ATTENTIONAL PROCESSING OF FACIAL EXPRESSIONS AND GAZE DIRECTION IN DEPRESSION AND FIRST-EPISODE PSYCHOSIS AS REFLECTED BY LPP MODULATION

Jonathan W. L. Kettle, Nicholas B. Allen

Abstract

Objective: Facial expressions communicate emotional states and regulate social bonds. An approach or avoidance-based valence might interact with direct or averted gaze to elicit different attentional allocation. These processes might be aberrant in major depression or first-episode psychosis and this requires empirical investigation.

Method: This study examined higher order, controlled attentional processing of emotional facial expressions (happy, neutral, angry and fearful), with direct or averted gaze, using electroencephalogram (EEG) measures of the face-elicited Late Positive Potential (LPP), in young people diagnosed with major depression or first-episode psychosis, compared with a healthy control group.

Results: In the control group, there was no evidence of increased attentional allocation to emotional facial expressions, or to facial expressions with a matching emotional expression and gaze direction. There was no evidence, in the depression or first-episode psychosis groups, for a threat-based, attentional hypersensitivity to fearful or angry facial expressions, nor for this effect to be potentiated in response to angry direct or fearful averted gaze faces. However, the absence of such effects could not be concluded due to sample size and the absence of stimulus arousal and valence ratings. Importantly, there was significantly increased attentional allocation in the first-episode psychosis group to facial expressions regardless of emotional expression or gaze direction, compared to both the depression and control group.

Conclusions: There might be an attentional hypersensitivity to facial expressions regardless of emotional expression or gaze direction in first-episode psychosis.

Key words: attention, facial expressions, late positive potential, depression, first-episode psychosis

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1. Introduction

Facial expressions are highly salient, socially and biologically significant stimuli (Palermo and Rhodes 2007), that represent a universal system of expressing emotion within social interactions (Jack et al. 2016). They serve vital informational, evocative and incentive functions (Keltner and Kring 1998), and the correct identification of facial expressions influences a range of important adaptive functions (Bora et al. 2006, Marsh et al. 2007). Broadly understood, facial expressions serve functions of emotional expression and the regulation of social bonds (Dimberg 1982, Hess and Fischer 2013).

1.1. Facial expression and gaze interaction

While faces convey rich information about the sender's age, gender, familiarity, emotional state, intentions and mental state, the direction of the eyes – that is, gaze – is of central importance in providing information relating to emotional and mental states (Baron-Cohen et al. 1997, Emery 2000). Within social dyadic interactions, direct (mutual gaze with each head directly facing the other) or averted gaze (the eyes and head both congruently oriented a significant degree to the left or right of the observer) may occur, which interacts with the facial expression to modulate the

intensity and meaning of the stimulus. Emotional facial expressions can be categorized on two basic dimensions of positive/approach or negative/withdrawal related behaviour (Davidson et al. 1990, Davidson and Hugdahl 1995). Studies have observed that a "matched" combination of direct eye gaze with an approach-based emotion (e.g., happiness or anger), or averted eye gaze with an avoidance-based emotion (e.g., fear), results in improved categorisation time, a higher likelihood of categorising an emotional expression correctly, and a heightened perceived emotional intensity of the expression (Adams Jr and Kleck 2003, Adams and Kleck 2005).

1.2. Attentional processing of facial expressions - the late positive potential (LPP)

One psychophysiological measure of attention to facial expressions is the late positive potential. The LPP (Bradley et al. 2007), maximal between 400 and 1000ms, is a relatively novel picture-elicited component and is potentiated over midline centro-parietal sites during extended passive viewing of unpleasant or pleasant relative to neutral pictures (Cuthbert et al. 2000, De Cesarei and Codispoti 2006, Keil et al. 2002, Keil et al. 2001, Schupp et al. 2000, Schupp et al. 2003). LPP amplitude has been found to be most potentiated in response to highly arousing, unpleasantly or pleasantly valenced pictures, such as pictures of mutilations, animal threat, opposite sex nudes or erotic couples (Schupp et al. 2000, Schupp et al. 2004a). LPP amplitude is modulated by the increased affective valence and increased arousal of the picture (Cuthbert et al. 2000, Schupp et al. 2000, Schupp et al. 2004b). It is proposed that potentiated LPP amplitude reflects facilitated, higher order controlled cognitive processing, stimulus evaluation and memory storage (Rosler 1986), which might be most pronounced for stimuli that have evolutionary significance such as those related to predation and reproduction (Bradley 2009). Thus the LPP is considered to reflect motivated attention to such significant stimuli (Hamm et al. 2003). Such a controlled attentional process, as measured by the LPP, might also be measured by other investigative techniques, such as visual scan path studies that assess overt, controlled attentional allocation to emotive or motivational stimuli (Savulich et al. 2012).

Empirical studies support the presence of a face-elicited LPP component, maximal between 600 to 1000ms, similar to that found in viewing of affective pictures, and that the LPP component is potentiated for emotional expressions of anger, happiness, fear, disgust, sadness or surprise (Eimer and Holmes 2007, Krolak-Salmon et al. 2001, Orozco and Ehlers 1998, Schupp et al. 2004c). Some studies have suggested this is underpinned by arousal ratings of faces (Eimer et al. 2003), others by valence ratings (Schupp et al. 2004b). One study has observed that mid-LPP amplitude (450ms to 650ms) increased with increasing degrees of affective expression of the face. Underlying this, the mid-LPP component was positively correlated with arousal ratings whereas the late-LPP component, which was significantly potentiated for angry compared to happy faces, was negatively correlated with valence ratings (that is, as faces moved from happy to angry in valence ratings the late-LPP component increased; Duval et al. 2013). Though not definitive, one possible explanation for this data is that the mid-LPP reflects stimulus arousal whereas the late-LPP reflects stimulus valence. The LPP component overall suggests increased higher order cognitive processing, evaluation and memory storage of pan-cultural facial expressions, according to both arousal and valence properties of the face.

No published studies have examined LPP modulation by facial expression and gaze direction. However, based upon the proposition that matched facial expressions will have heightened salience, it might be that matched facial expressions will recruit increased higher order cognitive processing, stimulus evaluation and memory storage, reflected in potentiated LPP amplitude.

Given the relative infancy of the research examining the LPP of attentional processing of facial expressions, and the interaction with gaze direction, it is not surprising that even less research has examined how this attentional process might be altered in people experiencing mental disorders. Any such abnormal processing of facial expressions might be involved in aspects of the symptoms and functional deficits associated with mental disorders.

1.3. Facial reactivity and attentional processing in major depression

It has been proposed that depression might involve a hypersensitivity to social stimuli (Allen and Badcock 2003, 2006). This contrasts with a hyporeactivity to positive stimuli but not a hyper-reactivity to negative stimuli in depression (Allen et al. 1999, Berenbaum and Oltmanns 1992, Canli et al. 2004, Rottenberg et al. 2002, Schaefer et al. 2006, Sloan et al. 2001). Allen and Badcock have posited that depression, and particularly anhedonia, has evolved within evolutionary pressures of social living to reduce threat to conspecifics and minimize the possibility of exclusion from the social group via a hypersensitivity to social stimuli, particularly relating to threat (with a hyporesponsivity to such stimuli remaining entirely feasible). The only study to have examined such sensitivity to facial expressions, a highly salient class of social stimuli, via attentional processing, measured by the LPP, in depressed individuals contradicts this proposal, as it observed an absence of the LPP potentiation to fearful or angry faces observed in the control group (Foti et al. 2010). This suggests possibly an absence of an increased higher order cognitive processing, evaluation and memory storage of threatening facial expressions during depression. However, it must be clearly noted that this cannot be reliably concluded due to the very minimal amount of research examining this issue. No study has examined whether such an attentional processing bias occurs differentially for matched approach or avoidant facial expressions in people diagnosed with major depressive disorder.

1.4. Facial reactivity and attentional processing in psychosis

Psychosis is hereafter used in this paper to include the range of psychotic disorder diagnoses in the Schizophrenia and Other Psychotic Disorders section in the Diagnostic and Statistical Manual (DSM) IV (American Psychiatric Association 2000), and in the sections Schizophrenia Spectrum and Other Psychotic Disorders, and Catatonia, of the DSM 5 (American Psychiatric Association 2013), excluding substance-induced psychotic disorders. Cognitive models have posited an attentional bias for threat-related stimuli as both a causal and maintaining factor in psychosis (Freeman et al. 2013, Freeman et al.

2002). Thus the term psychosis as used in this paper is not limited to schizophrenia. A review of cognitive, psychophysiological, neurocognitive and neuroimaging studies (Green and Phillips 2004) supported an attentional processing bias for threatening social stimuli (such as angry and fearful faces) in humans, with the proposal that such subtle alterations in a corresponding neural system might reflect abnormally heightened attentional processing of threat-based stimuli in clinical paranoia. However, a recent review, comprising mostly visual search, visual scan path and detection time studies in people with psychosis found evidence suggesting an avoidance processing bias rather than a hypervigilance (Savulich et al. 2012). Savulich and colleagues suggested that such eye tracking data might reflect overt attentional control away from threatening stimuli, in contrast to behavioural reaction time data that might reflect covert attentional processing. Disentangling the question of whether such avoidance might occur precisely because of a preceding hypervigilance is difficult without studies designed specifically for this purpose, for example with combined eye tracking and psychophysiological measures. Importantly, the studies reviewed by Savulich et al. did not use psychophysiological measures of attentional allocation.

Only two studies have examined an attentional bias while processing facial expressions in psychosis using the LPP (Bediou et al. 2007, Johnston et al. 2005). Bediou and colleagues observed an absence of an LPP waveform in participants diagnosed with schizophrenia, aged between 19 and 44 years. Johnston and colleagues did not observe an interaction between group and facial expression for the LPP in response to presentation of facial expressions for 400ms to 11 participants with remitted schizophrenia with an average age of 34 years (ranging between 18 and 45 years), compared to 15 healthy control participants. However, they did observe a significantly attenuated late positive potential component in the psychosis group compared to controls. Thus, this limited published data provide tentative support for a proposed reduction in higher order, attentional processing of facial expressions in psychosis, which is contrary to a synthesis of cognitive, psychophysiological, neurocognitive and neuroimaging studies (Green and Phillips 2004), but consistent with the proposal of avoidance of attentional processing of facial expressions (Savulich et al. 2012). Overall, drawing reliable theoretical conclusions regarding an attentional hypersensitivity or avoidance to facial expressions in psychosis is hampered by the very limited psychophysiological data available. Furthermore, neither Bediou et al., nor Johnston et al., explicitly tested LPP reactivity in a first-episode psychosis sample (as this study has), which introduces further confounds of the effects of number of psychotic episodes and duration of illness upon any such attentional processing biases. Lastly, no study has examined whether such an attentional processing bias occurs differentially for matched approach or avoidant facial expressions in people diagnosed with psychosis.

1.5. The present study

This study aims to examine attentional processing, via the LPP, while viewing facial expressions, as moderated by gaze direction, in individuals with major depressive disorder or first-episode psychosis, compared to individuals with no lifetime history of mental disorder.

In line with motivated attention to emotional stimuli (Hamm et al. 2003), it was hypothesized in the non-clinical group that face-elicited LPP amplitude will be significantly potentiated for happy, angry and fearful compared to neutral faces, reflecting increased motivated attention to emotional stimuli. Furthermore, it was hypothesized that this potentiation would be significantly greater for matched facial expressions, with happy and angry faces with direct compared with averted gaze, and fearful faces with averted compared with direct gaze.

In the depression group, consistent with attentional hypersensitivity to threatening, social stimuli during depressed states (Allen and Badcock 2003), it was hypothesized that face-elicited LPP amplitude will be significantly more potentiated compared to the non-clinical group for angry and fearful compared to neutral faces. In the psychosis group, consistent with a threat-based attentional processing bias (Freeman et al. 2013, Green and Phillips 2004), it was hypothesized that face-elicited LPP amplitude would be significantly more potentiated compared to the non-clinical group for angry and fearful compared to neutral facial expressions. Finally, it was hypothesized that in the depression and psychosis groups, the LPP potentiation for matched facial expressions (happy or angry with direct compared with averted gaze; fearful with averted compared with direct gaze) would be significantly greater compared to the non-clinical groups, again given the proposed attentional hypersensitivity to threatening, social stimuli in both disorders.

2. Method

2.1. Participants

Non-clinical participants in the control group were a sample drawn from the general community and clinical participants were drawn from the mood disorder (Youthscope) and first-episode psychosis (Early Psychosis Prevention and Intervention Clinic; EPPIC) clinics at Orygen Youth Health, Melbourne, Australia.

Non-clinical participants had no lifetime history of case level DSM-IV (American Psychiatric Association 2000) defined mood, psychotic, anxiety, eating, somatisation or substance use disorder. This was established using a screener version of the Structured Clinical Interview for Axis I DSM-IV Disorders (SCID-I; First et al. 2001) that was adapted to include questions assessing psychotic and mood disorders. Potential participants were also excluded if they were taking psychotropic medication.

Clinical participants were assigned to the depression or psychosis group based upon interviews using the SCID-I and reviews of clinical notes by two provisional psychologists enrolled in a graduate level Clinical Psychology training program, under the supervision of an experienced clinical psychologist. All SCID-I interviews were reviewed by both clinicians, in conjunction with clinical notes, to reach a diagnostic consensus. Participants were included in the depression group if they were experiencing Major Depressive Disorder (either current or in partial remission). Participants were included in the 'partial remission' category only if they were experiencing residual depressive symptoms following a major depressive episode, as opposed to having achieved full remission of depressive symptoms for less than two months' duration. Participants were excluded from the depression group if they were diagnosed with Bipolar Disorder (Type I or II), Major Depressive Disorder with Psychotic Features

or any DSM-IV defined psychotic disorder. Participants were included in the psychotic group if they currently met criteria for a DSM-IV defined psychotic disorder. Participants were not excluded from the psychotic group if they had significant, co-occurring mood symptoms or a diagnosable mood disorder. This would have created an unrepresentative psychosis sample, limiting the external validity results, given the high co-occurrence of depressive symptoms in psychotic disorders (Hafner et al. 2005), possible common etiological factors (Smith et al. 2006, Yung et al. 2007), and empirical evidence that depression is a symptom dimension present in first episode psychosis patients (McGorry et al. 1998). Participants were excluded from all three groups if they reported any history of neurological illness.

The final experimental sample consisted of 16 non-clinical, 14 depression and 15 psychosis participants. **Table 1** shows basic demographic information across and within groups.

The groups were equivalent on demographic measures, except for a significant preponderance of females in the depression group and males in the psychosis group. The supplementary table lists each clinical participant's group status, current and past DSM-IV diagnoses, and medication type and daily dosage at the time of testing. Chlorpromazine equivalents have been calculated (Andreasen et al. 2010, Leucht et al. 2015), with 25mg of Risperidone depot every two weeks equivalent to 2mg daily oral Risperidone used (Chue et al. 2005).

Table 2 shows the diagnostic composition and medication status of all clinical participants.

All participants in the depression group met criteria for Major Depressive Disorder. Six met full criteria at the time of testing and eight met criteria for 'in partial remission', the latter always reflecting residual depressive symptoms. No participant in the depression group had any lifetime history of diagnosable psychotic disorder. In the psychosis group, 12 of the 15 participants (80%) met criteria for schizophrenia or

Table 1. *Descriptive characteristics across and within experimental groups*

Measure	Across Groups N = 45	Non-Clinical n = 16	Depression n = 14	Psychosis n = 15	Statistic	p
Age	19.70 (3.15)	19.33 (2.92)	18.98 (2.88)	20.79 (3.60)	$F(2, 42) = 1.376$	0.264
Age range	15.51 to 26.88	15.54 to 24.31	15.51 to 25.14	15.68 to 26.88		
Gender	M = 22, F = 23	M = 8, F = 8	M = 3, F = 11	M = 11, F = 4	$\chi^2 (N = 45) = 7.820$	0.020*
Years education	12.09 (1.95)	12.50 (2.0)	11.36 (1.23)	12.33 (2.36)	$F(2, 42) = 1.489$	0.237
Lifetime diagnoses	1.53 (1.18)	----	2.21 (1.05)	2.53 (1.24)	$t(27) = 0.743$	0.464
Current diagnoses	1.11 (0.81)	----	1.86 (0.79)	1.60 (0.74)	$t(27) = 0.919$	0.366
Taking psychotropic medication	23	----	11	12	$\chi^2 (N = 29) = 0.009$	0.924

Note. Numbers are means with standard deviations in parentheses. M = males, F = females. * p < 0.05

Table 2. *Diagnostic and medication status for clinical participants*

ID	Group	Current DSM-IV Diagnoses	Past DSM-IV Diagnoses	Psychotropic Medication
103	Dep	MDD, Single Episode, Moderate		Fluoxetine 20mg
105	Dep	MDD, Single Episode, Continuous, Moderate	Panic Disorder without Agoraphobia	Venlafaxine 225mg
107	Dep	MDD, Recurrent, In Partial Remission; Social Phobia, Mild		None
108	Dep	MDD, Recurrent, Mild; Panic Disorder without Agoraphobia, Moderate		Fluoxetine 20 mg, Temazepam 10mg PRN
109	Dep	MDD, Single Episode, Moderate; PTSD, Mild; Anorexia Nervosa, Binge-eating/Purging Type, In Partial Remission		Sertraline 100mg, Quetiapine 200mg (Chlorpromazine equivalent 127mg)
111	Dep	MDD, Single Episode, In Partial Remission		Fluoxetine 30mg; Zolpidem 1mg PRN
112	Dep	MDD, Single Episode, In Partial Remission		Fluoxetine 20mg
113	Dep	MDD, Recurrent, Moderate; Panic Disorder with Agoraphobia, Mild; Social Phobia, Moderate	Bulimia Nervosa, Purging	None
114	Dep	MDD, Recurrent, In Partial Remission		Fluoxetine 20mg
115	Dep	MDD, Recurrent, In Partial Remission; Bulimia Nervosa, Purging Type, In Partial Remission	Panic Disorder with Agoraphobia	Sertraline 100mg
116	Dep	MDD, Recurrent, In Partial Remission; PTSD, Mild		Fluoxetine 30mg

Table 2. *Continue*

118	Dep	MDD, Recurrent, In Partial Remission; Social Phobia, Mild	Cannabis Dependence, Early Full Remission	None
119	Dep	MDD, Single Episode, In Partial Remission; OCD, Mild		Fluoxetine 40mg
120	Dep	MDD, Single Episode, Moderate; PTSD, Moderate; Alcohol Dependence, With Physiological Dependence, In Partial Remission	Cannabis Dependence, Sustained Full Remission	Fluoxetine 20 mg; Zolpidem 1mg
201	Psy	Schizophreniform Disorder, Provisional, With Good Prognostic Features	Cannabis Dependence, Sustained Full Remission; Ecstasy Dependence, Sustained Full Remission	Risperidone Consta 50mg (Chlorpromazine equivalent 341mg), Fluoxetine 20mg, Olanzapine 10 to 20mg wafers PRN (Chlorpromazine equivalent 241mg to 541mg PRN), Diazepam 10 mg PRN
202	Psy	Schizophrenia, Paranoid Sub-Type, Moderate; Panic Disorder without Agoraphobia, Mild; Alcohol Abuse	Amphetamine Dependence, Early Full Remission	Quetiapine 500 mg (Chlorpromazine equivalent 460mg)
204	Psy	Schizophrenia, Paranoid Sub-Type, Moderate, Continuous		Amisulpride 400mg (Chlorpromazine equivalent 254mg), Amitriptyline 125mg
205	Psy	Schizophrenia, Paranoid Sub-Type, Moderate, Continuous; Cannabis Dependence, With Physiological Dependence, Mild	Alcohol Dependence, Sustained Full Remission; Stimulant Dependence, Early Partial Remission; Opioid Dependence, Sustained Full Remission; Cocaine Dependence, Sustained Full Remission	Olanzapine 10mg (Chlorpromazine equivalent 241mg); Zolpidem 10mg
206	Psy	Schizophrenia, Single Episode in Partial Remission, Residual Type, Mild	Cannabis Dependence, Early Full Remission	None
207	Psy	Psychosis NOS	Cannabis Dependence, Early Partial Remission; Hallucinogen Abuse	Olanzapine 5mg (Chlorpromazine equivalent 90mg); Escitalopram 10mg
208	Psy	Schizophrenia, Paranoid Subtype, Moderate, Continuous; Cannabis Dependence, With Physiological Dependence, Moderate		None
209	Psy	Schizophrenia, Residual Type, Mild	Cannabis Dependence, Sustained Full Remission	Lithium Carbonate 950mg, Amisulpride 200mg (Chlorpromazine equivalent 97mg)
210	Psy	Schizophrenia, Paranoid Subtype, Moderate, Continuous	Cannabis Dependence, Early Full Remission	Fluoxetine 20mg; Risperidone 3mg (Chlorpromazine equivalent 255mg)
211	Psy	Schizophrenia, Severe, Paranoid Subtype, Continuous with Prominent Negative Symptoms; MDD, Recurrent, In Partial Remission	Cannabis Dependence, Early Full Remission	Clozapine 600mg (Chlorpromazine equivalent 768mg); Citalopram 60mg
212	Psy	Schizophrenia, Residual Subtype; Cyclothymic Disorder; Panic Disorder with Agoraphobia, Mild		None
213	Psy	Schizoaffective Disorder, Depressive Subtype, Moderate; PTSD, Moderate		Risperidone 2mg (Chlorpromazine equivalent 169mg); Citalopram 40 mg
214	Psy	Schizophrenia, Paranoid Subtype, Moderate		Olanzapine 15mg (Chlorpromazine equivalent 391mg); Diazepam 1mg PRN
215	Psy	Schizophrenia, Residual Type, Moderate, Single Episode with Prominent Negative Symptoms	Panic Disorder without Agoraphobia	Quetiapine 100mg (Chlorpromazine equivalent 16mg)
216	Psy	Psychotic Disorder NOS, Current; Agoraphobia without Panic Disorder, Mild		Risperidone 1mg (Chlorpromazine equivalent 82mg)

Note. Dep = Depression, Psy = Psychosis; MDD = Major Depressive Disorder; OCD = Obsessive Compulsive Disorder; PTSD = Post Traumatic Stress Disorder; NOS = Not Otherwise Specified

schizophreniform disorder. Two participants met criteria for psychotic disorder NOS and one for schizoaffective disorder, with this participant experiencing depressive but no manic episodes. All participants in the psychosis group were experiencing their first clinical episode of psychosis, with depressive disorders in the psychosis group only reaching diagnosable levels in three participants. Therefore, there was very minimal diagnostic overlap between the two groups.

Table 3 shows the mean total scores for depressive symptoms, positive and negative psychotic symptoms for both the depression and psychosis group.

As shown in **table 3**, there was no significant difference between the psychosis and depression group

and unusual thought content (no depression participants rated above 1 on these two levels). The psychosis and depression group were not differentiated by scores on the SANS scales. This is not surprising given phenomenological similarities of negative symptoms of anhedonia/asociality and avolition/apathy with depression, especially as low positive affect is at the core of anhedonia (Blanchard et al. 1994, Gooding et al. 2002). Negative symptoms and depression may also share underlying, causal mechanisms, such as demoralisation and a repetitive cycle of social withdrawal and diminished positive reinforcement from the environment, or poor self-esteem and a cognitive

Table 3. *Mean total and subscale scores for depressive, positive and negative symptoms within groups*

Measure	Non-Clinical Group $n = 16$	Depression Group $n = 14$	Psychosis Group $n = 15$	Statistic	p	Partial η²	Group Difference	Tukey p value
CESD Total	10.25 (6.12)	34.00 (10.25)	28.13 (10.92)	$F(2, 42) = 27.245$	< 0.001	0.565	NC < D	< 0.001[++]
							NC < P	< 0.001[++]
SANS								
Global affective flattening	---	0.50 (1.01)	1.00 (1.59)	$t(27) = 0.994$	0.329			
Global alogia	---	0.14 (0.52)	0.53 (1.01)	$t(27) = 1.307$	0.202			
Global avolition / apathy	---	1.65 (1.01)	1.53 (1.39)	$t(27) = 0.239$	0.813			
Global anhedonia / asociality	---	2.14 (1.23)	1.87 (1.12)	$t(27) = 0.631$	0.533			
Global attention	---	0.43 (0.94)	0.67 (0.81)	$t(27) = -0.731$	0.471			
BPRS								
Mean Positive Symptoms	---	1.10 (0.22)	2.38 (0.93)	Mann Whitney $U = 20.00$	< 0.001		P > D	
Grandiosity	---	1 (0)	1.20 (0.77)					
Hallucinations	---	1.29 (0.82)	3.87 (2.40)	Mann Whitney $U = 42.00$	0.002		P > D	
Unusual Thought Content	---	1 (0)	3.20 (1.51)					
Conceptual Disorganisation	---	1.15 (0.52)	1.27 (0.58)	Mann Whitney $U = 92.50$	0.363			

Note. CESD = Centre for Epidemiological Studies Depression Scale; SANS = Schedule for the Assessment of Negative Symptoms; BPRS = Brief Psychiatric Rating Scale – Expanded Version; NC = Non-Clinical group, D = Depression group, P = Psychosis group. Means are reported with standard deviations in brackets. * $p < 0.1$ ** $p < 0.05$ + $p < 0.01$ ++ $p < 0.001$

on total depression scores (both were significantly greater, however, than the non-clinical group). This is consistent with the elevated levels of depressive symptoms during first-episode psychosis (Hafner et al. 2005), common etiological and mutually reinforcing factors (Freeman and Garety 2003), as well as depression representing a continuous symptom dimension in first-episode psychosis (McGorry et al. 1998). No participant in the non-clinical group scored above 16, the cut-off for being at risk for major depression. The psychosis group was differentiated from the depression group by significantly greater total positive symptoms and hallucinations, as well as greater levels of grandiosity

bias towards negatively valenced information (Guillem et al. 2005, Morrison et al. 2004).

2.2. Design

The experimental paradigm employed a between-participants factor of group (non-clinical, depression, psychosis) and three within-participants factors of facial expression (happy, neutral, angry, fearful), gaze direction (0 or 30 degrees) and scalp site (Fz, Cz, Pz). The dependent, psychophysiological variable was face-elicited LPP amplitude (μV).

2.3. Measures

2.3.1. Symptom questionnaires.

2.3.1.1. The Centre for Epidemiological Studies Depression Scale (CESD).

The CESD (Radloff 1977) is a 20 item, self-report questionnaire measuring depression in an adult population. Respondents rate the degree to which they have experienced items relating to cognitive, somatic and affective aspects of depression in the last week on a four point Likert scale, ranging from 0 = 'Very little of the time/Rarely/Never' to 3 = 'Most or all of the time'. Research suggests forming a global depression score from this instrument by summing the 20 items (Skorikov and Vandervoort 2003). Radloff (1977) initially recommended that scores of 16 or greater suggested clinically significant depressive symptoms. However, contemporary research suggests that this may cause too many false positive cases in adolescent (Roberts et al. 1990) and university student samples (Santor et al. 1995), with some researchers suggesting a higher cut-off score of 24 in adolescent samples (Roberts et al. 1991). In Caucasian adults, the scale has shown good reliability, with Chronbach's alpha = .85, and based upon a cut-off score of 16, sensitivity and specify regarding major depression were above 80% and 90% respectively (Zimmerman and Coryell 1994). The CESD has been shown to evidence specificity and sensitivity equivalent to the Beck Depression Inventory-II (Beck et al. 1996) for current major depression as defined by the DSM-IV in university students aged 18 to 21 (Shean and Baldwin 2008). The reliability of the scale in this study was, in the non-clinical group, Chronbach's alpha = .684, in the depression group, Chronbach's alpha = .824, and in the psychosis group, Chronbach's alpha = .655.

2.3.2. Psychiatric interviews

2.3.2.1. The Structured Clinical Interview for DSM-IV Axis I Disorders.

The SCID-I (First et al. 2001) is a structured interview ascertaining psychiatric history and treatment, functioning and psychosocial context, and lifetime case level mental disorder as defined by the DSM-IV. Modules A to I were used in the interviewing of clinical participants (Module J, detailed 'Optional Disorders', was excluded). Non-clinical participants were interviewed with an overview section of the SCID-I that was adapted to cover screening questions for diagnoses contained in Modules A to I. Importantly, this screened for psychotic and mood disorders to enhance group differentiation. The SCID-I for DSM IV disorders has been shown to possess moderate to excellent inter-rater reliability (Lobbestael et al. 2011).

2.3.2.2. The Brief Psychiatric Rating Scale (BPRS) Expanded Version.

The BPRS Expanded Version (Lukoff et al. 1986) is a 24 item, semi-structured interview that assesses psychopathology, including psychotic symptoms, for a duration of time specified by the researcher. Positive psychotic symptoms over the two weeks prior to psychophysiological testing were assessed. The BPRS scales combine frequency and severity in the symptoms ratings, such that each item is rated on a 1 ('Not present') to 7 ('Extremely Severe') Likert scale, with the interviewer choosing the highest anchor point that applies to either frequency or severity. Regarding

validity, a meta-analysis of the BPRS factor structure (Shafer 2005) suggests a Positive Symptom subscale with four items: unusual thought content, conceptual disorganisation, hallucinations and grandiosity. The BPRS has shown good reliability coefficients across a range of studies (Hedlund and Vieweg 1980). This study used a mean score for Positive Symptoms, formed from averaging scores for each symptom dimension of unusual thought content, conceptual disorganisation, hallucinations and grandiosity (ranging from 1 to 7). The reliability of the four item total scale in this study in the psychosis group was Chronbach's alpha = .433. A four item, positive symptom score was used in this study rather than conducting the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen 1984), because this would have led to an excessively lengthy testing procedure.

2.3.2.3. The Scale for the Assessment of Negative Symptoms (SANS).

The SANS (Andreasen 1982) is a 25 item, semi-structured interview in which participants are rated on the severity of symptoms over the last two weeks on five subscales (Affective Flattening, Alogia, Avolition/ Apathy, Anhedonia/Asociality, Attention), using a six point Likert scale, where 0 = 'Not present' and 5 = 'Severe'. Items within each symptom dimension are rated from 0 to 5, and a global rating for each of the symptom dimensions is also made. Initial interviews with 26 patients diagnosed with schizophrenia who were admitted to the University of Iowa Psychiatry Hospital yielded Chronbach's alpha coefficients ranging from .632 to .814 for the five subscales, indicating questionable to good reliability. Multidimensional scaling has supported the validity of the SANS as a measure of negative symptoms in psychosis (Minas et al. 1992). The reliability of this scale in this study was, in the depression group, Chronbach's alpha = .844, and in the psychosis group, Chronbach's alpha= .910. Andreasen recommended using the global ratings of each symptom dimension (as opposed to summing items within each dimension, as item correlations with subscale scores suggested that tau-equivalence may not be present), and summing the global ratings to form a summary score that assesses the severity of the negative syndrome complex as a whole. The reliability of this summed scale total from the five global ratings in this study was, in the depression group, Chronbach's alpha = .618, and in the psychosis group, Chronbach's alpha= .691.

2.3.3. Stimuli

Stimuli were colour photographs of emotional facial expressions. Sixty-four colour pictures of male and female adult posers depicting 'neutral', 'happy', 'angry' and 'fearful' expressions were selected from a standardised set of facial affect pictures (Mazurski and Bond 1993). For half the neutral, happy, angry and fearful facial expressions the poser's expression was directed at the viewer (0-degree gaze), while on the other half the expression was turned away from the viewer (30-degree gaze). Each of four male and four female posers appeared in each of three of the four expression conditions (happy, neutral, angry) and the two gaze conditions (0-degree and 30-degree gaze). However, fearful pictures were not able to be precisely counterbalanced in this way. The original set of fearful faces contained only seven pictures of adults posing fearful facial expressions, with one female model and three male models. One picture of a female posing a

fearful facial expression at 0 degrees in the original set had an inter-rater agreement of only 53%, and thus this picture was excluded. Therefore, the eight fearful facial expressions at 0 degrees comprised eight male posers (two different models each repeated four times), and the eight fearful facial expressions at 30 degrees comprised two female posers (one model repeated twice) and six male posers (one model repeated four times and another model repeated twice). Across the 64 pictures, 59% were male pictures and five male models were used, with one appearing 2 times, two appearing 6 times, one appearing 10 times and one appearing 14 times. Four female models were used, with three appearing 6 times and one appearing 8 times. All pictures were selected on the basis of high inter-rater agreement for expression categorisation in the original validation of the series (Mazurski and Bond 1993). The 64 pictures were then doubled and arranged in 4 blocks of 32, such that each block included counterbalanced distributions of facial expression and gaze direction. However, due to the gender imbalance with fearful faces, each block contained a marginally different percentage of models and their gender. These four blocks were then arranged into two orders, one the reverse of the other. These two orders were then randomised within groups.

2.4. Apparatus, physiological measures and data reduction

Physiological measures were amplified through a Grass Model 12-A5 amplifier and recorded using a Grass Model 12 Neurodata Acquisition System linked to two interconnected IBM compatible Pentium-MMX 233 MHz computers. The experiment was conducted using the Visual Psychophysiological Monitor software package, Version 11.0 (Cook 2000). The pictures were presented by the 'slave' computer on a Sony Trinitron Multiscan 500PS 21-inch monitor screen. The monitor was placed at a distance of 1 metre from the participant so that the pictures covered approximately 20 degrees of visual angle. The other, 'master' computer controlled the timing of stimulus presentation and recorded all physiological data. Physiological data were analysed off-line.

2.4.1. Late Positive Potential

The electroencephalogram (EEG) was recorded from 3 midline sites based on the international 10-20 system – frontal (Fz), central (Cz), and parietal (Pz), with linked earlobes as the reference and forehead as the ground. Vertical electrooculography (EOG) was recorded from electrodes placed above and below the right eye, and horizontal EOG from electrodes placed at the outer canthi of each eye. These measures were used for correcting eyeblink and eye movement artefacts. The raw EEG and EOG signals were amplified by a factor of 10,000 and bandpass filtered for 0.1 to 30 Hz activity. Data were collected at 100Hz from 2000ms before picture onset through to the end of the 6000ms picture presentation period. Data chunks were then extracted in Neuroscan, from 150ms before picture onset to 1000ms after picture offset, and converted to μV from A/D units. EEG data were purified and analysed in Neuroscan v. 4.3. All channels were baseline corrected to the mean of the 150ms baseline period preceding picture onset. In order to correct for vertical ocular artefacts, the vertical EOG channel was then examined within each participant to identify an average eyeblink duration. An eye movement correction algorithm (Semlitsch et al. 1986) was then applied within participants to each trial

for each EEG channel. After this, each trial within each participant was inspected for saturation (trials in which EEG amplitude exceeded +- 75 μV) and saturated trials excluded. Event-related potential waveforms were then computed by averaging trials at each scalp site within conditions of facial expression and gaze direction.

A principal components analysis with Promax rotation (Kappa = 4) was run to determine areas from picture onset to 10,000ms post-picture onset with maximal variation, thereby indicating ERP components. Examination of the scree plot indicated that a five-component solution was the most appropriate solution to the data. The components were used to determine the time window for the LPP – 500 to 750ms post picture onset, similar to other studies examining timing of the picture elicited LPP (Schupp et al. 2000, Schupp et al. 2004c), and that straddles both the mid and late-LPP components of a recent face-elicited LPP study (Duval et al. 2013). The LPP was examined across Fz, Cz and Pz scalp sites and if any significant interaction occurred with scalp site and emotion, gaze or group, then this was followed up with further analyses within scalp sites.

2.5. Procedure

Non-clinical participants were recruited through advertisements placed in community newspapers and at community centres, combined with a 'snowballing' approach whereby participants were asked if they knew people who were amenable to being contacted about research participation. All clinical participants were recruited through initial discussions with treating case managers at Orygen Youth Health, Melbourne, Australia. The authors complied with APA ethical standards in the treatment of participants, and the research was carried out according to the National Statement on Ethical Conduct in Research Involving Humans (June 1999) produced by the National Health and Medical Research Council of Australia. The research was also approved by the Human Research Ethics Committee of The University of Melbourne as well as the Mental Health Research and Ethics Committee of Melbourne Health (MHREC 2004.079). The research was also conducted in accordance with the Helsinki Declaration of 1975, as revised in 2000.

Non-clinical participants were administered the SCID-I screener over the telephone, after giving verbal informed consent to participate in the research project. All participants individually attended the testing session at Orygen Youth Health. They read a plain language statement, gave written informed consent, and consent of a parent or legal guardian was obtained for participants aged under 18. Clinical participants were then administered the SCID-I, BPRS items and SANS before psychophysiological testing. Electrodes were then attached to participants' faces, heads and fingers. Participants then sat in a chair in a separate room, facing a computer screen. Participants were told to pay attention to the facial expressions that would be displayed on the computer screen. Then 128 pictures of facial expressions were presented on the computer screen. After the first or third blocks, participants completed the CESD, and were offered a 10-minute rest after the second block. The CESD was counterbalanced between the first and third blocks in order not to induce an order of presentation effect. Each picture was shown for 6000ms, followed by a 7000ms or 9000ms ITI in order to reduce the predictability of stimulus presentation. The next picture was presented after this ITI. After presentation of 128 pictures, the electrodes

were removed and participants were debriefed and reimbursed $50 for their involvement.

2.6. Statistical Analyses

Statistical significance levels used a criterion of $p \le 0.05$. Exact p values were reported to avoid dichotomous decision making and promote more precise interpretation of results (Wright 2003). Partial η^2 was reported to indicate effect sizes. Normality of variables was assessed by a combination of inspection of histograms and whether observed skewness exceeded a critical level of $3.29*\text{sqrt}(6/n)$. Univariate outliers were data points 3.29 standard deviations greater or lesser than the mean. Univariate outliers were brought in to be +- 0.1 unit greater or lesser than the next case in the distribution, such that the interval but not ordinal value of the case changed. Multivariate outliers were identified as cases with maximum Mahalanobis distance values exceeding a critical χ^2 value (df = number of independent variables, $p = 0.001$). When variable transformation or manipulation of outliers was necessary, rationale was given prior to that variable's analysis (Tabachnick and Fidell 1996).

Hypotheses were tested using a mixed factor, four-way analysis of variance (ANOVA; expression (4) x gaze (2) x group (3) x scalp site (3)) for LPP amplitude. Only significant main effects or interactions were followed-up with further ANOVAs or post-hoc comparison of means. Bonferroni post-hoc correction was applied to all comparisons between means following significant main effects in ANOVAs to reduce Type I error. For significant interactions, ANOVAs were run within the levels of one of the significant interaction factors.

3. Results

3.1. Late Positive Potential

No participant was excluded from face-elicited LPP analyses. The final group numbers for face-elicited ERP analyses were: non-clinical group $n = 16$, depression group $n = 14$, psychosis group $n = 15$. Face-elicited LPP waveforms for each expression by gaze condition within participants were based upon an average of 13.73 trials. LPP amplitudes for each facial expression, gaze direction and scalp site were normally distributed within all groups, with no univariate or multivariate outliers.

LPP amplitude was analysed using all the Fz, Cz and Pz scalp sites with a four-way mixed model ANOVA (Expression (4) x Gaze (2) x Group (3) x Scalp Site (3)). There was a significant main effect of site, Wilks' $\Lambda = .615$, $F(2, 41) = 12.853$, $p < 0.001$, partial $\eta^2 = 0.385$. LPP amplitude was significantly greater at the Pz compared to Cz ($p < 0.001$) and Fz ($p = 0.026$) sites, with no significant difference between Cz and Fz ($p = 1.0$). There was a significant main effect of group, $F(2, 42) = 6.501$, $p = 0.003$, partial $\eta^2 = 0.236$, with LPP amplitude significantly greater for the psychosis compared to non-clinical ($p = 0.004$) and also depression group ($p = 0.030$). There was no significant difference in LPP amplitude between the depression and non-clinical group ($p = 1.0$). **Figure 1** shows LPP amplitude by group within Fz, Cz and Pz.

There was no significant main effect for gaze, Wilks' $\Lambda = .951$, $F(1, 42) = 2.174$, $p = 0.148$, partial $\eta^2 = 0.049$, or expression, Wilks' $\Lambda = .911$, $F(3, 40) = 1.305$, $p = 0.286$, partial $\eta^2 = 0.089$. All possible interaction combinations amongst the four factors were

not significant: site by gaze, $F < 1$, site by expression, $F < 1$, site by group, Wilks' $\Lambda = .836$, $F(4, 82) = 1.915$, $p = 0.116$, partial $\eta^2 = 0.085$, site by gaze by expression, Wilks' $\Lambda = .831$, $F(6, 37) = 1.255$, $p = 0.302$, partial $\eta^2 = 0.169$, site by gaze by group, $F < 1$, site by expression by group, Wilks' $\Lambda = .725$, $F(12, 74) = 1.074$, $p = 0.394$, partial $\eta^2 = 0.148$, site by gaze by expression by group, $F < 1$, gaze by expression, Wilks' $\Lambda = .885$, $F(3, 40) = 2.256$, $p = 0.097$, partial $\eta^2 = 0.145$, gaze by group, $F < 1$, gaze by expression by group, Wilks' $\Lambda = .783$, $F(6, 80) = 1.731$, $p = 0.125$, partial $\eta^2 = 0.115$, expression by group, Wilks' $\Lambda = .790$, $F(6, 80) = 1.664$, $p = 0.141$, partial $\eta^2 = 0.111$.

4. Discussion

Hypotheses concerning motivated attention in non-clinical participants, and an attentional hypersensitivity to threatening, social stimuli in depression or first-episode psychosis, were not supported. Likewise, hypotheses of these processes being potentiated for matched facial expressions were not supported. An unpredicted result was observed indicating a significant group difference in LPP amplitude to facial expressions irrespective of emotional expression.

4.1. Attentional processing – the LPP

The hypothesis that LPP amplitude would be significantly potentiated in non-clinical participants for happy, angry and fearful faces was not supported, evident in the absence of a significant main effect of expression, or group by expression, or group by expression by gaze interaction, or any interaction of these combinations with scalp site. This is inconsistent with the motivated attention hypothesis (Lang et al. 1997), and studies that have observed either potentiated LPP amplitude to happy and angry facial expressions (Eimer and Holmes 2007) or preferentially to angry faces (Schupp et al. 2004c). This null result must be interpreted with caution. It could possibly suggest that, between 500 and 750ms post stimulus onset, non-clinical participants exhibited no motivated attention biases for processing emotional expressions. The hypothesis of this attentional bias being heightened for matched facial expressions in the non-clinical group was also not supported. This could possibly suggest an absence of recruitment of increased attention processing for matched facial expressions, contrary to the matched expressions hypothesis.

However, two methodological reasons might account for these two null results. LPP modulation by emotional expression or gaze direction is sensitive to stimulus arousal and valence (Duval et al. 2013, Eimer et al. 2003, Schupp et al. 2004c), and might require a particular arousal and valence threshold. No such ratings were measured in this study and a possible explanation is that these stimuli required greater arousal and valence properties to induce effects of emotional expression or gaze in a sample of this size. This proposition must be tested experimentally with valence and arousal ratings taken during experimentation. Secondly, these findings might reflect Type II error. Johnston et al. (2005) did observe a significant effect of emotional facial expression upon LPP amplitude with 26 participants, a smaller number than 45 participants in this study, which counters this explanation. However, a post-hoc sample size calculation was undertaken using an alpha level of .05, a statistical power of 0.8, and an anticipated Cohen's d of 0.8, which is a large effect

Figure 1. *Face-elicited LPP amplitude within frontal (Fz), central (Cz) and parietal (Pz) scalp sites, by groups, across emotional expression and gaze directions*
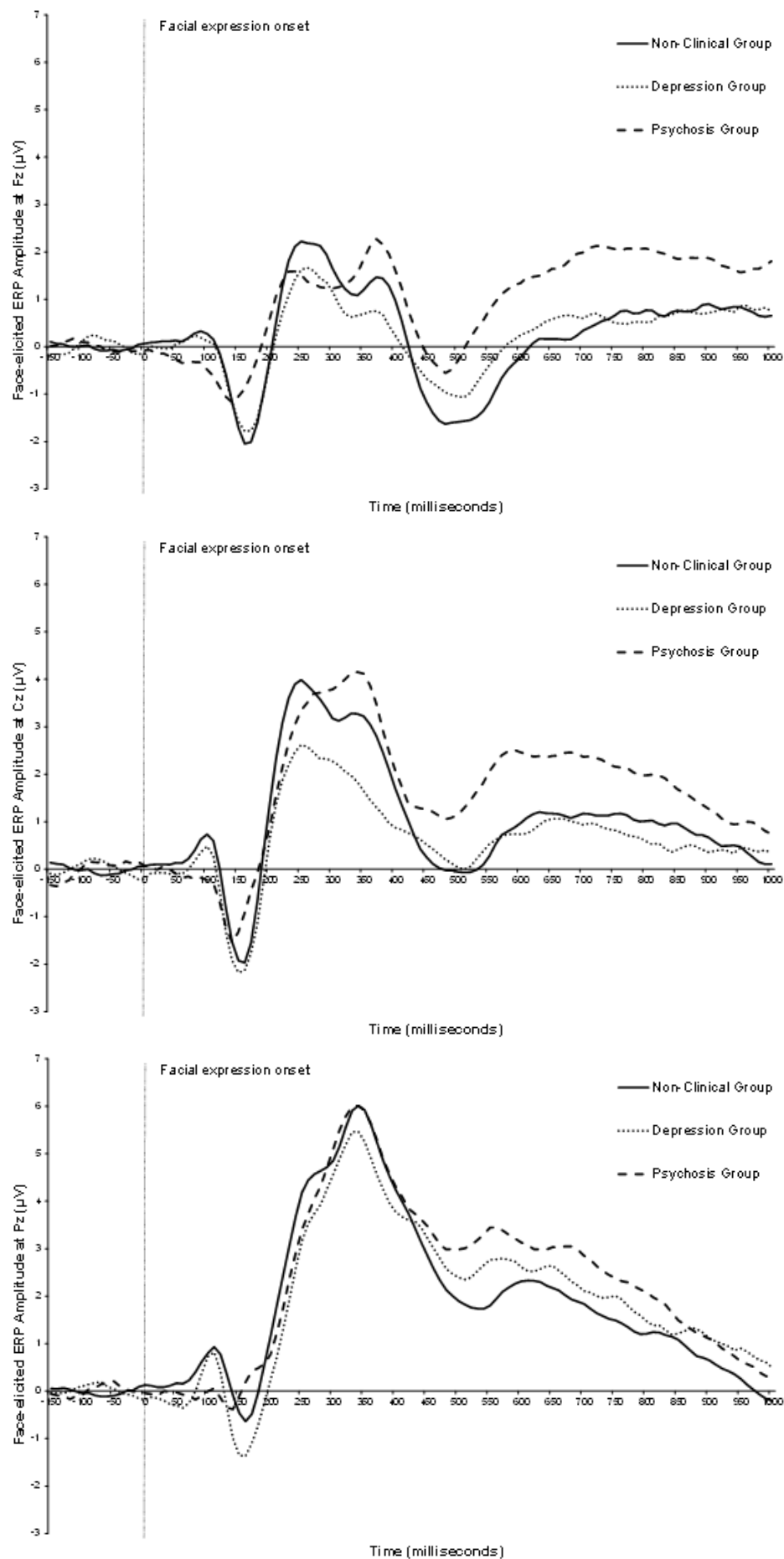
size (Cohen 1988). This yielded a required sample size for each group in this study of 21. This effect size was estimated from Johnston et al. (2005) finding a significant attenuation of LPP amplitude in their psychosis group of 11 participants, relative to a control group of 15 participants, and Bediou et al. (2007) observing significant effects with equal psychosis and control group sizes of 10 participants. Therefore, it is possible that this study observed a null effect due to reduced power and thus Type II error.

The hypothesis that LPP amplitude would be significantly more potentiated compared to the non-clinical group for angry and fearful faces, in both the depression and psychosis groups, was not supported. Likewise, the hypothesis of the potentiation of this effect occurring for matched angry and fearful faces in the depression and psychosis groups was not supported. This does not provide evidence to support a threat-related processing bias in depression or psychosis between 500 to 750ms post stimulus onset, inconsistent with the social risk hypothesis (Allen and Badcock 2006), and models of threat processing in psychosis (Freeman et al. 2013). The result also does not provide support for the potentiation of these effects with matched faces. However, the absence of these hypothesised effects in the clinical groups cannot be concluded. This is due to the aforementioned methodological reasons and also the absence of the demonstration of the experimental effect in the control group, against which the two clinical groups are compared.

Importantly, LPP amplitude was significantly potentiated for the psychosis group compared to both the depressed and non-clinical groups, across both emotional expressions and gaze directions. To the authors' knowledge, this effect has never been tested in a first-episode psychosis group. This study's result suggests that young people with first-episode psychosis afford increased, rather than decreased, higher order controlled cognitive processing, stimulus evaluation and memory storage, from 500 to 750ms post-stimulus onset, to facial expressions irrespective of emotional expression. Crucially, this occurs when compared to individuals of an equivalent age who either have no lifetime history of mental disorder or are diagnosed with major depression. That is, there might exist an attentional hypersensitivity to facial expressions specific to first-episode psychosis, regardless of emotional expression, that is also distinct from another psychopathological state – major depression. This might highlight the salience of facial expressions as a social emotional stimulus specific to first-episode psychosis.

This study's result is inconsistent with two other studies with adults with schizophrenia, that observed either an attenuated LPP or an absence of the LPP (Bediou et al. 2007, Johnston et al. 2005). These studies suggest attentional withdrawal from facial expressions in their clinical samples. However, both of these studies did not explicitly test first episode psychosis samples, and had different age ranges and means. The age range of the psychosis group for Bediou et al. was 19 to 44 years, and for Johnston et al. it was 18 to 45 years (with a mean age of 34 years), which is greater than the age range of 16 to 27 (with a mean age of 20.8 years) in this study's psychosis group. These sample differences introduce at least three differentiating variables that might account for the different face-elicited LPP amplitude results amongst the studies: age of participant, number of psychotic episodes, and duration of psychosis. Such potentiated LPP reactivity to facial expressions as a class of stimuli, as observed in this first-episode psychosis sample, could attenuate

as the number of psychotic episodes or duration of the psychotic illness progress. This requires empirical examination, preferably longitudinally. As mentioned, it cannot be excluded that the difference in results compared with Johnston et al. (2005), and Bediou et al. (2007), arises from differences in the age of the respective psychosis samples, as there is some evidence that LPP amplitude reduces with age (MacNamara et al. 2016)

The result of significantly potentiated LPP amplitude in the first-episode psychosis group to facial expressions regardless of emotional valence is significant when interpreted in a clinical staging model of psychotic illness (McGorry 2007, McGorry et al. 2006). An attentional hypersensitivity to facial expressions might characterise first-episode psychosis and an attentional hyposensitivity to facial expressions might characterise more severe, chronic stages or durations of psychosis. This effect might be concordant with other, more established psychophysiological correlates of facial-processing problems in psychosis. For example, it is generally established that face-elicited N170 amplitude at temporal and parieto-occipital sites is attenuated in schizophrenia patients (Bediou et al. 2007, Campanella et al. 2006, Herrmann et al. 2004), suggesting impaired structural encoding of facial expressions by attenuated early visual attention. However, preliminary evidence indicates potentiated N170 amplitude and hyperresponsivity to facial expressions in less chronic patients diagnosed with a range of psychotic disorders (Valkonen-Korhonen et al. 2005). It is possible that an initial hyperresponsivity to facial expressions in first-episode psychosis but hyporesponsivity in more chronic schizophrenia states could occur, perhaps because the clinical states differ by age, illness duration, number of episodes, hospitalizations, social impairment, unemployment, neurodegeneration, or persistent symptoms (McGorry et al. 2003, McGorry and Yung 2003). However, this tentative proposition must be investigated empirically.

4.2. Limitations and future research

The main limitations of the study were the absence of stimulus arousal and valence ratings, sample size, confounding clinical variables, and a gender imbalance in participants. Given the null and novel findings of this study in a first-episode psychosis group, the experiment needs to be repeated with similarly clinically defined (see below) control, depression and psychosis groups. Greater group sample sizes of at least 21 participants each can be used to improve statistical power, reduce the possibility of Type II error in statistical interactions and thereby provide more confidence in the interpretation of the absence of any hypothesised effects. Whilst a smaller sample size imposes limits on the data representing a normal distribution, importantly, variables did not violate the assumptions of normality, and there were no univariate or multivariate outliers. Non-parametric tests were not appropriate to use for data analysis because of the multifactor independent variable design. The same stimulus set can be used and arousal and valence ratings collected after each stimulus presentation. The latter will permit analysis of arousal and valence thresholds required to detect interaction effects between expression, gaze and group. Whilst the groups in this study were homogenous with respect to age, and there was no significant difference in age between groups, participants in this study were in young adulthood or mid to late adolescence (ages

ranging between 15 to 26, with a mean age of 19). Given that people aged between either 7 to 13, or 14 to 18, might be less sensitive than people aged over 18 to fearful or angry facial expressions (Thomas et al. 2007), this further reinforces repeated experimental research with a larger sample size to establish effects in this age group.

The psychosis and depression groups were clearly distinguished at a diagnostic level and also partially on a symptom based level, by positive psychotic symptoms but not significantly so by depressive symptoms. It is likely, but not certain, that differences between these two groups were due to psychotic and not depressive symptoms. Given that depression is a symptom dimension in first-episode psychosis (McGorry et al. 1998), and excluding psychotic participants with moderate to high depressive symptoms may therefore compromise external validity, future research could address this issue by testing more severely depressed individuals for the depression group. Similarly, whilst the psychosis participants were clearly symptomatic and receiving outpatient clinical treatment, a more severely symptomatic psychosis sample could also be tested to increase differentiation between the groups. Eleven patients in the depression sample, and four patients in the psychosis group, were taking selective serotonin reuptake inhibitors or serotonin-norepinephrine reuptake inhibitors at the time of testing. It is possible that this could have influenced fixation upon aspects of the facial expressions, as a recent paper indicates that seven days' prescription of 20mg of citalopram leads to reduced avoidance of ocular exploration of faces in volunteers high in neuroticism, though they were not clinically depressed (Simplicio et al. 2014). Depressed or psychotic patients could be recruited in a future study who were medication free; however, such a sample is very difficult to obtain in modern, public psychiatry in Melbourne, Australia. Lastly, the depression group had a significant majority of females, and the psychosis group a significant majority of males. Future research can test groups balanced with respect to gender.

4.3. Conclusions

It could not be concluded from the null results that, between 500 and 750ms post stimulus onset, healthy control participants do not exhibit motivated attention biases for processing 'novel' emotional expressions. Likewise, it could not be concluded that there is an absence of recruitment of increased attention processing for matched facial expressions. Whilst there was no evidence to support an attentional hypersensitivity to threatening, social stimuli during depressed states or first-episode psychosis, which was not potentiated for matched facial expressions, again the absence of these effects in a depressed or first-episode psychosis population could not be concluded. These null findings might be due to insufficient power to detect a significant effect, due to sample size or reduced arousal and valence properties of the stimulus set. Importantly, the results indicate an attentional hypersensitivity to facial expressions regardless of emotional expression or gaze direction that might be particular to first-episode psychosis.

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