and [Table 2.17](#). No significant overall group differences were found for either the percent or duration of fixations within the nose region. Irrespective of group, there was a significantly greater percentage of fixations within the nose region for fear or disgust compared to anger (EC5: Means = 24.89 vs. 21.95). There was also a significant group by emotion interaction for fixation duration (Table 2.17, GC1 by EC6:  $W^2 = 7.17$ ,  $p = 0.007$ ), whereby the psychosis group spent less time per fixation within the nose region when recognising fear versus disgust stimuli (Means = 277.17ms vs. 309.67ms), compared with the reverse pattern for the healthy controls (Means = 284.77ms vs. 245.95ms).

Two additional group by emotion interaction effects were also found, although only at a trend level of significance, for low versus high psychopathy traits for the percent (GC2 by EC4) and duration of fixations (GC2 by EC5). For the percent of fixations (Table 2.16), psychosis participants with high psychopathy traits (Means = 21.00 vs. 23.10) made less use of the nose region when categorising sad compared to other negative expressions, relative to the low psychopathy group (Means = 24.88 vs. 23.10). While for fixation duration (Table 2.17), psychosis participants with low psychopathy paid more attention to the nose when processing anger compared to fear or disgust (Means = 297.86ms vs. 282.37ms), relative to the high psychopathy group (Means = 265.11ms vs. 304.47ms).

Table 2.16: Fixations within nose region (Task A) – Mean (SD) percent within region by emotion and group, and associated analyses

Group	Fixations within nose region – Mean (SD) percent								
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall	
Healthy Control (N = 24)	23.78 (14.16)	25.01 (15.67)	23.21 (11.91)	22.37 (11.97)	25.79 (14.84)	27.43 (17.92)	23.48 (14.10)	24.44 (13.37)	
Psychosis - Low Psychopathy (N = 18)	26.72 (18.74)	24.88 (14.32)	22.73 (11.44)	21.81 (13.87)	22.68 (14.56)	24.80 (16.00)	20.80 (14.61)	23.50 (13.90)	
Psychosis - High Psychopathy (N = 19)	23.10 (14.39)	21.00 (13.35)	24.57 (11.81)	21.53 (11.99)	24.18 (14.02)	23.46 (13.50)	22.82 (12.57)	22.95 (12.03)	
<u>Group contrasts</u> (Between-groups)	<u>Emotion Contrasts</u> (Within-groups)		<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>		
	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	
GC1: Psychosis Vs. Not									
			EC1: Negative Vs. Positive or Neutral	-0.19	0.46 (0.497)	-0.51	3.16 (0.075)	0.28	1.25 (0.263)
Beta	W <sup>2</sup> (p)		EC2: Neutral Vs. Happy or Surprise	0.46	3.39 (0.066)	-0.36	2.08 (0.149)	-0.35	1.41 (0.235)
0.60	0.13 (0.716)		EC3: Surprise Vs. Happy	0.25	0.63 (0.427)	-0.08	0.07 (0.796)	-0.48	2.90 (0.088)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad	-0.06	0.05 (0.820)	0.00	0.00 (0.999)	-0.49	5.92 (0.015)#
Beta	W <sup>2</sup> (p)		EC5: Fear or Disgust Vs. Anger	-0.91	11.51 (0.001)**	-0.32	1.39 (0.238)	-0.20	0.48 (0.488)
1.48	0.82 (0.365)		EC6: Disgust Vs. Fear	-0.28	1.09 (0.296)	-0.13	0.22 (0.641)	0.23	0.83 (0.362)

Note. Grand Mean (SD) [N = 61] = 23.70 (12.92). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixations within Nose Region (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 2.17: Fixation durations within nose region (Task A) – Mean (SD) duration (ms) by emotion and group, and associated analyses

Group		Fixation durations within nose region – Mean (SD) duration (ms)							
		Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall
Healthy Control (N = 24)		267.87 (132.51)	247.44 (87.84)	247.20 (71.68)	269.76 (120.26)	284.77 (124.50)	245.95 (69.58)	251.09 (86.83)	259.84 (75.78)
Psychosis - Low Psychopathy (N = 18)		297.71 (172.35)	289.71 (197.46)	275.92 (146.27)	297.86 (190.88)	273.87 (124.43)	290.86 (155.88)	248.14 (117.99)	284.38 (149.55)
Psychosis - High Psychopathy (N = 19)		289.70 (104.09)	270.38 (105.31)	276.66 (79.26)	265.11 (88.89)	280.47 (124.36)	328.47 (112.56)	273.49 (92.01)	281.11 (78.47)
<u>Group contrasts</u> (Between-groups)		<u>Emotion Contrasts</u> (Within-groups)		<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>	
				Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not		EC1: Negative Vs. Positive or Neutral		-4.32	1.75 (0.186)	0.85	0.06 (0.802)	1.14	0.11 (0.737)
Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise		5.23	2.66 (0.103)	-2.61	0.63 (0.429)	-4.02	1.66 (0.198)
-11.56	0.91 (0.339)	EC3: Surprise Vs. Happy		4.92	1.83 (0.177)	0.49	0.02 (0.896)	-3.65	1.89 (0.169)
GC2: Low Vs. High Psychopathy (Continuous Contrast)		EC4: Other Negative Vs. Sad		-4.42	2.76 (0.097)	-1.56	0.39 (0.534)	-2.87	1.19 (0.276)
Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger		-1.84	0.36 (0.550)	2.58	0.70 (0.403)	-6.50	4.10 (0.043)#
1.25	0.00 (0.947)	EC6: Disgust Vs. Fear		-1.24	0.13 (0.714)	9.38	7.17 (0.007)*	-2.62	0.56 (0.435)

Note. Grand Mean (SD) [N = 61] = 273.58 (102.89). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixation Durations within Nose Region (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Scan-path performance for fixations within the mouth region, by group and emotion, for the percent and duration of fixations respectively, are detailed in [Table 2.18](#) and [Table 2.19](#). There were no significant overall group differences for either the percent or duration of fixations within the mouth region, although for percent of fixations to the mouth several main effects for emotion were apparent, with five of the six contrasts significant. Participants displayed significantly more fixations, making proportionately more use of the mouth when recognising negative compared to positive or neutral facial expressions of emotion (EC1: Means = 14.01 vs. 12.63). As well as when comparing: happy or surprise to neutral (EC2: Means = 13.17 vs. 11.54); surprise to happy (EC3: Means = 16.12 vs. 10.21); anger to fear or disgust (EC5: Means = 19.26 vs. 11.68); and disgust to fear (EC6: Means = 12.54 vs. 10.82). For the duration of fixations ([Table 2.19](#)), a trend level association was detected, with people tending to spend more time on the mouth when recognising sad relative to other negative emotions (EC4: Means = 324.39ms vs. 298.47ms). There were no significant group by emotion interactions, although there was one trend level association for fixation duration (GC2 by EC1). Psychosis participants with low psychopathy traits (Means = 356.04ms vs. 315.21ms) tended to spend longer looking at the mouth when viewing negative emotions compared to the high psychopathy group (Means = 279ms vs. 297.96ms).

Scan-path performance for fixations within non-feature regions, by group and emotion, for the percent and duration of fixations respectively, are detailed in [Table 2.20](#) and [Table 2.21](#). No significant overall group differences for either the percent or duration of fixations within non-feature regions were apparent. Overall, participants tended to have an increased proportion of fixations to non-feature areas for neutral compared to expressions of happy or surprise (EC2: Means = 26.13 vs. 24.01); and

Table 2.18: Fixations within mouth region (Task A) – Mean (SD) percent within region by emotion and group, and associated analyses

Group	Fixations within mouth region – Mean (SD) percent									
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall		
Healthy Control (N = 24)	9.99 (8.18)	12.11 (8.79)	15.33 (9.07)	19.05 (8.15)	10.28 (8.56)	12.04 (8.48)	10.70 (8.51)	12.78 (7.46)		
Psychosis - Low Psychopathy (N = 18)	7.89 (5.06)	12.76 (8.95)	16.98 (11.41)	16.99 (7.87)	9.00 (8.10)	12.31 (7.85)	12.14 (8.69)	12.58 (6.56)		
Psychosis - High Psychopathy (N = 19)	12.70 (12.15)	15.76 (12.56)	16.32 (11.00)	21.68 (9.18)	13.22 (12.37)	13.40 (12.24)	12.04 (11.28)	15.02 (10.47)		
<u>Group contrasts</u> (Between-groups)			<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>			
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)		
GC1: Psychosis Vs. Not			EC1: Negative Vs. Positive or Neutral		-0.69	10.72 (0.001)**	0.01	0.00 (0.960)	-0.29	1.51 (0.220)
	Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise		0.50	7.55 (0.006)*	0.08	0.22 (0.636)	0.13	0.72 (0.395)
	-0.51	0.27 (0.604)	EC3: Surprise Vs. Happy		-1.58	28.60 (<0.001)**	0.12	0.21 (0.651)	0.40	1.13 (0.287)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad		-0.25	1.16 (0.282)	-0.24	1.18 (0.278)	0.01	0.00 (0.960)
	Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger		2.34	52.68 (<0.001)**	0.08	0.06 (0.815)	0.09	0.07 (0.789)
	1.79	1.75 (0.186)	EC6: Disgust Vs. Fear		-0.46	5.17 (0.023)#	-0.01	0.00 (0.966)	0.07	0.11 (0.746)

Note. Grand Mean (SD) [N = 61] = 13.42 (8.22). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixations within Mouth Region (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 2.19: Fixation durations within mouth region (Task A) – Mean (SD) duration (ms) by emotion and group, and associated analyses

Group	Fixation durations within mouth region – Mean (SD) duration (ms)									
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall		
Healthy Control (N = 24)	316.47 (104.75)	310.37 (97.45)	307.16 (144.51)	284.74 (94.27)	266.19 (101.48)	284.21 (93.72)	305.18 (93.90)	289.40 (74.79)		
Psychosis - Low Psychopathy (N = 18)	333.52 (200.07)	376.67 (178.54)	297.84 (148.23)	331.69 (148.55)	342.79 (186.03)	373.41 (202.91)	314.28 (182.32)	332.77 (146.55)		
Psychosis - High Psychopathy (N = 19)	325.72 (123.60)	292.92 (111.05)	290.27 (88.00)	274.48 (108.44)	266.08 (92.83)	283.07 (102.87)	277.88 (106.06)	284.71 (78.57)		
<u>Group contrasts</u> (Between-groups)			<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>			
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)		
GC1: Psychosis Vs. Not			EC1: Negative Vs. Positive or Neutral		1.31	0.10 (0.752)	8.23	3.44 (0.064)	7.68	4.15 (0.042)#
	Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise		3.88	1.03 (0.312)	-1.48	0.14 (0.711)	4.87	1.57 (0.211)
	-7.91	0.44 (0.505)	EC3: Surprise Vs. Happy		6.74	2.10 (0.147)	-3.43	0.52 (0.473)	-1.56	0.11 (0.739)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad		8.70	3.85 (0.050)#	1.44	0.11 (0.741)	-4.92	0.93 (0.336)
	Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger		-1.38	0.10 (0.755)	3.49	0.80 (0.370)	10.60	3.19 (0.074)
	-15.69	0.62 (0.430)	EC6: Disgust Vs. Fear		-5.75	2.02 (0.155)	0.75	0.04 (0.835)	0.38	0.00 (0.954)

Note. Grand Mean (SD) [N = 61] = 300.74 (102.58). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixations Durations within Mouth Region (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

happy compared to surprise (EC3: Means = 25.40 vs. 22.62). There was a significantly increased percent of fixations to non-feature areas for sad when compared to other negative emotions (EC4: Means = 27.37 vs. 22.89); and for fear or disgust compared to anger (EC5: Means = 24.17 vs. 20.34). While the duration of fixations within non-feature areas were only significantly shorter for neutral expressions compared to happy or surprise (Table 2.21, EC2: Means = 221.60ms vs. 241.70ms).

There were also a number of group by emotion interaction effects for psychosis versus not, in relation to the percent of fixations within non-feature areas, one significant interaction (GC1 by EC3:  $W^2 = 6.99$ ,  $p = 0.008$ ) and two at trend level significance (GC1 by EC1; GC1 by EC6). The psychosis group (Means = 27.42 vs. 22.42) paid significantly more attention to non-feature areas when viewing happy compared to surprise, while the healthy controls (Means = 22.29 vs. 22.93) spent a similar percent in non-feature areas for both emotions. The psychosis group (Means = 26.17 vs. 25.19) also tended to have an increased but similar percentage of fixations to non-feature areas for both negative and positive or neutral expressions, while healthy controls (Means = 20.66 vs. 22.87) exhibited a slightly higher percent of fixations to non-feature areas only for positive or neutral emotions. In addition, the psychosis group (Means = 28.43 vs. 24.46) tended to have a greater percent of fixations to non-feature areas for disgust compared to fear, than the healthy controls (Means = 20.43 vs. 21.28). For low versus high psychopathy, a trend level group by emotion interaction for fixation duration was also found (GC2 by EC5). Psychosis participants with high psychopathy traits (Means = 212.43ms vs. 231.11ms) tended to spend less time per fixation in non-feature areas when viewing anger, with a lower mean fixation duration compared to fear or disgust, than those with low psychopathy traits (Means = 302.05ms vs. 291.87ms).

Table 2.20: Fixations within non-feature region (Task A) – Mean (SD) percent by emotion and group, and associated analyses

Group	Fixations within non-feature region – Mean (SD) percent									
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall		
Healthy Control (N = 24)	22.29 (23.93)	24.34 (22.40)	22.93 (22.30)	17.00 (15.47)	21.28 (23.19)	20.03 (23.49)	23.40 (20.59)	21.60 (21.11)		
Psychosis - Low Psychopathy (N = 18)	25.72 (21.20)	27.34 (23.74)	21.53 (22.03)	20.59 (18.96)	21.99 (22.67)	25.33 (22.91)	26.92 (24.21)	24.21 (21.34)		
Psychosis - High Psychopathy (N = 19)	29.03 (22.31)	31.24 (20.78)	23.27 (16.70)	24.31 (19.16)	26.81 (20.28)	31.37 (24.79)	28.83 (23.01)	27.84 (20.14)		
<u>Group contrasts</u> (Between-groups)			<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>			
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)		
GC1: Psychosis Vs. Not			EC1: Negative Vs. Positive or Neutral		0.35	1.56 (0.212)	0.60	5.24 (0.022)#	-0.40	1.90 (0.168)
	Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise		-0.66	5.45 (0.020)#	0.33	1.41 (0.235)	0.26	0.98 (0.323)
	-2.18	0.69 (0.407)	EC3: Surprise Vs. Happy		0.74	6.42 (0.011)#	-0.74	6.99 (0.008)*	0.06	0.03 (0.853)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad		1.47	22.14 (<0.001)**	0.11	0.14 (0.708)	0.13	0.18 (0.675)
	Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger		-1.18	12.56 (<0.001)**	0.04	0.02 (0.899)	0.09	0.12 (0.727)
	-0.48	0.03 (0.870)	EC6: Disgust Vs. Fear		-0.51	2.17 (0.141)	0.68	4.76 (0.029)#	-0.02	0.00 (0.967)

Note. Grand Mean (SD) [N = 61] = 24.31 (20.69). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixations within Non-feature Region (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 2.21: Fixation durations within non-feature region (Task A) – Mean (SD) duration (ms) by emotion and group, and associated analyses

Group	Fixation durations within non-feature region – Mean (SD) duration (ms)									
	Happy	Sad	Surprise	Anger	Fear	Disgust	Neutral	Overall		
Healthy Control (N = 24)	245.81 (88.69)	253.37 (79.86)	259.69 (83.08)	228.33 (85.28)	219.84 (58.50)	243.51 (101.42)	238.89 (76.78)	243.06 (69.71)		
Psychosis - Low Psychopathy (N = 18)	250.90 (138.48)	240.20 (154.57)	209.53 (75.00)	235.20 (123.90)	223.57 (112.74)	233.01 (101.20)	202.95 (70.73)	224.03 (74.01)		
Psychosis - High Psychopathy (N = 19)	233.01 (88.63)	234.59 (72.04)	245.17 (90.09)	212.43 (67.47)	218.88 (65.50)	243.33 (93.60)	217.20 (50.70)	230.87 (54.67)		
<u>Group contrasts</u> (Between-groups)			<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC6 interaction contrasts</u>		<u>GC2 by EC1 to EC6 interaction contrasts</u>			
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)		
GC1: Psychosis Vs. Not			EC1: Negative Vs. Positive or Neutral		1.27	0.18 (0.675)	3.71	1.80 (0.180)	-0.77	0.06 (0.802)
	Beta	W <sup>2</sup> (p)	EC2: Neutral Vs. Happy or Surprise		6.27	7.76 (0.005)*	-1.61	0.55 (0.460)	0.89	0.15 (0.696)
	6.24	0.53 (0.468)	EC3: Surprise Vs. Happy		0.79	0.04 (0.837)	-3.63	1.14 (0.286)	-5.60	1.02 (0.312)
GC2: Low Vs. High Psychopathy (Continuous Contrast)			EC4: Other Negative Vs. Sad		4.86	1.40 (0.236)	2.11	0.37 (0.543)	4.24	1.67 (0.196)
	Beta	W <sup>2</sup> (p)	EC5: Fear or Disgust Vs. Anger		-1.58	0.37 (0.544)	0.44	0.32 (0.859)	-5.03	4.26 (0.039)#
	6.46	0.49 (0.487)	EC6: Disgust Vs. Fear		-5.27	3.60 (0.058)	-0.85	0.10 (0.756)	-1.32	0.35 (0.554)

Note. Grand Mean (SD) [N = 61] = 233.65 (66.16). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 20 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixation Durations within Non-feature Region (N = 61 participants, across 7 emotions): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

#### 2.6.2.5 Relationships between Task A indices

As shown in the first row of [Table 2.22](#), there was no relationship between recognition accuracy and any of the scan-path parameters for the emotion recognition eye-tracking task. As anticipated, the scan-path indices were not independent. There was a strong inverse association between the number and duration of fixation indices ( $r = -0.80$ ). Scan-path length was influenced by all fixation duration indices (correlations ranging from  $r = -0.41$  to  $-0.56$ ), such that as fixation duration increased scan-path length reduced. Similarly, scan-path length was associated with all indices based on the number of fixations. There was also an association between duration of fixations across regions of interest, with a similar pattern for the mean duration of fixations across regions (ranging from  $r = -0.33$  to  $-0.53$ ).

Table 2.22: Correlations between Task A indices – Overall (427 measures per index; N = 61 participants, across 7 emotions).

<i>Task A index</i>	Number of fixations	Fixation duration	Distance between fixations	Scan-path length	Eye region percent fixations	Eye region fixation duration	Nose region percent fixations	Nose region fixation duration	Mouth region percent fixations	Mouth region fixation duration	Non-feature region percent fixations	Non-feature region fixation duration
Recognition accuracy	-0.07 (0.134)	0.06 (0.210)	-0.06 (0.228)	-0.06 (0.231)	-0.02 (0.762)	0.05 (0.337)	-0.02 (0.745)	-0.04 (0.379)	0.02 (0.669)	0.04 (0.387)	0.01 (0.779)	0.08 (0.090)
Number of fixations		-0.80 ( $<0.001$ )**	0.21 ( $<0.001$ )**	0.66 ( $<0.001$ )**	0.24 ( $<0.001$ )**	-0.64 ( $<0.001$ )**	-0.30 ( $<0.001$ )**	-0.69 ( $<0.001$ )**	-0.20 ( $<0.001$ )**	-0.66 ( $<0.001$ )**	0.07 (0.149)	-0.55 ( $<0.001$ )**
Fixation duration			-0.25 ( $<0.001$ )**	-0.56 ( $<0.001$ )**	-0.28 ( $<0.001$ )**	0.64 ( $<0.001$ )**	0.34 ( $<0.001$ )**	0.77 ( $<0.001$ )**	0.25 ( $<0.001$ )**	0.70 ( $<0.001$ )**	-0.08 (0.088)	0.56 ( $<0.001$ )**
Distance between fixations				0.68 ( $<0.001$ )**	-0.15 (0.003)*	-0.14 (0.004)*	-0.26 ( $<0.001$ )**	-0.15 (0.003)*	-0.04 (0.450)	-0.11 (0.027)#	0.31 ( $<0.001$ )**	-0.27 ( $<0.001$ )**
Scan-path length					-0.02 (0.709)	-0.41 ( $<0.001$ )**	-0.40 ( $<0.001$ )**	-0.47 ( $<0.001$ )**	-0.14 (0.005)*	-0.44 ( $<0.001$ )**	0.34 ( $<0.001$ )**	-0.44 ( $<0.001$ )**
Eye region percent fixations						-0.16 (0.001)**	-0.11 (0.018)#	-0.37 ( $<0.001$ )**	-0.33 ( $<0.001$ )**	-0.25 ( $<0.001$ )**	-0.65 ( $<0.001$ )**	-0.28 ( $<0.001$ )**
Eye region fixation duration							0.17 ( $<0.001$ )**	0.45 ( $<0.001$ )**	0.14 (0.004)*	0.51 ( $<0.001$ )**	-0.04 (0.385)	0.33 ( $<0.001$ )**
Nose region percent fixations								0.39 ( $<0.001$ )**	-0.01 (0.811)	0.17 (0.001)**	-0.54 ( $<0.001$ )**	0.05 (0.343)
Nose region fixation duration									0.16 (0.001)**	0.53 ( $<0.001$ )**	0.01 (0.870)	0.39 ( $<0.001$ )**
Mouth region percent fixations										0.26 ( $<0.001$ )**	-0.16 (0.001)**	0.17 ( $<0.001$ )**
Mouth region fixation duration											0.00 (0.992)	0.42 ( $<0.001$ )**
Non-feature region percent fixations												0.14 (0.004)*

Note. Pearson product-moment correlations, with two-tailed p values in brackets: # trend ( $p < 0.05$ ); \*  $p < 0.01$ ; \*\*  $p < 0.001$ .

## 2.7 Discussion

In the current study the vast majority of the psychosis group were in the remitted illness phase, with a mean age of 43.5 years, and an illness duration of 17.9 years. As anticipated, comparisons between the healthy controls and psychosis groups revealed significant illness related differences in neurocognitive functioning, psychiatric symptomatology and employment. There were, however, no sociodemographic group differences for age, gender, level of education, or pre-morbid IQ. Not dissimilar to the social isolation and unemployment challenges noted in the Australian National Survey of High Impact Psychosis (SHIP) project (Morgan et al., 2017), most of the psychosis group had never married (or lived in a de facto relationship, 91.9%), and a substantial number were currently living alone (35.1%) and unemployed (91.9%; with 86.5% receiving a disability support pension). In addition, when comparing the psychosis groups with low and high psychopathy traits on the PCL: SV, an increased incidence of offending was apparent among the high psychopathy group, primarily relating to increased offences against people and property. This is broadly consistent with research among offender populations, with psychopathic offenders reported as being more versatile in offending patterns, committing significantly more violent and non-violent offences (Kosson et al., 1990).

The level of impairment in neuropsychological functioning based on the RBANS total score among the psychosis groups (Mean = 82.11) was consistent with prior large Australian community and outpatient Mental Health Services samples (Loughland et al., 2010; Loughland, Lewin, Carr, Sheedy, & Harris, 2007), while performance by the healthy controls (Mean = 101.21) fell within expected normative population ranges. Current psychiatric symptoms, measured on the BPRS, were

predominantly in the ‘mild range’ (Mean 37.86), comparable to Australian and community psychosis samples more broadly (Loughland et al., 2010; McCleery et al., 2016). Substantial impairments in both global and social and occupational functioning were detected among the psychosis groups, ranging from ‘serious to moderate symptoms/impairment’ (American Psychiatric Association, 2000). This was consistent with the degree of impairment reported among other Australian psychosis studies (Loughland et al., 2010; McCabe et al., 2012; Moore, Green, & Carr, 2012), in this instance falling largely within the ‘medium’ functioning range (mean = 53.64) on the GAF (Loughland et al., 2007). Interestingly, the high psychopathy group exhibited poorer functioning in comparison to the low psychopathy group, perhaps in part, reflective of referral source differences (i.e., Psychiatric Rehabilitation, living in supported accommodation), and not inconsistent with a previously reported range in functioning among psychosis samples across different settings (Loughland et al., 2007).

Self-reported impairment in Quality of Life (AQoL-6D) was significantly elevated for both physical and psychological wellbeing (as well as overall) among the psychosis groups compared to healthy controls, who had similar scores to those reported in a large scale longitudinal Australian community cohort (Allen et al., 2013). Increased experience of childhood adversity (as measured on the CAQ) was detected among the psychosis groups, including abusive or dysfunctional parenting, loss, poverty or sexual abuse, and sibling loss; patterns which were not unlike those previously reported for psychosis (McCabe et al., 2012). However, a higher incidence of loss among our study sample was observed, which appears to be largely due to not restricting the documentation of loss to prior to the age of 18, in contrast to McCabe et al. (2012), who reported lower rates for loss up until the age of 18 years.

Consistent with research demonstrating high rates of comorbid personality disorder in schizophrenia using the IPDEQ (overall dimensional scores for: psychosis  $19.8/59 = 0.34$  vs. healthy controls  $10.0/59 = 0.17$ ; Moore et al., 2012), increased comorbid personality traits were detected among the psychosis groups compared to the healthy controls. Moreover, this was confirmed in the current study for all three personality disorder clusters (A, B & C), as well as overall. Personality disorder profiles among the healthy controls were also similar those reported in the Australian national mental health survey (Lewin et al., 2005). It has been suggested that high rates of comorbid personality disorders, and screening positive on multiple clusters, may impact on the emergence of clinical features and cognitive impairments in schizophrenia (Moore et al., 2012). In addition, whilst elevated among the psychosis groups, the rates of personality disorder were slightly lower than those reported among some other clinical groups, for example, individuals with depression and co-occurring alcohol misuse (McCarter et al., 2016).

Consistent with the primary study hypothesis and established findings (Chan et al., 2010; Kohler et al., 2010), performance on the emotion recognition task revealed that the psychosis groups displayed significantly poorer overall recognition accuracy compared to healthy controls. However, the secondary hypotheses were unsupported, in that elevated psychopathy traits among the psychosis group did not have a significant impact on performance accuracy. This is inconsistent with prior research indicating an association between psychopathy and emotion recognition deficits (Dawel et al., 2012; Wilson et al., 2011). However, mixed findings exist in the psychopathy literature, for example, some studies report an association between emotion recognition accuracy and psychopathy traits (Gillespie, Mitchell, Satherley, Beech, & Rotshtein, 2015a; Gillespie,

Rotshtein, Satherley, Beech, & Mitchell, 2015b; Prado, Treeby, & Crowe, 2015), while others have not (Gillespie et al., 2017b; Gillespie et al., 2015c). Consistent with the current study, no relationship between primary or secondary psychopathic traits and emotion recognition accuracy was reported among a community sample of male non-offenders (Gillespie et al., 2015c), or among violent offenders (Gillespie et al., 2017b). Although, in both studies primary psychopathic traits were associated with atypical visual scanning strategies (Gillespie et al., 2017b; Gillespie et al., 2015c).

While previous studies, including those by Gillespie and colleagues report high psychopathy traits as associated with reduced emotion recognition accuracy, in both community (Gillespie et al., 2015a) and violent offender samples (Gillespie et al., 2015b), this was not demonstrated among our psychosis groups. Not unlike these studies, static facial expression stimuli were utilised in the current series from the same stimulus set (Tottenham et al., 2009). The capacity to make direct comparisons between existing studies and the current sample of psychosis participants with elevated psychopathy traits is limited. However, inconsistent with findings from the current study, a previous study by Fullam and Dolan (2006b) examining the impact of psychopathy traits on emotion recognition in schizophrenia, found patients with a high number of psychopathic traits exhibited increased impairment in the recognition of sadness at the lowest intensity. While recognition accuracy for disgust was negatively related to severity of cognitive symptoms (Fullam & Dolan, 2006b). This study however, utilised morphed facial stimuli to create variable expression intensity, which may have contributed to increased task difficulty.

The level of emotion recognition impairment displayed among our psychosis groups, during a relatively low demand task, was in the moderate range, with an overall mean accuracy of 77.0% compared to 84.8% among the healthy controls. The extent of impairment exhibited accords with other psychosis research, and with expectations of less impaired processing of static compared to dynamic emotional stimuli among individuals with psychosis (Song et al., 2015). In addition, while performance accuracy significantly varied across emotion categories, there was no evidence to suggest that the impairment was emotion specific, with no group by emotion effects. This can be seen as consistent with previous meta-analyses confirming generalised deficits across emotion categories (Kohler et al., 2010). This is also in line with affect recognition performance among multi-episode schizophrenia samples, demonstrating a more generalised impairment in later illness stages (Romero-Ferreiro et al., 2016).

Irrespective of group, emotion recognition accuracy was significantly reduced for negative versus positive or neutral emotions; neutral versus happy or surprise; surprise versus happy; other negative emotions versus sad; fear or disgust versus anger; and fear versus disgust. These emotion specific differences are generally consistent with research indicating for example, that happy facial expressions are easier for people to recognise, and identified more rapidly than other expressions even at low intensity levels among clinical samples (Kohler et al., 2004; Loughland et al., 2002b; Williams et al., 2001). Similarly, consistent with our findings, lower accuracy in the ability to recognise expressions of fear has been reported among clinical and offender samples (Gillespie et al., 2017b).

Furthermore, analyses of misattribution only revealed one significant group difference, with a tendency for the psychosis group with high psychopathy not to make the same misattribution of “sad as disgust” as the healthy controls or the psychosis group with low psychopathy. The psychosis group with high psychopathy tended to mistake “sad as neutral”, implying that they may not have been picking up on the intensity of emotion displayed. Consistent with Kee et al. (2006), misattributions among the psychosis group were distributed across emotion categories. This is contrary to studies suggesting a negativity bias involving the misattribution of neutral stimuli in psychosis (Edwards et al., 2001), or differentially amongst those with high psychopathy traits (Eisenbarth et al., 2008). However, face recognition tasks that are more challenging due to factors such as reduced stimulus presentation time, are likely to impact on performance and limit the generalisability of findings across studies.

An examination of the accompanying scan-path indices during the emotion recognition task did revealed some atypical scan-path results. Regardless of group, for the temporal scan-path indices, there were significant differences across emotion categories, including increased fixations of shorter duration for neutral expressions compared to happy or surprise, and for fear compared to disgust, as well as increased fixations for fear or disgust versus anger. More importantly, at a group level, psychosis participants tended to exhibit shorter fixation duration for positive or neutral expressions compared to negative emotions. Consistent with previous eye-tracking studies (Loughland et al., 2002a, 2002b; Streit et al., 1997; Williams et al., 2001), a longer duration of fixations for negative emotions among the psychosis group could be seen to demonstrate impaired visual scanning performance (on this temporal scan-path parameter). While psychosis participants with high psychopathy traits also exhibited

shorter fixation durations for fear stimuli compared to disgust. Which is broadly consistent with the reduced overall dwell time previously reported among community (Gillespie et al., 2015c) and offender samples with high psychopathy traits (Gillespie et al., 2017b).

On the spatial indices, while there were no group or interaction effects, as a measure of attentional processing, emotion specific differences were demonstrated. These entailed a significant increase in the distance between fixations and the total scan-path length for positive or neutral compared to negative emotions. This is potentially related to increased expressivity, in accordance with the literature demonstrating emotion specific differences in the importance of particular facial areas, such as, in the recognition of surprise by observing wide-open eyes and mouth (Hoffmann et al., 2013). Other emotion specific differences, such as significantly longer scan-paths for fear (compared to disgust), and the tendency for longer scan-paths for neutral (compared to happy and surprise), and sad (compared to other negative) stimuli, could perhaps relate to an increased difficulty associated with recognising these particular emotions. This is also consistent with evidence suggesting that negative and positive expressions involve different processing styles, for example, sad expressions require greater sequential processing of features, than happy expressions, which rely more on configurational processing (Loughland et al., 2002b; McKelvie, 1995).

Analyses considering regions of interest revealed additional atypical scan-path findings. Firstly, regardless of group, the proportion of fixations significantly differed for all three salient feature regions, with the largest percentage of fixations to the eyes, followed by the nose and mouth. There were also significant overall differences in the

average duration of fixations, with fixation times for the eyes and mouth having a similar duration, followed by the nose and then other facial regions. An examination of scan-path performance, utilising the percent and duration of fixations within the eye region, revealed that (irrespective of group) there were a significantly higher percent of fixations to the eyes for other negative emotions compared to sad, and for fear compared to disgust. In terms of group differences, the psychosis group tended to pay less attention to the eyes than healthy controls, when processing happy expressions (compared to surprise). Consistent with findings from previous eye-tracking studies in psychosis (Loughland et al., 2002b). Similarly, psychosis participants with high psychopathy traits spent significantly less time per fixation within the eye region when processing fear (compared to disgust). Which is consistent with previous reports of reduced attention to the eyes, among both children and adolescent with high callous unemotional psychopathy traits (Dadds et al., 2008; Dadds et al., 2006), and adult males with high psychopathy traits (Gillespie et al., 2017b; Gillespie et al., 2015c) when processing facial expression depicting fear.

For percent and duration of fixations within the nose region, irrespective of group, there was a significantly greater percentage of fixations within the nose region for fear or disgust compared to anger. A significant group by emotion interaction for fixation duration demonstrated that the psychosis group spent significantly less time in the nose region when recognising fear compared to disgust, in comparison to the healthy controls who did the opposite. Psychosis participants with high psychopathy traits also tended to make less use of the nose region when categorising sad compared to other negative expressions, with a lower percent of fixations than the low psychopathy group. While for fixation duration, psychosis participants with low psychopathy paid

more attention to the nose when processing anger (compared to fear or disgust) than the high psychopathy group.

For the percent of fixations to the mouth, participants (irrespective of group) made proportionately more use of the mouth when recognising negative compared to positive or neutral facial expressions; similarly, when comparing happy or surprise to neutral, surprise to happy, anger to fear or disgust, and disgust to fear. While for the duration of fixations, participants tended to spend proportionately more time fixating on the mouth when recognising sad relative to other negative emotions. As, previously mentioned this is likely to be also largely consistent with the differing processing strategies used for different emotions. There was only one group by emotion interaction, with psychosis participants with low psychopathy tending to spend longer looking at the mouth (increased duration of fixations) when viewing negative compared to positive or neutral emotions, than the high psychopathy group.

Conversely, analyses examining the percent and duration of fixations to non-feature areas revealed several interesting scan-path results. Participants tended to spend an increased proportion of fixations in non-feature areas for neutral compared to expressions of happy or surprise, and happy in comparison to surprise; as well as a significantly increased percent of fixations to non-feature areas for sad compared to other negative emotions, and for fear or disgust compared to anger. Overall, the duration of fixations within non-feature areas were only significantly shorter for neutral expressions in comparison to happy or surprise. Group by emotion interactions demonstrated that the psychosis group paid significantly more attention to non-feature areas when viewing happy compared to surprise expressions. This increased percent of

fixations, around 5% more to non-feature regions than healthy controls, could in part explain the 6.6% less fixations to the eyes observed when psychosis participants viewed happy facial expressions. The psychosis group also tended to have an increased but similar percentage of fixations to non-feature areas for negative and positive or neutral expressions, as opposed to healthy controls who exhibited a slightly higher percent of fixations to non-feature areas for positive or neutral expressions. These findings are largely consistent with previous eye-tracking studies that have reported reduced attention to salient features, during facial emotion processing in schizophrenia (Loughland et al., 2002a, 2002b; Williams et al., 2001). Psychosis participants with high psychopathy traits also tended to have an increased percentage of fixations to non-feature areas for disgust (compared to fear) than healthy controls; as well as tending to spend less time in non-feature areas when viewing anger, with a lower mean fixation duration compared to fear or disgust than the psychosis participants with low psychopathy traits.

An examination of the relationship between visual scanning performance and recognition accuracy revealed no associations between any of the scan-path indices and accuracy. This is inconsistent with studies reporting an association between atypical scan-paths and emotion recognition performance (Loughland et al., 2002a, 2002b; Williams et al., 2001). However, the relevant literature contains mixed findings, with some studies reporting deficits in both facial emotion recognition performance and eye-movement abnormalities but no significant associations between the two (Streit et al., 1997). Similarly, in the current study, some atypical scan-path patterns were exhibited among the psychosis group, as well as differentially for those with high psychopathy traits, but were not found to be significantly associated with recognition accuracy.

In the current study, while high psychopathy traits among the psychosis group were not associated with recognition accuracy, as noted some differential atypical scanning patterns were exhibited. These included: significantly shorter fixation durations; less time per fixation within the eye region when processing fear (compared to disgust); a tendency to pay less attention to the nose when recognising sad (compared to other negative expressions); an increased percent of fixations to non-feature areas when processing disgust (compared to fear); and spending less time in non-feature areas when viewing anger (compared to fear or disgust), with a lower mean fixation duration. Based on these findings, we are unable to provide strong support for either of the two major etiological theories addressing different mechanisms as to how facial emotion expressions are processed among individuals with high psychopathy traits. However, with regard to a possible amygdala-mediated deficit (*Integrated Emotions System*) (Blair, 2006) in psychopathy, while emotion recognition deficits for fear and sad facial expressions were not found to be associated with high psychopathy traits among the psychosis group, some atypical visual scanning patterns were observed for fear stimuli (i.e., shorter fixation durations), indicative of reduced allocation of attention.

Furthermore, also providing some support for an alternate theory of an attention mediated deficit (*response modulation hypothesis*) (Newman et al., 2010), some attentional or visual processing abnormalities were exhibited across emotions, with reduced attention to salient features (i.e., eyes when processing fear), and paying attention to information in the periphery (i.e., non-feature areas when processing disgust). Additionally, there was some evidence suggesting that subtle but important information was not noticed (i.e., misattribution of sad as neutral), indicative of possible dysfunction in higher order cognitive processes among the psychosis group with high

psychopathy traits. Visual processing also varied by emotion, consistent with finding by Munneke et al. (2018) that top down attentional influences may not determine the processing of all types of facial expressions in psychopathy. However, when considering the clinical utility of the current study, it must be noted that the generalisability of these findings are not without limitations. Whilst diagnostic complexity is apparent among a group of psychosis participants with low to high psychopathy traits, those in the high psychopathy traits group did not all meet the formal diagnostic criteria for psychopathy, making direct comparisons with the psychopathy literature difficult.

## **Chapter 3 – Study 2: Emotion Induction**

### **3.1 Introduction**

The first study provided evidence of impaired emotion recognition and some atypical scan-path patterns in psychosis, but emotion perception performance was not differential by high psychopathy traits. Emotion refers to a collection of psychological states, that includes the subjective experience, expressive behaviour (i.e., facial, bodily, verbal), as well as peripheral physiological responses (e.g., heart rate, respiration) (Gross & Barrett, 2011). Therefore, the conscious experience of emotion is not limited to identification or emotion recognition, but also involves the subjective experience (i.e., valence and arousal) (Hoppenbrouwers et al., 2016a). It remains unclear if the subjective experience of emotion or mood induction performance differs among this clinical group. The main objective of study 2 was to explore how emotional stimuli are processed when individuals are not asked to categorise or identify the emotion displayed (i.e., emotion recognition as in Task A study 1) but rather to feel or experience that emotion during an emotion induction task. Of particular interest is how performance, in terms of mood induction and visual perceptual scanning, is differentially impacted by the emotional content being displayed, and whether aspects of clinical presentation have an additional impact on performance (i.e., symptoms, psychopathy traits, mood responsivity and emotion regulation).

### **3.2 Symptoms, emotion regulation and emotion experience in psychosis**

Psychotic disorders are complex; schizophrenia for example, is characterised by several independent symptom domains, specifically positive, disorganised and negative symptom clusters, which can restrict individuals in social engagement (Shayegan &

Stahl, 2005) and impact negatively on quality of life (Eack & Newhill, 2007; Eack, Newhill, Anderson, & Rotondi, 2007; Nevarez-Flores et al., 2018).

### *3.2.1 Symptoms and emotion regulation*

Most individuals with psychosis present a mixed syndrome. Individuals experiencing paranoid (positive) symptoms have been reported to exhibit impaired judgement and reasoning (Iqbal et al., 2000), thought to contribute to emotion perception problems (Combs & Gouvier, 2004). Interestingly, prior research has shown that in the absence of pronounced acute symptoms, performance in social judgements in schizophrenia improves (Hall et al., 2004). However, even when positive symptoms are reduced, a marked impairment in the ability to derive accurate social information remains (Shayegan & Stahl, 2005), as do associated deficits in visual scan-path performance (Streit et al., 1997). Impaired emotional functioning is a prominent feature of psychosis, with negative symptoms, including flat affect, contributing to debilitation and treatment resistance (Gur et al., 2006). Severity of symptoms, particularly negative symptoms, have been found to correlate with facial emotion processing, specifically emotion recognition deficits; for example, increased recognition accuracy for happy faces is associated with less severe negative symptoms (Heimberg et al., 1992; Nieman et al., 2005; Sachs et al., 2004; Turetsky et al., 2007).

Flattened affect and other negative symptoms also have the potential to impact on emotion regulation, which has been defined as the ability to change or regulate (i.e., up or down) your emotions (Marra, 2004). More than simply the modulation of emotional arousal, emotion regulation involves an awareness, understanding and acceptance of emotions and an ability to act in desired ways regardless of emotional

state (Gratz & Roemer, 2004). Emotion regulation is typically conceptualised as distinct from emotion generation, involving a separate set of processes that either stop an emotion from launching or prevent it from being expressed once triggered, via cortical modulation of subcortical circuits (Gross & Barrett, 2011). However, some perspectives do not regard them as distinct constructs, with the boundaries between emotion generation and regulation blurred (Gross & Barrett, 2011). The impact of emotion regulation on facial emotion processing has not been extensively explored in the psychosis literature. However, given the importance of others' facial expressions in helping to regulate reactions to them (Schwartz, Mastropalo, Rosse, Mathis, & Deutsch, 2006), emotion regulation is likely to have important consequences for facial affect recognition and emotion processing.

The term '*alexithymia*' is often used clinically to describe abnormalities of affect regulation, such as difficulties in recognising, identifying and describing one's own emotions (Bagby & Taylor, 1997) and may be present in psychosis as well as a range of psychiatric and neurological disorders (van der Velde et al., 2013). Individuals with alexithymia may also show specific inability to communicate emotions despite the experience of emotion being intact (Kilstrom, Mulvaney, Tobia, & Tobis, 2000; Wout, Aleman, Kessels, Laroi, & Kahn, 2004). Neuropsychological studies have shown that emotion processing by individuals with alexithymia is associated with corresponding brain alterations in the amygdala, insula and cingulate cortex, involving reduced activation in the emotional attention system in cognitive emotional processing areas (van der Velde et al., 2013).

### *3.2.1 Experience of emotion*

Impaired emotion processing in schizophrenia is not limited to emotion recognition but relates to the experience and expression of emotion as well (Kohler & Martin, 2006). Patients with schizophrenia have been found to be less accurate in imitating and producing facial expressions than healthy controls (Schwartz et al., 2006). While in terms of expressivity, an apparent negative facial affectivity bias has been reported in schizophrenia, where facial expressions during clinical interviews were dominated by expressions of disgust and contempt; a bias which remaining relatively stable over time (Fatouros-Bergman et al., 2012). However, while reduced expressiveness and perception are observed, ratings of emotional experience appear to be normal (Gur et al., 2006; Kring et al., 1993). A meta-analysis by (Cohen & Minor, 2010), which included 26-emotion induction studies among individuals with schizophrenia and healthy controls, suggests that the ability to experience hedonic emotion in schizophrenia is preserved, finding no differences in subjective hedonic reactions to stimuli. Moreover, patients with schizophrenia also exhibited strong aversive emotions, when processing laboratory stimuli considered pleasant/neutral by others (Cohen & Minor, 2010). Consequently, a disconnect between the subjective experience and expressive display of emotion is indicated (Lindner et al., 2016).

Studies among patients with flattened affect, have also reported that moderate to severe affect flattening uniquely predicts emotion processing task performance, revealing greater impairments in both emotion recognition and emotion intensity (Gur et al., 2006). In evoking facial expressions, affective flattening and inappropriate affect is also evident in schizophrenia (i.e., displaying neutral or non-target expressions) (Kohler et al., 2008). One study, examining visual scanning behaviour in schizophrenia during

facial affect recognition, has reported patients with flattened affect showed selective scan-path characteristics, involving looking less frequently at the eyes, and spending longer looking at the regions in between the eyes and the centre of the face (Streit et al., 1997). Visual scanning associated with negative symptoms has previously been described as ‘staring’ or ‘minimal scanning’, or in this study as narrow and restricted scanning associated with the presence of flattened affect (Streit et al., 1997). However, affective flattening was not found to correlate with affect recognition performance (Streit et al., 1997).

Evidence from early structural and functional imaging studies, has revealed neurocognitive abnormalities, such as volume reduction (Aleman & Kahn, 2005) and attenuated response in the amygdala, during sad mood induction among individuals with schizophrenia (Schneider et al., 1998). Errors in fear detection have also been associated with higher activation in the amygdala and abnormal superior temporal connectivity, with activation correlating with severity of flattened affect in schizophrenia (Leitman et al., 2008). A recent neuroimaging study indicates that there is an association between affective flattening in schizophrenia and amygdala responsivity in processing threat related facial expressions (Lindner et al., 2016). Amygdala hyper-responsivity to unmasked fearful faces is suggested as a potential functional characteristic of schizophrenia, while amygdala hyper-responsivity to masked fearful faces appears to be a specific characteristic of patients with affective flattening (Lindner et al., 2016).

In addition, with respect to comorbidity, there is evidence of relatively high rates of personality disorder in schizophrenia, and these personality features can impact on

both clinical and cognitive characteristics (Moore et al., 2012), as well as emotion perception and experience. Personality traits are enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personal contexts (Lynam & Gudonis, 2005).

### **3.3 Symptoms, emotion regulation and emotion experience in psychopathy**

Psychopathy, described as a form of personality disorder, represents an enduring pattern of inner experience and behaviour that deviates markedly from cultural and societal expectations (Lynam & Gudonis, 2005). Psychopathic traits, including callous-unemotional and impulsive antisocial components, are associated with two core impairments: reduced empathic responding to others distress; and deficits in decision making and in reinforcement learning (Blair, 2013). These core impairments are likely to have an impact on emotion regulation and the experience of emotion. Alexithymia has also commonly been described among offenders, and found to correlate positively with psychopathy (Gori et al., 2014), suggesting a difficulty with not only recognising but also feeling emotion.

#### *3.3.1. Symptoms and emotion regulation*

Psychopathy traits are detectible early in childhood, persist into adulthood, and interfere with socialisation (Lynam & Gudonis, 2005). *Behaviourally* - the psychopath has been described as an impulsive risk-taker involved in a variety of criminal activities; *interpersonally* - as grandiose, egocentric, manipulative, forceful and cold-hearted; and *affectively* - as displaying shallow emotions, being unable to maintain close relationships, and lacking empathy, anxiety and remorse (Lynam & Gudonis, 2005). From a theoretical standpoint, the affective factor is of particular interest because it uniquely identifies

psychopathy from other forms of antisocial behaviour, and appears to have a strong genetic basis (Dawel et al., 2012). The emotional or affective dimension gives rise to insensitive/shalldownness coupled with low anxiety, while from a biological perspective, hypo-reactivity to aversive stimuli is apparent, as evidenced by subcortical activation (Moreira et al., 2014). Considering cognitive dimensions, deficits also exist in alternative thinking and locus of control, as well as cognitive distortions (i.e. denial, minimising) (Moreira et al., 2014). The behavioural dimension of aggressiveness, impulsivity and manipulation is associated with elevated high-risk behaviours, including alcohol and drug misuse, risky sexual behaviours, and offending (Kosson, Lorenz, & Newman, 2006; Walsh et al., 2007).

As a result of these characteristics, psychopaths may have difficulty regulating emotions (Blair et al., 2004). Emotional expressions also have a communicatory function, with impairments in processing displays of emotion impacting on the rapid communication of valence information between individuals, which is required for decision making and behaviour modifications appropriate to the social environment (Blair, 2003). According to the *violence inhibition mechanism* proposed by Blair (1995), displays of distress do not inhibit aggressive behaviour in psychopathy, as they normally would in healthy individuals, which is thought to be related to emotion processing deficits. Evidence suggested as support for this theory includes reduced physiological (Benning, Patrick, & Iacono, 2005) and cortical activation (i.e., amygdala and limbic structures) (Kiehl et al., 2001), as well as reduced behavioural responses to negative emotions such as fear and sadness (Blair, Colledge, & Mitchell, 2001b). Findings from meta-analyses of emotion recognition also support, emotion specific deficits (i.e. fear and sadness) (Blair et al., 2004); however, these are mixed, with others reporting a more

generalised impairment across emotions (Dawel et al., 2012; Wilson et al., 2011). Some studies have found no deficit (Glass & Newman, 2006), with others finding emotion decoding improved with increased emotional detachment (Habel et al., 2002).

Research undertaken among violent offenders with psychopathy provides some additional neurological insights (Gregory et al., 2012). Reactive and instrumental aggression among this subgroup has previously been associated with dysfunction within the vmPFC, known to regulate emotional reactivity to perceived threats and modulate behaviour accordingly, as well as abnormalities within the amygdala (Blair & Cipolotti, 2000). Deficits in aversive conditions, reinforcement learning, and recognition of fearful facial expressions in psychopathy have also been associated with dysfunction in both regions (Birbaumer et al., 2005; Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Herpertz et al., 2001; Patrick, 1994). More recently, gray matter volume reductions have been found in areas implicated in empathic processing, moral reasoning and pro-social emotions [i.e., anterior rostral prefrontal cortex (Brodmann area 10); and temporal poles (Brodmann area 20/38)], suggested as potentially contributing to the social behaviour abnormalities observed specifically among violent offenders with psychopathy (Gregory et al., 2012).

### 3.3.2 *Experience of emotion*

Psychopaths are typically callous, shallow and superficial, and do not experience insight into or empathy for those their behaviour affects (Blair et al., 2004; Hart et al., 2003). The lack of emotional insight or empathy has led to investigations around how emotion is experienced. Empathy is a complex construct involving affective, motivational and cognitive components, defined as a social-emotional response that is

induced by the perception of another person's affective state (Decety et al., 2014). It is regarded as a fundamental component of the emotional experience, playing a vital role in social interactions (Decety et al., 2014). Increased empathy has also previously been shown to be associated with improved emotion recognition (Wilson et al., 2011).

A study by Dolan and Fullam (2004), among individuals with antisocial personality disorder and psychopathy, found subtle deficits in mentalising ability in addition to emotion recognition deficits, although theory of mind was relatively intact. For example, concerning empathy, a lack of concern for potential impacts on victims was identified rather than an inability to take a victims perspective *per se*, which may have an adaptive role in criminal behaviour. Another study by Bertone et al. (2017) assessed empathic cognition and empathic emotion (using a Hinting Task, Reading the Mind in the Eyes Test and Cambridge Mind Reading Test), comparing social cognition among male offenders with antisocial personality disorder or psychosis. They found that patients with psychosis exhibited deficits in both social reasoning and emotion recognition. On the other hand, those with antisocial personality disorder only showed reduced emotion recognition performance; however, psychopathy traits were not assessed (Bertone et al., 2017).

A recent meta-analysis, assessing the conscious experience of emotions in psychopathy, reported moderate effect sizes for a reduction in the experience of happiness (0.19), while the experience of anger was increased (-0.15); contrary to expectations, there were no significant findings for fear (Hoppenbrouwers et al., 2016a). The authors also noted previous inconsistencies in meta-analysis findings for emotion recognition (Dawel et al., 2012; Marsh & Blair, 2008; Wilson et al., 2011); historical

conceptualisations of psychopathy, involving fearlessness as one factor behind callous and antisocial behaviour; coupled with neurological and physiological evidence for fear deficits; as well as differing methods and measurements, which may have led to the term fear being used too generically in the literature (Hoppenbrouwers et al., 2016a). In addition, definitional differences were evident across studies among both forensic and community samples, involving categorical versus dimensional approaches to measuring psychopathy traits along a continuum. Hoppenbrouwers et al. (2016a) took the approach of collapsing the existing evidence into separate subcomponents, as well as limiting the inclusion of studies to adult samples, and including emotion recognition as well measures of valence. In relation to fear, their findings indicated that individuals with psychopathy do exhibit deficits in threat detection and responsivity, but evidence for the reduced subjective experience of fear was less clear (Hoppenbrouwers et al., 2016a).

Studies targeting the conscious subjective experience of emotions have largely relied on self-report measures, and have predominately focused on fear. Essentially, it has yet to be clearly established whether individuals with psychopathy have problems in the subjective or conscious experience of emotions (i.e. valence, recognition), such as fear, or whether fearlessness for example is mainly attributed to disturbed autonomic bodily responsivity to threat (i.e. skin conductance, heart rate) (Hoppenbrouwers et al., 2016a).

While the amygdala has been implicated in autonomic responses to threatening stimuli, another meta-analysis focusing on the neurological findings found little evidence of amygdala activation during the conscious experience of fear (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012). Furthermore, for all emotion categories,

there is little evidence that discrete emotions can be consistently and specifically localized to distinct brain regions (Lindquist et al., 2012). Instead, the neurological evidence supports a psychological constructionist approach, involving a set of interacting brain regions in basic psychological operations of both an emotional and non-emotional nature, which are active during emotion experience and perception (Lindquist et al., 2012).

Whilst outside of the scope of the immediate study, a number of studies have attempted to address the neurobiological underpinnings. Fear processing in psychopathy has received the most attention, with the key components involved in both autonomic threat processing, and the conscious experience examined (Hoppenbrouwers et al., 2016a). A large body of work has addressed the involvement of the amygdala, but other brain regions including the hippocampus, thalamus and prefrontal cortex (vmPFC) are thought to be involved, with functional and structural impairments posited in an impaired threat processing network in psychopathy (see review: Hoppenbrouwers et al., 2016a). The insula and (dorsal) anterior cingulate cortex are also important regions in the subjective experience of emotion, within an “*amygdala centred threat network*” model (Bechara & Naqvi, 2004; Hoppenbrouwers et al., 2016a). It is believed that environmental information processed by the sensory systems (i.e., thalamus) relays input to areas involved in threat detection (i.e. amygdala), initiating autonomic threat responses (i.e., heart rate, skin conductivity, pupil dilation), with feedback projected to the insula and anterior cingulate cortex, leading to visceral awareness (Bechara & Naqvi, 2004; Hoppenbrouwers et al., 2016a).

Of particular relevance here, are the few studies that have explored the conscious or subjective experience of emotion in psychopathy during emotion processing tasks. However, a number of studies have previously explored psychophysiological responses, confirming reduced autonomic responsivity to emotional material in psychopathy (Kirsch & Becker, 2007; Patrick, Cuthbert, & Lang, 1994). A study by Eisenbarth et al. (2008) of psychopathic women included three emotion processing tasks, one of which required participants to rate valence (i.e. how positive or negative the picture was), as well as arousal (i.e. how arousing the picture was). Psychopathic individuals were found to respond less strongly to all emotional stimuli, making less positive ratings for happy expressions and rated less arousal for angry expressions compared to healthy controls, indicating subjective differences exist not only those previously observed at a physiological level (Eisenbarth et al., 2008).

Another study, examining the experience and suppression of emotion among violent offenders, found that individuals with higher psychopathy scores were more cardiovascular responsive (i.e. increased heart rate) when processing negative images of people in a variety of scenes, suggesting a possible rewarding aspect of processing otherwise normally unpleasant material (Casey, Rogers, Burns, & Yiend, 2013). When requested to experience emotion, by '*getting into the feeling*' of the emotion conveyed, this was combined with an absence of subjective self-report differences. On the other hand, individuals with higher affective psychopathic traits (PCL: Factor 1) showed reduced responsiveness to negative images. Consequently, the authors suggested that these physiological differences, in the absence of subjective self-report differences in experience, may have important clinical implications related to normalising emotion

processing among psychopathic offenders, and potentially for improving treatment outcomes (Casey et al., 2013).

### **3.4 Aims and hypotheses**

This second study sought to extend previous research on emotion processing deficits in psychosis by examining emotion induction, or the ability to invoke an emotional response using facial expressions of emotion. In addition, differential processing patterns based on the type of emotion processing task were able to be examined by comparing the visual scanning strategies utilised here (i.e., when asked to “feel” or invoke emotions) with those used when requested to “recognise” or categorise emotions (as examined in the previous Chapter 2 - Task A). The psychosis literature provides evidence of impairments in both emotion recognition and intensity (Kohler et al., 2010; Kurtz & Richardson, 2012), although the actual experience of emotion may be intact (Gur et al., 2006). While the psychopathy literature also indicates deficits in emotion recognition and intensity (Dawel et al., 2012; Wilson et al., 2011), findings around the conscious experience of emotion appears to be less clear, with some evidence of reduced experience for specific emotions (Hoppenbrouwers et al., 2016a).

The primary hypotheses for this study are: 1) Compared to a healthy control group, individuals with psychosis will exhibit reduced emotion responsivity in terms of their ability to feel or evoke emotion, as evidenced by lower mood variation (using an abbreviated version of the profile of mood states (POMS)) and a more restricted pattern of visual scanning; and 2) Psychosis participants with a higher number of psychopathic traits will perform more poorly than those with low psychopathy traits during an emotion induction task, exhibiting lower mood responsivity as well as more restricted

visual scanning. Secondary analyses will also consider the impact of difficulties in emotion regulation on task performance.

### **3.5 Method**

#### *3.5.1 Participants*

All experimental studies in this dissertation were undertaken consecutively during the same structured interview. Participant details for the entire study sample of 61 participants were provided in Chapter 2: Section 2.5.1. In brief, there were 24 healthy control participants and 37 community mental health outpatients with a psychotic disorder, with the latter divided into two groups comprising 18 psychosis participants with low psychopathy traits and 19 with high psychopathy traits based on their PCL: SV total scores.

#### *3.5.2 Measures*

All measures utilised in the study have already been described elsewhere (see Chapter 2: Section 2.5.2 for full details).

#### *3.5.3 Facial expression stimuli*

The stimuli utilised during the visual-cognitive eye-tracking Task B examining emotion induction, consisted of 40 colour images, portraying facial expressions, with equal numbers of male and female actors depicting one of four emotions. To allow for some variation in expressiveness, images were selected for moderate to high interrater agreement for categorisation of each emotion (mean overall validation score = 0.81, SD = 0.20) from the same standardised set of facial expressions detailed in chapter 2, Section 2.5.3 for the emotion recognition Task A (NimStim: Tottenham et al., 2002;

Tottenham et al., 2009; <http://www.macbrain.org/resources.htm>). However, none of the images presented during Task A were utilised in Task B. All four categories of stimuli selected had validity data that indicated a moderate-high mean proportion correct for the chosen facial expressions, which included happy (Mean = 0.96, SD = 0.034), sad (Mean = 0.84, SD = 0.16), anger (Mean = 0.86, SD = 0.16), and fear (Mean = 0.57, SD = 0.15). All images had a resolution of 506 × 650 pixels, displayed within equivalent parameters on a computer screen, in order to maintain a similar set location of facial features, such as eyes and mouth across all stimuli presentations.

#### *3.5.4 Eye tracking apparatus*

Full details on the View Point eye tracker and visual scanning technology (Eye-link: 1000; SR Research Ltd, Ontario, Canada) utilised to record eye movements have been provided in Chapter 2, Section 2.5.4.

#### *3.5.5 Procedures*

All three experimental studies were undertaken consecutively, with most procedural details already documented in Chapter 2: Section 2.5.5. However, details specific to the second study (Task B) are outlined here, which involved a visual-cognitive eye movement-recording task exploring emotion induction. All participants took part in Task B, during which 40 facial expression stimuli (in total) were presented in four blocks, each consisting of a series of 10 images (5 male and 5 female) depicting similar emotions presented in a fixed random order. These images depicted one of four different categories of facial affect: happy, sad, anger, and fear. The task utilised a 4-block randomisation so that any possible effects due to the order of emotions displayed could be accounted for during the outcome analyses. Several constraints influenced the

ordering of emotion blocks: firstly, each trial block began with a different emotion; secondly, the categories of happy (H) or sad (S) never followed each other; and thirdly, anger (A) and fear (F) never followed each other. The resulting order options were: 1) S A H F; 2) H A S F; 3) A S F H; or 4) F S A H. Each participant completed one of these trial sequences, and within each sequence, all facial emotion stimuli were presented in the same predetermined randomised order.

An abbreviated version of the Profile of Mood States (POMS) questionnaire (McNair et al., 2005) was presented on a computer screen (see [Appendix H](#)), in conjunction with the emotion induction task. Participants completed POMS ratings in order to provide a baseline measure of mood state prior to the commencement of the emotion induction task, as well as a measure of variation in mood state after exposure to each block of facial expression stimuli. The POMS identifies and assesses mood state across 6 dimensions including; Tension; Depression; Anger; Vigour; Fatigue; and Confusion; and it also provides a total mood disturbance score (McNair et al., 2005). Importantly, ratings are provided in relation to how a person is feeling “*Right Now*” on a 5-point Likert scale for each item (ranging from 0 = not at all, to 4 = extremely). The abbreviated version used to monitor variation in mood state consisted of 12-items selected from the 65-item version (McNair et al., 2005), whereby two adjectives were chosen to most closely represent each of the 6 domains.

Task B utilised the same desk, computer, chin rest configuration, standard SR Eye Link calibration, validation and eye-movement scan-path recording procedures as those outlined earlier for the emotion recognition Task A. All participant instructions were displayed centrally on a computer screen. On commencement of Task B, the

following statements were displayed and read aloud: *“I’m going to show you some pictures of people expressing different emotions. Look carefully at each picture while it is presented and draw on your personal experiences to try to feel that emotion”*.

Immediately prior to the exposure of the first stimulus block, participants were asked to rate the intensity of their current mood on the POMS using the following instructions:

*“Below is a list of words that describe feelings that people have. Please read each word carefully. Then tell me the number that best describes how you feel RIGHT NOW”*.

Participants were given as much time as they required to provide a verbal response to each of the 12 items, which was manually recorded by the experimenter (see [Appendix J](#) for full task instructions).

At the commencement of each stimulus trial block, the first image was presented when a participant fixated on a centrally located dot (3 cm diameter) on the computer screen for more than 1000ms. The 10 facial expression stimuli within each series were displayed for 6 seconds each, taking 60 seconds in total, while continuous eye-movement recordings were obtained. Immediately following the visual scan-path recording a screen displaying the POMS questionnaire appeared and participants reported their current mood state, which was manually recorded for each of the 12 items by the experimenter. Subsequently, mean scores on the POMS were calculated for five subscales: Tension (T); Depression (D); Anger (A); Vigour (V); and Fatigue/Confusion combined; as well as Total Mood Disturbance at baseline; and Mood Variation (TDAV) based on changes in Tension, Depression, Anger and Vigour following each block of emotion stimuli. The entire task, including four stimulus blocks, one baseline, and four subsequent mood state measures, took approximately 10 minutes to complete.

Following the emotion induction Task B, and prior to the delayed recall component of the face recognition and working memory Task C, participants completed a number of self-report questionnaires. One of these measures was the comprehensive Difficulties in Emotion Regulation Scale (DERS), which provided a multidimensional assessment of emotion regulation and dysregulation. The DERS was scored using the original 6-factor solution reported by Gratz and Roemer (2004), which has also been used among clinical populations, including an Australian psychosis sample (Ayre, 2013). A total DERS score was calculated (by summing the individual items), together with scores for the six subscales: non-acceptance of emotional responses; difficulties in engaging in goal-directed behaviour; impulse control difficulties; lack of emotional awareness; limited access to emotion regulation strategies; and lack of emotion clarity. Relationships between emotion regulation on the DERS and variation in mood state on the POMS were also examined.

In addition, during the development of Task B, a pilot study among 11 healthy control participants was undertaken in order to appraise procedural aspects of the task. The pilot successfully demonstrated the feasibility of undertaking a novel emotion induction task, with changes in emotion responsivity apparent among healthy controls whilst concurrently recording visual-scan-path strategies (see [Appendix K](#) for details).

### *3.5.6 Eye movement parameters*

Full details on the scan-path parameters available for extraction have been provided in Chapter 2: Section 2.5.6. In brief, the scan-path parameters utilised for analyses in this study were the two temporal indices, which included the mean number of fixations (count) and the average duration of fixations (time/ms).

### 3.5.7 Data analysis

Data coding and analyses were undertaken using SPSS Statistical Software (Version 24.0; SPSS, Armonk, NY, USA). Multiple regression analyses, with psychopathy included as a continuous contrast, were utilised to examine group differences in emotion regulation on the Difficulties in Emotion Regulation Scale (DERS) and disturbances in mood state on the Profile of Mood States (POMS) at baseline. In order to examine changes in POMS ratings during the emotion induction Task B, separate multiple linear regression analyses were conducted for each scale, with mood change scores as the dependent variable (post-presentation minus pre-presentation), two predictors (orthogonal contrasts) assessing group differences, and three covariates (dummy variables) accounting for stimulus order effects. A mood variation score was also calculated for each participant as their average squared change score across the Tension (T), Depression (D), Anger (A) and Vigour (V) scales.

In relation to the psychometric properties of the abbreviated POMS scale, internal consistency was demonstrated based on the 302 sets of ratings obtained during Task B (N = 61 participants by 5 repeated POMS applications). A Cronbach's alpha of 0.779 was obtained for the total mood disturbance score (12 items), with reasonably comparable Cronbach's alphas for the four two-item subscales (Tension, 0.630; Depression, 0.661; Anger, 0.862; and Vigour, 0.747) and the four-item Fatigue/Confusion subscale (0.801).

Analyses of mood variation and scan-path indices by emotion and group were undertaken using a Generalised Linear Model with a series of planned orthogonal contrasts. As detailed in [Table 3.1](#), eleven planned orthogonal contrasts were defined

comprising two between-group contrasts (GC1 and GC2), three within-group emotion contrasts (EC1 to EC3), and six interaction or product contrasts (GC1 x EC1 to EC3, and GC2 x EC1 to EC3). Three dummy variables (covariates) were also included to account for stimulus order effects. Contrast coefficients were standardised (i.e., weighted mean of zero, and standard deviation of 1.000). Pearson product-moment correlations (using two-tailed significance tests) were used to examine relationships between total mood disturbance prior to Task B, mood variation and scan-path indices from Task B, and selected Task A emotion recognition accuracy and scan-path indices. As a partial control for the number of statistical tests conducted, the threshold for statistical significance was set at  $p < 0.01$  for all analyses, although statistical trends ( $p < 0.05$ ) are also noted.

Table 3.1: *Planned contrasts (Task B) – Standardised coefficients*

<i>Group Contrasts (Between-groups)</i>	Healthy Control (N = 24)		Psychosis Groups	
			Low Psychopathy (N = 17)	High Psychopathy (N = 19)
GC1: Psychosis Vs. Not	1.222		-0.815	-0.815
GC2: Low Vs. High Psychopathy (Continuous Contrast)	0.000		-0.138 to -1.98	0.112 to 2.181
<i>Emotion Contrasts (Within-groups)</i>				
	Happy (H)	Sad (S)	Anger (A)	Fear (F)
EC1: Negative Vs. Positive	1.72	-0.576	-0.576	-0.576
EC2: Other Negative Vs. Sad	0.000	1.630	-0.815	-0.815
EC3: Fear Vs. Anger	0.000	0.000	1.411	-1.411
<i>Interaction Contrasts</i>				
GC1 x EC1 to EC3 (Three Contrasts)				
GC2 x EC1 to EC3 (Three Contrasts)				
<i>Dummy Variables (Covariates)</i>				
	SAHF (N = 15)	HASF (N = 17)	ASFH (N = 14)	FSAH (N = 14)
Dummy Variable 1	1.728	-0.576	-0.576	-0.576
Dummy Variable 2	-0.627	1.587	-0.627	-0.627
Dummy Variable 3	-0.551	-0.551	1.809	-0.551

*Note. Eleven planned orthogonal contrasts were defined, comprising two between-group contrasts (GC1 and GC2), three within-group emotion contrasts (EC1 to EC3) and six interaction or product contrasts (GC1 x EC1 to EC3, and GC2 x EC1 to EC3). Three dummy variables (covariates) were also included to account for stimulus order effects. Contrast coefficients have been standardised (i.e., weighted mean of zero, and standard deviation of 1.000).*

## 3.6 Results

### 3.6.1 Emotion regulation difficulties and profile of mood state

As detailed in [Table 3.2](#), multiple regression analyses examining self-reported difficulties in emotion regulation (DERS) revealed the psychosis group were experiencing increased difficulty on all six domains, with significantly higher mean scores on four domains and the total score. These included non-acceptance of emotional responses ( $t_{(58)} = -2.90, p < 0.01$ ); difficulties engaging in goal-directed behaviour ( $t_{(58)} = -3.61, p < 0.001$ ); impulse control difficulties ( $t_{(58)} = -3.82, p < 0.001$ ); limited access to emotion regulation strategies ( $t_{(58)} = -5.24, p < 0.001$ ); and the total score ( $t_{(58)} = -4.84, p < 0.001$ ). A trend level difference was also detected for lack of emotional clarity ( $t_{(58)} = -2.19, p = 0.032$ ). The largest group differences between healthy controls and the psychosis groups were for limited access to emotion regulation strategies (Means = 10.21 vs. 17.22), and the total score (Means = 59.33 vs. 81.70). For low versus high psychopathy traits only one significant difference was apparent ( $t_{(58)} = -3.11, p < 0.010$ ), with the low psychopathy group reporting increased difficulty on the non-acceptance of emotional responses domain compared with the high psychopathy group (Means = 14.67 vs. 10.53). Correlations between all six sub-domains and the total DERS score were moderate to high (ranging from  $r = 0.46$  to  $0.87, p < 0.001$ ).

Prior to undertaking the visual-cognitive emotion induction Task B, participants were also asked to rate their mood “right now” on the Profile of Mood States (POMS), to provide a baseline measure of any mood disturbance. The psychosis group reported a significantly higher level of mood disturbance on the Tension sub-domain ( $t_{(58)} = -2.92, p < 0.01$ ) as well as Total Mood Disturbance ( $t_{(58)} = -3.06, p < 0.01$ ), and tended to have increased levels for Depression ( $t_{(58)} = -2.24, p = 0.029$ ) at baseline (see Table 3.2). No

Table 3.2: *Emotion measures: difficulties in emotion regulation and pre Task B profile of mood state.*

<i>Measures</i>	Healthy Control N=24 Mean (SD)	Psychosis Groups		R <sup>2</sup>	Comparisons	
		Low Psychopathy N=18 Mean (SD)	High Psychopathy N= 19 Mean (SD)		Psychosis Vs. Not	Low Vs. High Psychopathy <sup>a</sup>
<i>Difficulties in Emotion Regulation Scale (DERS)</i>						
<i>Non acceptance of emotional responses (NONACCEPTANCE)</i>	9.63 (2.63)	14.67 (5.22)	10.53 (3.44)	0.24	t <sub>(58)</sub> =-2.90,p<0.01*	t <sub>(58)</sub> =-3.11,p<0.01*
<i>Difficulties engaging in goal-directed behaviour (GOALS)</i>	9.92 (3.83)	16.17 (4.08)	12.16 (4.98)	0.23	t <sub>(58)</sub> =-3.61,p<0.001**	t <sub>(58)</sub> =-1.96,p=0.055
<i>Impulse control difficulties (IMPULSE)</i>	7.92 (2.19)	12.50 (4.60)	11.16 (4.73)	0.21	t <sub>(58)</sub> =-3.82,p<0.001**	t <sub>(58)</sub> =-0.88,p=0.383
<i>Lack of emotional awareness (AWARENESS)</i>	13.96 (5.19)	14.94 (5.30)	18.21 (5.46)	0.10	t <sub>(58)</sub> =-1.90,p=0.062	t <sub>(58)</sub> =1.58,p=0.119
<i>Limited Access to emotion regulation strategies (STRATEGIES)</i>	10.21 (2.36)	18.00 (6.34)	16.47 (6.25)	0.33	t <sub>(58)</sub> =-5.24,p<0.001**	t <sub>(58)</sub> =-1.02,p=0.310
<i>Lack of emotional clarity (CLARITY)</i>	7.71 (3.14)	9.44 (2.45)	9.37 (3.00)	0.08	t <sub>(58)</sub> =-2.19,p=0.032 <sup>#</sup>	t <sub>(58)</sub> =-0.23,p=0.822
<i>Total Score</i>	59.33 (13.90)	85.72 (16.97)	77.89 (21.88)	0.30	t <sub>(58)</sub> =-4.84,p<0.001**	t <sub>(58)</sub> =-1.22,p=0.229
<i>Profile of Mood States (POMS)</i>						
<i>Tension</i>	0.27 (0.42)	0.56 (0.70)	0.87 (0.62)	0.15	t <sub>(58)</sub> =-2.92,p<0.01*	t <sub>(58)</sub> =1.38,p=0.172
<i>Depression</i>	0.00 (0.00)	0.11 (0.37)	0.37 (0.64)	0.11	t <sub>(58)</sub> =-2.24,p=0.029 <sup>#</sup>	t <sub>(58)</sub> =1.41,p=0.163
<i>Anger</i>	0.00 (0.00)	0.03 (0.12)	0.26 (0.71)	0.03	t <sub>(58)</sub> =-1.37,p=0.175	t <sub>(58)</sub> =0.32,p=0.752
<i>Vigour</i>	1.85 (0.97)	1.75 (0.79)	1.42 (0.90)	0.04	t <sub>(58)</sub> =1.16,p=0.253	t <sub>(58)</sub> =-1.02,p=0.310
<i>Fatigue and Confusion</i>	0.43 (0.44)	0.51 (0.58)	0.88 (0.73)	0.08	t <sub>(58)</sub> =-1.78,p=0.081	t <sub>(58)</sub> =1.33,p=0.190
<i>Total Mood Disturbance Score</i>	0.55 (0.27)	0.66 (0.32)	0.97 (0.41)	0.18	t <sub>(58)</sub> =-3.06, p<0.01*	t <sub>(58)</sub> =1.94,p=0.058

Note. Low Psychopathy < 11 on PCL: SV; <sup>a</sup> tested as a continuous contrast within a multiple regression analysis; <sup>#</sup> trend (p < 0.05); \* p < 0.01; \*\* p < 0.001

significant POMS differences for low versus high psychopathy traits were apparent at baseline.

### *3.6.2 Emotion induction performance –Task B*

#### *3.6.2.1 Mood variation based on POMS change scores*

As detailed in [Table 3.3](#), multiple regression analyses revealed marked overall induction effects for Task B following the presentation of happy facial affect stimuli. There were significant reductions (based on post-presentation minus pre-presentation change scores) on the POMS Tension ( $t_{(54)} = -3.71$ ,  $p < 0.001$ ), Anger ( $t_{(54)} = -2.90$ ,  $p = 0.005$ ), and Fatigue/Confusion ( $t_{(54)} = -3.20$ ,  $p = 0.002$ ) domains, as well as significant overall Mood Variation (i.e., deviation from zero) based on the TDAV change scores ( $t_{(54)} = 2.96$ ,  $p = 0.005$ ). A trend for increased scores on Vigour ( $t_{(54)} = 2.44$ ,  $p = 0.018$ ) was also detected, although the capacity for change (i.e., an increase) when presented with happy stimuli was probably reduced, given the relatively high mean levels of Vigour at baseline (see Table 3.2). However, there were no significant group differences in these analyses and the net induction effects obtained were relatively small, in the order of change on 1 out of 8 items on the POMS scale (overall Mean Mood Variation TDAV change = 0.36).

Similarly, as detailed in [Table 3.4](#), there was significant overall Mood Variation following the presentation of sad facial expressions ( $t_{(54)} = 4.26$ ,  $p < 0.001$ ), with trend level changes on the Depression ( $t_{(54)} = 2.27$ ,  $p = 0.028$ ) and Vigour ( $t_{(54)} = -2.62$ ,  $p = 0.011$ ) subscales, regardless of group. A reciprocal relationship between Depression and Vigour was apparent, whereby as Depression (sadness) increased, Vigour (happiness) reduced (overall Mean change: Depression = 0.10 and Vigour = -0.18). While there

Table 3.3: Change in POMS ratings following presentation of Happy stimuli (Task B) by group

<i>POMS scale</i>	Overall N= 60 Mean (SD)	Healthy Control N= 24 Mean (SD)	Psychosis Groups		R <sup>2</sup>	Overall Induction effect	Comparisons	
			Low Psychopathy N= 17 Mean (SD)	High Psychopathy N= 19 Mean (SD)			Psychosis Vs. Not	Low Vs. High Psychopathy <sup>a</sup>
Tension (T)	-0.23 (0.50)	-0.13 (0.27)	-0.23 (0.44)	-0.37 (0.72)	0.117	t <sub>(54)</sub> =-3.71, p<0.001**	t <sub>(54)</sub> =1.27, p=0.210	t <sub>(54)</sub> =0.13, p=0.897
Depression (D)	-0.08 (0.50)	-0.04 (0.14)	-0.03 (0.54)	-0.18 (0.71)	0.027	t <sub>(54)</sub> =-1.24, p=0.222	t <sub>(54)</sub> =0.52, p=0.605	t <sub>(54)</sub> =-0.74, p=0.462
Anger (A)	-0.15 (0.38)	-0.08 (0.28)	-0.09 (0.40)	-0.26 (0.45)	0.060	t <sub>(54)</sub> =-2.90, p=0.005*	t <sub>(54)</sub> =1.00, p=0.324	t <sub>(54)</sub> =-0.10, p=0.992
Vigour (V)	0.25 (0.83)	0.29 (0.66)	0.18 (0.88)	0.26 (1.01)	0.133	t <sub>(54)</sub> =2.44, p=0.018#	t <sub>(54)</sub> =0.18, p=0.858	t <sub>(54)</sub> =0.01, p=0.995
Fatigue/Confusion	-0.18 (0.43)	-0.08 (0.18)	-0.12 (0.29)	-0.36 (0.67)	0.086	t <sub>(54)</sub> =-3.20, p=0.002*	t <sub>(54)</sub> =1.32, p=0.193	t <sub>(54)</sub> =-1.00, p=0.322
Mood variation based on TDAV changes <sup>b</sup>	0.36 (0.95)	0.17 (0.37)	0.36 (1.25)	0.61 (1.13)	0.059	[t <sub>(54)</sub> =2.96, p=0.005*]	t <sub>(54)</sub> =-1.26, p=0.212	t <sub>(54)</sub> =0.33, p=0.743

Note. Low Psychopathy < 11 on PCL: SV; separate multiple (linear) regression analyses were conducted for each Profile of Mood States (POMS) scale, with mood change scores as the dependent variable (Post-presentation minus pre-presentation), two predictors (orthogonal contrasts) assessing group differences, and three covariates (dummy variables) accounting for stimulus order effects; <sup>a</sup> tested as a continuous contrast; <sup>b</sup> a mood variation score was calculated for each participant as their average squared change score across the T, D, A and V scales; # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 3.4: Change in POMS ratings following presentation of Sad stimuli (Task B) by group

POMS scale	Overall N= 60 Mean (SD)	Healthy Control N= 24 Mean (SD)	Psychosis Groups		R <sup>2</sup>	Overall Induction effect	Comparisons	
			Low Psychopathy N= 17 Mean (SD)	High Psychopathy N= 19 Mean (SD)			Psychosis Vs. Not	Low Vs. High Psychopathy <sup>a</sup>
Tension (T)	-0.09 (0.56)	-0.19 (0.36)	0.03 (0.60)	-0.08 (0.73)	0.109	t <sub>(54)</sub> =-1.18, p=0.243	t <sub>(54)</sub> =-1.12, p=0.270	t <sub>(54)</sub> =-0.85, p=0.397
Depression (D)	0.10 (0.33)	0.10 (0.21)	0.00 (0.31)	0.18 (0.45)	0.041	t <sub>(54)</sub> =2.27, p=0.028#	t <sub>(54)</sub> =0.10, p=0.919	t <sub>(54)</sub> =1.19, p=0.239
Anger (A)	-0.02 (0.38)	-0.13 (0.27)	0.03 (0.45)	0.08 (0.42)	0.153	t <sub>(54)</sub> =-0.35, p=0.732	t <sub>(54)</sub> =-1.99, p=0.051	t <sub>(54)</sub> =1.02, p=0.314
Vigour (V)	-0.18 (0.54)	-0.21 (0.67)	-0.18 (0.43)	-0.16 (0.47)	0.063	t <sub>(54)</sub> =-2.62, p=0.011#	t <sub>(54)</sub> =-0.29, p=0.771	t <sub>(54)</sub> =-0.06, p=0.955
Fatigue/Confusion	-0.01 (0.29)	0.00 (0.26)	0.01 (0.30)	-0.04 (0.33)	0.076	t <sub>(54)</sub> =-0.16, p=0.887	t <sub>(54)</sub> =0.13, p=0.896	t <sub>(54)</sub> =-1.07, p=0.290
Mood variation based on TDAV changes <sup>b</sup>	0.23 (0.40)	0.19 (0.48)	0.21 (0.30)	0.29 (0.38)	0.031	[t <sub>(54)</sub> =4.26, p<0.001**]	t <sub>(54)</sub> =-0.47, p=0.639	t <sub>(54)</sub> =0.44, p=0.661

Note. Low Psychopathy < 11 on PCL: SV; separate multiple (linear) regression analyses were conducted for each Profile of Mood States (POMS) scale, with mood change scores as the dependent variable (Post-presentation minus pre-presentation), two predictors (orthogonal contrasts) assessing group differences, and three covariates (dummy variables) accounting for stimulus order effects; <sup>a</sup> tested as a continuous contrast; <sup>b</sup> a mood variation score was calculated for each participant as their average squared change score across the T, D, A and V scales; # trend (p<0.05); \* p < 0.01; \*\* p < 0.001.

were no significant group differences for psychosis or low versus high psychopathy traits, when presented with sad stimuli the psychosis group tended to report increased Anger in comparison to a reduction on this domain among the healthy controls (Means = 0.06 vs -0.13;  $t_{(54)} = -1.99$ ,  $p = 0.051$ ).

Following the presentation of Angry facial expressions, as detailed in [Table 3.5](#), there were significant overall mood changes on the Anger ( $t_{(54)} = 3.01$ ,  $p = 0.004$ ) and Vigour ( $t_{(54)} = -3.30$ ,  $p = 0.002$ ) domains, as well as Mood Variation based on TDAV changes ( $t_{(54)} = 5.67$ ,  $p < 0.001$ ). While there were no significant group differences, there was a trend level association for low versus high psychopathy traits on the Vigour domain ( $t_{(54)} = -2.34$ ,  $p = 0.023$ ), with the low psychopathy group not reporting any change while the high psychopathy group reported reduced Vigour (Means = 0.00 vs. -0.37), as did the healthy controls (Mean = -0.27). Similarly, as detailed in [Table 3.6](#), following the presentation of facial expressions of Fear, significant overall Mood Variation was apparent ( $t_{(54)} = 2.93$ ,  $p = 0.005$ ), as well as trend level mood changes on the Vigour domain ( $t_{(54)} = -2.60$ ,  $p = 0.012$ ). However, there were no significant findings for Tension, or any group differences.

### 3.6.2.2 Mood variation by emotion and group

Generalised Linear Model analyses of Mood variation by emotion and group, involving eleven planned orthogonal contrasts (for group, emotion, and interaction effects), while controlling for stimulus order effects, revealed no significant differences. As evidenced by the low mean mood variation scores summarised in [Table 3.7](#) (grand mean = 0.27), the induction effects in Task B were relatively modest. Indeed, mood variation scores of zero were recorded for 31.7% of participants viewing the Happy and

Table 3.5: Change in POMS ratings following presentation of Anger stimuli (Task B) by group

POMS scale	Overall N= 60 Mean (SD)	Healthy Control N= 24 Mean (SD)	Psychosis Groups		R <sup>2</sup>	Overall Induction effect	Comparisons	
			Low Psychopathy N= 17 Mean (SD)	High Psychopathy N= 19 Mean (SD)			Psychosis Vs. Not	Low Vs. High Psychopathy <sup>a</sup>
Tension (T)	0.12 (0.47)	0.10 (0.36)	0.09 (0.51)	0.16 (0.58)	0.076	t <sub>(54)</sub> =1.88, p=0.065	t <sub>(54)</sub> =-0.13, p=0.896	t <sub>(54)</sub> =0.20, p=0.845
Depression (D)	0.00 (0.32)	0.00 (0.21)	0.00 (0.31)	0.00 (0.44)	0.020	t <sub>(54)</sub> =-0.01, p=0.990	t <sub>(54)</sub> =-0.02, p=0.982	t <sub>(54)</sub> =0.38, p=0.704
Anger (A)	0.17 (0.42)	0.19 (0.36)	0.15 (0.46)	0.16 (0.47)	0.044	t <sub>(54)</sub> =3.01, p=0.004*	t <sub>(54)</sub> =0.33, p=0.740	t <sub>(54)</sub> =-0.58, p=0.565
Vigour (V)	-0.23 (0.54)	-0.27 (0.51)	0.00 (0.56)	-0.37 (0.52)	0.176	t <sub>(54)</sub> =-3.30, p=0.002*	t <sub>(54)</sub> =-0.69, p=0.491	t <sub>(54)</sub> =-2.34, p=0.023#
Fatigue/Confusion	0.02 (0.31)	-0.03 (0.17)	0.01 (0.32)	0.09 (0.41)	0.031	t <sub>(54)</sub> =0.52, p=0.607	t <sub>(54)</sub> =-1.05, p=0.298	t <sub>(54)</sub> =0.17, p=0.869
Mood variation based on TDAV changes <sup>b</sup>	0.22 (0.30)	0.16 (0.20)	0.21 (0.29)	0.29 (0.40)	0.105	[t <sub>(54)</sub> =5.67, p<0.001**]	t <sub>(54)</sub> =-1.06, p=0.295	t <sub>(54)</sub> =0.84, p=0.406

Note. Low Psychopathy < 11 on PCL: SV; separate multiple (linear) regression analyses were conducted for each Profile of Mood States (POMS) scale, with mood change scores as the dependent variable (Post-presentation minus pre-presentation), two predictors (orthogonal contrasts) assessing group differences, and three covariates (dummy variables) accounting for stimulus order effects; <sup>a</sup> tested as a continuous contrast; <sup>b</sup> a mood variation score was calculated for each participant as their average squared change score across the T, D, A and V scales; # trend (p<0.05); \* p < 0.01; \*\* p < 0.001.

Table 3.6: Change in POMS ratings following presentation of Fear stimuli (Task B) by group

POMS scale	Overall N= 60 Mean (SD)	Healthy Control N= 24 Mean (SD)	Psychosis Groups		R <sup>2</sup>	Overall Induction effect	Comparisons	
			Low Psychopathy N= 17 Mean (SD)	High Psychopathy N= 19 Mean (SD)			Psychosis Vs. Not	Low Vs. High Psychopathy <sup>a</sup>
Tension (T)	0.08 (0.51)	0.02 (0.31)	0.06 (0.66)	0.18 (0.58)	0.071	t <sub>(54)</sub> =1.28, p=0.205	t <sub>(54)</sub> =-0.69, p=0.491	t <sub>(54)</sub> =0.45, p=0.654
Depression (D)	0.01 (0.42)	-0.04 (0.20)	0.18 (0.56)	-0.08 (0.45)	0.070	t <sub>(54)</sub> =0.24, p=0.813	t <sub>(54)</sub> =-0.76, p=0.453	t <sub>(54)</sub> =-1.65, p=0.105
Anger (A)	0.06 (0.46)	0.02 (0.10)	0.18 (0.66)	0.00 (0.53)	0.171	t <sub>(54)</sub> =1.16, p=0.251	t <sub>(54)</sub> =-0.53, p=0.601	t <sub>(54)</sub> =-1.86 p=0.068
Vigour (V)	-0.20 (0.67)	-0.21 (0.67)	-0.32 (0.86)	-0.08 (0.45)	0.193	t <sub>(54)</sub> =-2.60, p=0.012#	t <sub>(54)</sub> =0.00, p=0.997	t <sub>(54)</sub> =1.70, p=0.095
Fatigue/Confusion	0.06 (0.30)	0.04 (0.16)	0.13 (0.42)	0.03 (0.33)	0.135	t <sub>(54)</sub> =1.75, p=0.086	t <sub>(54)</sub> =-0.45, p=0.658	t <sub>(54)</sub> =-1.44, p=0.156
Mood variation based on TDAV changes <sup>b</sup>	0.28 (0.81)	0.16 (0.48)	0.50 (1.36)	0.25 (0.36)	0.138	[t <sub>(54)</sub> =2.93, p=0.005*]	t <sub>(54)</sub> =-1.03, p=0.308	t <sub>(54)</sub> =-1.30, p=0.201

Note. Low Psychopathy < 11 on PCL: SV; separate multiple (linear) regression analyses were conducted for each Profile of Mood States (POMS) scale, with mood change scores as the dependent variable (Post-presentation minus pre-presentation), two predictors (orthogonal contrasts) assessing group differences, and three covariates (dummy variables) accounting for stimulus order effects; <sup>a</sup> tested as a continuous contrast; <sup>b</sup> a mood variation score was calculated for each participant as their average squared change score across the T, D, A and V scales; # trend (p<0.05); \* p < 0.01; \*\* p < 0.001.

Table 3.7: Mood variation by emotion and group (Task B) – Mean (SD) mood variation scores and associated analyses

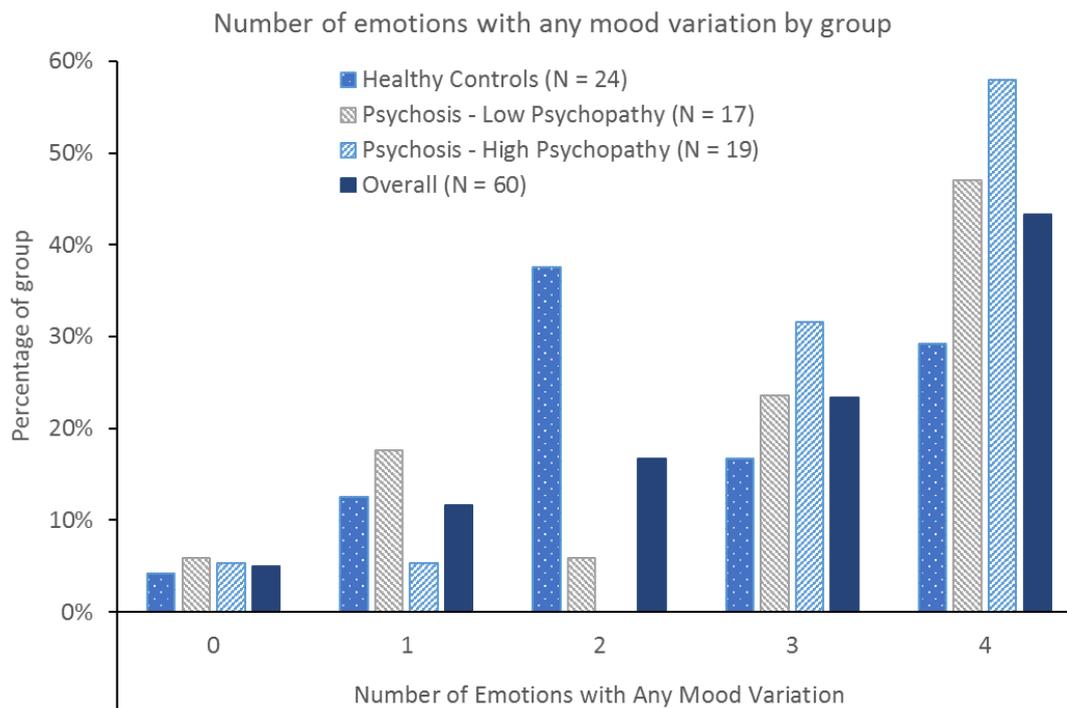
Group		Mood variation – Mean (SD) mood variation scores based on POMS changes							
		Happy	Sad	Anger	Fear	Overall			
Healthy Control (N = 24)		0.17 (0.37)	0.19 (0.48)	0.16 (0.20)	0.16 (0.48)	0.17 (0.35)			
Psychosis - Low Psychopathy (N = 17)		0.36 (1.25)	0.21 (0.30)	0.21 (0.29)	0.50 (1.36)	0.32 (0.74)			
Psychosis - High Psychopathy (N = 19)		0.61 (1.13)	0.29 (0.38)	0.29 (0.40)	0.25 (0.36)	0.36 (0.47)			
<u>Group contrasts</u> (Between-groups)		<u>Emotion Contrasts</u> (Within-groups)		<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC3 interaction contrasts</u>		<u>GC2 by EC1 to EC3 interaction contrasts</u>	
				Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not		EC1: Negative Vs. Positive		0.05	1.90 (0.168)	-0.04	1.77 (0.183)	0.02	0.25 (0.619)
Beta	W <sup>2</sup> (p)	EC2: Other Negative Vs. Sad		-0.01	0.24 (0.623)	0.02	1.15 (0.283)	0.03	0.93 (0.334)
-0.08	1.95 (0.163)	EC3: Fear Vs. Anger		-0.02	0.55 (0.457)	0.02	0.62 (0.431)	0.06	1.57 (0.210)
GC2: Low Vs. High Psychopathy (Continuous Contrast)									
Beta	W <sup>2</sup> (p)								
-0.01	0.01 (0.924)								

Note. Mood variation scores were calculated for each participant as their average squared change score (Post-presentation minus pre-presentation) across the Profile of Mood States (POMS) Tension (T), Depression (D), Anger (A) and Vigour (V) scales.

Grand Mean (SD) [N = 60] = 0.27 (0.52). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 11 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Mood variation (N = 60 participants, across 4 emotions), which also included three covariates (dummy variables) accounting for stimulus order effects: # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Fear stimuli, 28.3% for the Sad stimuli, and 20.0% for the Anger stimuli. However, as illustrated in [Figure 3.1](#), across the four Task B emotions, most participants displayed some mood variation (No emotions: 5.0%; One: 11.7%; Two: 16.7%; Three: 23.3%; and Four emotions: 43.3%).

While no main effects were apparent (Table 3.7), among the healthy controls mood variation was relatively similar across all four categories of facial expression stimuli (Mean TDAV mood variation scores ranging from 0.16 to 0.19). On the other hand, for the psychosis group, there were slightly increased fluctuations in mood variation across emotional stimuli (Mean mood variation scores ranging from 0.21 to



0.61).

Figure 3.1 *Number of emotions with any mood variation by group*

### 3.6.2.3 Scan-path performance for temporal indices – Task B

Scan-path performance on the two temporal parameters extracted during the emotion induction Task B are detailed in [Table 3.8](#) and [Table 3.9](#), respectively. Similar

Table 3.8: Fixation counts (Task B) – Mean (SD) number of fixations by emotion and group, and associated analyses

Group		Fixation counts – Mean (SD) number of fixations							
		Happy	Sad	Anger	Fear	Overall			
Healthy Control (N = 24)		139.58 (41.23)	145.38 (45.81)	135.42 (43.33)	146.63 (47.82)	141.75 (40.32)			
Psychosis - Low Psychopathy (N = 17)		137.82 (40.70)	133.82 (35.64)	136.35 (31.49)	141.00 (29.30)	137.25 (29.77)			
Psychosis - High Psychopathy (N = 19)		137.89 (41.05)	148.21 (46.68)	157.76 (39.35)	153.37 (42.81)	147.78 (39.87)			
<u>Group contrasts</u> (Between-groups)		<u>Emotion Contrasts</u> (Within-groups)		<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC3 interaction contrasts</u>		<u>GC2 by EC1 to EC3 interaction contrasts</u>	
				Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not		EC1: Negative Vs. Positive		-2.40	3.11 (0.078)	0.91	0.53 (0.468)	-0.77	0.32 (0.573)
Beta	W <sup>2</sup> (p)	EC2: Other Negative Vs. Sad		-0.66	0.26 (0.612)	1.99	2.17 (0.141)	-0.21	0.03 (0.865)
-0.26	0.00 (0.954)	EC3: Fear Vs. Anger		-1.81	1.82 (0.177)	-1.74	1.44 (0.230)	1.01	0.86 (0.354)
GC2: Low Vs. High Psychopathy (Continuous Contrast)									
Beta	W <sup>2</sup> (p)								
5.29	1.40 (0.236)								

Note. Grand Mean (SD) [N = 60] = 142.38 (37.10). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 11 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixation counts (N = 60 participants, across 4 emotions), which also included three covariates (dummy variables) accounting for stimulus order effects: # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 3.9: Fixation duration (Task B) – Mean (SD) fixation duration (ms) by emotion and group, and associated analyses

Group	Fixation duration – Mean (SD) fixation duration (ms)							
	Happy	Sad	Anger	Fear	Overall			
Healthy Control (N = 24)	425.24 (152.34)	419.30 (191.69)	431.56 (203.57)	431.66 (229.39)	426.94 (179.76)			
Psychosis - Low Psychopathy (N = 17)	422.80 (170.72)	395.89 (113.58)	402.19 (109.03)	367.28 (107.13)	397.04 (99.30)			
Psychosis - High Psychopathy (N = 19)	400.47 (179.39)	362.73 (141.08)	334.51 (129.93)	346.38 (131.23)	361.52 (122.90)			
<u>Group contrasts</u> (Between-groups)	<u>Emotion Contrasts</u> (Within-groups)	<u>Emotion main effect contrasts</u>		<u>GCI by EC1 to EC3 interaction contrasts</u>		<u>GC2 by EC1 to EC3 interaction contrasts</u>		
		Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	
GC1: Psychosis Vs. Not	EC1: Negative Vs. Positive	10.80	2.82 (0.093)	-9.74	2.99 (0.084)	-3.41	0.29 (0.589)	
Beta	W <sup>2</sup> (p)	EC2: Other Negative Vs. Sad	1.80	0.11 (0.743)	-5.51	0.90 (0.343)	2.76	0.31 (0.580)
21.60	1.35 (0.245)	EC3: Fear Vs. Anger	2.79	0.26 (0.613)	-2.50	0.17 (0.680)	-6.38	2.10 (0.148)
GC2: Low Vs. High Psychopathy (Continuous Contrast)								
Beta	W <sup>2</sup> (p)							
-18.74	1.57 (0.210)							

Note. Grand Mean (SD) [N = 60] = 397.75 (143.70). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 11 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixation duration (N = 60 participants, across 4 emotions), which also included three covariates (dummy variables) accounting for stimulus order effects: # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Generalised Linear Model analyses, involving the same set of eleven planned contrasts revealed no significant overall group differences for either the number or duration of fixations, nor were there any scan-path differences across categories of emotional stimuli, or any group by emotion interactions. For Task B, the Grand Mean number of fixations was 142.38, with a corresponding mean fixation duration of 397.75ms (across the 60sec presentations of 10 stimuli per emotion category).

#### *3.6.2.4 Relationships between Task B indices*

As shown in [Table 3.10](#), Total Mood Disturbance on the POMS prior to undertaking Task B was associated with emotion regulation difficulties on the DERS total score ( $r = 0.35$ ,  $p < 0.001$ ). There was also a trend level association between Total Mood Disturbance at baseline and Task B Mood Variation based on TDAV changes on the POMS ( $r = 0.15$ ,  $p = 0.021$ ). In terms of the Task B scan-path indices, there was a relationship between Mood Variation and both the number ( $r = -0.24$ ,  $p < 0.001$ ) and duration of fixations ( $r = 0.19$ ,  $p = 0.004$ ), whereby fewer fixations and a longer mean duration per fixation were associated with increased Mood Variation. There was also the same strong inverse association, noted in Task A, between the Task B number of fixations and duration of fixations indices ( $r = -0.82$ ,  $p < 0.001$ ). The relationships between temporal scan-path parameters across tasks revealed significant moderate associations between Task A and Task B for the number and duration of fixation indices (see the right hand columns of Table 3.10). However, within Task A, for the same four categories of emotion, there was a trend level negative association ( $r = -0.13$ ,  $p = 0.045$ ) between recognition accuracy and fixation count, with higher accuracy associated with fewer fixations.

To further examine changes in tracking performance between Task B and Task A, fixation count and fixation duration difference scores (Task B minus Task A) were calculated for each participant for the four emotions common to both tasks. Task B fixation counts per emotion (see [Table 3.8](#)) were divided by 10 prior to these calculations to account for the number of face stimuli per emotion. [Figure 3.2](#) illustrates the mean differences in tracking performance between tasks by emotion and group. Similar Generalised Linear Model analyses to those reported in [Tables 3.8](#) and [3.9](#) were conducted, with these difference scores as the dependent variables (although full details are not reported here). Once again, there were no statistically significant differences between groups, or emotions, or any significant interaction contrasts. However, there was a substantial overall reduction in the number of fixations per face stimulus in Task B (Mean difference: -3.76;  $W^2 = 106.41$ ,  $p < 0.001$ ) and a corresponding increase in the mean fixation duration (Mean difference: 94.67ms;  $W^2 = 52.06$ ,  $p < 0.001$ ). There was also a trend level group effect (GC1:  $W^2 = 4.14$ ,  $p = 0.042$ ), with healthy controls tending to have a more marked fixation duration increase in Task B relative to the psychosis groups (Mean differences: 129.55 vs. 71.08ms).

Table 3.10: Selected correlations with Task B indices – Overall (240 measures per index; N = 60 participants across 4 emotions).

<i>Task index</i>	POMS Total Mood Disturbance <sup>a</sup>	Task B Mood Variation based on TDAV changes <sup>b</sup>	Task B Number of fixations	Task B Fixation duration	Task A Recognition accuracy	Task A Number of fixations	Task A Fixation duration
DERS Total score	0.35 (<0.001)**	0.10 (0.131)	-0.10 (0.115)	-0.04 (0.541)	-0.05 (0.486)	-0.09 (0.186)	0.14 (0.036)#
POMS Total Mood Disturbance		0.15 (0.021)#	-0.10 (0.123)	-0.07 (0.305)	-0.10 (0.112)	-0.02 (0.757)	-0.08 (0.233)
Task B Mood Variation based on TDAV changes			-0.24 (<0.001)**	0.19 (0.004)*	0.01 (0.937)	-0.16 (0.013)*	0.10 (0.140)
Task B Number of fixations				-0.82 (<0.001)**	-0.10 (0.115)	0.60 (<0.001)**	-0.53 (<0.001)**
Task B Fixation duration					0.13 (0.052)	-0.45 (<0.001)**	0.50 (<0.001)**
Task A Recognition accuracy						-0.13 (0.045)#	0.11 (0.094)
Task A Number of fixations							-0.78 (<0.001)**

Note. Pearson product-moment correlations, with two-tailed *p* values in brackets; <sup>a</sup> POMS Total Mood Disturbance score pre task B; <sup>b</sup> a mood variation score was calculated for each participant as their average squared change score across the POMS Tension (T), Depression (D), Anger (A) and Vigour (V) scales; # trend ( $p < 0.05$ ); \*  $p < 0.01$ ; \*\*  $p < 0.001$ .

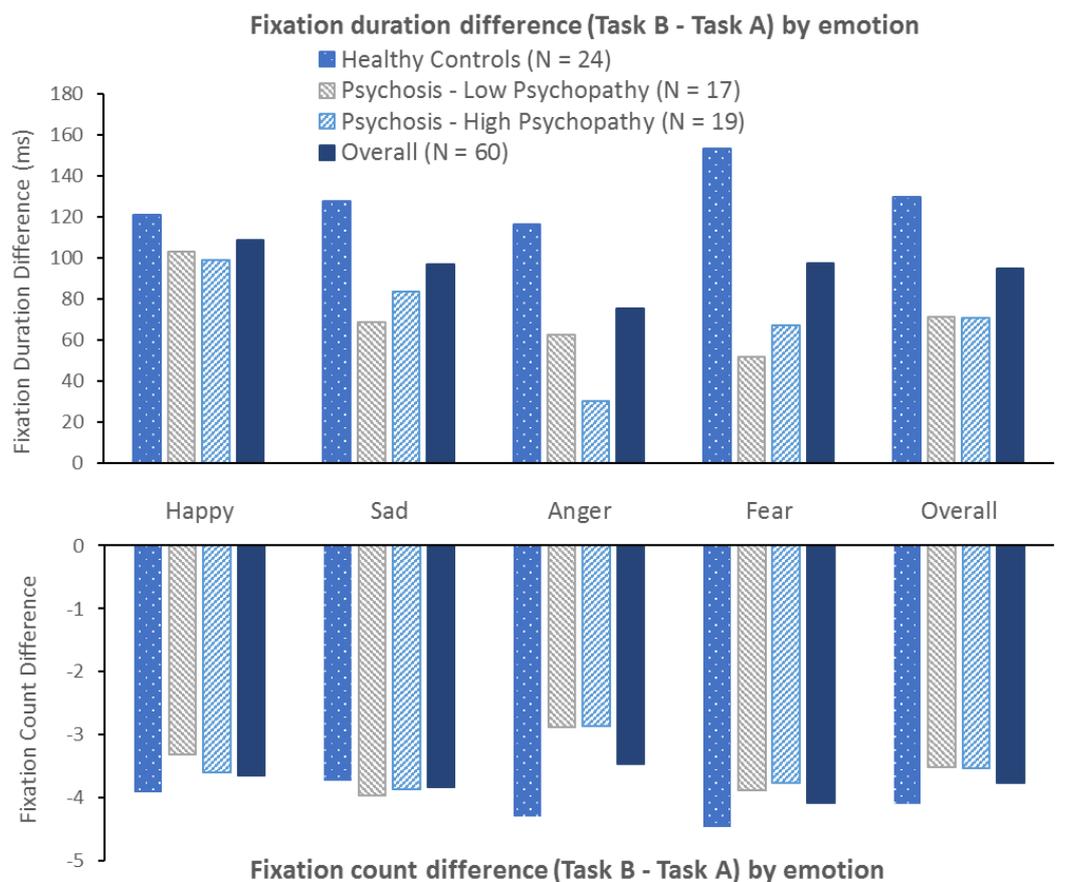


Figure 3.2 Tracking performance between Task B and Task A, by emotion and group

### 3.7 Discussion

In comparison to the healthy controls, an increased difficulty in emotion regulation as measured on the DERS was apparent among the psychosis groups (means = 59.33 vs. 81.70). The level of emotion dysregulation reported among the psychosis group with low psychopathy traits was very similar on all six domains to that reported among another Australian community schizophrenia sample (Ayre, 2013). The psychosis groups had significantly higher means for non-acceptance of emotional responses, difficulties engaging in goal-directed behaviour, impulse control difficulties, limited access to emotion regulation strategies, the total score, and a tendency for an increased lack of emotional clarity. For low versus high psychopathy traits, only one

significant difference was apparent, with the psychosis group with low psychopathy reporting increased difficulty on the non-acceptance of emotional responses domain. The healthy controls in this study reported slightly lower levels of emotion regulation difficulties in comparison to another Australian community sample (Mean = 66.53; Ayre, 2013), although not substantially dissimilar.

At baseline, prior to undertaking the visual-cognitive emotion induction task, the psychosis groups reported significantly higher levels of mood disturbance on the tension domain, as well as total mood disturbance on the POMS, and tended to have increased levels for depression. There were no differences in mood state between the low and high psychopathy groups at baseline. Total mood disturbance on the POMS at a baseline was associated with emotion regulation difficulties on the DERS total score. The existence of mood disturbance differences at baseline is consistent with research around the subjective experience of emotion, with regard to an individual homeostatic baseline, influenced by life events and experiences (Hoppenbrouwers et al., 2016a). For example, the subjective experience of emotion may be lower (e.g., fear in psychopathy) but deviations from baseline may still occur, with relative mood changes similar to individuals from non-clinical samples (Hoppenbrouwers et al., 2016a).

Variations in mood state using the POMS “right now” instrument were successfully demonstrated during the emotion induction task across multiple ratings (every 2-3 minutes), following the presentation of a series of facial expression stimuli among both the healthy control and psychosis group participants. However, the mean mood variation scores obtained were indicative of a modest induction effect, consistent with the type of static facial expression stimuli utilised in laboratory-based settings. In

addition, pre-existing relatively high baseline levels on the Vigour domain may have potentially limited the capacity for substantial variation in positive mood (e.g., item-9 Cheerful) given POMS ratings utilised a 5-point Likert scale.

Following the presentation of happy facial expressions, while there were no apparent group differences, global induction effects were obtained with significant reductions on the Tension, Anger, and Fatigue/Confusion domains, on overall Mood Variation, as well as a trend for increased Vigour. Although, as already mentioned, the capacity for change in Vigour was likely reduced, given the relatively high mean level at baseline. Similarly, following the presentation of sad facial expressions, significant overall Mood Variation occurred. There were trend level changes in Depression and Vigour, and a reciprocal relationship emerged, whereby, as Depression (sadness) increased Vigour (happiness) reduced. Of interest, while not reaching significance, when presented with sad stimuli the psychosis group tended to report increased Anger, while healthy controls reported reduced Anger.

Once again, while there were no significant group differences, following the presentation of angry facial expressions, there were significant overall changes on the Anger and Vigour domains, as well as in overall Mood Variation. A trend level difference was found for the psychosis group, with the low psychopathy group not reporting any change in Vigour while the high psychopathy and healthy controls reported reduced Vigour. Similarly, following the presentation of facial expressions of Fear, while no group differences emerged, there was significant overall Mood Variation, as well as trend level changes in Vigour. However, there were no significant mood changes in Tension or Depression. Perhaps this is not surprisingly, as it is unlikely that

substantial mood variation would have been apparent on some associated POMS items; for example, when utilising static facial stimuli in the context of a (safe) laboratory setting, and enquiring about how “anxious” or “terrified” you feel right now (items 11 or 12). However, across the four emotions utilised, the majority of participants did actually display some mood variation.

Analyses of mood variation by emotion and group, controlling for stimulus order, revealed no significant group differences in mood induction performance for Task B, suggesting the ability to experience or induce a change in mood state is not impaired in psychosis *per se*. This finding is broadly consistent with previous research suggesting the subjective or conscious experience of emotion in psychosis is intact (Cohen & Minor, 2010; Gur et al., 2006; Kring et al., 1993). Although it is worthy of note that the psychosis group at baseline reported elevated total mood disturbance in comparison to the healthy controls, as well as Tension, and slightly elevated scores on the Depression domain. These higher scores could have led to a decreased capacity for mood state changes on the POMS following the presentation of particular facial expression stimuli, such as sad on the depression domain. Moreover, a trend level association between Total Mood Disturbance at baseline and overall Task B Mood Variation on the POMS was found. In addition, while there were no main effects for mood variation by group, the healthy controls did tend to respond with changes in mood variation of a similar magnitude regardless of the emotion category presented. They changed in both a positive and negative direction on the POMS domains simultaneously, in a manner that was consistent with the category of emotional stimuli presented. This is perhaps suggestive of a higher order cognitive shift in mood state in relation to the induction task being undertaken (i.e., responding based on perceived

stimulus characteristics expectations, as opposed to the genuinely felt emotions), which was not as apparent among the psychosis groups. The psychosis groups tended to exhibit increased fluctuations in mood variation, or mood volatility across all categories of emotional stimuli and on all POMS domains, irrespective of the emotion presented.

An examination of the accompanying visual scan-path performance, recorded simultaneously during the emotion induction task, revealed no atypical scan-path patterns among the psychosis groups. There were no group differences for either the number or duration of fixations, across categories of emotional stimuli, or any group by emotion interactions. However, regardless of group, there were significant associations between eye-tracking performance and the magnitude of emotion induction. The length of stimulus presentation during the emotion induction task allowed for extended processing and did not require rapid decision making (since it involved ten static facial stimuli per emotion category presented for 6000ms each, with eye-movement recordings obtained over 60 seconds). Consequently, this task could therefore be seen as having a relatively low level of cognitive demand or difficulty, which traditionally is generally not the case in studies examining facial emotion processing; for example, tasks typically involve a brief stimulus presentation window (i.e., ranging from 1000-6000ms) followed immediately by an identification or discrimination decision.

Regardless of group, for the Task B scan-path indices a relationship was found between mood variation and both the number and duration of fixations, whereby, fewer fixations and a longer mean duration per fixation were associated with increased Mood Variation. The same strong inverse association noted between the temporal scan-path parameters in Task A, as well as moderate associations between Task A and Task B for

the number and duration of fixation indices were apparent. However, in Task A, for the same four categories of emotion (Happy, Sad, Anger, Fear) there was a trend level negative association between recognition accuracy and fixation count, with higher accuracy associated with fewer fixations. Interestingly, analyses undertaken using fixation count and duration difference scores between Task B and Task A, in order to examine changes in eye-tracking performance across tasks, revealed differential scan-path strategies were used in the emotion recognition and induction tasks (for the same categories of emotion). While no significant group, emotion or interaction effects were evident, there was a substantial overall reduction in the number of fixations per face stimulus in Task B and a corresponding increase in the mean fixation duration. These findings suggests that fewer fixations (around 3.76 fewer), of longer duration (approximately 94.7ms longer), may be advantageous strategies in feeling (experiencing) or inducing mood. On the other hand, this more restricted scanning strategy (in terms of the number of fixation points) may be disadvantageous in correctly recognising or categorising emotions. In addition, healthy controls tended to have a more marked fixation duration increase in Task B relative to the psychosis groups.

It is also worthy of note that mood variation, whilst modest in scale, was successfully demonstrated amongst the majority of participants. However, the self-report POMS instrument utilised during this task may have limited utility in detecting more subtle induction effects for particular categories of facial emotion stimuli. For example, there was evidence of floor effects (e.g., Sad) due to pre-existing levels of mood disturbance at baseline among the psychosis participants, as well as ceiling effects (e.g., Happy) with relatively high levels reported on the Vigour domain at baseline. This potentially restricts the capacity for detection of subtle mood variation or induction

effects, and finding group differences, especially in the context of using five-point Likert scales.

The findings from this current study do not support the suggestion from the psychopathy literature of a reduced experience of happy and increased experience of anger (Hoppenbrouwers et al., 2016a). However, a similar absence of subjective self-report differences during an emotion experience task by Casey et al. (2013) among individuals with high psychopathy traits has been reported; although, the stimuli utilised were emotional scenes including people not faces expressing emotion. The authors suggest the possible normalising of subjective experience as having clinical and treatment implications among this population, as their findings were combined with increased autonomic arousal (i.e. cardiovascular response) for negative emotions. A similar absence of differences in self-reported valence and arousal ratings among psychopathy groups (e.g., high compared to low psychopathy) when ratings the emotional content of stimuli, has also previously been reported (Patrick et al., 1993). Although, direct comparisons to the current study group of psychosis participants with coexisting psychopathy traits is limited.

## **Chapter 4 – Study 3: Face Recognition and Working Memory**

### **4.1 Introduction**

The ability to perceive and induce emotional states involve a myriad of social and neurocognitive process. The existing literature provides solid evidence of a relationship between neurocognitive and social-cognitive functioning and patient outcomes in psychosis. Consequently, it is important to address how higher order cognitive processing may influence facial emotion processing within the current study sample of individuals with psychosis and coexisting psychopathy traits. Given that both emotion and visual processing differences are documented, it may be difficult to disentangle the potential linkages with neurocognitive, attentional, and memory factors within the context of social-cognitive processing of facial emotional stimuli. Therefore, the visual-cognitive task reported in this chapter was restricted to images displaying a neutral emotion, thereby placing particular emphasis on face recognition and visual scanning performance, and associations with working memory (via comparisons between immediate and delayed recall components of the task). Throughout this chapter expressions such as “cognitive” and “neuro-cognitive” functioning are used relatively interchangeably, largely reflective of variations in the studies being referenced.

### **4.2 Cognition and face processing in psychosis**

Impairments in cognitive functioning in psychosis fall broadly into two areas, neurocognition and social cognition. Neurocognitive dysfunction is a central feature, with impairments present in the majority of people with schizophrenia (Seidman & Mirsky, 2017). Core neurocognitive deficits include learning and memory impairments (Rajji et al., 2009; Salavati et al., 2015), with a broad range of impairments impacting on a large number of domains (Harvey & Rosenthal, 2018), including: verbal (Brébion,

David, Jones, & Pilowsky, 2004; Tracy et al., 2001); working (Bowie & Harvey, 2005; Fuller et al., 2009; Lee & Park, 2005; Silver, Feldman, Bilker, & Gur, 2003; Zanello, Curtis, Badan Bâ, & Merlo, 2009) and episodic memory (Bowie & Harvey, 2005); attention (Gold, Wilk, McMahon, Buchanan, & Luck, 2003); visuospatial ability (Takahashi et al., 2005); learning (Kurtz et al., 2001); processing speed; executive functioning; and certain language skills (Bowie & Harvey, 2005). Domains less impacted include vocabulary and information skills, and word recognition reading (Harvey et al., 2006a). Performance has also been shown to be less impaired in recognition memory, during episodic memory tests compared to rate of learning and free recall, and in verbal fluency, more than other verbal skills such as naming (Harvey & Rosenthal, 2018).

With respect to severity, processing speed, followed by episodic and working memory deficits, are reported to be most impaired (Dickinson & Harvey, 2009), and attention is also particularly affected (Reichenberg et al., 2010). While the profile of impairments exhibited are substantial, they are not consistent with cortical dementia, such as that seen in Alzheimer's disease, with rapid forgetting absent in psychosis and delayed recall performance unaffected for successfully encoded information (Harvey & Rosenthal, 2018). The profile of cognitive and functional impairments have been described as similar to accelerated or exaggerated aging, whereby performance of individuals with psychosis is not unlike healthy controls 30 plus years older, occurring substantially earlier than anticipated (Harvey & Rosenthal, 2018). The magnitude of deficits (ranging from 1-2 SD) across domains can be considerable in comparison to healthy controls (Georgiades et al., 2017; Gold & Harvey, 1993).

A familial transmission aspect of memory and attention deficits has also been suggested, due to detection among first-degree relatives (Sponheim, Steele, & McGuire, 2004). Impaired working memory has been suggested as a potential endophenotypic marker for schizophrenia, due to performance deficits (Park & Gooding, 2014), as well as fMRI evidence of alterations in default mode network (DMN) activity and functional connectivity among both patients and their first-degree relatives, indicating possible genetic transmission of this trait abnormality (Whitfield-Gabrieli et al., 2009). Alterations in DMN activity in the parahippocampal gyrus is also linked to working memory performance among first-degree relatives (Seidman et al., 2014).

Neurodevelopmental models propose that core cognitive deficits occur due to abnormal brain development, with abnormal processes evident long before illness onset (Rapoport, Giedd, & Gogtay, 2012), leading to problems in acquiring cognitive abilities (Bora & Murray, 2014). Further decline and structural brain changes years after illness onset, are seen as support for neurodegenerative models (Rund, 2009). In opposition to these models, longitudinal studies reveal stability of neurocognitive deficits and even small improvements, providing strong evidence against schizophrenia being neurodegenerative (Bora & Murray, 2014; Szoke et al., 2008), including a recent study of first-episode patients followed up over 10 years (Rund et al., 2016). However, the possibility of neurodegeneration or neuroprogression cannot be ruled out entirely (Rund et al., 2016), given the decline in neurocognitive functioning reported among older populations (Harvey et al., 1999). Moreover, studies demonstrating that cognitive and functional impairment worsens around illness onset, in relation to premorbid functioning, imply that deficits are not purely due to developmental disabilities (Harvey & Rosenthal, 2018).

Neurocognitive deficits, such as those associated with attention and memory, have also been shown to be present during the prodromal phase (Caspi et al., 2003) and at illness onset (Hill, Schuepbach, Herbener, Keshavan, & Sweeney, 2004). They appear to be independent of symptoms, being present with only minimal psychotic symptoms (Reichenberg et al., 2009), and have been shown to remain relatively stable (Bonner-Jackson et al., 2010; Rund et al., 2016). However, increased severity has been reported with younger age of illness onset (Rajji et al., 2009) and greater deterioration in older age (Harvey et al., 1999; Harvey & Rosenthal, 2018). Some previous studies have suggested associations between symptoms and neurocognition (Ventura, Thames, Wood, Guzik, & Helleman, 2010), in particular negative symptoms (Bora, Yucel, & Pantelis, 2009; Meyer et al., 2014), but findings are mixed, with others suggesting weak (Rund et al., 2007) or no relationships (Harvey, Green, Bowie, & Loebel, 2006b; Rund et al., 2016; Rund et al., 2007). Associations between psychotic relapse, treatment resistance and longitudinal cognitive and functional changes are also apparent (Harvey & Rosenthal, 2018). In addition, comorbid conditions such as metabolic syndrome (Boyer et al., 2014; Lindenmayer et al., 2012) and substance abuse can impact on cognitive functioning (Harvey & Rosenthal, 2018).

More broadly, the neurotoxicity hypothesis proposes that psychotic states may have a toxic effect on the brain (Rund, 2014). Brain volume and gray matter changes have been detected early in the illness (Arango et al., 2008; Pantelis et al., 2005) and at chronic stages (Hulshoff Pol & Kahn, 2008; van Haren, Schnack, Cahn, & et al., 2011). Some previous studies report increased psychotic episodes related to progressive gray matter loss (Cahn et al., 2006; van Haren et al., 2007), as well as ventricular enlargement in chronic patients (Davis et al., 1998). One longitudinal imaging study

reported that excessive cortical thinning across the illness course, particularly in frontal and temporal regions associated with poorer functional and symptomatic outcome, as well as typical antipsychotic use (van Haren et al., 2011). On the other hand, improved verbal memory and learning at 1-2 year follow-up in early psychosis has been associated with fewer relapses (Rund et al., 2007). This longitudinal study of first-episode psychosis also demonstrated that relapse and treatment response (remission) within the first year of illness significantly predicted longer-term neurocognitive functioning at 10-year follow-up (Barder et al., 2013; Rund et al., 2016). On the basis of these studies, limited evidence for the neurotoxicity hypothesis exists, with both the duration of untreated psychosis and symptoms unrelated to the neurocognitive trajectory, and with stability in functioning over time; moreover, early clinical course has been shown to be the strongest predictor of longer-term neurocognitive outcome (Rund et al., 2016).

Of clinical importance, difficulties in social and occupational functioning and quality of life are associated with neurocognitive deficits in psychosis (Cohen, Forbes, Mann, & Blanchard, 2006; Green, Kern, Braff, & Mintz, 2000; Hofer et al., 2005). Neurocognitive functioning is a key determinant of recovery of everyday and work functioning, and factors predictive of outcome include: working memory; attention and early perceptual processing; verbal memory; and processing speed (Nuechterlein et al., 2011; Seidman & Mirsky, 2017). A number of previous studies confirm that individuals with schizophrenia who show improved aspects of neurocognition (i.e., working memory, attention, verbal learning, and executive function) are likely to obtain better vocational and quality of life outcomes (Green & Mandal, 2002; Shayegan & Stahl, 2005). Furthermore, impairments in functional capacity are not associated with illness

chronicity or length of treatment, being present in first episode patients, with psychosis patients in their 20s performing more poorly on measures of everyday functioning skills than healthy controls in their 60s and 70s (Harvey & Rosenthal, 2018).

Social-cognitive deficits are distinct from neurocognition and social skills but are also overlapping (Mancuso et al., 2011), with dysfunction in attention, memory systems, language and perceptual mechanisms seen as “*cold cognition*” and abnormalities in emotion processing seen as “*hot cognition*” (Chung & Barch, 2011; Mathews & Barch, 2010; Niznikiewicz et al., 2013). Social cognition draws on both sets of processes, being the ability to express attitudes and intentions and predict and interpret those in others; for example, social cognition involves recognising non-verbal social cues such as emotion from a person’s face and making decisions in social situations (Niznikiewicz et al., 2013). Impairments in emotion processing are prominent in psychosis, stable across illness phases (Green et al., 2012; Horan et al., 2012), and negatively associated with outcomes (Hoertnagl & Hofer, 2014). They are also predictive of clinical and functional outcomes, including community (McCleery et al., 2016), social and occupational functioning, and independent living ability (Hooker & Park, 2002; Horan et al., 2012; Kee et al., 2003; Penn et al., 2000a; Weiss et al., 2006).

#### *4.2.1 Face recognition performance*

Studies examining face recognition among individuals with schizophrenia have reported global deficits (Addington & Addington, 1998; Heinisch, Wiens, Grundl, Juckel, & Brune, 2013; Lee, Kwon, Shin, Lee, & Park, 2007; Zhang, Zhu, Xu, Jia, & Liu, 2012), as well as a specific self-face recognition impairment that is associated with positive symptoms (Irani et al., 2006; Kircher et al., 2000). Using a matching task,

Martin et al. (2005) found reduced recognition accuracy, which was considered to be indicative of a deficit in face identity processing. More recently, using an object processing task She et al. (2017) reported significantly lower accuracy for both face identity and affect, suggestive of face specific visual processing deficits in schizophrenia.

Face recognition involves both physiological and psychological processes, across visual, cognitive and affective domains (Chen et al., 2009). These include cognitive domains of attention, memory and processing speed, all of which are known to be impaired in schizophrenia and impact on face processing. Such associations also make it difficult to ascertain whether impairments are attributable to confounding factors such as attention and perceptual deficits (Bortolon et al., 2015). Existing research indicates a strong relationship between impairments in face processing and neurocognitive impairments: in face recognition, across the domains of attention and working memory (Addington & Addington, 1998; Chen et al., 2009); and for facial emotion processing, in attention, working, verbal and spatial memory, as well as language abilities (Addington & Addington, 1998; Huang & Hsiao, 2017; Kohler et al., 2000; Sachs et al., 2004; Schneider et al., 1995).

Chen et al. (2009) examined visual and cognitive processing of faces as well as non-face visual objects, which revealed that patients with schizophrenia display: reduced accuracy in visual detection of configural facial images; moderately degraded performance in the perceptual discrimination of identity (varied via morphing); and significantly impaired working memory for faces compared to healthy controls. Performance was also shown to be degraded on non-face versions of the tasks, but was

not associated with face recognition performance, with the researchers suggesting greater signal strength may be required for the visual and cognitive processing of facial information (Chen et al., 2009). Structural imaging studies have also found a reduced volume of gray matter in the fusiform gyrus and adjacent areas (FFA), the cortical systems for visual and cognitive processing of facial information in schizophrenia (Lee, Shenton, Slaisbury, & et al., 2002) regarded as crucial in face identification and recognition (Kanwisher & Yovel, 2006). However, mixed findings have been found from functional imaging studies of face working memory tasks, including altered (Yoo, Choi, Juh, & et al., 2005) and comparable (Yoon, D'Esposito, & Carter, 2006) responses to healthy controls. This suggests that, some neurophysiological processes involved in face recognition may be deficient but others may be preserved in schizophrenia, highlighting the need for sophisticated study designs to tease out the complex intricate visual and cognitive processes that are involved (Chen et al., 2009).

Emerging research continues to attempt to disentangle the complex interrelationships between clinical symptoms, neurocognition and facial emotion processing, to determine the extent of their contributions to social functioning in schizophrenia (Huang & Hsiao, 2017). One such study, by Huang and Hsiao (2017), found that years of education, younger age, sustained attention, better working memory and facial emotion recognition were all significantly associated with global social functioning in schizophrenia. These findings are consistent with prior research supporting the important contribution of neurocognitive abilities to social functional outcomes, in particular working memory and attention (Milev, Ho, Arndt, & Andreasen, 2005; Pan, Chen, Chen, & Liu, 2009). While facial emotion processing has been

extensively explored, face processing *per se* has received less attention (Bortolon et al., 2016).

#### *4.2.2 Face recognition: working memory for faces and visual scanning in psychosis*

In terms of visual processing deficits, working memory has been shown to play a key role in higher-level visual tasks and is strongly correlated with overall cognitive ability (Luck & Vogel, 2013). Working memory has been described as an active short-term system of limited capacity, that temporarily maintains information and supports thought processes by providing an interface between perception, long-term memory and action (Baddeley, 2003; Park & Gooding, 2014). Likewise, visual working memory has been defined as “the active maintenance of visual information to serve the needs of ongoing tasks” (Luck & Vogel, 2013). With respect to visual scanning, information is acquired during short fixations (around 200-500ms) separated by saccadic eye-movements that temporarily suppress processing and shift retinal image (Luck & Vogel, 2013). Visual working memory is seen as necessary to bridge the temporal and spatial shifts created by eye movements, linking pre- and post-saccade representations at different retinal locations (Luck & Vogel, 2013). More broadly, research has shown that the target of the eye-movement is stored in visual working memory and compared to newly fixated objects (Hollingworth, Richard, & Luck, 2008). Furthermore, eye movements may be biased towards objects that match the contents of current visual working memory (Mannan, Kennard, Potter, Pan, & Soto, 2010), with saccades becoming faster for target matches (Hollingworth, Matsukura, & Luck, 2013).

Visual working memory capacity differs across both individuals and groups, and is known to be impaired among individuals with psychosis (Gold et al., 2006; Gold et

al., 2003; Hahn et al., 2010; Leonard et al., 2013; Mayer, Fukuda, Vogel, & Park, 2012). Apparent differences in storage capacity (i.e., number of items held in memory) account for substantial reductions in intellectual functioning, while variations in the ability to use memory capacity efficiently also exists (Luck & Vogel, 2013). In schizophrenia, intact attentional selection for visual working memory storage has been demonstrated in some studies, with patients showing the same ability as healthy controls to exclude irrelevant distractors, suggesting attention is not globally impaired (Gold et al., 2006). However, ERP studies have revealed contralateral delay amplitude decreases in schizophrenia, when multiple items must be stored reflecting a specific impairment in the ability to distribute attention more broadly, potentially hyper-focusing on a smaller number of items (Leonard et al., 2013). Visuospatial working memory tasks also suggest that salient information can result in impaired filtering or effortful control of attention (Hahn et al., 2010). Slowed disengagement, or impaired contingent attentional capture, are suggestive of a selective impairment of top-down attentional control in schizophrenia, which may impair working memory encoding (Mayer et al., 2012).

Attaining detailed information about visual working memory impairments in psychosis may assist in understanding the real world clinical implications of cognitive impairments, such as when viewing dynamic faces or highly salient distractors (Luck & Vogel, 2013). It may also be crucial for informing cognitive remediation strategies; for example, if impaired perceptual encoding rather than memory retention is suggested, targeted behavioural training may be focused on top-down attentional selection aimed at enhancing working memory encoding (Mayer et al., 2012). Rather than simply documenting that deficits in working memory exist, whether due to reduced storage slots or an inability to gate distractors, it is important to explore the specific

components, such as encoding, maintenance, and manipulation, as well as how attention interacts with working memory (Park & Gooding, 2014). All have important implications for remediation work, and in clarifying the underlying casual mechanisms for behavioural impairments (Park & Gooding, 2014). Over the last decade, a range of social-cognitive interventions have been developed, some targeting specific social-cognitive domains, while others are more broad-based, combining neurocognitive training, with both achieving promising gains in social functioning (Tan et al., 2018). Although findings are inconsistent, the sustainability of positive social-cognitive gains are thought to be related to neurocognitive functional gains (Tan et al., 2018).

Research exploring the relationships between visual scan-path performance and social-cognitive functioning in psychosis has emphasised understanding the interactions between cognition and facial emotional processing (Becerril & Barch, 2010). Importantly, deficits can originate from perceptual low-level stages of visual exploration or higher-level cognitive stages (Bortolon et al., 2016). Eye tracking methodologies allow a natural continuous record of face processing, including the location, number and duration of fixations to relevant areas (such as facial features) to be tracked, utilising face recognition tasks involving both passive exploration and active decision making (Bortolon et al., 2016). Eye tracking face perception studies have shown that healthy individuals tend to focus on relevant or salient facial features, such as the eyes and mouth (Walker-Smith, Gale, & Findlay, 1977). On the other hand, individuals with schizophrenia tend to exhibit more restricted scan-path strategies, with fewer fixations of longer duration (Bortolon et al., 2016; Loughland et al., 2002a; Williams, Loughland, Gordon, & Davidson, 1999), as well as reduced saccades or avoidance of relevant features (Loughland et al., 2002a; Williams et al., 1999).

Subsequent studies have demonstrated that individuals with schizophrenia can direct attention to relevant facial features during active task conditions, such as when requested to recognise a face in comparison to a passive free viewing condition (Bortolon et al., 2016; Delerue, Laprevote, Verfaillie, & Boucart, 2010). For example, Delerue et al. (2010) did not find any significant group differences on temporal or spatial visual scan-path measures between individuals with schizophrenia or healthy controls during active tasks. Bortolon et al. (2016) examined processing of self, famous and unknown faces during both active and passive tasks, reporting that atypical visual scanning involved fewer fixations of longer duration in comparison to healthy controls (Bortolon et al., 2016). However, during tasks involving low-level memory and processing speed demands, individuals with schizophrenia demonstrated the ability to focus attention on relevant facial features in a similar way to controls, with no apparent performance differences in recognising faces, irrespective of the identity of the face or type of task (active or passive) (Bortolon et al., 2016).

Whether visual processing deficits in schizophrenia are general, across different types of stimuli or specific to faces remains under debate (She et al., 2017). Some studies report abnormal visual scanning patterns that are not stimulus specific, indicating a general impairment of eye-movement patterns in schizophrenia (Benson et al., 2012). For example, visual exploration of scenes has been shown to be impaired, implicating low-level visual processing deficits more broadly as a possible trait marker in schizophrenia (Beedie et al., 2011).

A review of behavioural, neuroimaging and neurophysiological studies was undertaken by Bortolon et al. (2015), seeking to address whether non-emotional aspects

of face processing are impaired. This review indicated that at a behavioural level visual perception deficits may not be specific to faces, and that, when accompanied by increased cognitive and perceptual demands, there was evidence of impaired second order configural processing (Bortolon et al., 2015). Neural and neurophysiological evidence implies that impaired earlier levels of visual processing are present prior to illness onset, possibly involving deficits in the interaction of the magnocellular and parvocellular pathways impacting on further processing (Bortolon et al., 2015; Butler et al., 2009). Consequently, Bortolon et al. (2015) propose that face perception deficits may not be a specific marker and may result from deficits in earlier visual processing. However, others challenge the view that there is a general deficit in visual processing in schizophrenia. For example, She et al. (2017) used visual search tasks concentrating on detection and identification, rather than matching tasks containing a general mnemonic component across stimuli (and shown to be impaired in schizophrenia). They found face evidence supporting face specific perception deficits (for identity and affect) but normal form perception, supporting a specific but not a general impairment in visual processing (She et al., 2017).

#### **4.3 Cognition and face processing in psychopathy**

There is limited research literature about the aspects of cognitive functioning associated with psychopathy traits that might influence face processing among individuals with psychosis. The two main cognitive impairments detailed in the literature relate primarily to empathic dysfunction, and impaired decision-making associated with the callous-unemotional and impulsive-antisocial components of psychopathy (Blair, 2013). While the psychopathy literature has tended to emphasize emotion-processing deficits, information-processing deficits have also been investigated

(Hiatt & Newman, 2006). Impaired executive functioning has been implicated in reduced behaviour control, with impulsivity a strong risk factor for violence among offending populations more broadly (Slotboom et al., 2017).

Specific cognitive processing deficits involving, involving dual-task attention, and behavioural inhibition have been outlined in psychopathy (Hiatt & Newman, 2006). While visual attention described as a critical element for cognitive control and goal directed behaviour, has been explored among violent offender populations (Slotboom et al., 2017). Specifically in psychopathy, interpersonal and affective traits have been related to improved or superior selective attention, while impulsive and antisocial traits have been associated with poorer attentional performance (Baskin-Sommers et al., 2012; Baskin-Sommers et al., 2011). Specific attentional abnormalities, in top-down (e.g. goal directed) attention and selection history, but not in bottom-up (e.g. stimulus driven) attention have also been reported among offender and community samples (Hoppenbrouwers et al., 2016b; Hoppenbrouwers et al., 2015)

Neurobiological evidence related to reduced empathic responding to the distress of others, has been associated with reduced amygdala responsiveness (Blair, 2013). While deficits in aspects of decision-making, specifically reinforcement learning (related to the impulsive-antisocial component of psychopathy), suggests functional impairments associated with dysfunction in the vmPFC and striatum (Blair, 2013). Developments in the understanding of the cognitive neuroscience of psychopathic traits as a result of recent functional imaging studies continues (Blair, 2013), however, a detailed understanding of the relationships between cognitive and emotional deficits in psychopathy has not yet been attained. There is a need to acquire a further

understanding of the cognitive mechanisms associated with face perception in psychopathy, as well as relationships for specific traits. More broadly, research exploring the relationships between personality traits and cognition is ongoing area of research, that aims to measuring the extent to which between-individual variation in a cognitive ability predicts or is predicted by, between-individual variation in a behavioural traits (Griffin, Guillette, & Healy, 2015).

#### *4.3.1 Face recognition performance*

The research base examining face processing in psychopathy has been primarily undertaken in the area of emotion recognition, focusing on the ability to recognise emotion (as detailed in Chapter 2, section 2.3) but also some research on the experience emotion (as detailed in Chapter 3, section 3.3), with a large body of research focussing on fear. Existing meta-analyses make note of the paucity of studies including the full range of emotions, for example, (Hoppenbrouwers et al., 2016a) when examining the conscious experience of emotion was unable to include the facial expressions of disgust, due to the few number of studies of sufficient quality available. Of particular relevance to this current study, neutral faces were not included in any of the existing meta-analysis exploring facial emotion processing (Dawel et al., 2012; Hoppenbrouwers et al., 2016a; Wilson et al., 2011). There appears to be a paucity of research in the area of face or recognition or identity processing among individuals with psychopathy.

#### *4.3.2 Face recognition: working memory for faces and visual scanning in psychopathy*

As noted, there is limited research specifically examining face recognition in psychopathy, while numerous studies have investigated the identification of facial emotion. In addition, while a few eye-tracking studies have explored the associated visual scanning strategies when processing facial expressions of emotion using eye-tracking technologies, these emotion recognition tasks have not included neutral face stimuli (Gillespie et al., 2017b; Gillespie et al., 2015c). Some studies however, have investigated attentional processes during facial emotion processing. One such study by Edalati, Walsh, and Kosson (2016) examining attentional bias to emotional faces did include neutral face stimuli. Using the emotion dot probe task, they found attentional bias among male inmates with psychopathy but contrary to expectations, toward happy versus neutral faces, which was attributable to factor 1 psychopathy traits (Edalati et al., 2016). Inconsistent and mixed findings, cast doubt on the generalisability of facial emotion findings but also highlighting the need for continued investigations in order to increase our understanding of the exact mechanisms involved.

#### **4.4 Aims and hypotheses**

This final study aimed to extend visual-cognitive and face processing research in psychosis by assessing immediate and delayed memory for faces, using neutral facial expressions. Firstly, this study sought to ascertain the extent of any impairments in face recognition among individuals with psychosis, while considering the impact of co-existing psychopathy traits. The second aim was to examine more broadly how higher order neurocognitive processes impact on the processing of facial emotion stimuli, irrespective of emotion; that is, when viewing faces depicting neutral facial expressions, what associations are detectable between visual scan-path, attentional, and memory

indices. The final goal was to further explore whether visual scanning strategies differ across emotion processing tasks; for example, are any differential processing strategies revealed by comparing participant performance when asked to “remember” a face versus “recognise” or “feel” emotion (examined in previous Chapters 2 and 3 - Task A and B). Neurocognitive impairments across a number of domains have been confirmed in the psychosis literature, including verbal and working memory, attention, visuospatial ability and learning, with core deficits in memory and attention present at illness onset (Brébion et al., 2004; Fuller et al., 2009; Heinrichs & Zakzanis, 1998; Lee & Park, 2005; Silver et al., 2003; Tracy et al., 2001). While in psychopathy cognitive deficits in executive functioning associated with impairments in attention, and decision making are indicated (Hiatt & Newman, 2006) (Hoppenbrouwers et al., 2016b; Hoppenbrouwers et al., 2015).

The primary hypotheses for this study are: 1) Compared to a healthy control group, individuals with psychosis will exhibit reduced face recognition accuracy, in terms of their immediate and delayed ability to recall faces, and a more restricted pattern of visual scanning; and 2) Psychosis participants with a higher number of psychopathy traits will perform more poorly, exhibiting poorer recall accuracy for faces as well as more restricted visual scanning. It was also generally anticipated that cognitive functioning will be positively associated with face recognition accuracy.

## **4.5 Method**

### *4.5.1 Participants*

As noted previously, all experimental studies in this dissertation were undertaken consecutively, during the same structured interview. Full participant details

for the study sample of 61 participants were provided in Chapter 2: Section 2.5.1. In brief, there were 24 healthy control participants and 37 community mental health outpatients with a psychotic disorder, with the latter divided into two groups comprising 18 psychosis participants with low psychopathy and 19 with high psychopathy traits.

#### *4.5.2 Measures*

All measures utilised in this study have been described elsewhere (please see Chapter 2: Section 2.5.2 for comprehensive details).

#### *4.5.3 Face stimuli*

The facial expression stimuli utilised during the face recognition and working memory Task C consisted of 28 colour images, with equal numbers of male and female actors portraying neutral expressions. Images were selected from three stimulus sets, which included four from the NimStim standardised set of facial expressions (Tottenham et al., 2009; <http://www.macbrain.org/resources.htm>) detailed previously in chapters 2 and 3 (Sections 2.5.3 and 3.5.3) for Task A and B, although none of the images presented in the prior tasks were utilised in Task C. In order to have sufficient numbers of neutral stimuli, six images were also selected from the standardised Ekman set of facial affect (Ekman & Matsumoto, 1993-2004), and a further 18 from the Centre for Brain and Mental Health Research (CBMHR) image set, developed locally by researchers at the CBMHR Visual-cognitive laboratory (unpublished, stimulus set). As illustrated in [Figure 4.1](#), stimulus images were selected two at a time from the same stimulus set (i.e., as a notional pair) based on, shared common elements (including age range, gender, and relatively similar features such as eye colour, hair colour/style), thereby comprising a total of 14 target and 14 non-target

(distractor) stimuli. All images had a resolution of  $506 \times 650$  pixels, displayed within equivalent parameters on a computer screen, in order to maintain a similar set location of facial features, such as eyes and mouth across all stimuli presentations.

### CBMHR image set

T10 and N11



T9 and N7



### Ekman image set

T2 and N1



T13 and N13



*Image set not approved for online publication subject to copyright*

Figure 4.1 *Examples of neutral facial expressions utilised as target and non-target stimuli in Task C, extracted from the CBMHR set; stimuli utilised from the Ekman (Ekman & Matsumoto, 1993-2004) and NimStim sets (Tottenham et al., 2009) were not approved for publication.*

#### 4.5.4 Eye tracking apparatus

Full details on the View Point eye tracker and visual scanning technology (Eye Link: 1000; SR Research Ltd, Ontario, Canada) utilised to record eye movements have been provided in Chapter 2, Section 2.5.4.

#### 4.5.5 Procedures

As noted previously, all three experimental studies were consecutive, with substantive procedural details provided in Chapter 2: Section 2.5.5. Specific details pertaining to the final study are provided here, which involved a visual-cognitive eye movement-recording task exploring face recognition and working memory.

All participants took part in the final face recognition and working memory Task C, during which selected subsets of the 28 neutral facial expression stimuli were presented across three phases: acquisition; immediate; and delayed recall (see [Table 4.1](#) for stimulus sequence). During the acquisition phase, visual scan-paths were recorded while participants viewed 14 target faces depicting neutral emotion (7 male and 7 female), presented in a pre-randomised order. The following instructions were displayed on a computer screen and read aloud: *“During this task I am going to show you a series of faces, one at a time, I would like you to try to remember them. At the end of the task, I will show you some more pictures of faces and ask you to recall which ones you have already seen”* (see [Appendix L](#) for task instruction screens). Approximately 45 seconds later (i.e., at the conclusion of the acquisition phase) the immediate recall phase began, which comprised a second series of 14 faces, including 7 target faces presented during acquisition and 7 new non-target (distractor) images (see [Figure 4.1](#) for examples of target and non-target distractor stimuli). The following instructions were displayed at the start of this phase and read aloud: *“Now I am going to show you some more pictures of faces, one at a time. I want you to look at each face carefully. Say YES if the face is one that I asked you to remember or NO if it is not”*. Approximately 25 minutes later, at the delayed recall phase, a third series of 14 faces were presented which included the remaining 7 target faces presented at acquisition and 7 new non-target (distractor)

images. Face recognition accuracy and visual scan-path performance was recorded concurrently. During the time delay between the immediate and delayed recall phases, participants were asked to complete a set of standardised self-report questionnaires, as detailed in Chapter 2: Section 2.5.5.

Table 4.1: *Task C trial and stimulus sequence*

Acquisition		Immediate Recall			Delayed Recall		
Trial	Target stimulus	Trial	Target stimulus	Non-Target stimulus	Trial	Target stimulus	Non-Target stimulus
1	T1	15	T11		29		N8 (T12)
2	T2	16	T13		30	T8	
3	T3	17		N1 (T2)	31		N9 (T4)
4	T4	18		N2 (T3)	32	T10	
5	T5	19		N3 (T5)	33	T2	
6	T6	20	T5		34		N10 (T14)
7	T7	21		N4 (T1)	35		N11 (T10)
8	T8	22	T3		36		N12 (T6)
9	T9	23		N5 (T7)	37	T6	
10	T10	24		N6 (T11)	38	T4	
11	T11	25		N7 (T9)	39		N13 (T13)
12	T12	26	T7		40	T14	
13	T13	27	T1		41		N14 (T8)
14	T14	28	T9		42	T12	

*Note. Values in brackets following each Non-target stimulus identify the Target stimulus with which it most shared common elements (e.g., gender, age range, facial features i.e., eye colour, hair colour/style, drawn from same stimulus set).*

Task C utilised the same identical desk, computer, chin rest configuration, standard SR Eye Link calibration, validation and eye-movement recording procedures as outlined earlier for the visual-cognitive Tasks A and B. At the commencement of each Task C phase, the first image was displayed when a participant fixated on a centrally located dot (3 cm diameter) on the computer screen for more than 1000ms.

Neutral facial expression stimuli were presented for 3000ms each (which was half the Task A presentation time, see Section 2.5.5), during which eye-movement recordings were obtained. At the immediate and delayed recall phases, a response screen was displayed after each image, which stated: “*Say YES if the face is one that I asked you to remember or NO if it is not*”. Participants were given as much time as they required to provide a verbal response, which the experimenter recorded manually before cueing the next image. The Task C elements, including the presentation of the target stimuli at acquisition, and repeat presentation of selected targets and new non-target (distractor) stimuli at immediate and delayed recall, took approximately 5 minutes in total, although the entire task was undertaken over 25-30 minutes.

Cognitive performance on the WTAR and RBANS collected prior to the visual-cognitive eye-tracking tasks was examined in relation to recall accuracy and visual scan-path performance. The total standardised (UK) score on the WTAR was utilised (Wechsler, 2001), while the RBANS was scored to obtain a mean Total Scale score providing an indication of a global level of cognitive functioning, as well as mean index scores, providing an indication of cognitive functioning in the domains of Immediate Memory; Visuospatial/Constructional abilities; Language; Attention; and Delayed Memory (Randolph et al., 1998).

#### *4.5.6 Eye movement parameters*

Full details on the scan-path parameters available for extraction have been provided in Chapter 2: Section 2.5.6. In brief, the scan-path parameters utilised for analyses in this study were: two temporal indices, the mean number of fixations (count)

and average duration of fixations (time/ms); and two spatial indices, the mean distance between fixations (mm), and overall scan-path length (mm).

#### 4.5.7 Data analysis

Data coding and analyses were undertaken using SPSS Statistical Software (Version 24.0; SPSS, Armonk, NY, USA). Multiple regression analyses with psychopathy tested as a continuous contrast were utilised to examine group differences in cognitive functioning on the WTAR and RBANS, as well as scan-path performance during the acquisition phase, representing the initial presentation of target stimuli. Pearson product-moment correlations (with two-tailed significance tests) were used to examine relationships between Task C acquisition phase scan-path indices (including the number, duration and distance between fixations, and scan-path length) and the same Task A scan-path indices for *neutral stimuli*.

Analyses of face recognition accuracy and scan-path indices by task component and group were undertaken using a Generalised Linear Model with a series of planned orthogonal contrasts. As detailed in [Table 4.2](#), eleven planned orthogonal contrasts were defined, comprising two between-group contrasts (GC1 and GC2: psychosis vs. not and low vs. high psychopathy); three within-group task-related contrasts (TC1 to TC3: non-target vs. target stimuli, delayed vs. immediate recall, and interaction); and six interaction or product contrasts (GC1 x TC1 to TC3, and GC2 x TC1 to TC3). Contrast coefficients were standardised (i.e., weighted mean of zero, and standard deviation of 1.000). Chi-square tests were also utilised to assess associations between recall accuracy and selected stimulus characteristics (e.g., whether or not a paired target or non-target distractor had occurred earlier in the stimulus block). Relationships between Task C

face recognition accuracy, cognitive functioning and scan-path indices, as well as selected Task A accuracy and scan-path indices, were also examined using Pearson product-moment correlations (involving two-tailed significance tests). As a partial control for the number of statistical tests conducted, the threshold for statistical significance was set at  $p < 0.01$  for all analyses, although statistical trends ( $p < 0.05$ ) are also noted.

Table 4.2: *Planned contrasts (Task C) – Standardised coefficients*

<i>Group Contrasts (Between-groups)</i>	Healthy Control (N = 24)	Psychosis Groups		
		Low Psychopathy (N = 18)	High Psychopathy (N = 19)	
GC1: Psychosis Vs. Not	1.240	-0.804	-0.804	
GC2: Low Vs. High Psychopathy (Continuous Contrast)	0.000	-0.099 to -2.085	0.144 to 2.154	
<i>Task Contrasts (Within-groups)</i>	Immediate Recall		Delayed Recall	
	Targets	Non-targets	Targets	Non-targets
TC1: Non-target Vs. Target	1.000	-1.000	1.000	-1.000
TC2: Delayed Vs. Immediate	1.000	1.000	-1.000	-1.000
TC3: TC1 by TC2 Interaction	1.000	-1.000	-1.000	1.000
<i>Interaction Contrasts</i>				
GC1 x TC1 to TC3 (Three Contrasts)				
GC2 x TC1 to TC3 (Three Contrasts)				

*Note. Eleven planned orthogonal contrasts were defined, comprising two between-group contrasts (GC1 and GC2), three within-group task-related contrasts (TC1 to TC3) and six interaction or product contrasts (GC1 x TC1 to TC3, and GC2 x TC1 to TC3). Contrast coefficients have been standardised (i.e., weighted mean of zero, and standard deviation of 1.000).*

## 4.6 Results

### 4.6.1 Cognitive functioning: premorbid IQ and neuropsychological functioning

As illustrated in [Table 4.3](#), multiple regression analyses examining cognitive functioning revealed no significant group differences in premorbid IQ based on performance on the Wechsler Test of Adult Reading (WTAR). However, current neurocognitive functioning on the Repeatable Battery for Assessment of Neuropsychological functioning (RBANS) was significantly lower among the psychosis group ( $t_{(58)} = 6.22$ ,  $p < 0.001$ ), with a mean total scale score of 82.11 (SD = 12.17) compared to 101.21 (SD = 10.95) among the healthy controls. In addition, all of the RBANS index subscales revealed significantly lower mean performance among the psychosis group compared to healthy controls: Immediate memory (Means: 74.22 vs. 94.75); Visual-constructional ability (Means: 100.00 vs. 111.13); Language (Means: 90.68 vs. 100.29); Attention (Means: 86.62 vs. 103.71); and Delayed Memory (Means: 81.38 vs. 96.17). While at a trend level of significance ( $t_{(58)} = -2.33$ ,  $p=0.023$ ), the psychosis - high psychopathy group exhibited increased impairment in Delayed Memory (Means: 74.11 vs. 89.06) compared with the psychosis – low psychopathy group. However, it is worthy of note that only two members of the low psychopathy group had poorer Delayed compared to Immediate Memory performance on the RBANS. As noted previously, the WTAR and RBANS total scores were also moderately correlated ( $r = 0.44$ ,  $p < 0.001$ ).

Table 4.3: *Cognitive functioning: premorbid IQ and neurocognitive functioning*

<i>Measure</i>	Healthy Control	Psychosis Groups		$R^2$	Comparisons	
	N=24 Mean (SD)	Low Psychopathy N=18 Mean (SD)	High Psychopathy N= 19 Mean (SD)		Psychosis Vs. Not	Low Vs. High Psychopathy <sup>a</sup>
Wechsler Test of Adult Reading (WTAR)						
<i>Total standardised score</i>	101.29 (11.08)	97.44 (12.39)	92.32 (17.70)	0.58	$t_{(58)}=1.77, p=0.082$	$t_{(58)}=-0.62, p=0.535$
Repeatable Battery for Assessment of Neuropsychological Status (RBANS)						
<i>Immediate memory</i>	94.75 (13.92)	77.22 (13.91)	71.37 (14.45)	0.36	$t_{(58)}=5.54, p<0.001^{**}$	$t_{(58)}=-1.08, p=0.284$
<i>Visual-constructional</i>	111.13 (11.26)	99.22 (15.65)	100.74 (17.77)	0.13	$t_{(58)}=2.86, p<0.01^*$	$t_{(58)}=0.43, p=0.666$
<i>Language</i>	100.29 (8.70)	91.06 (11.06)	90.32 (12.09)	0.17	$t_{(58)}=3.48, p<0.001^{**}$	$t_{(58)}=0.26, p=0.796$
<i>Attention</i>	103.71 (12.64)	88.83 (16.17)	84.53 (14.89)	0.26	$t_{(58)}=4.48, p<0.001^{**}$	$t_{(58)}=-0.31, p=0.758$
<i>Delayed Memory</i>	96.17 (12.76)	89.06 (12.30)	74.11 (18.89)	0.25	$t_{(58)}=3.69, p=0.001^{**}$	$t_{(58)}=-2.33, p=0.023^{\#}$
<i>Total scale score</i>	101.21 (10.95)	85.11 (11.90)	79.26 (12.03)	0.41	$t_{(58)}=6.22, p<0.001^{**}$	$t_{(58)}=0.94, p=0.350$

Note. Low Psychopathy < 11 on PCL: SV; <sup>a</sup> tested as a continuous contrast within a multiple regression analysis; <sup>#</sup> trend ( $p < 0.05$ ); \*  $p < 0.01$ ; \*\*  $p < 0.001$

## 4.6.2 Face recognition performance – Task C

### 4.6.2.1 Acquisition phase: scan-path performance on initial presentation of targets

During the acquisition phase of Task C participants were instructed to remember a series of target faces displaying neutral expressions. Multiple regression analyses examining scan-path performance across groups, as shown in [Table 4.4](#), revealed no significant group differences during the acquisition phase on either the temporal or the spatial indices. As previously reported for tasks A and B, and displayed in [Table 4.5](#), scan-paths during the acquisition phase demonstrated the same strong inverse relationship between the number and duration of fixations ( $r = -0.85$ ,  $p < 0.001$ ), as well as a strong association for the distance between fixations and the overall scan-path length ( $r = 0.88$ ,  $p < 0.001$ ). Scan-path length was also moderately correlated with the number ( $r = 0.47$ ,  $p < 0.001$ ) and duration of fixations ( $r = -0.58$ ,  $p < 0.001$ ). When comparing scan-paths indices obtained during the acquisition phase for Task C and during emotion recognition of neutral stimuli in Task A, eye-tracking performance on all four scan-path indices were significantly related (see the right hand columns of Table 4.5). This included moderate associations for: the number ( $r = 0.64$ ,  $p < 0.001$ ) and duration ( $r = 0.50$ ,  $p < 0.001$ ) of fixations; the distance between fixations ( $r = 0.45$ ,  $p < 0.001$ ); and scan-path length ( $r = 0.59$ ,  $p < 0.001$ ); indicative of a reasonably similar or consistent pattern of eye-tracking strategies across tasks.

### 4.6.2.2 Face recognition accuracy

Analyses of face recognition accuracy were undertaken using a Generalised Linear

Table 4.4: Acquisition phase (Task C): Scan-path performance during the initial presentation of target stimuli

Scan-path indices	Healthy Control	Psychosis Groups		R <sup>2</sup>	Comparisons	
	N=24 Mean (SD)	Low Psychopathy N=18 Mean (SD)	High Psychopathy N= 19 Mean (SD)		Psychosis Vs. Not	Low Vs. High Psychopathy <sup>a</sup>
Acquisition phase						
Fixation count	9.39 (1.77)	8.65 (2.16)	9.36 (1.75)	0.02	t <sub>(58)</sub> =0.78,p=0.441	t <sub>(58)</sub> =0.81,p=0.420
Fixation duration (ms)	322.67 (111.47)	362.65 (141.95)	299.60 (56.83)	0.04	t <sub>(58)</sub> =-0.29,p=0.772	t <sub>(58)</sub> =-1.46,p=0.150
Distance between fixations (mm)	37.66 (11.36)	44.32 (47.89)	37.47 (18.94)	0.06	t <sub>(58)</sub> =-0.44,p=0.661	t <sub>(58)</sub> =-1.79,p=0.079
Scan-path length (mm)	318.48 (148.59)	299.68 (235.01)	303.78 (164.86)	0.02	t <sub>(58)</sub> =0.35,p=0.729	t <sub>(58)</sub> =-0.95,p=0.348

Note. Low Psychopathy < 11 on PCL: SV; <sup>a</sup> tested as a continuous contrast within a multiple regression analysis; # trend ( $p < 0.05$ ); \*  $p < 0.01$ ; \*\*  $p < 0.001$ .

Table 4.5: Acquisition phase: correlations between Task C and selected Task A scan-path indices for neutral stimuli (N = 60 participants)

Task index	Task C Fixation duration	Task C Distance between Fixations	Task C Scan-path length	Task A -Neutral Number of fixations	Task A -Neutral Fixation duration	Task A - Neutral Distance between fixations	Task A - Neutral Scan-path length
Task C Number of fixations	-0.85 (<0.001)**	0.10 (0.442)	0.47 (<0.001)**	0.64 (<0.001)**	-0.59 (<0.001)**	-0.08 (0.525)	0.26 (0.045)#
Task C Fixation duration		-0.32 (0.014)#	-0.58 (<0.001)**	-0.44 (<0.001)**	0.50 (<0.001)**	0.16 (0.216)	-0.14 (0.287)
Task C Distance between fixations			0.88 (<0.001)**	0.31 (0.018)#	-0.38 (0.003)*	0.45 (<0.001)**	0.55 (<0.001)**
Task C Scan-path length				0.43 (<0.001)**	-0.51 (<0.001)**	0.36 (0.005)*	0.59 (<0.001)**

Note. Pearson product-moment correlations, with two-tailed p values in brackets; # trend ( $p < 0.05$ ); \*  $p < 0.01$ ; \*\*  $p < 0.001$ .

Model with a series of planned orthogonal contrasts. As detailed in [Table 4.6](#), there was a trend level significant group main effect for recognition accuracy (GC1:  $W^2 = 5.56$ ,  $p = 0.018$ ). Overall, healthy controls tended to be more accurate at recognising faces than the psychosis group (Means = 79.91 vs. 75.48). Main effects for task components revealed that accuracy for faces was significantly higher for Target compared to Non-Target stimuli (TC1:  $W^2 = 33.95$ ,  $p < 0.001$ ; Means = 84.78 vs. 69.67). Accuracy was also significantly higher at Immediate compared to Delayed recall (TC2:  $W^2 = 65.27$ ,  $p < 0.001$ ; Means = 83.84 vs. 70.61), irrespective of group.

Trend level group by task interaction contrasts revealed that healthy controls tended to differ from the psychosis group on performance for Target compared to Non-Target stimuli (GC1 by TC1:  $W^2 = 5.26$ ,  $p = 0.022$ ). Healthy controls were more accurate than the psychosis group when the face was a target (Means = 91.07 vs. 80.70) but not when the face was a Non-Target (Means = 68.75 vs. 70.27), with a mean drop in accuracy of 22.32% compared to only a 10.42% drop among the psychosis group. A group by task interaction at trend level significance (GC1 by TC3:  $W^2 = 5.53$ ,  $p = 0.019$ ) was also apparent, revealing that the magnitude of the group performance differences for Target and Non-Target stimuli were not the same for Immediate and Delayed recall. Among the psychosis group, performance differences revealed accuracy between Target vs. Non-Target stimuli was more marked at Immediate Recall (Means: Immediate = 13.90 vs Delayed = 6.95), while for the healthy controls performance accuracy between Targets and Non-Targets was more marked in the Delayed Recall condition (Means: Immediate = 16.67 vs Delayed = 27.98). No performance differences were apparent for low versus high psychopathy.

Table 4.6: Face recognition accuracy (Task C) – Mean (SD) percent correct by task component and group, and associated analyses

Group		Face recognition accuracy – Mean (SD) percent correct						Overall	
		Immediate Recall			Delayed Recall				
		Targets	Non-targets	Aggregate	Targets	Non-targets	Aggregate		
Healthy Control (N = 24)		96.43 (8.68)	79.76 (13.27)	88.09 (8.34)	85.71 (16.32)	57.74 (19.97)	71.73 (9.76)	79.91 (7.13)	
Psychosis - Low Psychopathy (N = 18)		86.51 (11.46)	72.22 (15.08)	79.97 (6.23)	75.40 (19.48)	64.29 (16.43)	69.84 (11.64)	74.60 (7.13)	
Psychosis - High Psychopathy (N = 19)		89.47 (11.51)	75.94 (22.36)	82.33 (13.12)	71.43 (15.06)	68.42 (21.07)	69.92 (9.40)	76.32 (8.27)	
<u>Group contrasts</u> (Between-groups)		<u>Task Contrasts</u> (Within-groups)		<u>Task main effect contrasts</u>		<u>GC1 by TC1 to TC3 interaction contrasts</u>		<u>GC2 by TC1 to TC3 interaction contrasts</u>	
				Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not		TC1: Non-target Vs. Target		7.57	33.95 (<0.001)**	2.91	5.26 (0.022)#	-0.58	0.18 (0.672)
Beta	W <sup>2</sup> (p)	TC2: Delayed Vs. Immediate		6.63	65.27 (<0.001)**	1.27	2.62 (0.106)	-0.30	0.13 (0.715)
2.18	5.56 (0.018)#	TC3: TC1 by TC2 Interaction		-0.06	0.00 (0.952)	-2.24	5.53 (0.019)#	-0.33	0.18 (0.668)
GC2: Low Vs. High Psychopathy (Continuous Contrast)									
Beta	W <sup>2</sup> (p)								
1.09	1.89 (0.169)								

Note. Grand Mean (SD) [N = 61] = 77.22 (7.72). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 11 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Facial Recognition Accuracy (N = 61 participants, across 4 task components): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Given the extent to which misidentification of non-target stimuli impacted face recognition performance, particularly in the Delayed Recall condition, further exploration of accuracy for individual target and non-target distractor pairs was undertaken. As shown in [Figure 4.2](#) (Immediate Recall) and [Figure 4.3](#) (Delayed Recall), participants were generally less accurate at delayed compared to immediate recall. Reduced accuracy for non-target distractor stimuli was more marked among healthy controls in the delayed recall condition. The four Target stimuli (T7, T9, T6, T12) with the poorest accuracy performance within their respective sets, all had a Non-

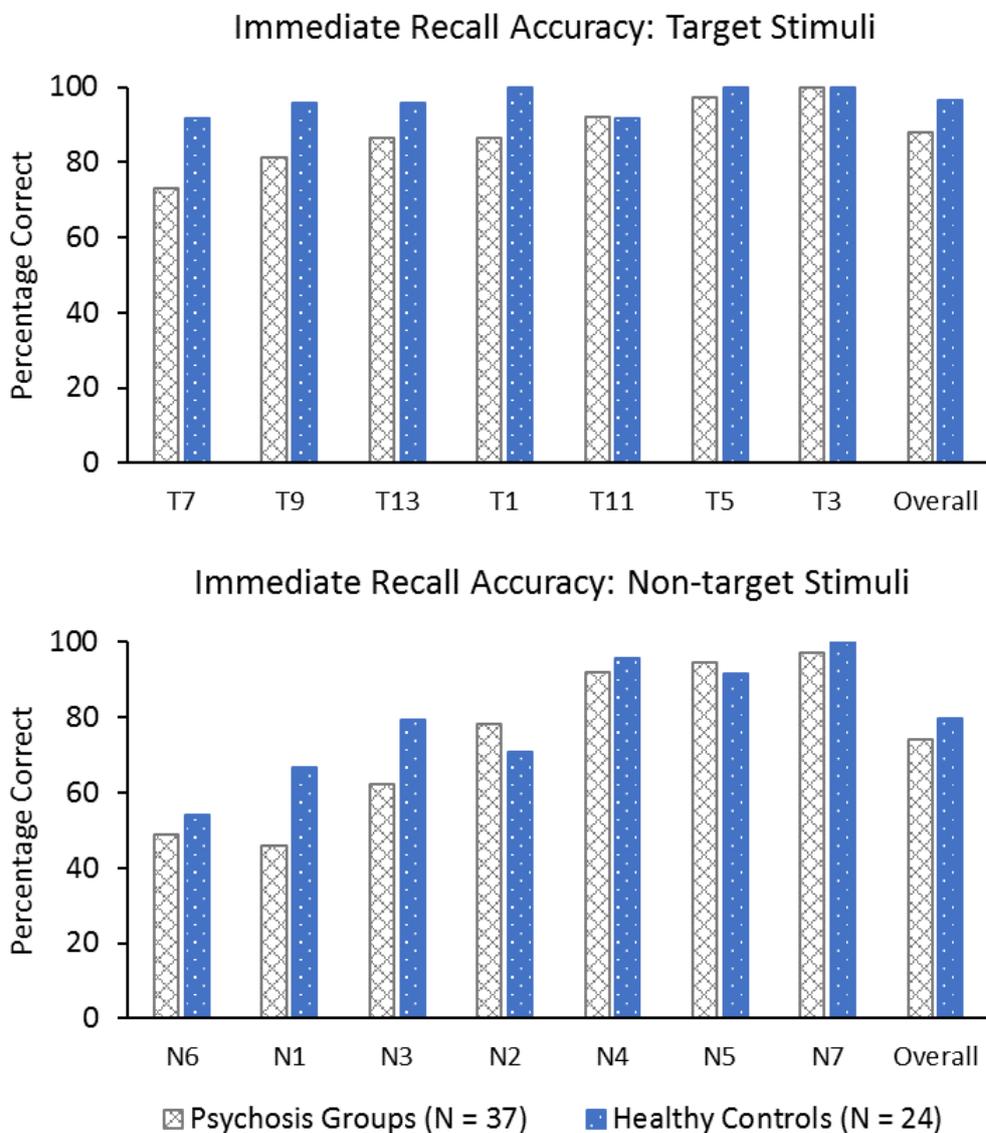


Figure 4.2 Immediate recall accuracy for individual target and non-target stimuli (Task C) for psychosis groups (N = 37) and healthy controls (N = 24)

Target distractor stimuli (pair) presented earlier in the same stimulus recall set.

All Target and Non-Target stimuli pairs were selected from the same source (i.e., NimStim, Ekman, or CBMHR, see Section 4.5.3). Consequently, some non-central or peripheral visible features may have influenced misidentification errors for Non-Target stimuli. For example, The Non-Target stimuli with poorer accuracy scores (N6, N1, N9, N10, N11) may have shared elements, such as identical shirts, t-shirts or capes (see [Figure 4.1](#)), potentially contributing to increased (misidentification) error rates.

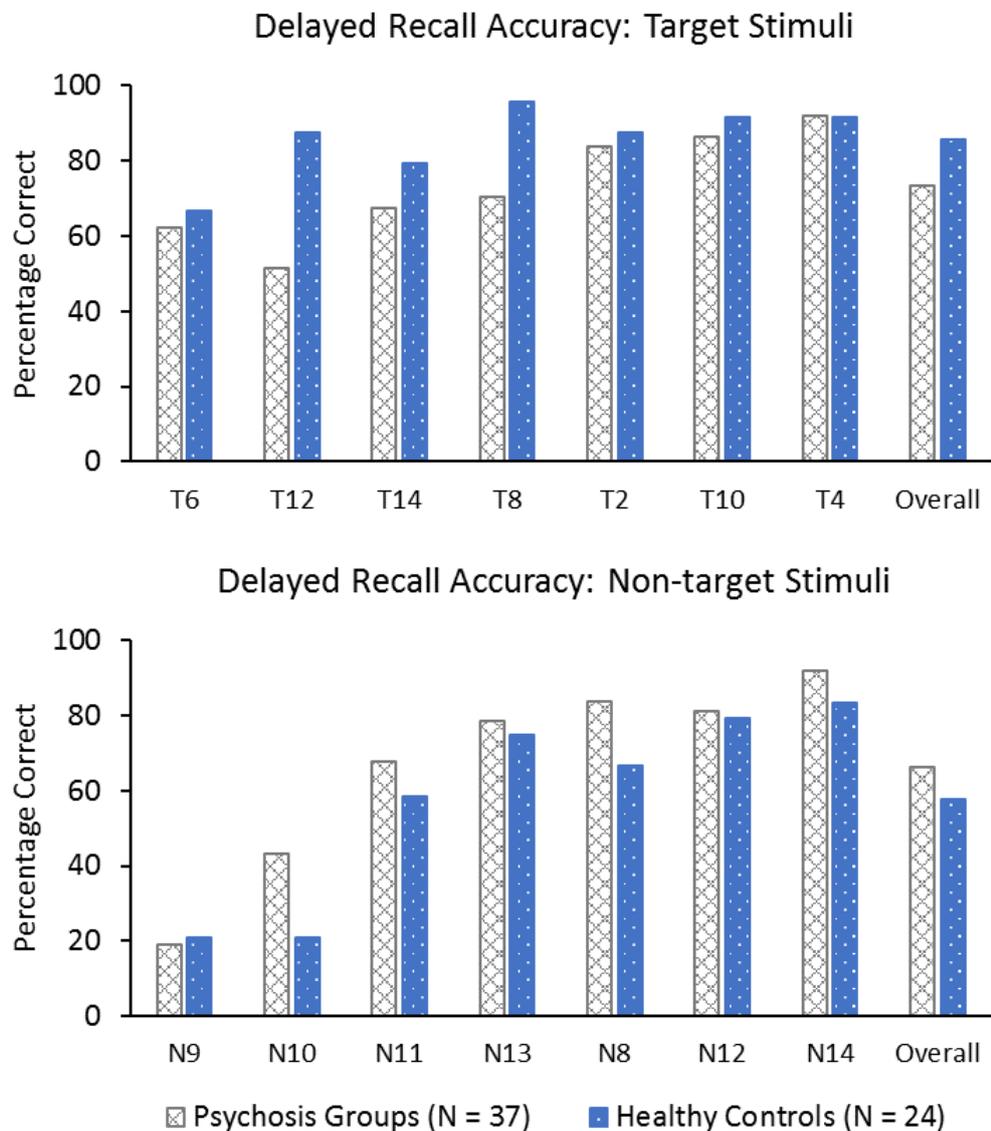


Figure 4.3 Delayed recall accuracy for individual target and non-target stimuli (Task C) for psychosis groups (N = 37) and healthy controls (N = 24)

#### 4.6.2.3 *The influence of within set distraction on recall accuracy*

To further examine the influence of potential distraction factors in Task C, all stimuli were classified by whether or not they had a paired target or non-target distractor earlier in the same stimulus block. Twelve of the 28 stimuli had such potential distraction influences: Immediate Recall – Targets (T5, T3, T7, T1, T9); Immediate Recall – Non-targets (N6); Delayed Recall – Targets (T6, T4, T14, T12); Delayed Recall – Non-targets (N11, N14). As illustrated in [Figure 4.4](#), overall accuracy rates were not impacted by distraction status within the Immediate Recall – Targets set (No earlier pair: 91.0% accuracy; Yes, paired stimulus earlier in set: 91.5% accuracy;  $\chi^2 = 0.03$ ,  $p = 0.870$ ). Within the Immediate Recall – Non-targets set (No: 80.6%; Yes: 50.8%;  $\chi^2 = 25.68$ ,  $p < 0.001$ ) and the Delayed Recall – Targets set (No: 84.7%; Yes: 73.4%;  $\chi^2 = 7.89$ ,  $p = 0.005$ ) having an earlier potential distraction stimulus within the set was associated with lower accuracy. On the other hand, non-targets within the final set were relatively more likely to have been viewed as targets (i.e., wrongly identified) when there was no earlier distractor, but likely to have been correctly rejected when their paired target stimulus had already been seen and could be ruled out of consideration (No: 57.7%; Yes: 76.5%;  $\chi^2 = 12.83$ ,  $p < 0.001$ ). Although overall accuracy was lower among the psychosis groups, in broad terms, both the healthy controls and the psychosis groups were influenced in similar ways by potential distraction effects.

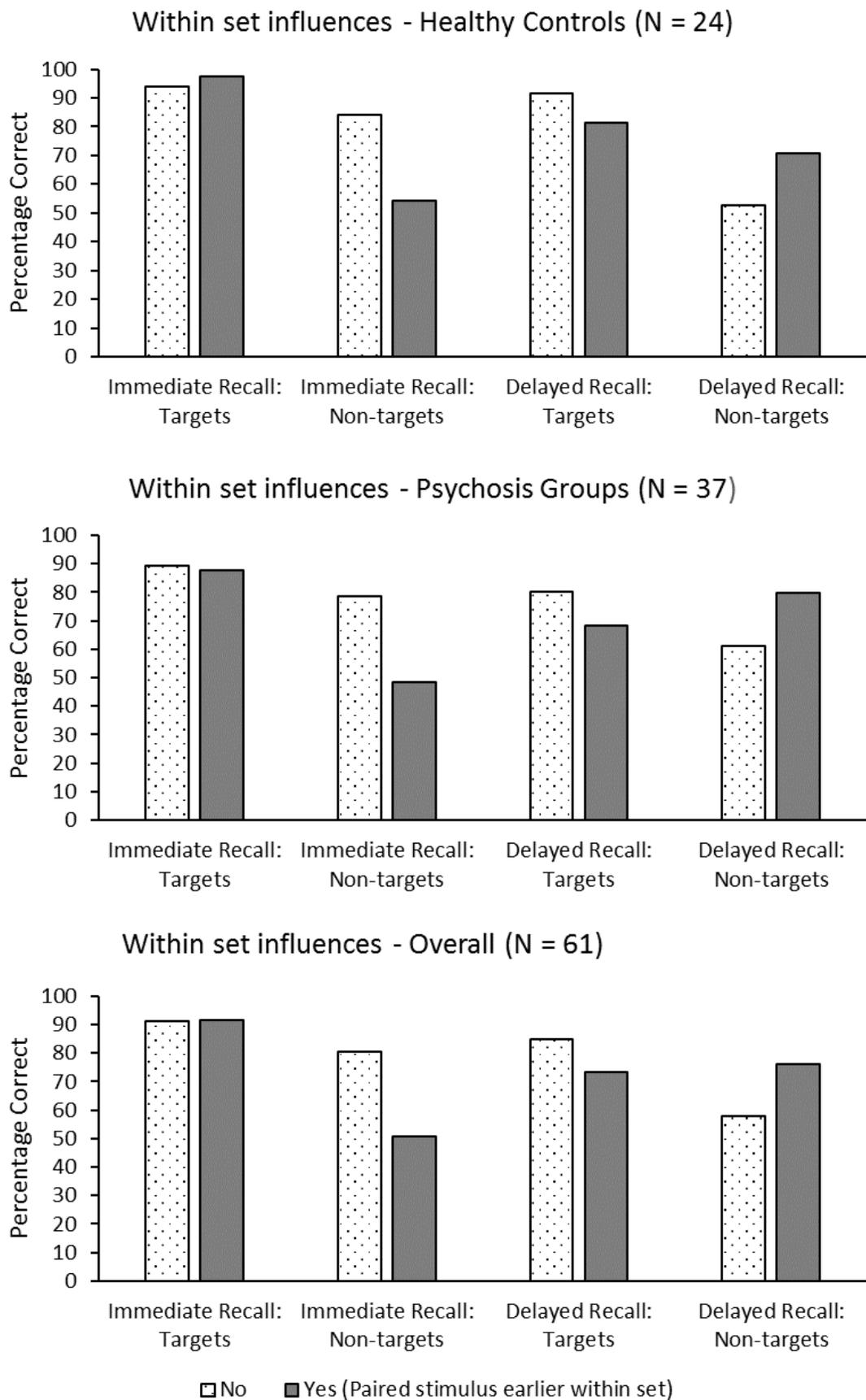


Figure 4.4 Influence of within set distractors on Recall accuracy for target and non-target stimuli (Task C) for psychosis groups (N = 37) and healthy controls (N = 24)

#### 4.6.2.4 Relationships between face recognition accuracy and cognitive functioning

As shown in [Table 4.7](#), analyses of relationships between neurocognitive functioning and Task C performance revealed that improved accuracy during the Immediate recall phase was associated with Visuospatial Constructional ability ( $r = 0.40$ ,  $p < 0.001$ ), as well as Immediate Memory ( $r = 0.26$ ,  $p = 0.046$ ), and Total RBANS Scale score ( $r = 0.25$ ,  $p = 0.049$ ). Better performance for Non-targets in the Immediate Recall phase was associated with higher Visuospatial Constructional ability ( $r = 0.37$ ,  $p = 0.003$ ), and consequently with overall accuracy. On the other hand, several RBANS subscale scores were associated with poorer performance for Non-targets in the Delayed Recall phase. Most notably, higher Attention scores ( $r = -0.36$ ,  $p = 0.005$ ), were associated with poorer performance. Likewise, higher Total RBANS Scale scores ( $r = -0.29$ ,  $p = 0.022$ ), Language ( $r = -0.28$ ,  $p = 0.028$ ) and Visuospatial Constructional ability scores ( $r = -0.26$ ,  $p = 0.044$ ) were associated with poorer performance during the Delayed Recall task for Non-target images, suggesting that these neurocognitive abilities contributed to misidentification of Non-Target stimuli. The differential correlations between Target and Non-Target stimuli in the Delayed Recall Phase are reflective of the inverse correlation between recall performance for Target and Non-target stimuli in this phase ( $r = -0.42$ ,  $p < 0.001$ ), whereas there was no corresponding association during the Immediate Recall phase ( $r = -0.01$ ,  $p = 0.971$ ).

Correlations examining overall accuracy for Task C (last column in [Table 4.7](#)) suggest that higher Immediate Memory scores on the RBANS ( $r = 0.26$ ,  $p = 0.042$ ), particularly at Immediate Recall, and premorbid IQ on the WTAR ( $r = 0.27$ ,  $p = 0.038$ ) were associated with improved performance (particularly for Targets at Delayed Recall). In addition, analyses of relationships between face recognition accuracy in Task

Table 4.7: Correlations between Task C accuracy, neurocognitive functioning, & Task A accuracy for neutral stimuli (N = 61 participants).

Task Index	Immediate Recall Accuracy			Delayed Recall Accuracy			Overall
	Targets	Non-targets	Aggregate	Targets	Non-targets	Aggregate	
WTAR	0.07	0.10	0.15	0.27	0.05	0.28	0.27
Total Standard Score	(0.591)	(0.446)	(0.241)	(0.038) <sup>#</sup>	(0.702)	(0.027) <sup>#</sup>	(0.038) <sup>#</sup>
RBANS	0.09	0.23	0.26	0.25	-0.06	0.15	0.26
Immediate Memory	(0.473)	(0.074)	(0.046) <sup>#</sup>	(0.057)	(0.623)	(0.238)	(0.042) <sup>#</sup>
RBANS	0.15	0.37	0.40	0.19	-0.26	-0.09	0.20
Visuospatial Constructional	(0.242)	(0.003) <sup>*</sup>	(<0.001) <sup>**</sup>	(0.151)	(0.044) <sup>#</sup>	(0.508)	(0.116)
RBANS	0.10	-0.03	0.01	0.12	-0.28	-0.17	-0.09
Language	(0.464)	(0.811)	(0.924)	(0.365)	(0.028) <sup>#</sup>	(0.193)	(0.473)
RBANS	0.16	0.07	0.15	0.19	-0.36	-0.18	-0.02
Attention	(0.217)	(0.617)	(0.250)	(0.142)	(0.005) <sup>*</sup>	(0.165)	(0.856)
RBANS	0.12	0.03	0.09	0.22	-0.13	0.07	0.10
Delayed Memory	(0.377)	(0.830)	(0.501)	(0.087)	(0.303)	(0.619)	(0.445)
RBANS	0.16	0.19	0.25	0.27	-0.29	-0.05	0.13
Total Scale Score	(0.205)	(0.150)	(0.049) <sup>#</sup>	(0.035) <sup>#</sup>	(0.022) <sup>#</sup>	(0.733)	(0.306)
Task A – Neutral emotion recognition Accuracy	0.42 (<0.001) <sup>**</sup>	-0.02 (0.881)	0.21 (0.105)	0.25 (0.049) <sup>#</sup>	-0.21 (0.109)	0.02 (0.864)	0.15 (0.236)

Note. Pearson product-moment correlations, with two-tailed p values in brackets; <sup>#</sup> trend ( $p < 0.05$ ); <sup>\*</sup>  $p < 0.01$ ; <sup>\*\*</sup>  $p < 0.001$ .

C and neutral emotion recognition accuracy in Task A, revealed Task A accuracy was associated with Task C recall accuracy for Targets at Immediate ( $r = 0.42, p < 0.001$ ) and delayed recall ( $r = 0.25, p < 0.049$ ). Higher RBANS Total Scale scores ( $r = 0.28, p = 0.029$ ) and Language scores ( $r = 0.28, p = 0.031$ ) were also associated with increased Task A emotion recognition accuracy for *neutral stimuli*.

#### 4.6.2.5 Recall phase (Task C): scan-path performance for temporal indices

Scan-path performance for the two temporal parameters extracted during the recall phase of the face recognition Task C are detailed in [Tables 4.8](#) and [4.9](#). Similar Generalised Linear Model analyses to those undertaken for accuracy, involving the same set of eleven planned contrasts, revealed no significant overall group differences for either the number or duration of fixations. There were significant main effects for task on both scan-path indices when processing Target versus Non-Target stimuli. Irrespective of group, there were significantly fewer fixations for Target compared to Non-Target stimuli (Table 4.8, TC1:  $W^2 = 8.82, p = 0.003$ ; Means = 9.41 vs. 9.73); and longer durations per fixation (Table 4.9, TC1:  $W^2 = 7.37, p = 0.007$ ; Means = 288.95ms vs. 277.09ms).

#### 4.6.2.6 Recall phase (Task C): scan-path performance for spatial indices

Generalised Linear Model analyses of scan-path performance on the two spatial parameters extracted during the recall phase of the face recognition Task C, which comprised the mean distance between fixations and total scan-path length, are detailed in [Tables 4.10](#) and [4.11](#). There were no group, task component differences, or interaction effects for these scan-path indices in the recall phase data.

Table 4.8: Fixation counts (Task C) – Mean (SD) number of fixations by task component and group, and associated analyses

Group	Fixation counts – Mean (SD) number of fixations							
	Immediate Recall			Delayed Recall			Overall	
	Targets	Non-targets	Aggregate	Targets	Non-targets	Aggregate		
Healthy Control (N = 24)	9.59 (2.20)	10.27 (1.89)	9.93 (1.95)	9.69 (1.70)	10.10 (1.47)	9.89 (1.49)	9.91 (1.56)	
Psychosis - Low Psychopathy (N = 18)	9.34 (2.49)	9.42 (2.78)	9.38 (2.59)	9.13 (2.36)	9.38 (2.37)	9.25 (2.32)	9.32 (2.18)	
Psychosis - High Psychopathy (N = 19)	9.15 (1.78)	9.44 (1.70)	9.29 (1.67)	9.41 (2.09)	9.52 (1.95)	9.47 (1.95)	9.38 (1.54)	
<i>Group contrasts</i> (Between-groups)	<i>Task Contrasts</i> (Within-groups)	<i>Task main effect contrasts</i>		<i>GC1 by TC1 to TC3 interaction contrasts</i>		<i>GC2 by TC1 to TC3 interaction contrasts</i>		
		Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	
GC1: Psychosis Vs. Not	TC1: Non-target Vs. Target	-0.16	8.82 (0.003)*	-0.09	2.37 (0.124)	-0.02	0.14 (0.708)	
	Beta	W <sup>2</sup> (p)	0.00	0.00 (0.997)	0.02	0.02 (0.884)	-0.21	2.73 (0.098)
	0.28	1.70 (0.193)	TC3: TC1 by TC2 Interaction	-0.03	0.53 (0.466)	-0.03	0.75 (0.386)	-0.02
GC2: Low Vs. High Psychopathy (Continuous Contrast)	Beta	W <sup>2</sup> (p)						
	0.07	0.06 (0.800)						

Note. Grand Mean (SD) [N = 61] = 9.57 (1.75). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 11 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Fixation Counts (N = 61 participants, across 4 task components): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.



Table 4.10: Distance between fixations (Task C) – Mean (SD) distance (mm) by task component and group, and associated analyses

Group	Distance between fixations – Mean (SD) distance (mm)							
	Immediate Recall			Delayed Recall			Overall	
	Targets	Non-targets	Aggregate	Targets	Non-targets	Aggregate		
Healthy Control (N = 24)	48.40 (26.02)	50.09 (24.78)	49.24 (25.25)	42.78 (10.29)	43.57 (11.79)	43.18 (10.50)	46.21 (16.65)	
Psychosis - Low Psychopathy (N = 18)	44.99 (18.02)	52.01 (26.82)	48.50 (22.16)	44.01 (15.93)	48.55 (24.94)	46.28 (19.03)	47.39 (17.03)	
Psychosis - High Psychopathy (N = 18)	58.70 (85.21)	50.38 (37.91)	54.54 (61.16)	53.15 (50.67)	43.55 (18.71)	48.35 (33.71)	51.44 (46.94)	
<u>Group contrasts</u> (Between-groups)	<u>Task Contrasts</u> (Within-groups)		<u>Task main effect contrasts</u>		<u>GC1 by TC1 to TC3 interaction contrasts</u>		<u>GC2 by TC1 to TC3 interaction contrasts</u>	
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not		TC1: Non-target Vs. Target	0.31	0.04 (0.843)	-0.75	0.34 (0.558)	3.24	1.81 (0.178)
Beta	W <sup>2</sup> (p)	TC2: Delayed Vs. Immediate	2.48	2.49 (0.114)	0.45	0.10 (0.757)	0.16	0.01 (0.934)
-1.62	0.24 (0.625)	TC3: TC1 by TC2 Interaction	-0.37	0.58 (0.445)	0.11	0.08 (0.783)	0.42	0.54 (0.464)
GC2: Low Vs. High Psychopathy (Continuous Contrast)								
Beta	W <sup>2</sup> (p)							
2.32	0.18 (0.674)							

Note. Grand Mean (SD) [N = 60] = 48.13 (28.84). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 11 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Distance between Fixations (N = 60 participants, across 4 task components): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

Table 4.11: Scan-path length (Task C) – Mean (SD) total scan-path length (mm) by task component and group, and associated analyses

Group	Scan-path length – Mean (SD) total scan-path length (mm)							
	Immediate Recall			Delayed Recall			Overall	
	Targets	Non-targets	Aggregate	Targets	Non-targets	Aggregate		
Healthy Control (N = 24)	412.87 (317.32)	453.36 (305.27)	433.12 (309.58)	368.32 (134.25)	393.47 (124.64)	380.89 (125.69)	407.01 (202.70)	
Psychosis - Low Psychopathy (N = 18)	354.56 (173.35)	411.56 (256.09)	383.06 (210.13)	330.12 (127.41)	391.36 (256.25)	360.74 (181.10)	371.90 (161.36)	
Psychosis - High Psychopathy (N = 18)	351.50 (223.11)	382.07 (209.82)	366.78 (212.76)	411.02 (394.91)	353.56 (179.15)	382.29 (279.58)	374.54 (244.26)	
<u>Group contrasts</u> (Between-groups)	<u>Task Contrasts</u> (Within-groups)		<u>Task main effect contrasts</u>		<u>GC1 by TC1 to TC3 interaction contrasts</u>		<u>GC2 by TC1 to TC3 interaction contrasts</u>	
			Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)	Beta	W <sup>2</sup> (p)
GC1: Psychosis Vs. Not	TC1: Non-target Vs. Target		-13.13	3.62 (0.057)	-2.67	0.20 (0.652)	13.03	2.05 (0.152)
Beta	W <sup>2</sup> (p)	TC2: Delayed Vs. Immediate	10.93	0.77 (0.380)	12.28	0.83 (0.363)	-18.64	3.23 (0.072)
16.50	0.40 (0.526)	TC3: TC1 by TC2 Interaction	-8.02	1.97 (0.161)	3.37	0.50 (0.480)	-6.48	0.71 (0.401)
GC2: Low Vs. High Psychopathy (Continuous Contrast)								
Beta	W <sup>2</sup> (p)							
8.44	0.07 (0.786)							

Note. Grand Mean (SD) [N = 60] = 386.73 (202.46). Regression weights (Betas), Wald Chi-squares (W<sup>2</sup>) and associated p values are reported for the 11 predictors (orthogonal contrasts) included in the Generalised Linear Model analysis for Scan-path Length (N = 60 participants, across 4 task components): # trend (p < 0.05); \* p < 0.01; \*\* p < 0.001.

#### 4.6.2.7 Relationships between recall correctness and scan-path performance

For Target stimuli, as displayed in [Table 4.12](#), scan-path indices from the recall phase were not associated with recall accuracy, suggesting that at recall scanning was less purposeful or perhaps subsidiary. However, acquisition scan-path indices indicate that more fixations ( $r = 0.17, p < 0.001$ ), of shorter duration ( $r = -0.13, p < 0.001$ ), lead to increased recall accuracy for targets. The same pattern of associations between scan-path parameters at acquisition and recall phases were apparent (see Table 4.12). This included: the strong inverse relationship between number and duration of fixations (acquisition,  $r = -0.74, p < 0.001$ ; delayed,  $r = -0.79, p < 0.001$ ); and distance between fixations and scan-path length (acquisition,  $r = 0.66, p < 0.001$ ; delayed,  $r = 0.76, p < 0.001$ ); which were already noted previously in the visual-cognitive eye-tracking tasks. In addition, significant correlations between the same scan-path indices at acquisition and recall, ranging from  $r = 0.16$  to  $0.37$ , indicate that reasonably consistent scanning strategies were utilised.

Interestingly, as displayed in [Table 4.13](#), Non-Target stimuli appear to be tracked in a relatively similar way to their paired targets, with the patterns of association between the scanning indices from the acquisition and recall phases being very comparable in both the target and non-target analyses. As expected, there was a lack of crossover associations with acquisition and recall accuracy, given that the Non-Target stimuli were different and had no relationship to the Target. Reflective of uncertainty, increased errors for Non-Targets co-occurred with a tendency to have increased fixations.

Table 4.12: Correlations between Task C scan-path indices for targets (854 measures,  $N = 61$  participants, across 14 target stimuli).

Task Index	Acquisition Phase			Recall Phase				
	Fixation Duration	Distance between Fixations	Scan-path Length	Number of Fixations	Fixation Duration	Distance between Fixations	Scan-path Length	Recall Correctness
Acquisition: Number of fixations	-0.74 ( $<0.001$ )**	0.10 (0.003)*	0.41 ( $<0.001$ )**	0.37 ( $<0.001$ )**	-0.32 ( $<0.001$ )**	0.04 (0.260)	0.15 ( $<0.001$ )**	0.17 ( $<0.001$ )**
Acquisition: Fixation duration		-0.17 ( $<0.001$ )**	-0.38 ( $<0.001$ )**	-0.22 ( $<0.001$ )**	0.23 ( $<0.001$ )**	-0.05 (0.180)	-0.10 (0.006)*	-0.13 ( $<0.001$ )**
Acquisition: Distance between fixations			0.66 ( $<0.001$ )**	-0.01 (0.841)	-0.07 (0.062)	0.03 (0.353)	0.012 (0.735)	0.03 (0.366)
Acquisition: Scan-path length				0.12 (0.001)*	-0.22 ( $<0.001$ )**	0.12 (0.001)*	0.16 ( $<0.001$ )**	0.08 (0.025)#
Recall: Number of fixations					-0.79 ( $<0.001$ )**	-0.03 (0.340)	0.38 ( $<0.001$ )**	0.01 (0.737)
Recall: Fixation duration						-0.17 ( $<0.001$ )**	-0.42 ( $<0.001$ )**	0.00 (0.991)
Recall: Distance between fixations							0.76 ( $<0.001$ )**	0.00 (0.948)
Recall: Scan-path length								-0.02 (0.650)

Note. Pearson product-moment correlations, with two-tailed  $p$  values in brackets; # trend ( $p < 0.05$ ); \*  $p < 0.01$ ; \*\*  $p < 0.001$ .

Table 4.13: *Correlations between Task C scan-path indices for non-targets and with acquisition indices for their paired target stimuli (854 measures, N = 61 participants, across 14 non-target stimuli).*

<i>Task Index</i>	Recall Phase				
	Number of Fixations	Fixation Duration	Distance between Fixations	Scan-path Length	Recall Correctness
Acquisition – Paired target stimulus: Number of fixations	0.39 ( $<0.001$ )**	-0.39 ( $<0.001$ )**	0.07 (0.034) <sup>#</sup>	0.22 ( $<0.001$ )**	0.00 (0.990)
Acquisition – Paired target stimulus: Fixation duration	-0.23 ( $<0.001$ )**	0.28 ( $<0.001$ )**	-0.09 (0.010) <sup>#</sup>	-0.16 ( $<0.001$ )**	-0.02 (0.482)
Acquisition – Paired target stimulus: Distance between fixations	0.01 (0.839)	-0.12 (0.001)*	0.11 (0.002)*	0.06 (0.094)	0.06 (0.118)
Acquisition – Paired target stimulus: Scan-path length	0.15 ( $<0.001$ )**	-0.29 ( $<0.001$ )**	0.17 ( $<0.001$ )**	0.20 ( $<0.001$ )**	0.07 (0.056)
Recall: Number of fixations		-0.77 ( $<0.001$ )**	0.05 (0.135)	0.42 ( $<0.001$ )**	-0.08 (0.025) <sup>#</sup>
Recall: Fixation duration			-0.28 ( $<0.001$ )**	-0.49 ( $<0.001$ )**	0.02 (0.666)
Recall: Distance between fixations				0.87 ( $<0.001$ )**	0.05 (0.172)
Recall: Scan-path length					0.00 (0.939)

*Note. For these analyses, each non-target stimulus was able to be paired with a target stimulus, being the stimulus with which it most shared common elements (e.g., gender, age range, hair style, drawn from same stimulus set). Pearson product-moment correlations, with two-tailed p values in brackets; <sup>#</sup> trend ( $p < 0.05$ ); \*  $p < 0.01$ ; \*\*  $p < 0.001$ .*

## 4.7 Discussion

While there were no group differences in level of education or premorbid IQ, there were significant neurocognitive impairments on the RBANS among the psychosis group that were in accordance with the research literature. These included: deficits in memory and general neurocognitive performance (Iverson, Brooks, & Haley, 2009; Loughland et al., 2010; Loughland et al., 2007; Wilk et al., 2004); as well as in attention (Iverson et al., 2009; Loughland et al., 2010; Wilk et al., 2004), language, and visual-constructional ability (Wilk et al., 2004). The largest impairments in current functioning were for immediate and delayed memory, and overall neuropsychological functioning, which were not unlike an Australian volunteer schizophrenia register sample (Mean RBANS total score = 88.72; Loughland et al., 2007). However, the psychosis participants in this study recruited from community mental health services exhibited significant impairments across all RBANS domains, which were not dissimilar to a US normative sample of (inpatient and outpatient) health service patients (Mean RBANS total score = 70.54; Wilk et al., 2004). The RBANS total scale score (Mean = 82.11) was around 1 SD below the standardised non-clinical population (Mean = 100; Randolph et al., 1998), indicating that the neuropsychological deficits observed here fall somewhere in between these two published samples. This is also consistent with the notion that a severity/functioning gradient exists across recruitment settings (Loughland et al., 2004b), with memory impairments a core feature (Loughland et al., 2007; Rajji et al., 2009; van Os & Kapur, 2009).

The visual scanning indices provide accompanying behavioural data during the acquisition phase of the face recognition Task C, when participants were required to remember a series of 14 target faces displaying neutral expressions. Interestingly, this

revealed no apparent visual scan-path differences between the groups, which is consistent with one study finding that during active tasks (such as deciding whether a face is known or unknown) there were no significant differences in terms of temporal or spatial scan-path indices between individuals with schizophrenia or healthy controls (Delerue et al., 2010). However, this is inconsistent with previous eye tracking studies reporting evidence of atypical restricted scan-paths for emotional and neutral faces. These studies found fewer fixations of longer duration among individuals with schizophrenia during face recognition tasks (Bortolon et al., 2016; Loughland et al., 2002a; Williams et al., 1999) and while viewing emotional faces (Nikolaides et al., 2016). However, in the current study, consistent visual scanning strategies were detected, with similar relationships between the temporal and spatial eye-tracking indices to those noted in previous tasks. Further evidence of similar scanning strategies being utilised across tasks, comes from comparisons between Task C acquisition and Task A emotion recognition scan-path indices for neutral stimuli; in this instance, eye-tracking performance on each of the indices was significantly correlated, with moderate associations ranging from 0.45 to 0.64. Evidence of consistent visual scanning strategies across face and emotion recognition tasks, could also broadly be seen as supportive of models proposing, “*Integrated processing*” of facial identity and emotion (Yankouskaya et al., 2014a).

In terms of task performance at recall, and consistently with prior studies (Martin et al., 2005; She et al., 2017), the psychosis group tended to be less accurate than healthy controls at recognising faces. Irrespective of group, accuracy significantly improved for target faces, and was better at immediate recall. Furthermore, trend level interaction contrasts revealed that healthy controls were more accurate than the psychosis group when a face was a target but not when the face was a non-target. Among the psychosis

groups, the performance difference between target and non-target faces was more marked at immediate recall, while for the healthy controls this accuracy difference was more marked at delayed recall. These findings are generally consistent with previous studies, confirming moderately degraded perceptual discrimination and impaired working memory for faces, among individuals with schizophrenia in comparison to healthy controls (Chen et al., 2009).

The misidentification of non-target stimuli significantly affected recognition performance, particularly during the delayed recall condition. Face stimuli with the poorest performance accuracy, all had a non-target distractor stimuli (pair) presented earlier in the same recall stimulus set. Given that the distractor stimuli (pair) came from identical stimulus sets (CBMHR (unpublished); Ekman & Matsumoto, 1993-2004; Tottenham et al., 2009), visible similarities in peripheral non-feature areas may have contributed to overall misidentification error rates (i.e., shirt, t-shirt, cape – as can be seen in [Figure 4.1](#)). The presence of an earlier distractor stimulus within the same set significantly lowered recall accuracy for non-target faces at immediate recall, and target faces at delayed recall. Non-targets in the delayed recall condition were more likely to be wrongly identified as a target face when there was no earlier distractor but correctly rejected when the target stimulus had already been seen and ruled out. However, while overall accuracy was reduced among the psychosis groups, both the healthy control and psychosis groups appeared to have been influenced in similar ways by potential distractors. The pattern of associations obtained tended to indicate that *face processing decisions were indeed being influenced by the target stimuli* (including their more peripheral characteristics), although recall performance overall was poorer among the psychosis groups.

With respect to the impact of neurocognition on task performance, improved recognition accuracy of target faces at immediate recall was associated with better visuospatial constructional ability, immediate memory and overall neuropsychological functioning, while improved performance for non-targets (i.e., correctly discriminating) was associated with higher visuospatial constructional ability. However, the inverse relationship was apparent at delayed recall, with several neurocognitive domains associated with a decline in performance for non-target faces (including higher attention scores, overall neuropsychological functioning, language, and visuospatial constructional ability). Improved visuospatial constructional and attentional capacity significantly contributed to misidentification of non-target stimuli at delayed recall. This was likely to be due, in part, to the attention paid to the peripheral similarities of the distractor stimuli, which increased the likelihood of misidentification among both the psychosis and healthy control participants. Overall, face recognition accuracy was associated with better immediate memory and higher premorbid IQ.

In terms of immediate recall, during this face recognition task, visual working memory capacity is of particular importance. Existing research evidence demonstrates that a strong relationship exists between working memory and visual attention processes, in that what we have in mind can determine where and to which stimulus features we attend (Mannan et al., 2010). Visual working memory capacity has also previously been shown to be strongly correlated with overall cognitive ability (Luck & Vogel, 2013), with substantial differences across groups, including a reduced capacity among individuals with schizophrenia in comparison to healthy controls (Gold et al., 2003). The previously observed links between impaired visual working memory capacity and poorer task

performance are consistent with the findings from the current face recognition study, particularly during the immediate recall phase, as well as overall. With respect to potential cognitive treatment approaches among individuals with schizophrenia, these findings also support suggestions that improving or normalising visual working memory capacity could help reduce overall cognitive deficits (Luck & Vogel, 2013). Some researchers suggest that differences are reflected in storage capacity, while others indicate variations may exist in the ability to use memory capacity more efficiently (Luck & Vogel, 2013). Those with higher levels of cognitive functioning are also more likely to use improved strategies.

In addition, the current findings are consistent with the literature around episodic and working memory impairments in psychosis (Bowie & Harvey, 2005; Dickinson & Harvey, 2009; Fuller et al., 2009; Harvey & Rosenthal, 2018; Lee & Park, 2005; Loughland et al., 2007; Silver et al., 2003; Zanello et al., 2009). Overall face recognition performance was reduced among the psychosis groups, particularly at immediate recall, indicative of poorer working memory. However, the difference in performance accuracy exhibited by the psychosis groups between immediate and delayed recall was of a similar magnitude to that of the health controls. That is, rapid forgetting was not demonstrated at delayed recall, which is consistent with the prior psychosis literature (Harvey & Rosenthal, 2018), and suggests that when information on facial identity was successfully encoded it was largely able to be recalled.

When considering performance across tasks, face recognition accuracy in Task C for target faces at immediate and delayed recall was significantly associated with emotion recognition accuracy for neutral faces in Task A, suggestive of potentially similar processing strategies. Higher overall neurocognitive functioning and improved language

ability were also associated with increased emotion recognition accuracy for neutral faces in Task A.

An examination of the accompanying visual scan-path performance during the recall phases of Task C also revealed no significant group differences, on either the temporal or spatial scan-path indices. Again, this is inconsistent with prior eye-tracking face recognition studies reporting restricted scan-paths for neutral faces, involving fewer fixations of longer duration among individuals with schizophrenia (Bortolon et al., 2016; Loughland et al., 2002a; Williams et al., 1999). However, it is consistent with a study by Delerue et al. (2010), who also found no significant temporal or spatial visual scan-path differences between individuals with schizophrenia and healthy controls while undertaking an active face recognition task in contrast to undertaking a passive free viewing task. Similarly, using a visual search paradigm, She et al. (2017) found that face recognition accuracy was only impaired under certain circumstances. Their task used neutral faces, in which one face was presented as the target followed by two or four faces for 600ms, and participants needed to indicate if the target stimuli was present. Accuracy was only significantly lower among the schizophrenia participants when the set size included 2 not 4 facial stimuli (She et al., 2017).

Interestingly, and in line with these findings, a study investigating neural activity related to processing emotional and non-emotional facial information (i.e., tasks matching facial emotion, identity, and complex visual patterns) reported that neural activation patterns in schizophrenia patients and healthy controls were distinctly different while processing affect-related facial information but not for non-emotional facial features (Quintana et al., 2011). During emotion matching, orbital frontal cortex

and left amygdala activations were apparent among the healthy controls but not in the schizophrenia patients (Quintana et al., 2011). Comparisons between emotion versus identity matching revealed, activation of the fusiform and middle temporal gyri, left superior temporal gyrus, and right inferior and middle frontal gyrus among the healthy controls, while only activation in the middle and inferior frontal gyri, the frontal operculi and the right insular cortex was found in the schizophrenia patients (Quintana et al., 2011). The authors suggested that these findings imply schizophrenia patients and healthy controls may utilize different neural networks when processing facial emotional information but less so in matching facial identity (Quintana et al., 2011).

In the current study, regardless of group, significantly fewer fixations of longer duration were recorded when participants viewed target faces compared to non-targets. Visual scanning performance at recall was also not associated with face recognition accuracy. However, acquisition scan-path indices were associated, with increased fixations of shorter duration resulting in significantly improved recall accuracy for target faces. The suggestion that the acquisition scan-paths were more purposeful than the recall scan-paths is perhaps broadly consistent with literature around eye-movements towards objects already stored in visual working memory. For example, attention and gaze control can be influenced, with even simple saccades becoming faster when the current saccade target matches the contents of visual working memory (Hollingworth et al., 2013).

There was also consistency of visual scanning strategies across phases for target faces, with the same pattern of associations between scan-path indices at acquisition and recall. This included a strong inverse relationship between the number and duration of

fixations, and a strong positive association between the distance between fixations and scan-path length. In addition, non-target faces appeared to be processed in a similar way to their paired targets, with comparable patterns of association between indices at acquisition and recall. However, reflective of how participants dealt with uncertainty (given that all non-target stimuli were novel), increased errors for non-target faces co-occurred with a tendency to have increased fixations. This can also be seen to be consistent with previous research indicating visual working memory representations are compared to newly fixated objects (Hollingworth et al., 2008), and that eye-movements may be biased towards objects that match the current contents of working memory (Mannan et al., 2010). The importance of visual working memory may even extend even to basic operations of the oculomotor system (Hollingworth et al., 2013).

No atypical visual scanning differences for psychosis were apparent during the face recognition Task C. However, associations between neurocognitive impairments and both face (Task C) and emotion recognition performance (Task A), highlight the importance of undertaking comprehensive neuropsychological investigations among individuals with psychosis. This recommendation is further reinforced by the known impact of neurocognitive deficits among psychosis populations on social and role functioning (Seidman & Mirsky, 2017), coupled with promising results from targeted cognitive remediation (Hooker et al., 2014; Seidman & Mirsky, 2017) and broad-based neurocognitive interventions (Tan et al., 2018). The current study demonstrates the importance of immediate memory, in terms of visual working memory capacity during a higher order visual task, and the findings are consistent with research evidence supporting a strong relationship between working memory and visual attention processes (Mannan et al., 2010). More broadly, monitoring changes in

neuropsychological functioning is likely to have important clinical implications, not only around illness progression but also in detailing any progress following cognitive, psychological or pharmacological interventions.

Worthy of mention, while the RBANS was utilised in this study, having been extensively used in Australian schizophrenia research, currently the MATRICS would be seen as the preferred cognitive battery (Nuechterlein et al., 2008). This is largely due to the more restricted domains assessed using the RBANS. For example, as displayed in [Table 4.3](#), there was limited differentiation between the groups on the RBANS Visuospatial Constructional domain. Future research utilising the MATRICS consensus cognitive battery could potentially assist in elucidating additional findings related to visual learning, speed of processing and working memory.

## **Chapter 5 – Synthesis of key findings of theoretical and clinical relevance**

### **5.1 Introduction**

The main objective of this clinical dissertation was to extend the existing research evidence base about facial expression (emotion/face) processing deficits in individuals with psychosis, by undertaking a series of visual scanning eye-tracking tasks that examined the relative contributions of psychosis and coexisting psychopathy traits. An offender sample with psychosis was purposefully recruited, representing a group of individuals who are otherwise difficult to engage but often excluded from traditional research designs. Such psychosis subgroups are likely to have neurocognitive and comorbid complexities that are evident on presentation, to experience ongoing difficulties in social and occupational functioning, and to have poorer long-term outcomes.

### **5.2 Summary of results**

#### *5.2.1 Study 1 – Emotion recognition*

As anticipated, consistent with part one of the hypotheses, an overall impairment in emotion recognition accuracy among the psychosis group was apparent. Psychosis participants displayed significantly poorer recognition accuracy compared to healthy controls; however, there was no evidence to suggest that the impairment was emotion specific. In addition, no relationships between emotion recognition accuracy and any of the visual scan-path parameters were found; however, in partial support of the hypotheses, some atypical scan-path patterns were exhibited in Task A. For the temporal scan-path indices, psychosis participants tended to exhibit a shorter duration of fixations for positive or neutral expressions compared to negative emotions, and among those with high psychopathy traits a shorter fixation duration for fear stimuli compared

to disgust. No group differences, or interactions by emotion, were apparent for the spatial indices. However, as a measure of attentional processing, the task successfully demonstrated emotion specific differences in visual scan-path indices, irrespective of group.

Analyses of scan-paths within regions of interest revealed additional atypical scan-path results. The psychosis group tended to pay less attention to the eyes when processing happy expressions (compared to surprise) than healthy controls, with a reduced percentage of fixations to the eye region. Psychosis participants with high psychopathy traits spent significantly less time within the eye region when processing fear (compared to disgust), with a shorter duration of fixations. The psychosis group also spent significantly less time on the nose region, with shorter fixation durations when recognising fear (compared to disgust) than healthy controls. Psychosis participants with high psychopathy traits also tended to make less use of the nose region, with a smaller percentage of fixations when categorising sad (compared to other negative expressions). Psychosis participants with low psychopathy tended to pay relatively more attention to the nose, with increased fixation durations when processing anger (compared to fear or disgust). They also tended to spend longer looking at the mouth when viewing negative emotions than the high psychopathy group.

Conversely, analyses looking at the percent and duration of fixations to non-feature areas revealed some interesting results. The psychosis group paid significantly more attention to non-feature areas when viewing happy expressions (compared to surprise). This increase, of around 5% more fixations than controls, could help in explaining the 6.6% decrease in fixations to the eyes observed among the psychosis

group when recognising happy expressions. Likewise, there was an increased percentage of fixations for negative expressions, and slightly less but still higher for positive or neutral expressions, compared to healthy controls who only exhibited a slight increase to non-feature areas for positive or neutral expressions. Psychosis participants with high psychopathy tended to have an increased percentage of fixations to non-feature areas for disgust (compared to fear), and tended to spend less time fixating in non-feature areas when recognising anger (compared to fear or disgust), with a lower mean fixation duration.

### *5.2.2 Study 2 – Emotion induction*

The psychosis groups reported a significantly increased difficulty in emotion regulation and total mood disturbance at baseline; however, contrary to the hypotheses, during the emotion induction task a broadly similar level of mood variation was reported among both the psychosis and healthy control groups. Overall, the induction effects were modest, consistent with the presentation of static facial expression stimuli in a laboratory-based setting. The majority of participants displayed some mood variation across all four emotions. Following the presentation of happy stimuli, global induction effects included significant reductions in Tension, Anger, Fatigue/Confusion, overall Mood Variation, and a trend for increased Vigour on the POMS domains. Following the presentation of sad stimuli, there was significant overall Mood Variation and trend level changes in Depression and Vigour. Interestingly, while only a trend, the psychosis group reported increased Anger after viewing sad expressions, as opposed to healthy controls whose Anger ratings reduced. Significant overall changes in Anger, Vigour, and overall Mood Variation were also evident following the presentation of angry expressions; as well as a trend level difference for the psychosis group with low

psychopathy, who reported no change on the Vigour domain, while the other groups reported a reduction. Significant overall Mood Variation and trend level Vigour changes were also apparent following the presentation of fear stimuli.

Analyses of mood variation by emotion and group, whilst controlling for stimulus order, revealed no significant group differences in mood induction performance. These findings indicate that the ability to experience emotion was not impaired in the psychosis group. However, worthy of note, healthy controls tended to respond with variations in mood of a similar magnitude in both a positive and negative direction on the POMS domains, consistent with the category of emotional stimuli presented. On the other hand, the psychosis groups tended to show increased fluctuations in mood or volatility across emotion categories on all the POMS domains.

Again contrary to the hypotheses, an examination of the accompanying visual scan-paths in Task B revealed no atypical scan-path patterns among the psychosis groups. There were no group differences for the number or duration of fixations, across emotion categories or any group by emotion interactions. Visual scanning performance was significantly associated with the magnitude of emotion induction, regardless of group. A relationship between mood variation and both the number and duration of fixations was found, whereby, fewer fixations and a longer mean duration per fixation were associated with increased Mood Variation.

Interestingly, analyses examining changes in eye-tracking performance across tasks, using fixation count and duration difference scores between Task B and Task A, revealed differential scan-path strategies were used in the emotion recognition and

induction tasks (for the same categories of emotion). While there were no significant group differences, there was a substantial reduction in the number of fixations per face stimulus in Task B and a corresponding increase in the mean fixation duration. The findings from this study suggest that fewer fixations (around 3.76 fewer), of longer duration (approximately 94.7ms longer), may be advantageous strategies in feeling (experiencing) or inducing mood, while this more restricted scanning strategy (i.e., of fewer fixation points) may be disadvantageous in correctly recognising emotions. In addition, healthy controls tended to have a more marked fixation duration increase in Task B relative to the psychosis groups.

### *5.2.3 Study 3 – Face recognition and working memory*

In partial support of the study hypotheses, the psychosis group tended to be less accurate than healthy controls at recognising faces. Overall accuracy was better for target faces and at immediate recall, with the misidentification of non-target faces significantly related to a decline in performance accuracy, particularly at delayed recall. All groups were influenced in a similar way by distractors images, which introduced an additional cognitive load to the task, in terms of having to correctly identify a paired stimulus as not being a target face. Improved recognition of target faces at immediate recall was associated with better visuospatial constructional ability, immediate memory and overall neuropsychological functioning, and correctly responding to non-targets with increased visuospatial constructional ability. However, at delayed recall the inverse relationship was true, with declining performance for non-target faces associated with improved attention, as well as overall neuropsychological functioning, language, and visuospatial constructional ability. Overall face recognition performance was associated with better immediate memory and higher premorbid IQ.

Contrary to the study hypotheses, visual scanning parameters in Task C, recorded during both the acquisition and recall phases, revealed no overall differences between the health control and psychosis groups, or any atypical scan-path strategies. A comparison of scan-path indices revealed similar eye-tracking patterns and consistency of visual scanning strategies across social-cognitive tasks, with significant relationships between indices within (Task C acquisition & recall) and across tasks (Task C & A). Irrespective of group, visual scan-path performance was sensitive to task components at recall, with significantly fewer fixations of longer duration exhibited when participants viewed target faces. Acquisition but not recall visual scan-path indices were significantly associated with recall performance, indicating a greater number of fixations of shorter duration resulted in significantly increased accuracy for target faces. In addition, non-target stimuli were tracked in a similar way to their paired targets at recall, although, perhaps reflective of uncertainty, increased errors for non-target faces co-occurred with a tendency to have increased fixations. This indicates that elements of processing that are shown to be beneficial in one task set may not be in another (i.e., distractor stimuli in Task C).

Worthy of note, performance accuracy for target faces in Task C was related to facial emotion recognition accuracy in Task A, as were associated scan-path indices, suggesting that visual scanning strategies utilised when recognising a person displaying a neutral facial expression were not dissimilar to those utilised when identifying or recognising that neutral emotion. That is, consistent visual scanning strategies were evident, involving a pattern of fixations to salient facial features, but these strategies also varied according to the particular expression set.

### 5.3 Key task performance indices across all three social cognition tasks.

A number of key indices selected from Tasks A, B and C were utilised to examine social cognition performance across tasks. As detailed in [Tables 5.1a, b & c](#) (comprising one sub-table per study group), individual participant scores on eight task indices were coded into quartiles, from Q1 (lowest quartile) to Q4 (highest quartile); numerical values of 0 to 3 were assigned to these quartiles for purposes of deriving simple aggregate indices (see footnote to Table 5.1c for further information).

Summations for Task A, B and C indices were moderately correlated (A with B:  $r = 0.26$ ,  $p = 0.043$ ; A with C:  $r = 0.39$ ,  $p = 0.002$ ; and B with C:  $r = 0.35$ ,  $p = 0.006$ ). An Aggregate Index based on all 8-task indices was also categorised by quartiles to aid discussion of individual profiles. Mean (SD) scores obtained on this Aggregate Index [healthy controls, 13.63 (4.40); psychosis – low psychopathy, 10.83 (3.31); psychosis – high psychopathy, 11.00 (4.57); and psychosis groups combined, 10.92 (3.95)], revealed a trend level difference for psychosis status,  $t_{(58)} = 2.48$ ,  $p = 0.016$ , indicative of poorer social-cognitive performance across tasks.

These individual participant profiles facilitated an examination of possible subsets within groups. As shown in [Table 5.1a](#), based on the Aggregate Index of task performance, a large proportion of healthy controls (11/16) were in the top quartile (Q4). However, two healthy controls were also among the poorest performers across social cognition tasks (2/11), falling in the bottom quartile (Q1). As detailed in [Table 5.1b & c](#), the psychosis group with low psychopathy, only had one participant (1/16) in the top quartile (Q4) on the Aggregate Index of task performance, while the psychosis group with high psychopathy had four high performance participants (4/16).

Table 5.1a: *Healthy Control (HC) task performance profiles for participants (N = 24) based on quartiles for 8 task indices*

Group / Participant	Aggregate Index: Sum of 8 Indices	Task A			Task B		Task C		
		Index 1: Recognition accuracy	Index 2: Number of fixations	Index 3: % Fixations in feature areas	Index 4: Lower mood variation	Index 5: Shorter fixation duration	Index 6: Immediate recall accuracy	Index 7: Delayed recall accuracy	Index 8: Acquisition no. of fixations
HC 1	21 (O4)	O2	O4	O4	O3	O4	O4	O4	O4
HC 2	19 (O4)	O4	O3	O4	O2	O4	O4	O2	O4
HC 3	19 (O4)	O4	O3	O4	O4	O3	O4	O2	O3
HC 4	19 (O4)	O3	O4	O3	O3	O4	O3	O3	O4
HC 5	17 (O4)	O3	O4	O4	O2	O4	O3	O1	O4
HC 6	17 (O4)	O2	O3	O4	O4	O3	O4	O2	O3
HC 7	17 (O4)	O4	O3	O1	O4	O2	O4	O4	O3
HC 8	16 (O4)	O4	O4	O3	O3	O1	O3	O3	O3
HC 9	16 (O4)	O4	O2	O3	O2	O3	O4	O4	O2
HC 10	16 (O4)	O3	O2	O3	O4	O3	O4	O2	O3
HC 11	15 (O4)	O2	O4	O2	O3	O4	O2	O2	O4
HC 12	13 (O3)	O3	O3	O3	O3	O2	O4	O1	O2
HC 13	13 (Q3)	O1	O3	O3	O4	O3	O2	O1	O4
HC 14	13 (Q3)	O3	O2	O1	O3	O3	O4	O2	O3
HC 15	13 (Q3)	O1	O1	O4	O4	O3	O2	O2	O4
HC 16	12 (Q3)	O2	O1	O2	O4	O2	O4	O2	O3
HC 17	11 (O2)	O2	O2	O4	O3	O1	O2	O4	O1
HC 18	11 (Q2)	O4	O2	O1	O2	O1	O4	O3	O2
HC 19	11 (Q2)	O4	O1	O1	O1	O1	O4	O4	O3
HC 20	10 (Q2)	O4	O2	O4	O1	O1	O3	O2	O1
HC 21	10 (Q2)	O1	O2	O3	O2	O1	O4	O3	O2
HC 22	9 (Q2)	O4	O2	O3	O2	O2	O1	O2	O1
HC 23	7 (O1)	O4	O1	O2	O1	O1	O3	O2	O1
HC 24	2 (Q1)	O2	O1	O1	O1	O1	O1	O2	O1

Note. See footnote Table 5.1c

Table 5.1b: *Psychosis - Low Psychopathy (P-LP) task performance profiles for participants (N = 18) based on quartiles for 8 task indices*

Group / Participant	Aggregate Index: Sum of 8 Indices	Task A			Task B		Task C		
		Index 1: Recognition accuracy	Index 2: Number of fixations	Index 3: % Fixations in feature areas	Index 4: Lower mood variation	Index 5: Shorter fixation duration	Index 6: Immediate recall accuracy	Index 7: Delayed recall accuracy	Index 8: Acquisition no. of fixations
P-LP 1	21 (Q4)	Q4	Q3	Q4	Q4	Q3	Q4	Q3	Q4
P-LP 2	14 (O3)	O4	O4	O2	O3	O4	O1	O1	O3
P-LP 3	14 (O3)	O1	O4	O2	O3	O4	O1	O3	O4
P-LP 4	12 (O3)	O4	O3	O2	O1	O2	O2	O3	O3
P-LP 5	12 (O3)	O4	O2	O1	O2	O3	O2	O3	O3
P-LP 6	12 (O3)	O1	O4	O2	O3	O3	O1	O2	O4
P-LP 7	12 (Q3)	Q2	Q2	Q3	Q4	Q3	Q1	Q3	Q2
P-LP 8	11 (O2)	O2	O1	O3	O2	O3	O2	O3	O3
P-LP 9	10 (O2)	O3	O2	O3	O3	O2	O3	O1	O1
P-LP 10	10 (O2)	O2	O2	O3	O3	O2	O2	O2	O2
P-LP 11	10 (O2)	O2	O1	O2	O2	O2	O3	O4	O2
P-LP 12	9 (Q2)	Q4	Q1	Q4	Q1	Q1	Q2	Q2	Q2
P-LP 13	9 (Q2)	Q3	Q4	Q1			Q3	Q2	Q2
P-LP 14	9 (Q2)	Q3	Q1	Q2	Q2	Q2	Q3	Q3	Q1
P-LP 15	8 (O1)	O2	O3	O1	O2	O2	O3	O2	O1
P-LP 16	8 (Q1)	O3	O1	O2	O4	O1	O2	O2	O1
P-LP 17	7 (Q1)	O2	O1	O4	O1	O1	O2	O3	O1
P-LP 18	7 (Q1)	Q1	Q3	Q1	Q4	Q2	Q2	Q1	Q1

Note. See footnote Table 5.1c

Table 5.1c: *Psychosis - High Psychopathy (P-HP) task performance profiles for participants (N = 19) based on quartiles for 8 task indices*

Group / Participant	Aggregate Index: Sum of 8 Indices	Task A			Task B		Task C		
		Index 1: Recognition accuracy	Index 2: Number of fixations	Index 3: % Fixations in feature areas	Index 4: Lower mood variation	Index 5: Shorter fixation duration	Index 6: Immediate recall accuracy	Index 7: Delayed recall accuracy	Index 8: Acquisition no. of fixations
P-HP 1	20 (Q4)	Q2	Q4	Q4	Q3	Q4	Q4	Q3	Q4
P-HP 2	19 (Q4)	Q3	Q4	Q2	Q4	Q4	Q4	Q2	Q4
P-HP 3	17 (Q4)	Q4	Q3	Q3	Q1	Q4	Q4	Q2	Q4
P-HP 4	15 (Q4)	Q3	Q4	Q1	Q3	Q4	Q2	Q2	Q4
P-HP 5	14 (Q3)	Q2	Q3	Q4	Q2	Q3	Q2	Q3	Q3
P-HP 6	13 (Q3)	Q3	Q3	Q2	Q1	Q2	Q4	Q3	Q3
P-HP 7	13 (Q3)	Q2	Q4	Q1	Q2	Q4	Q3	Q1	Q4
P-HP 8	12 (Q3)	Q1	Q2	Q3	Q2	Q4	Q2	Q3	Q3
P-HP 9	11 (Q2)	Q2	Q3	Q3	Q1	Q2	Q4	Q2	Q2
P-HP 10	9 (Q2)	Q3	Q2	Q4	Q2	Q1	Q1	Q2	Q2
P-HP 11	9 (Q2)	Q1	Q1	Q4	Q2	Q1	Q4	Q2	Q2
P-HP 12	9 (Q2)	Q1	Q4	Q1	Q1	Q2	Q4	Q3	
P-HP 13	9 (Q2)	Q4	Q1	Q1	Q2	Q4	Q1	Q3	Q1
P-HP 14	9 (Q2)	Q1	Q2	Q1	Q3	Q4	Q2	Q2	Q2
P-HP 15	8 (Q1)	Q2	Q3	Q2	Q1	Q3	Q2	Q1	Q2
P-HP 16	7 (Q1)	Q3	Q4	Q1	Q1	Q2	Q1	Q1	Q2
P-HP 17	7 (Q1)	Q1	Q2	Q2	Q1	Q3	Q2	Q2	Q2
P-HP 18	5 (Q1)	Q1	Q1	Q2	Q2	Q1	Q1	Q4	Q1
P-HP 19	3 (Q1)	Q1	Q1	Q3	Q1	Q1	Q1	Q2	Q1

Note. Eight indices were used to characterise individual participant performance across the set of tasks: Task A – Overall recognition accuracy, number of fixations, and percentage of fixations in feature areas; Task B – Lower mood variation (based on POMS changes), and shorter fixation durations, which were effectively re-interpreted as indicative of lower overall mood intensity or volatility (as opposed to poorer induction of specific moods); Task C – Immediate recall accuracy; delayed recall accuracy, and number of fixations during the acquisition phase. Individual participant's scores for these indices were coded into quartiles, from Q1 (lowest quartile) to Q4 (highest quartile), with numerical values of 0 to 3 also assigned to these quartiles for purposes of deriving simple aggregate indices.

Within the bottom quartile (Q1), four of the low psychopathy (4/11) and five of the high psychopathy (5/11) group were among the poorer performers across tasks. Worthy of note, the psychosis group with low psychopathy tended to have greater numbers of poorer performers (Q1-Q3), while there was greater variability among the high psychopathy group, with participants well represented across all four quartiles (Q1-Q4).

#### **5.4 Key findings and contribution to the literature**

In accord with the substantive literature in this area, the first study was able to demonstrate emotion recognition impairments in Task A, with significantly reduced accuracy among the psychosis group. However, contrary to expectations, there was no differential impact of coexisting high psychopathy traits. Some atypical visual scan-paths were apparent among the psychosis groups, as well as among the group with high psychopathy traits, but visual scanning strategies were not associated with performance accuracy. The second study demonstrated that emotion induction was achievable among both the psychosis and healthy control groups, with similar levels of mood variation across groups in Task B. No atypical visual scan-path patterns were exhibited in Task B among the psychosis groups, or among those with high psychopathy traits. However, differential scan-path strategies were observed across tasks A and B, suggesting that visual information processing when asked to feel or experience emotions is likely to be different to that required to recognise or categorise emotions. Study 3 revealed that accuracy tended to reduce among the psychosis groups during a face identification task utilising neutral emotion images (Task C). On the other hand, the capacity to recall faces following delayed recall remained relatively intact, with no sign of rapid forgetting among the psychosis groups and no apparent visual scanning deficits. As anticipated, performance accuracy overall was associated with cognitive functioning,

and specifically with better immediate memory and higher premorbid IQ. However, elevated psychopathy traits amongst the psychosis group were not associated with face recognition performance.

In considering variations in difficulty or neurocognitive demands across tasks, the easiest or least cognitively taxing task was the emotion induction Task B, in which participants were asked to view a series of images (displayed for 60 sec/6000ms each) expressing emotion and to try to feel the emotion displayed. By contrast, in the emotion recognition Task A, participants were asked to recognise one of seven emotions displayed, requiring higher level processing (i.e., attention, visuospatial ability), as they needed to match the facial emotion displayed to their own pre-existing representation or categorisation for that emotion in order to decide which emotion was randomly displayed (within 6000ms). However, Task C was possibly the most difficult, requiring the greatest cognitive load, in that the time allowed to acquire information (i.e., remember a face), as well as the time to provide a decision at recall (i.e., whether they had seen it before), was half that provided in tasks A & B for each stimulus (i.e., 3000ms vs. 6000ms per stimuli). This task required the capacity for not only increased attention, processing speed and visuospatial ability, but working memory in order to undertake accurate decision-making. In addition, task difficulty increased further with the introduction of similar distractor images (paired stimuli). However, it is worthy of mention that the largest performance deficits obtained in this series of tasks still related to the emotion recognition Task A, in that recognition accuracy was significantly impaired among the psychosis group and was accompanied by atypical patterns of visual scanning in comparison to the healthy controls.

Whilst not definitive, based on the current psychosis sample's performance accuracy and visual scanning, coupled with an exploration of their performance profiles across tasks, the results obtained imply that there is a specific facial emotion perception and processing deficit, rather than a more general face-processing deficit. However, only some aspects of visual processing associated with facial expressions of emotion were impaired among participants with a psychotic disorder. Notwithstanding findings from the current study provide some support for research suggesting that deficits in facial affect processing are a potential cognitive marker (She et al., 2017). However, eye-tracking research suggesting that individuals with schizophrenia have different eye movement patterns (e.g., longer fixations and less saccades) compared to healthy controls (Benson et al., 2012) was unable to be confirmed in the current study. However, as already noted, some evidence of atypical visual scanning was apparent across emotion categories among the psychosis group during facial emotion recognition. Similarly, eye tracking studies showing restricted attention to relevant or salient aspects of face stimuli in schizophrenia (Loughland et al., 2002a, 2002b) were only partially supported, being found only for some emotion categories during emotion recognition among the psychosis group, and those with high psychopathy traits.

In addition to the above, some schizophrenia research has reported a smaller visual span compared to healthy controls, whereby patients attend to a smaller region of visual field during a single eye fixation under some search conditions, and lack flexibility to modulate visual span contingent on task demands, potentially leading to poor search performance (Elahipanah, Christensen, & Reingold, 2010, 2011). Across the three tasks utilised in the current study, the visual scan path length was not significantly different among the psychosis group. However, each task did control for

initial eye-movement, due to participants being required to fixate on a centrally located dot for 1000ms prior to the face stimuli being presented, restricting initial differential eye-movement patterns on commencement of each trial. It is also important to point out that given the identical administration protocol across tasks, atypical scan-path patterns were still found during the facial emotion recognition task, consistent with previous research.

The findings obtained from the current study do not support a more general impairment in visual processing in schizophrenia, with no atypical eye-movement parameters exhibited during visual scanning in two of the three active tasks, utilising facial emotion stimuli with identical configural properties. However, that said, this is implied rather than conclusive evidence, given the absence of non-face stimuli in the current series, and utilisation of static rather than dynamic face stimuli, limiting generalisability. Recent research evidence suggests, increased facial emotion processing deficits may be exhibited among individuals with psychosis and psychopathy, during task conditions utilising dynamic facial stimuli, in natural settings, or real world contexts (Cigna et al., 2017; Dowiasch et al., 2016; Sasson, Pinkham, Weittenhiller, Faso, & Simpson, 2016).

Overall, the current study does highlight a core deficit in facial emotion recognition processing in psychosis (Task A), as well as a tendency for this to generalise to a difficulty with utilising neutral emotion face stimuli (Task C), in the absence of deficits in the ability to experience emotion (Task B). In contrast to the original hypothesised expectations, coexisting high-levels of psychopathy traits do not appear to make a significant independent contribution to the level of facial emotion

processing deficits exhibited. However, the relationships between social cognitive impairments and psychopathy may differ among individuals meeting formal diagnostic criteria for psychopathy (i.e., at or above the cut-off score on the PCL-R). In addition, further research is required to address any associations with distinct facets of psychopathy.

In broad terms, findings from the current series of tasks are consistent with prior research in schizophrenia supporting the significant contributions of facial emotion recognition and neurocognitive abilities to global social functioning, including the roles played by working memory and attention (Huang & Hsiao, 2017; Milev et al., 2005; Pan et al., 2009). Emerging psychosis research continues to attempt to disentangle the complex interrelationships between clinical symptoms, neurocognition and facial emotion processing, in determining the extent of their contribution to social functioning in schizophrenia (Huang & Hsiao, 2017). Similar complex associations have been reported in other clinical populations, such as among individuals with bipolar disorder, where social cognition has been found to be associated with neurocognitive functioning but not with emotion regulation (Van Rheenen, Meyer, & Rossell, 2014). Within the context of the current study, which sought to address the impact of higher levels of coexisting psychopathy traits, no support was found for the proposition that psychopathy traits make an independent contribution to facial emotion processing deficits in psychosis. However, as the current psychosis sample experienced predominately remitted and mild symptoms, and exhibited a moderate range of psychopathic traits, further exploration among psychopathic populations considering the independent contribution of psychosis is warranted (i.e., including distinct facets, or specific psychopathy traits).

## 5.5 Strengths and limitations of the current research

This study investigated the level of social-cognitive impairment during facial emotion processing across multiple tasks within a psychosis sample, considering coexisting psychopathy traits. Whilst a clinical comparison group of psychopaths meeting full criteria for psychopathy was not recruited, based on performance across the three tasks utilised in this study (examining emotion recognition, invoking or experiencing emotion, and face recognition and working memory), the level of emotion processing impairments exhibited appears to be largely characteristic of psychotic illness more broadly. That is, elevated psychopathy traits did not make a significant independent contribution to the level of impairment in emotion processing displayed. In the context of this study, our findings suggest that individuals with psychosis and coexisting high levels of psychopathy are just as likely to benefit from social-cognitive treatment modalities. In short, based on the extent of the deficits, they should be included in remediation opportunities rather than excluded due to diagnostic complexity, which anecdotally can be the case. While psychopathy is a low prevalence disorder, it is costly at a societal level, disproportionately accounting for societal burden; consequently, improved treatment options have important long-term implications.

More generally, it is worthy of note that emotion processing studies traditionally only consider one diagnostic construct, in this case either psychosis or psychopathy, when exploring social-cognitive impairment. Given that psychosis is evident across a spectrum of disorders (e.g., schizophrenia, schizoaffective and bipolar disorder), it is important for studies to try to adequately address aspects of specificity of the impairment displayed. Ideally, future studies should include additional clinical samples simultaneously, such as affective disorder, rather than limiting opportunities for making direct performance

comparisons only to healthy control participants. This current study sought to address the impact of psychopathy traits. Emotion-processing research is emerging that considers additional clinical subgroups, including personality disorders, and this may help to quantify the extent of neuro- and social-cognitive impairments and how best to address them.

This current research study has some limitations that may directly influence the generalisability of findings. Firstly, the sample size was relatively small, due to difficulties related to the recruitment of the low prevalence disorders of interest. Whilst some significant and trend level atypical scan-path findings were apparent, sample size limitations may have restricted the capacity to fully address the extent of impairments displayed. Similarly, while psychopathy is referred to as a dimensional construct, the relationships between psychopathy and social cognitive impairments may change as the number of traits increase closer to the cut-off score for psychopathy (on the PCL:SV). Previous studies have had similar limitations with respect to sample size and inclusion of mild levels of psychopathy traits (Gillespie et al., 2015c). Therefore, further studies among individuals meeting criteria for psychopathy, as well as among larger sub-clinical samples, considering the associations between specific facets of psychopathy, are warranted given some specific findings related for example to the affective component (factor 1) of psychopathy being reported in the literature.

With regard to emotional stimuli used in this study, while static stimuli of similar configural properties allowed for an examination of visual scan-path strategies to be undertaken across three different social-cognitive tasks, they were relatively simple images free of context. Similar criticism about reliance on static stimuli could be made of

the other visual-scanning studies examining face and emotion perception in psychopathy (Gillespie et al., 2017b; Gillespie et al., 2015c). Further research on facial emotion processing and social interactions, in more natural settings, utilising dynamic stimuli may reveal additional important finding related to impaired social cognition in both psychosis and psychopathy.

This study investigated the level of social-cognitive impairment within a psychosis sample, whilst considering coexisting psychopathy traits; however, as previously noted, the study design did not incorporate a clinical group meeting full diagnostic criteria for psychopathy. Therefore, direct comparisons to some of the psychopathy literature are limited. Furthermore, the current study sample was comprised of individuals living in the community, potentially limiting the generalisability to studies undertaken within forensic settings. In relation to the largely null findings obtained for psychopathy, sample size, stimulus type, and the mild range of psychopathy traits have been noted, however, on the other hand existing psychopathy studies have not adequately assessed the influence of psychiatric illness. Given the negligible impact of high psychopathy traits found within the current psychosis sample, future studies utilising *psychopathic samples* should diligently document any current or prior history of mental illness, particularly any psychotic symptoms, and account for these factors in any outcome analyses.

In spite of these limitations, the current study has important clinical and research implications, being one of very few studies to have considered the impact of coexisting psychopathy traits on social-cognitive functioning in psychosis, specifically in terms of facial emotion processing and visual scanning strategies. Challenges related to undertaking research among this population, include difficulties associated with engaging

a complex diagnostic group, who are often transient and poorly engaged with services. With a substantial proportion of the existing psychopathy research undertaken within forensic and institutionalised samples, at the high end of the psychopathy spectrum rather than within community samples. This is most likely, in part due to difficulties engaging these individuals in research, but also the stigma associated with disclosure. Individuals with psychosis and coexisting psychopath traits, represent an important clinical population, typically experiencing high levels of unmet need, with significant social functioning impairments in the community, and often have limited access to interventions within existing mental health services.

The current study design provided a substantial advantage, in that the same cohort of participants completed all three visual-cognitive tasks, allowing inter-relationships between tasks to be examined in detail for the entire sample. All participants undertook the tasks in the same administration order, which had the potential to introduce possible carry over effects for the type of visual processing strategies utilised across tasks. However, as the configural properties of the stimuli utilised were the same in all tasks, and variations in eye-tracking strategies by task and across emotional stimuli type were obtained, such potential carry over effects were likely to have been inconsequential.

Overall, the current study found deficits in facial emotion recognition processing (Task A), intact emotional experience (Task B), and a tendency for reduced face identification in psychosis (Task C), irrespective of the level of psychopathy traits. No atypical scan-path parameters were exhibited on two of the three visual-cognitive task utilising face stimuli, challenging the viewpoint that schizophrenia is associated with a

general deficit in visual processing. Further studies are required to clarify the underlying mechanisms and specific stages of visual processing impacted in psychosis.

## **5.6 Implications of these research findings**

This study extended existing facial expression processing research in schizophrenia by examining the impact of coexisting psychopathy, as well as considering the association of clinical symptoms, emotion regulation and neurocognitive functioning. This study was one of the first to use visual scanning techniques to examine the relationship between psychosis, coexisting psychopathy traits and facial emotion perception deficits. Previous emotion recognition findings among an inpatient forensic sample of schizophrenia patients with comorbid psychopathy were not able to be replicated (Fullam & Dolan, 2006b). Individuals with a psychotic disorder and higher psychopathy traits (based on PCL: SV scores) did not demonstrate greater impairment in emotion recognition accuracy. Similarly, visual scan-paths, examining the neurocognitive strategies used in visual information processing during emotion recognition, induction and working memory tasks did not reveal differential eye-tracking strategies among the psychosis group with high psychopathy traits. Nonetheless, an examination of the visual-cognitive and face perception deficits among a sub-group of individuals with a psychotic disorder and coexisting high psychopathy traits, utilising relatively complex task designs, did assist in increasing our understanding of the underlying mechanisms, as well as providing further insight into diagnostic and physiological issues. Hopefully, these findings fulfil the overarching aim of adding to investigations examining relationships between deficits in face perception and social-cognition in schizophrenia.

## 5.7 Future research directions

Clinical implications include an increased understanding of facial emotion processing deficits in psychosis, potentially leading to additional treatment advances and specifically to cognitive remediation aimed at improving social and occupational functional outcomes among individuals with complex diagnostic issues. In relation to coexisting psychopathy traits, the findings support (or, at least, are not inconsistent with) treatment options focusing on combined neurocognitive and emotion recognition training, to assist in obtaining sustained improvements in social functional outcomes. While a larger study sample may have allowed for additional subgroup analyses, based on the current findings facial emotion-processing performance is not significantly poorer among psychosis participants with higher psychopathy traits. This has important clinical implications, as complex, comorbid diagnostic issues are often seen as an indicator of increased debilitation, and potentially a limiting factor in terms of treatment options and anticipated functional gains. However, based on these research findings, in all likelihood individuals with psychosis and coexisting high psychopathy traits have the capacity to receive similar benefits from social-cognitive based treatment modalities, to those with low psychopathy traits.

While outside the scope of this dissertation, a burgeoning area worthy of continued consideration, relates to the ever-advancing range of computing technologies. This includes but is not limited to, the increased availability and portability of eye-tracking hardware and software, face recognition software, virtual reality and gaming technologies. These all represent potential areas of further treatment focus. A review of eHealth treatments currently used in forensic mental health settings, identified multiple beneficial technologies (i.e., virtual reality, web based and videoconferencing

interventions) across a number of treatment domains, including some targeting social interactions, emotional reactivity and violence; although evaluations of effectiveness and intervention tailoring to specific needs are ongoing (Kip, Bouman, Kelders, & van Gemert-Pijnen, 2018).

In relation to psychopathy traits, treatment evaluation may have important societal implications, as some evidence suggests there is a potential to improve or worsen outcomes (i.e. learning to manipulate the system) (Moreira et al., 2014). For example, the suggestion that an enhanced ability to perceive vulnerability in others by improved emotion recognition could be counterproductive among this population (Wilson et al., 2011). This notion largely relates to the premise that some traits may be adaptive in particular contexts, such as in competitive environments where a non-empathic, aggressive approach may be optimal (Moreira et al., 2014). Where ‘*successful psychopaths*’ are seen to have both interpersonal and affective traits, but less antisocial behaviour than criminal psychopaths often seen as the ‘*unsuccessful psychopaths*’ (Moreira et al., 2014). Up until recently, it was not possible to adapt social-cognitive training to online environments, and to a range of treatment settings, thereby facilitating better targeting of clinical populations with known impairments, including individuals with psychosis and complex comorbidities (i.e. high psychopathy traits).

If clinicians are able to achieve improved therapeutic outcomes among individuals with psychosis, particular when complex comorbidities are present, extending the evidence based knowledge around emotion processing, including the basic mechanisms behind facial emotion recognition and experience, is essential to translational work aimed at addressing clinical needs, specifically related to enduring

social-cognitive impairments among this population. In addition, coupled with further neuropsychological research among complex clinical groups, may assist in elucidating further information on the mechanisms involved in functional disturbances. As well as potentially confirming behavioural findings, such as those around the subjective experience of emotion, which is likely to be of clinical significance. In addition, whilst a potentially contentious issue, rather than relying on the limitations of diagnostic constructs, potential implications related to specific clinical phenotypes are worthy of continued investigation.

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## Appendices

### Appendix A: Participant information sheet



Attachment 1 – Version 5 – 27/06/12  
Facial expression processing in  
individuals with a mental illness

**Associate Professor Carmel Loughland**  
Centre for Translational Neuroscience and Mental Health  
University of Newcastle  
McAuley Centre, The Mater  
Ph: (02) 40335722  
Fax: (02) 40335692  
E-mail: [Carmel.Loughland@newcastle.edu.au](mailto:Carmel.Loughland@newcastle.edu.au)

#### Information Statement for the Research Project

#### Facial expression processing in individuals with a mental illness Version 5, dated 27/06/2012

You are invited to take part in the research project named above which is being conducted by Ketrina Sly as part of her PhD in Clinical Psychology Degree under the supervision of Associate Professor Carmel Loughland from the Centre for Translational Neuroscience and Mental Health, Associate Professor Mick Hunter, from the School of Psychology at the University of Newcastle, and Associate Professor Terry Lewin from the Centre for Translational Neuroscience and Mental Health.

#### **Why is the research being done?**

This project will study how mental illness, personality traits, symptoms, and cognitive functioning might affect a person's ability to perceive other people's emotions. Cognitive functioning refers to your ability to perform or complete tasks involving particular strategies used to work problems through. Previous research has shown that some individuals with a mental illness and those with particular personality traits have problems perceiving and interpreting some facial displays of emotion. Understanding what causes these problems may lead to the development of programs that help people improve their social and employment outcomes.

#### **Who can participate in the research?**

We are seeking people aged 18-65 years who fit one of the following criteria:

- 1) People who have been in contact with HNE Mental Health or the Newcastle Mental Health Court Liaison (NMHCL) service, have a history of offending behaviour and have been diagnosed with a mental illness; or
- 2) People who have been diagnosed with schizophrenia; or
- 3) People without a personal or family history of mental health problems to provide a community control sample.

People who have experienced a severe head injury, have severe intellectual disability, a diagnosis of bipolar disorder or a substance-induced psychosis will not be able to participate in the study.

#### **What choice do you have?**

Participation is completely voluntary. Whether you decide to participate or not, your decision will not disadvantage you in any way or affect your access to services or treatment. If you do consent to participate, you may withdraw from the project at any time without giving a reason. If you decide to withdraw, your request will be respected and, upon receipt of written request, your stored personal information will be destroyed.

#### **What would you be asked to do?**

If you agree to participate, you will be asked to do the following:

- 1) To complete a clinical assessment, including two brief questionnaires. Interview questions are of a general nature and do not ask direct questions about specific dates or times of any previous offending behaviour. Questions will be asked about your mental health and well-being, emotions, personality, and mood. You should not give specific details of a criminal activity or incident otherwise the researchers are obligated to report it to the relevant authorities. The interview and assessments may take up to 2.5 hours to complete. You will also be asked to allow a limited period of access to your current medical record held by the Hunter New England Mental Health Service. This would be for a maximum of 3 months to enable the researchers to clarify the nature of your mental illness.

- 2) To complete three face emotion-processing tasks. In each task, faces will be presented using a computer screen. Your eye movement will be recorded at the same time using a non-invasive technique, involving recording your eye movements using an eye tracker. This device consists of a chin support and infrared low light camera that records retinal and corneal reflections from your eye. In the first task, you will be asked to interpret the emotion presented on the faces. In the second task, you will be asked you to rate how you feel when looking at the faces. In the third task you will be asked to recall which faces you have already seen. These three tasks will take about 25 minutes in total.

Regular breaks will be provided during the interview and tasks. All assessments will take place at the Centre for Translational Neuroscience and Mental Health (CTNMH) at the Mater. People will be reimbursed for their time and any travel expenses after they have completed the study.

**What are the risks and benefits of participating?**

There are no known risks associated with taking part in this project. There will be no benefit to you for participating in this research.

**How will your privacy be protected?**

People entering the study will be given a unique ID code and all their personal information will be stored using this code. The data will be stored on a password-protected computer and paper copies of your interview will be stored in a locked filing cabinet at the Centre for Translational Neuroscience and Mental Health for the duration of the project. Only the research team will have access this information. Your data will be kept in locked storage for a period of five years, after which it will be destroyed.

**How will the information collected be used?**

Group data will be presented in a thesis to be submitted by Ketrina Sly as part of her PhD in Clinical Psychology degree at the University of Newcastle. Data may also be reported in scientific journals or at conferences. However, no personal data will be used in any of the reports or publications. All participants will be given a one-page summary of the results at the completion of the study, along with a contact number for those people wishing to ask any questions.

**What do you need to do to participate?**

Please read this information statement and be sure you understand it before you consent to participate. If there is anything you do not understand, or you have any questions, contact the researcher. If you would like to participate, please tell a staff member and they will pass your contact details on to Ketrina Sly. She will telephone you to arrange an appointment time convenient to you to attend the Centre. You will be asked to complete a consent form on the day of testing.

**Further information**

If you would like any further information about this study, please contact Associate Professor Carmel Loughland or Ketrina Sly on 40335707.

Thank you for your time and consideration of this invitation.

Yours sincerely,

Associate Professor Carmel Loughland  
Chief Investigator

Ketrina Sly  
PhD Student

**Complaints about this research**

This project has been approved by the Hunter New England Human Research Ethics Committee of the Hunter New England Health, Reference 07/02/21/5.05.

Should you have concerns about your rights as a participant in this research, or you have a complaint about the manner in which the research is conducted, it may be given to the researcher, or, if an independent person is preferred, to Dr Nicole Gerrand, Professional Officer (Research Ethics), Hunter New England Human Research Ethics Committee, Hunter New England Health, Locked Bag 1, New Lambton NSW 2305, telephone (02) 49 214950, email [Nicole.Gerrand@hnehealth.nsw.gov.au](mailto:Nicole.Gerrand@hnehealth.nsw.gov.au)

## Appendix B: Participant consent form

Attachment 2 – Version 4 – 27/06/12  
Facial expression processing in  
individuals with a mental illness



**Associate Professor Carmel Loughland**  
Centre for Translational Neuroscience and Mental Health  
University of Newcastle  
McAuley Centre, The Mater  
Ph: (02) 40335722  
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E-mail: [Carmel.Loughland@newcastle.edu.au](mailto:Carmel.Loughland@newcastle.edu.au)

### Consent Form for the Research Project

Facial expression processing in individuals with a mental illness

Version 4, dated 27/06/2012

Prepared by Ketrina Sly

I.....  
(print name)

of.....  
(print address)

*Agree to participate in the above research project examining facial expression processing among individuals with a mental illness, and give my consent freely.*

*I acknowledge that I have read the participant information sheet, and I understand that the project will be conducted as described in the information Statement, a copy of which I have retained.*

*I have had the opportunity to ask questions relating to the project and had all questions answered to my satisfaction.*

*I understand that I can withdraw from the project at any time and do not have to give any reason for withdrawing.*

*I consent to complete a clinical interview and three emotion processing tasks, and where applicable I give permission for limited access to my current medical record held by Hunter New England Mental Health Service.*

*I understand that my personal information will remain confidential to the researchers, and that research data gathered from the results of this study may be published, provided that I cannot be identified.*

*I have the opportunity to have questions answered to my satisfaction.*

**Please Print Name:** \_\_\_\_\_

**Signature:** \_\_\_\_\_ **Date:** \_\_\_\_/\_\_\_\_/\_\_\_\_

## Appendix C: Participant flyer



**LOCATION**  
McAuley Centre, The Mater  
Waratah NSW 2298  
Ph: (02) 403 35690  
Fax: (02) 403 35692

### **Centre for Translational Neuroscience & Mental Health Participants Needed for Facial expression processing Study**

#### **Why is the research being done?**

To study how mental illness, personality traits, symptoms, and cognitive functioning might affect a person's ability to perceive other people's emotions.

#### **Who can participate in the research?**

We are seeking people aged 18-65 years who fit one of the following criteria:

- 1) People who have a history of offending behaviour and have been diagnosed with a mental illness; or
- 2) People who have been diagnosed with schizophrenia; or
- 3) People without a personal or family history of mental health problems to provide a community control sample.

#### **What would you be asked to do?**

- 1) To complete a clinical assessment, including two brief questionnaires.
- 2) To complete 3 face emotion-processing tasks.

The interview and assessments may take up to 2.5 hours to complete. Regular breaks will be provided. All assessments will take place at the Centre for Translational Neuroscience and Mental Health (CTNMH) at the Mater.

People will be reimbursed for their time and any travel expenses after they have completed the study.

#### **What do you need to do to participate?**

If you would like to participate, please contact Ketrina Sly on 40335707 to arrange an appointment time convenient to you to attend the Centre.

#### **Further information**

If you would like any further information about this study, please contact Associate Professor Carmel Loughland or Ketrina Sly on 40335707.

This research is being conducted by Ketrina Sly as part of her PhD in Clinical Psychology Degree under the supervision of Associate Professor Carmel Loughland from the Centre for Translational Neuroscience and Mental Health, Associate Professor Mick Hunter, from the School of Psychology at the University of Newcastle, and Associate Professor Terry Lewin from the Centre for Translational Neuroscience and Mental Health.

Thank you for your time and consideration of this invitation.

Associate Professor Carmel Loughland  
Chief Investigator

Ketrina Sly  
PhD Student

Appendix D: Recruitment video



### **Facial Expression Processing Study**

Ketrina Sly is a Research & Information Officer for HNE Mental Health. Ketrina is undertaking a study to explore how mental illness, personality traits, symptoms, and cognitive functioning might affect a persons ability to perceive other people's emotions. Ketrina is recruiting participants for this study who have a major mental illness and a record of offending behaviour in the past 2 years. Please contact

[Online Video-Clip link](#)

## Appendix E: Ethics approval notification HNE HREC



**Health**  
Hunter New England  
Local Health District

15 March 2013

A/Professor Carmel Loughland  
Centre for Translational Neuroscience & Mental Health  
McAuley Centre  
Calvary Mater Newcastle

Dear Professor Loughland

**Re: Facial Expression Processing in Individuals with a Mental Illness (07/02/21/5.05)**

Thank you for submitting the renewal application for the above project which was considered by the Hunter New England Human Research Ethics Committee at its meeting held on **15 March 2013**. This Human Research Ethics Committee is constituted and operates in accordance with the National Health and Medical Research Council's *National Statement on Ethical Conduct in Human Research 2007* and the *CPMP/ICH Note for Guidance on Good Clinical Practice*.

I am pleased to advise that the Hunter New England Human Research Ethics Committee has granted ongoing ethical approval of the protocol: **Facial Expression Processing in Individuals with a Mental Illness**

Approval from the Hunter New England Human Research Ethics Committee for the above protocol is given for a maximum of **3** years from the date of this letter, after which a renewal application will be required if the protocol has not been completed.

The *National Statement on Ethical Conduct in Human Research 2007*, which the Committee is obliged to adhere to, include the requirement that the committee monitors the research protocols it has approved. In order for the Committee to fulfil this function, it requires:

- A report of the progress of the above protocol be submitted at 12 monthly intervals. Your review date is **March 2014**. A proforma for the annual report will be sent two weeks prior to the due date.
- A final report must be submitted at the completion of the above protocol, that is, after data analysis has been completed and a final report compiled. A proforma for the final report will be sent two weeks prior to the due date.
- All variations or amendments to this protocol, including amendments to the Information Sheet and Consent Form, must be forwarded to and approved by the Hunter New England Human Research Ethics Committee prior to their implementation.
- The Principal Investigator will immediately report anything which might warrant review of ethical approval of the project in the specified format, including:

Hunter New England Human Research Ethics Committee  
(Locked Bag No 1)  
(New Lambton NSW 2305)  
Telephone (02) 49214 950 Facsimile (02) 49214 818  
Email: hnehrec@hnehealth.nsw.gov.au  
[http://www.hnehealth.nsw.gov.au/research\\_ethics\\_and\\_governance\\_unit](http://www.hnehealth.nsw.gov.au/research_ethics_and_governance_unit)

- any serious or unexpected adverse events

- Adverse events, however minor, must be recorded as observed by the Investigator or as volunteered by a participant in this protocol. Full details will be documented, whether or not the Investigator or his deputies considers the event to be related to the trial substance or procedure.
- Serious adverse events that occur during the study or within six months of completion of the trial at your site should be reported to the Professional Officer of the Hunter New England Human Research Ethics Committee as soon as possible and at the latest within 72 hours.
- Copies of serious adverse event reports from other sites should be sent to the Hunter New England Human Research Ethics Committee for review as soon as possible after being received.
- Serious adverse events are defined as:
  - Causing death, life threatening or serious disability.
  - Cause or prolong hospitalisation.
  - Overdoses, cancers, congenital abnormalities whether judged to be caused by the investigational agent or new procedure or not.
  - Unforeseen events that might affect continued ethical acceptability of the project.
- If for some reason the above protocol does not commence (for example it does not receive funding); is suspended or discontinued, please inform Dr Nicole Gerrand, the Manager, Research Ethics and Governance Unit as soon as possible.

The Hunter New England Human Research Ethics Committee also has delegated authority to approve the commencement of this research on behalf of the Hunter New England Local Health District. This research may therefore commence.

Should you have any queries about your project please contact Dr Nicole Gerrand as per the contact details at the bottom of the page. The Hunter New England Human Research Ethics Committee Terms of Reference, Standard Operating Procedures, membership and standard forms are available from the Hunter New England Area Health Service website:

Internet address: [http://www.hnehealth.nsw.gov.au/research\\_ethics\\_and\\_governance\\_unit](http://www.hnehealth.nsw.gov.au/research_ethics_and_governance_unit)

Please quote **07/02/21/5.05** in all correspondence.

The Hunter New England Human Research Ethics Committee wishes you every success in your research.

Yours faithfully

For: Associate Professor M Parsons  
Chair  
Hunter New England Human Research Ethics Committee

Hunter New England Human Research Ethics Committee  
(Locked Bag No 1)  
(New Lambton NSW 2305)  
Telephone (02) 49214 950 Facsimile (02) 49214 818  
Email: [hnehrec@hnehealth.nsw.gov.au](mailto:hnehrec@hnehealth.nsw.gov.au)  
[http://www.hnehealth.nsw.gov.au/research\\_ethics\\_and\\_governance\\_unit](http://www.hnehealth.nsw.gov.au/research_ethics_and_governance_unit)

## Appendix F: Ethics registration of approval University of Newcastle HREC

### HUMAN RESEARCH ETHICS COMMITTEE



#### Notification of Expedited Approval

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To Chief Investigator or Project Supervisor:	Doctor Carmel Loughland
Cc Co-investigators / Research Students:	Conjoint Associate Professor Mick Hunter Conjoint Associate Professor Terry Lewin Ms Ketrina Sly
Re Protocol:	Facial expression processing in individuals with a mental illness
Date:	09-Aug-2012
Reference No:	H-425-0407

---

Thank you for your **Variation** submission to the Human Research Ethics Committee (HREC) seeking approval in relation to a variation to the above protocol.

Variation to;

1. Expand the source of recruitment to other clinical services including Acute and Community Mental Health,
2. To increase the reimbursement amount to \$80 to allow an equal opportunity for participants residing in outlying suburbs to be able to participate in the study,
3. Information Statement (Version 5 dated 27 June 2012)
4. Consent Form (Version 4 dated 27 June 2012) and
5. Study Flyer (version 2 dated 27 June 2012).

Your submission was considered under **Expedited Review of External Approval** review by the Chair/Deputy Chair.

I am pleased to advise that the decision on your submission is **External HREC Approval Noted effective 09-Aug-2012**.

The full Committee will be asked to ratify this decision at its next scheduled meeting. A formal *Certificate of Approval* will be available upon request.

Professor Allyson Holbrook  
Chair, Human Research Ethics Committee

*For communications and enquiries:*  
Human Research Ethics Administration

Research Services  
Research Integrity Unit  
HA148, Hunter Building  
The University of Newcastle  
Callaghan NSW 2308  
T +61 2 492 18999  
F +61 2 492 17164  
[Human-Ethics@newcastle.edu.au](mailto:Human-Ethics@newcastle.edu.au)

*Linked University of Newcastle administered funding:*

Funding body	Funding project title	First named investigator	Grant Ref
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Appendix G: Recruited sample characteristics

Supplementary Table S1: *Recruited sample by Age, Gender and Group*

Age Category (years)	Healthy Control		Psychosis	
	Male	Female	Male	Female
29-30	1	0	2	1
30-34	3	1	6	0
35-39	5	0	4	0
40-44	3	2	4	1
45-49	0	1	5	2
50-54	4	1	2	3
55-59	1	0	4	1
60 +	1	1	2	0
Total	18	6	29	8

Appendix H: Abbreviated Profile of Mood States (POMS) 12 item version.

Measure as displayed on screen during the emotion induction visual-cognitive eye-tracking task (Task B).

Below is a list of words that describe feelings that people have. Please read each word carefully. Then tell me the number that best describes how you feel <b>RIGHT NOW</b> .						
		Not at all	A little	Moderately	Quite a bit	Extremely
<b>1.</b>	<b>Tense</b>	0	1	2	3	4
<b>2.</b>	<b>Angry</b>	0	1	2	3	4
<b>3.</b>	<b>Worn out</b>	0	1	2	3	4
<b>4.</b>	<b>Confused</b>	0	1	2	3	4
<b>5.</b>	<b>Sad</b>	0	1	2	3	4
<b>6.</b>	<b>Energetic</b>	0	1	2	3	4
<b>7.</b>	<b>Unable to concentrate</b>	0	1	2	3	4
<b>8.</b>	<b>Annoyed</b>	0	1	2	3	4
<b>9.</b>	<b>Cheerful</b>	0	1	2	3	4
<b>10.</b>	<b>Exhausted</b>	0	1	2	3	4
<b>11.</b>	<b>Anxious</b>	0	1	2	3	4
<b>12.</b>	<b>Terrified</b>	0	1	2	3	4

## Appendix I: Emotion recognition Task A – Participant instructions

Displayed on screen during the emotion recognition visual-cognitive eye-tracking task.

I'm going to show you some pictures of people expressing different emotions.

After each picture the following choices will appear:

**AFRAID HAPPY NO EMOTION SAD SURPRISED DISGUSTED ANGRY**

Look carefully at each picture while it is presented and  
then choose the emotion that best describes that picture.

We are going to start with a calibration.

**Are you ready?**

## **Calibration**

Look at the dot until you hear a click and  
wait until the next dot appears before you move your eyes

Don't try to guess where the dot is going to move  
We need to make sure the computer is recording each time you fixate on the dot

**AFRAID HAPPY NO EMOTION SAD SURPRISED DISGUSTED ANGRY**

Appendix J: Emotion induction Task B – Participant instructions

Displayed on screen during the emotion induction visual-cognitive eye-tracking task.

Now I'm going to show you some pictures of people expressing different emotions.

**Look carefully at each picture while it is presented and  
draw on your personal experiences to try to feel that emotion.**

After each set of pictures, a brief questionnaire will appear  
on the screen asking you to rate how you are feeling "right now".

Are you ready?

Below is a list of words that describe feelings that people have. Please read each word carefully.  
Then tell me the number that best describes how you feel **RIGHT NOW**.

	Not at all	A little	Moderately	Quite a bit	Extremely
<b>1. Tense</b>	0	1	2	3	4
<b>2. Angry</b>	0	1	2	3	4
<b>3. Worn out</b>	0	1	2	3	4
<b>4. Confused</b>	0	1	2	3	4
<b>5. Sad</b>	0	1	2	3	4
<b>6. Energetic</b>	0	1	2	3	4
<b>7. Unable to concentrate</b>	0	1	2	3	4
<b>8. Annoyed</b>	0	1	2	3	4
<b>9. Cheerful</b>	0	1	2	3	4
<b>10. Exhausted</b>	0	1	2	3	4
<b>11. Anxious</b>	0	1	2	3	4
<b>12. Terrified</b>	0	1	2	3	4

## Appendix K: Study development - visual cognitive eye tracking tasks

Extracted from - Poster presented at the ASPR Conference, Newcastle, Australia.

**Centre for Brain and Mental Health Research**

University of Newcastle, AUSTRALIA



### FACIAL EXPRESSION PROCESSING IN SCHIZOPHRENIA: ASSOCIATIONS WITH PSYCHOPATHY, SYMPTOMATOLOGY AND EMOTION RECOGNITION – STUDY DEVELOPMENT

**Ketrina Sly<sup>1</sup>, Carmel Loughland<sup>1,2</sup>, Terry Lewin<sup>1,2</sup>, Mick Hunter<sup>1</sup>.**

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<sup>2</sup>Schizophrenia Research Institute (SRI), AUSTRALIA

[ketrina.sly@hnehealth.nsw.gov.au](mailto:ketrina.sly@hnehealth.nsw.gov.au)

#### BACKGROUND:

Individuals with schizophrenia are known to exhibit marked deficits in facial expression perception<sup>1,2</sup>. Similarly, people with a history of psychopathy exhibit emotion recognition difficulties, particularly in the recognition of negative valenced emotions, such as fear and anger<sup>3,4</sup>. However, the relative contribution of psychosis and psychopathy traits to visual processing deficits remains unclear. The aim of this study is to extend previous research on emotion processing deficits in schizophrenia by assessing the contribution of coexisting psychopathy traits, as well as relationships with symptomatology, emotion dysregulation, and overall cognitive functioning. It is hypothesized that subjects with schizophrenia would exhibit a more restricted pattern of visual scanning, poorer overall emotion recognition accuracy and lower emotion responsivity, and that psychopathic traits would make an independent contribution to performance deficits.

#### PURPOSE:

This poster outlines the initial phase of the study in terms of task development. Eye-tracking tasks were devised to assess facial affect processing among individuals with schizophrenia or another major mental illness, and varying levels of coexisting psychopathy traits. Whilst previous research has demonstrated impairments in emotion recognition performance among both individuals with schizophrenia and psychopathy, eye-tracking performance and emotion responsivity has not been investigated among this population. In preparation for the main study, the feasibility of undertaking an eye-tracking task with an emotion induction component was therefore explored.

#### METHOD:

In the main study, clinical participants with a history of offending behaviour and a major mental illness will be recruited through a Mental Health Court Liaison service. For the pilot, a convenience sample of healthy control participants was recruited. Facial affect processing was assessed in terms of visual scanning, emotion recognition and emotion induction, using two separate visuo-cognitive eye tracking tasks. Both tasks involve the presentation of face stimuli, consisting of colour pictures of male and female actors depicting specific expressions<sup>5</sup>. In each task, face stimuli are presented for 6 seconds and eye movement recorded using a View Point eye tracker (60hz sampling frame). The optic assembly consists of an infrared low light camera, with retinal and corneal reflections recorded to obtain the point of fixation (error of resolution less than 0.5 degrees). A procedure for calibration of eye fixation position was conducted prior to recording. Eye tracker software ensures that stimuli appear only when the participant has maintained fixation on a centrally presented fixation point for 1000ms, thereby controlling for the initial direction of retinal attention.

In the first task, scan-paths were recorded while participants viewed 28 separate face stimuli (14 M & 14 F) depicting 7 emotions (neutral, happy, sad, surprise, angry, fear and disgust). In the second task, participants viewed separate series of 10 faces (5 M & 5 F) depicting four different categories of facial affect (happy, sad, anger or fear) presented randomly (40 images in total). Participants were instructed as follows: "During this task, I am going to show you a series of images. I would like you to use the images to feel the emotion displayed". Prior to and after exposure to each block of stimuli, participants were asked to rate the intensity of their emotion using an abbreviated version of the Profile of Mood States (POMS), which included a selected range of mood domains. The POMS presents a series of adjectives rated on 5-point Likert scales (0='not at all' to 4='extremely') to describe a participants mood "right now", and is a validated measure sensitive enough to detect changes in mood state<sup>6</sup>.

## RESULTS:

Pilot findings are reported from 11 healthy controls (mean age=37.9 years). For the emotion recognition task, both recognition accuracy and tracking parameters are reported. A total of 6 scan-path parameters were extracted, including two temporal indices (average duration and number of fixations), and two spatial indices (median distance between fixations, raw scan-path length), and two attention measures (feature vs non-feature areas). The mean recognition accuracy was 92.2%. Patterns of misattribution were consistent with earlier studies (e.g., surprise for fear, disgust for sad). Figure 1 illustrates the scan-path parameters recorded for the healthy controls. On average, there were 19.79 fixations per person, with an average duration of 260ms. The mean distance between fixations was 46.42mm, and mean scan-path length was 864.24mm. 78.92% of the time (4735ms) participants attended to the face, with 70.65% of the time (4239ms) within feature areas.

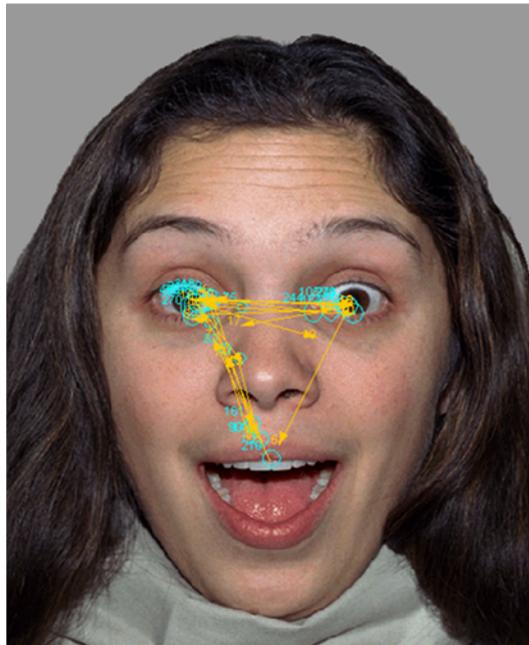


FIGURE 1: EMOTION RECOGNITION TASK - SCAN PATHS

Figure 2 displays the stimuli used in one component of the emotion induction task. We were able to successfully demonstrate change in ratings of emotion on the POMS. As illustrated in Figure 3, the largest change in POMS total Mood Disturbance (TMD) ratings occurred following the presentation of images depicting a happy facial affect [ $t_{(10)}=4.23$ ,  $p=0.002$ ], followed by anger [ $t_{(10)}=0.46$ ,  $p=0.04$ ].



FIGURE 2: EMOTION INDUCTION IMAGES

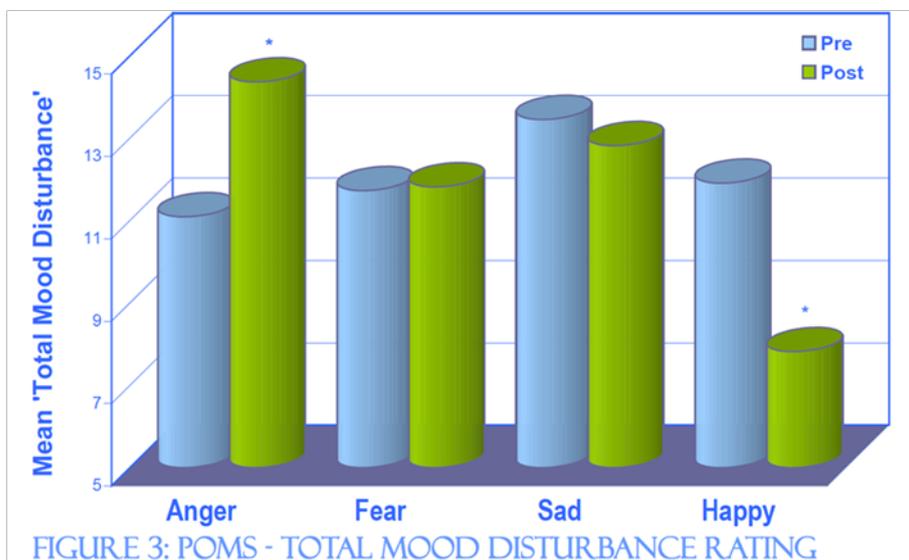


FIGURE 3: POMS - TOTAL MOOD DISTURBANCE RATING

## CONCLUSIONS:

- Patterns of misattribution during emotion recognition were consistent with previous studies.
- The temporal, spatial and attentional scan-path parameters obtained were all within expected range.
- A significant change in emotion responsivity during a novel emotion induction task was apparent following the presentation of images depicting happy and angry facial affect.
- The feasibility of incorporating a visuo-cognitive eye tracking task with an emotion induction component was demonstrated.
- This study will be one of the first to examine the relationship between schizophrenia and the impact of psychopathy on face processing using visual scanning techniques.

## REFERENCES:

1. Hooker, C., & Park, S. (2002). Emotion processing and its relationship to social functioning in schizophrenia patients. *Psychiatry Research*, 112(1), 41-50.
2. Kohler, CG & Brennan, AR (2004). Recognition of facial emotions in schizophrenia. *Current Opinion in Psychiatry*, 17(2) 81-86.
3. Fullam, R. & Dolan, M. (2006). Emotional information processing in violent patients with schizophrenia: association with psychopathy and symptomatology. *Psychiatry Research*, 141(1), 29-37.
4. Weiss, E.M., Kohler, C.G., Nolan, K.A., Czobor, P., Volavka, J., Platt, M.M., et al. (2006). The Relationship between history of violent and criminal behaviour and recognition of Facial expression of emotions in men with schizophrenia and schizoaffective disorder. *Aggressive Behaviour*, 32(3), 187-194.
5. Tottenham, N., Borscheid, A., Ellertsen, K., Marcus, D.J., Nelson, C.A. (2002). Categorization of Facial Expressions in Children and Adults: Establishing a Larger Stimulus Set. Poster presented at the Cognitive Neuroscience Society annual meeting, San Francisco
6. McNair, D. M., Lorr, M., & Derogpleman, L. F. (1971). *The Manual for the Profile of Mood States (POMS)*. San Diego, CA: Educational and Industrial Testing Services.

Appendix L: Face recognition and working memory Task C – Participant instructions

Displayed on screen during the face recognition visual-cognitive eye-tracking task.

During this task I am going to show you a series of faces, one at a time,  
I would like you to try to remember them.

At the end of the task I will show you some more pictures of faces  
and ask you to recall which ones you have already seen.

We are going to start with a calibration.

**Are you ready?**

## **Calibration**

Look at the dot until you hear a click and  
wait until the next dot appears before you move your eyes

Don't try to guess where the dot is going to move  
We need to make sure the computer is recording each time you fixate on the dot

Instructions used for immediate recall stimuli set, were repeated for delayed recall set.

Now I am going to show you some more pictures of faces, one at a time.

I want you to look at the face on each page carefully.

Say **Yes** if the face is one that I asked you to remember or **No** if it is not.

Say **Yes** if the face is one that I asked you to remember or **No** if it is not.

## **Glossary of Terms**

aINS	Anterior insula
AQoL6D	Assessment of Quality of Life
BPRS	Brief Psychiatric Rating Scale
CAQ	Childhood Adversity Questionnaire
CBMHR	Centre for Brain and Mental Health Research
CTNMH	Centre for Translational Neuroscience and Mental Health (Previous name for the CBMHR)
DERS	Difficulties in Emotion Regulation Scale
DMN	Default Mode Network
DSM-IV	Diagnostic and Statistical Manual of Mental Disorder, Fourth edition
DSM-5	Diagnostic and Statistical Manual of Mental Disorder, Fifth edition
ERPs	Event related potentials
FFA	Fusiform face area
fMRI	Functional Magnetic Resonance Imaging
GAF	Global Assessment of Functioning
hMNS	human Mirror Neuron System
HNE-LHD	Hunter New England Local Health District
HNE-MH	Hunter New England Mental Health
ICD-10	International Statistical Classification of Diseases and related health problems, 10 <sup>th</sup> Edition
IFG	Inferior frontal gyrus
IPDEQ	International Personality Disorder Examination Questionnaire
MRI	Magnetic Resonance Imaging
NMHCL	Newcastle Mental Health Court Liaison

OFC	Orbitofrontal cortex
PANSS	Positive and Negative Syndrome Scale
PCL	Psychopathy Checklist
PCL-R	Psychopathy Checklist - Revised Version
PCL: SV	Psychopathy Checklist - Screening Version
PD	Personality Disorder
POMS	Profile of Mood States
pSTS	Posterior superior temporal sulcus
RBANS	Repeatable Battery for Assessment of Neuropsychological Status
SANS	Scale for the Assessment of Negative Symptoms
SAPS	Scale for the Assessment of Positive Symptoms
SCID	Structured clinical interview for DSM-IV
SOFAS	Social Occupational Functioning Scale
sMRI	Structural Magnetic Resonance Imaging
STS	Superior temporal sulcus
TAT	Thematic Apperception Test
ToM	Theory of Mind
TPJ	Temporal-parietal occipital junction
vmPFC	ventromedial prefrontal cortex
WTAR	Wechsler Test of Adult Reading