Electrophysiological Correlates of Emotional Face Processing in Typically Developing Adults and Adults with High Functioning Autism

by

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Abstract

Emotional expressions have been found to affect various event-related potentials (ERPs). Furthermore, socio-emotional functioning is altered in individuals with autism, and a growing body of neuroimaging and electrophysiological evidence substantiates underlying neural differences for face processing in this population. However, relatively few studies have examined the time-course of emotional face processing in autism. This study examined how implicit (not the intended focus of attention) versus explicit (the intended focus of attention) presentations of emotion differentially influenced the processing of fearful, sad, and happy facial expressions relative to neutral expressions. Study 1 was conducted in a sample of typically developing (TD) young adults. Study 2 compared a group of high-functioning young adults with autism to a new group of age-, gender-, and IQ-matched TD controls. Results from both studies supported the prediction that ERP components would be differently modulated as a function of emotion relevance and emotional expression, suggesting different emotions and implicit/explicit presentations are processed in partially separable neural networks. Both studies found that sad faces uniquely modulated the P150, VPP, EAP, and LPP in TD adults. This unique response to sad faces was found as early as 150ms post-stimulus onset over frontal electrodes, suggesting early, relatively automatic recognition of sad faces. It was also found over posterior sites as indexed by the LPP, reflecting more conscious appraisal of the emotion. In individuals with autism, sad faces did not elicit distinctive effects. Rather, happy faces uniquely modulated the P150 and N4a, suggesting that this positive social emotion was most salient to adults with autism whereas sad faces, which may communicate a social error message and elicit empathy, were most salient to TD adults. Both experiments provide support for a neural network that is sensitive to socially relevant information and provide important clues for underlying neural differences that may contribute to difficulties with socio-emotional functioning so commonly reported in autism.

Keywords: Event-related potentials; Autism; Adolescents and young adults; Emotional face processing.

Dedication

To my grandparents.

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General Introduction

Emotional face processing has become an increasingly popular area of research over the past two to three decades. Furthermore, socio-emotional functioning is commonly reported as being an area of difficulty in individuals with autism, and a growing body of neuroimaging and electrophysiological evidence suggests that faces may be processed differently in autism compared to their typically developing (TD) peers. Although there is a growing interest in how the human brain processes emotional facial expressions and numerous studies have helped develop our understanding of the time course and brain regions involved in processing emotional faces, several important questions remain.

Specific questions that have been raised in the literature and were of interest to this project, included determining if and how implicit and explicit presentations of emotional facial expressions (happy, sad, fearful, and neutral) differently modulated event-related potentials (ERP) of interest, whether or not different emotions differently modulated these ERP effects, and whether or not the face-sensitive N170 component was modulated by emotional expressions. The goals of the first study were, firstly, to address some of these questions and, secondly, to test the effectiveness of a newly developed paradigm, to determine if it would be a useful experiment with which to examine the electrophysiological correlates of emotional face processing in autism.

To examine these questions, a well-controlled paradigm was developed that permitted examination of implicit and explicit emotional expression processing. Emotional faces were selected from the Karolinska Directed Emotional Faces dataset (Lundqvist, Flykt, & Öhman, 1998). The facial expressions most reliably identified by three members of the Laboratory for Affective and Developmental Neuroscience were selected for use in this paradigm. A small, coloured square was then centrally positioned on the nose of each face. The use of the same stimuli minimizes extraneous variables and helps isolate differences of interest (Luck, 2005)—namely the difference in behavioural and electrophysiological responses to emotional faces when they are not the focus of attention (implicit condition) compared to when they are the focus of attention (explicit condition).As mentioned, the second goal of the first study was to determine how the stimuli in this paradigm modulated ERP components of interest, which included those

that have previously been identified in the literature as being sensitive to faces, emotional expressions, or socially salient information. The objective was to then use this same experiment to elucidate the time course of emotional face processing in high-functioning young adults with autism and to compare them to a group of well-matched typically developing control participants. Participants in the second study were matched on variables that have previously been identified as important factors to consider in autism research. These demographic variables included: chronological age; gender, verbal ability; nonverbal ability; intellectual ability, and overall level of self-reported mood symptoms (specifically, symptoms common to anxiety and depression).

Emotional face processing can be seen as a proxy for investigating social-emotional functioning. Functionally, difficulties with social communication and socio-emotional functioning are hallmarks of autism (American Psychological Association [APA], 2000). Since Kanner's (1943) and Asperger's (1944; translated by Frith, 1991) initial descriptions of autism, this has been of great interest to autism researchers. Although there is much interest in emotional face processing in autism, relatively few ERP studies have been conducted to elucidate the time course of emotional face processing in autism. Furthermore, those studies that have been published have generally focused on early ERPs recorded over posterior electrode sites, most notably the P1 and N170. Given a plethora of recent data from neuroimaging studies and the special focus of our laboratory on regulatory processes in the frontal lobes, the current experiments were particularly interested in exploring how emotional faces modulated ERP effects over the frontal scalp.

Chapter 1.
The Electrophysiological Correlates of Implicit and Explicit Emotional Face Processing in Young Adults

Faces are an integral part of human interactions. They capture the interest and attention of typically developing humans from birth (Morton & Johnson, 1991), and throughout the lifespan (Darwin, 1872/1998). Emotionally expressive faces, in particular, are an integral part of socio-emotional communication and provide essential information about another person's emotional state and communicate important information about the person and the environment (Dolan, 2002; Ekman, Friesen, & Ellsworth, 1972). As such, emotionally expressive faces provide a biologically relevant, highly focused (as opposed to pictorial scenes; Williams, Palmer, Liddell, Le Song, & Gordon, 2006), and salient (Eimer & Holmes, 2007) means with which to study the neural mechanisms of emotion processing.

Many studies have investigated the neural network underlying emotional processing by measuring brain responses to emotionally salient stimuli. Such studies have revealed a complex and interconnected network of brain areas that work together to process emotional information. A meta-analyses of nearly 2000 imaging studies showed that emotional processing is supported by a diffuse neural network that included posterior, anterior, and medial brain regions (Kober, Barrett, Joseph, Bliss-Moreau, Lindquist, & Wager, 2008). Specifically, the following posterior brain areas were consistently involved: lateral occipital gyri; right ventral temporo-occipital cortex (including the fusiform gyrus), lateral temporal cortex (including the superior temporal sulcus (STS), primary visual cortex, and posterior cingulate cortex (PCC). These brain areas are generally believed to be important for the initial perceptual analysis of faces (specifically the inferior occipital cortex; Rossion, Caldera, Seghier, Schuller, Lazeyras, & Mayer, 2003), analysis of the structural properties of the faces (the fusiform gyrus; Gobbini & Haxby, 2006; Hoffman & Haxby, 2000), and in processing the dynamic aspects of faces, such as facial expressions (STS); Allison, Puce, & McCarthy, 2000). Outside primary and association visual cortex, frontal brain regions were also consistently recruited across studies, including the right frontal operculum, bilateral inferior frontal gyri, the left

middle frontal gyrus, anterior cingulate cortex (ACC) and dorsal-medial pre-frontal cortex (PFC; Kober, et al., 2008).

As mentioned, limbic structures were also actively recruited in tasks of emotion processing, including the amygdala, parts of the thalamus, lateral hypothalamus, and the ventral striatum (Kober et al., 2008). This meta-analysis further showed that multiple regions of the frontal cortex co-activated with regions of the limbic system, suggesting important connections between these brain regions for emotion processing. Aspects of the PFC, in combination with the amygdala, are believed to coordinate a rapid evaluation of the emotional significance of facial expressions (Adolphs, 2003; Eimer & Holmes, 2007); whereas, the ACC and more posterior aspects of the PFC have been linked to a conscious appraisal of emotional facial expressions (Adolphs, 2003; Eimer & Holmes, 2007). Similarly, posterior regions in visual cortices were also found to be functionally connected to regions of the paralimbic cortex (Kober et al., 2008). In sum, Kober et al's meta-analysis (2008) suggests that emotion processing is a multifaceted process requiring a complex interconnected neural network composed of several brain regions working in concert to rapidly process facial expressions.

Electroencephalography (EEG) offers a particularly powerful means with which to study affective processing in the human brain (Batty & Taylor, 2003; Eimer & Holmes, 2002, 2007; Williams et al., 2006), recording neural responses with very high temporal resolution, on the order of one millisecond (ms). Such high sensitivity is very well suited to investigating the time course of affective face processing as this is believed to be processed rapidly in the brain. Rapid decoding of facial expression is important to subserve adaptive social and situational behaviour (Carretie & Iglesias, 1995; Eimer, Kiss, & Holmes, 2008; Williams et al., 2006).

Several ERP components have been identified as being sensitive to emotional facial expressions. Several authors have reported an early fronto-central positivity peaking between 110-150ms after stimulus onset that is modulated by emotion (Eimer & Holmes, 2002). Most authors have recorded this broadly over fronto-central regions and reported an enhanced positivity in response to fearful compared to neutral faces (Batty & Taylor, 2003; Eimer & Holmes, 2002; Eimer et al., 2008; Holmes, Winston, & Eimer, 2005; Rossignol, Philippot, Douilliez, Crommelinck, & Campanella, 2004; Williams et al., 2006).

Significant differences between fearful and neutral faces were identified 120ms post-stimulus onset in Eimer and Holmes' 2002 study. This peak positivity was distributed fronto-centrally and showed that emotional facial expressions were analysed rapidly and affected cortical processing at very early latencies (Eimer & Homes, 2002, 2006). This initial peak positivity is believed to reflect initial rapid detection of facial expression in prefrontal areas of the brain that play an important role in detecting emotionally relevant information (Eimer & Holmes, 2007).

Studies have also found a slightly later prolonged positivity occurring between 200-300ms. Taake, Jaspers-Fayer, and Liotti (2009), the so-called Early Anterior Positivity (EAP), that was sensitive to emotionally salient stimuli (in this study, threat vs. neutral words). Implicitly presented threatening words elicited a significantly greater EAP compared to neutral words. Li et al (2007) and Pauli, Amrhein, Muhlberger, Dengler & Wiedeman (2005) identified a similar sustained ERP effect in the 300-500 ms time window, with threat words again eliciting an enhanced positivity compared to neutral words between 300-500ms and 200-400ms post-stimulus onset, respectively. However, in contrast to the finding that the EAP is uniquely sensitive to implicit presentations of emotions, some studies have found that when emotions were not the spatial focus of attention, early effects to emotional expressions have been eliminated entirely over the frontal scalp (Eimer, Holmes, & McGlone, 2003; Holmes, Vuilleumier, & Eimer, 2003), suggesting that at least at some level attentional resources are required to process emotion as indexed by ERP modulations over the frontal scalp.

The EAP is typically followed by a later, broadly distributed, sustained positivity that starts as early as 250ms post-stimulus onset and stays online for the duration of the analysis interval (Eimer & Holmes, 2002, 2006). The Late Positive Potential (LPP), recorded over anterior and posterior scalp regions, respectively are believed to reflect higher level analysis of the emotional information, including evaluating the meaning and significance of this information (Eimer & Holmes, 2002, 2006). This sustained positivity has also been observed in response to emotional information other than faces (Cuthbert, Schupp, Bradly, Birbaumer, & Lang, 2000), in line with neuroimaging evidence that lateral and medial PFC play an important role in evaluating the meaning of emotionally salient information and is not uniquely sensitive to faces (Eimer & Holmes, 2007).

Also of interest to this study were the P1, the face-sensitive N170, recorded over occipito-temporal scalp regions, and the N170's proposed inverse dipole component, the vertex positive potential (VPP; Schacht & Sommer, 2009), recorded centrally over more anterior electrode sites.

The P1 reflects early visual processing and is typically recorded over the occipital scalp. More specifically, the P1 is believed to reflect coarse perceptual discrimination of visual signals. It, however, has also been shown to be sensitive to attention (Taylor, 2002), information from eyes and eye gaze (Taylor, Edmonds, McCarthy, & Allison, 2001; Senju, Tojo, Yaguchi, & Hasegawa, 2009) and emotional expression (Batty & Taylor, 2003; Utama, Takemoto, Koike, & Nakmura, 2009). More specifically, a number of studies have reported an increase in P1 amplitude to negative or disliked relative to neutral faces (Kolassa & Miltner, 2006; Pizzagalli, Lehmann, Hendrick, Regard, Pascual-Marqui, & Davidson, 2001; Pourtois, Dan, Grandjean, Sander, & Vuilleumier, 2005); although, P1 modulations to facial expression may reflect differences in the visual properties of the different facial expressions rather than a sensitivity to emotional expression per se.

The modulation of the N170 by emotional expression is particularly contested. The N170 is an ERP component sensitive to faces and recorded over occipito-temporal sites between 140-200ms post-stimulus onset (Bentin, Allison, Puce, Perez, & McCarthy, 1996). The N170 is traditionally conceptualized as reflecting structural encoding of facial features (Bentin et al., 1996). Bruce and Young (1986) proposed a dissociable neural network for the processing of facial identity and facial expression, supported by a number of lesion studies. Damage to specific brain regions in the occipito-temporal cortex can impair a patient's ability to recognise facial identity without impairing the patient's ability to read facial expression (Etcoff, 1984; Posamentier & Abdi, 2003; Tranel & Damasio, 1988; Young, Newcombe, de Haan, Small & Hay, 1993). inversely, one study found that patients lost their ability to read emotional information from faces but maintained their ability to recognise facial identity (Hornak, Rolls, & Wade, 1996).

This model of suggests that basic facial processing and emotional facial processing are dissociable skills. This traditional viewpoint would thus suggest that the N170, which reflects structural encoding of facial features and is not sensitive to subtle changes in

faces (e.g., facial expressions) should not be modulated by emotional expression. However, thus far studies examining the effect of emotion on the N170 have yielded mixed results and whether emotional expressions differentially modulate ERP effects remains unclear.

Many studies have failed to find an effect of emotion on the N170. Using a 1-back design in which participants had to attend to the emotional expression, identity, or orientation of the face, Ashley, Vuilleumier, and Swick (2004) presented upright and inverted emotional faces and houses to older adults. This study did not find an effect of emotion on the face-sensitive N170 component. An earlier study employing a very similar n-back design, with upright and inverted fearful and neutral faces and upright houses, reported the same set of results, finding that emotional faces failed to modulate the N170 (Eimer & Holmes, 2002). Likewise, using an odd-ball paradigm in which participants had to either identify a target chair (Holmes, Winston, & Eimer, 2005) or a target happy or fearful face (Rossignol, Philippot, Douilliez, Crommelink, & Campanella, 2004), two groups failed to find an effect of emotion on the face-sensitive N170 component. These findings are consistent with intracranial recordings showing that face-specific potentials are only modulated by affect at longer latencies (Puce, Allison, & McCarthy, 1999).

By contrast to the above designs, in which emotion was presented explicitly, or was the target of attention, several studies have found that the N170 is modulated by emotional facial expressions (Batty & Taylor, 2003; Caharel, Courtay, Bernard, Lalonde, & Rebaï, 2005; Kolassa & Miltner, 2006; Krombholz, Schaeger, & Bouscein, 2007; Rossignol et al., 2005; Williams et al., 2006). Batty and Taylor (2003) used a *passive* viewing paradigm to present expressive faces displaying one of six basic emotions (fearful, sad, angry, disgusted, happy, and surprised), or a neutral expression. Batty and Taylor (2003) reported that the N170 was modulated by emotion, an effect that was driven by fearful faces eliciting a larger peak response than any other facial expression, including neutral. Another study (Rossignol et al., 2005) also reported that fearful faces elicited a greater peak N170 negativity than neutral faces. Williams et al (2006) used a similar passive viewing paradigm, and found that both happy and fearful faces elicited a greater negativity compared to neutral faces.

In sum, the above findings generally suggest that implicitly presented or passively viewed affective faces elicit enhanced neural response compared to neutral stimuli and suggest that the N170 is modulated by implicit presentations of emotional faces. It is important to note, however, that a handful of studies have also reported modulation of N170 using *explicit* paradigms (Caharel et al., 2005; Kolassa & Miltner, 2006; Krombholz et al., 2007). Nonetheless, the implicit-explicit distinction seems to be an important factor when considering the effect of emotion on the N170. To my knowledge, this prediction has not been examined conclusively in a single study.

Various tasks have been used to study emotional face processing. As mentioned, a distinction made in the literature that was of interest to this study is the potential difference in neural responses to implicitly versus explicitly presented emotional stimuli. It has been previously proposed that whether or not the emotion is the focus of attention affects which regions of the brain are recruited to support emotion processing (Williams et al., 2006) and can differentially modulate ERP effects, as was briefly reviewed above in relation to early frontal effects and the N170. More specifically, implicit emotional processing is suggested to take place subcortically, whereas explicit processing- directly taxing attentional resources- is thought to take place in cortical regions (Critchley et al., 2000; Gur, Gunning-Dixon, Bilker, & Gur, 2002; Gorno-Tempini et al., 2001; Iidaka et al., 2001; Scheuerecker et al., 2007; Taake et al., 2009). More specifically, a direct feed-forward pathway, or a fast-processing route, has been proposed. This model hypothesizes that subcortical limbic structures, such as the amygdala and thalamus, rapidly send forward signals of threat directly to regions of the frontal cortex when emotion is presented implicitly (Williams et al., 2006). In contrast, when the emotion is the focus of attention (explicit presentation) its' processing is thought to engage neocortical networks such as the PFC typically involved in attention, working memory, and top-down regulatory processes (Williams et al., 2006). Thus, it has been proposed that implicit and explicit emotional processing are supported by distinct, or at least partially distinct, neural networks.

Partial support for this model comes from reaction time, functional magnetic resonance imaging (fMRI) and EEG data. In a study examining reaction times to implicit versus explicit emotional face processing, a distinction was found in how quickly adults responded to different expressions in the implicit versus explicit conditions (Williams,

Mathersul, Palmer, Gur, Gur, & Gordon, 2009). Specifically, in the explicit condition, responses were most accurate for happy faces and slowest for fearful faces whereas the reverse was true in the implicit condition with accuracy being lowest for happy faces and response times fastest to fearful faces (Williams et al., 2009). The authors proposed that these findings reflected the different ways in which the priority for threat-related signals is processed, with an automatic priority being assigned to unexpected fearful stimuli that are not the current focus of attention, and a more controlled elaboration of the threat signals when they are consciously or overtly attended (Williams et al., 2009). This pattern of results could be interpreted as further supporting the theory that there is more limbic system involvement to implicitly presented emotions, especially negative emotions, and more cortical involvement, which ultimately dampens the limbic system response and slows the behavioural reaction times for explicit presentations of emotions (Williams et al., 2006).

Neuroimaging studies have also found that partially distinct networks support the processing of implicit and explicit presentations of emotion. Specifically, studies have shown that when emotional stimuli were presented implicitly, subcortical limbic regions, including the amygdala, thalami, hippocampi, and paralimbic cortical regions (ventro-lateral and dorso-medial PFC, including the ACC) were recruited (Critchley et al., 2000; Gur et al., 2002; Gorno-Tempini et al., 2001; Iidaka et al., 2001; Scheuerecker et al., 2007; Taake et al., 2009). Fearful faces in particular elicited activation in these subcortical limbic brain regions (Cornwell, Carver, Coppola, Johnson, Alvarez, & Grillona, 2008; Gorno-Tempini et al., 2001; Guyer et al., 2008; Iidaka et al., 2001; Vuilleumier, Armony, Clarke, Husain, Driver, & Dolan, 2002; Williams et al., 2005). In contrast, when stimuli were presented explicitly, activation from these subcortical regions were absent or recruited to a significantly lesser extent (Critchley et al., 2000) and neocortical regions (dorsolateral PFC and dorsal ACC) were recruited to a greater extent (Scheuerecker et al., 2007).

Additional evidence for a dissociation in processing of emotion comes from EEG studies employing subliminal vs. supraliminal passive presentations of facial expressions (Liddell, Williams, Rathjen, Shevrin, and Gordon, 2004). This study found that fearful faces triggered an enhanced N2 in the subliminal condition. This result was interpreted as reflecting an automatic orienting response that is triggered when emotions are not

consciously perceived. This study also found that fearful faces elicited an enlarged parietal P3 in the supraliminal condition, believed to reflect a more conscious integration of the emotional content (Liddell et al., 2004).

By contrast to the latter findings, Eimer et al (2008) examined how fearful faces presented for varying amounts of time (from subliminal to supraliminal) modulated *anterior* ERP effects. They rather found that when fearful and neutral faces were presented for varying durations, it was not the length of presentation time that differentially modulated early anterior ERP components, but whether or not the fearful face was correctly identified by participants. This experiment failed to find an effect of emotion when fearful faces were not correctly identified. This result was interpreted in a different manner from Williams and colleagues, in that they imply that early frontal emotion modulation is present only when the facial expressions are the focus of attention or consciousness (Eimer et al., 2008) and they are ascribable to a fast cortical mechanism, possibly due to a very rapid feedforward sweep from early visual areas to frontal and temporal neocortical areas (Brosch, Pourtois, & Sander, 2010).

Alternatively, some EEG studies have failed to find a difference in how explicitly and implicitly presented emotional faces modulated the N170, Early Posterior Negativity (EPN, likely involving an inverse dipole to the EAP (Junghöfer, Peyk, Flaisch, & Schupp, 2006), and the LPP (Frühholz, Jellinghau, & Herrmann, 2011). These findings suggest that the brain processes emotional facial expressions similarly regardless of presentation type and regardless of whether or not the emotion is the intended focus of attention.

Another question of interest to the present project was whether or not different emotions distinctly modulate ERP effects of interest. Eimer, Holmes, and McGlone's block design EEG study (2003) found that six basic emotional expressions (anger, disgust, sadness, happiness, fear, and surprise) all modulated the prolonged frontal effect (180-1000ms; N4a) similarly. The similarity in ERPs for all six basic emotional expressions was interpreted as indicating that emotionally relevant information delivered by facial expressions elicits similar neocortical activation and suggests all facial expressions are processed similarly by the neocortex (Eimer et al., 2003; Eimer & Holmes, 2007). Williams et al's 2006 study also failed to find differences in how positive and negative

emotions modulated that N170, further suggesting the brain processes emotions similarly.

By contrast, several studies have reported different ERP modulations in response to different emotional expressions (Batty & Taylor, 2003; Morel, Ponz, Mercier, Vuilleumier, & George, 2008; Pourtois, Grandjean, Sander, & Vuilleumier, 2004; Taake et al., 2009). Specifically, Batty and Taylor's 2003 study found that different emotional facial expressions differentially modulated the N170. Specifically, disgust and sadness elicited a delayed peak latency compared to neutral, happy, and surprised faces. Furthermore, fearful faces eliciting a greater peak negativity than other expressions. Analysis of a prolonged fronto-central components, from 270-420ms post-stimulus onset (which I will call the N4a), revealed a greater sustained positivity to neutral compared to all other facial expressions and a significantly smaller response to angry faces. Pourtois et al (2004) found that fearful faces elicited great peak amplitudes in early ERP effects (P1 and C1) than neutral and happy faces, which was interpreted as reflecting differences in how the brain processes negatively and positively valenced emotional expressions. Likewise, an EEG/ magnetoencephalography (MEG) study found early and late ERP/MEG differences in how the brain processed fearful, happy, and neutral faces (Morel et al., 2008), again suggesting a possible difference in how the brain processes positively versus negatively valenced emotions.

It is evident from the research to date that significant questions persist with regard to how the brain processes emotion. Specific areas of ambiguity that were of interest to this study included how the brain processes implicit and explicit presentations of emotions, how and if different emotional expressions modulate ERP effects, and if the N170 is modulated by emotional expressions. The goals of this project were to examine, in a single study, the effect of implicitly and explicitly presented emotional facial expressions on early and late ERP components known to be sensitive to visual, facial and/or emotional processing, using a well-controlled paradigm.

Behaviourally, in the explicit condition, it was hypothesized that participants would have longer reaction times to fearful faces compared to neutral, sad, and happy faces, whereas, in the implicit condition, it was hypothesized that fearful faces would elicit the fastest reaction times, consistent with Williams et al's results (2009) and the negativity

bias reported in the literature (Öhman, Lundquist, & Esteves, 2001; Smith, Cacioppo, Larsen, & Chartrand, 2003). Overall, reaction times were expected to be faster to implicit presentations of emotion as this was an easier task.

In terms of electrophysiological effects, the goal of this study was to clarify if ERP effects (specifically the P1, N170, VPP, P150, and EAP) were differentially modulated by implicit versus explicit presentations of emotion. If they are differentially modulated, this would support evidence of partially separable neural networks for processing emotion. However, if no clear distinctions are discovered, it would suggest that the neocortex processes emotional faces similarly regardless of whether or not they are the focus of attention.

Another goal of this study was to determine if and how happy, sad, fearful, and neutral faces differently modulated early ERP effects, which will provide insight into the underlying neural networks supporting emotion processing in the brain. Previous ERP research has consistently shown that different emotions modulate the LPP similarly; however fMRI research shows that different emotions are differently processed in the brain and some ERP studies have found evidence of different modulations by emotion (Batty & Taylor, 2003; Taake et al., 2009).

The final goal of this study was to clarify if the face-sensitive N170 component was modulated by emotion, pitting Bruce and Young's classic view of structural facial encoding and supporting evidence (Ashley, Vuilleumier, & Swick, 2004; Eimer & Holmes, 2002; Holmes, Winston, & Eimer, 2005; Puce, Allison, & McCarthy, 1999; Rossignol, Philippot, Douilliez, Crommelink, & Campanella, 2004) against studies which have reported modulations of the N170 by emotional expressions (Batty & Taylor, 2003; Caharel et al., 2005; Kolassa & Miltner, 2006; Krombholz, et al., 2007; Rossignol et al., 2005; Williams et al., 2006).

Based on research to date, it was specifically hypothesized that implicit presentations of fearful faces would elicit earlier and greater peak negativity in the right N170 and VPP compared to sad, happy, and neutral faces. Based on fMRI research showing that sad faces in particular elicit robust activitations in prefrontal cortex, sad expressions were expected to give raise to a greater peak positivity in the P150 and a greater overall

sustained positivity in the EAP compared to fearful, happy, and neutral faces. Explicit presentations of sad, happy, and fearful faces were expected to elicit a greater sustained positivity in the LPP compared to neutral expressions, as has previously been reported in ERP research. Emotional facial expressions were not expected to differently modulate the P1, since typically P1 effects are observed more when emotional stimuli are expected in advance- such as in block designs.

Method

The Simon Fraser University (SFU) Research Ethics Board approved this experiment. All participants gave their written informed consent before participating in the study and received course credit for their participation.

Participants

After blink rejection was completed, 16 participants were included in the analyses for the explicit condition (M_{age}=19.7 years; 50% female; 100% right-handed) while 15 were kept for implicit condition analyses (M_{age}=18.6 years; 53.33% Female; 100% right-handed). One subject in the implicit condition was dropped for excessive blinks. All participants were recruited through Simon Fraser University's (SFU) research participation system for Psychology 100 and 200 level students and received course credit for their participation. All participants were screened for history of neurological and psychiatric conditions, such as epilepsy, brain injury, depression and anxiety disorders, using a medical screening questionnaire developed in the lab and potential subjects with such histories were not included in the study.

Materials

A variant of the emotional Stroop task was used to identify differences in the neural mechanisms of emotional face processing to implicit versus explicit emotional processing. Emotional face stimuli were selected from the Karolinska Directed Emotional Faces dataset (Lundqvist et al., 1998; 13 male faces and 15 female faces; four expressions: happy, sad, fearful, and neutral), and altered so that the face was centred on a black background square and the hair was masked. Each emotional face had a coloured square (red, blue, green, or yellow), superimposed on the nose, in the same

position as the central fixation cross that was presented during the inter-stimulus interval (ISI).

Procedure

Participants completed a practice block comprised of different faces than were used in the actual trials, and were required to reach 80% accuracy before advancing to the actual trials. Using Presentation software, stimuli were presented in a pseudo-randomized order, constrained so that no more than three stimuli with the same emotion, gender, or colour were presented in a row. In order to minimize eye movements, participants were asked to keep their eyes on a central fixation (i.e., "+") on the computer screen throughout the experiment.

There were 100 stimuli per emotion/colour, resulting in a total of 400 stimuli. Faces were presented for 200ms, followed by a randomized ISI of 1700-2200ms (see Figure 1.1). Participants completed two tasks: an implicit task in which they were asked to attend to the colour of the square and an explicit emotion recognition task in which participants were asked to identify the emotional facial expression. In the implicit task, participants were asked to indicate the colour of the square on the nose by pressing one of the four shoulder buttons on a gamepad controller (Logitech, Romanel-sur-Monges, Switzerland). During the explicit task, participants identified the emotion displayed by the face again by pressing one of the same four shoulder buttons on the same gamepad controller. Each experiment was divided into four approximately five-minute blocks. After each block, participants were permitted to rest between 30-60 seconds, or longer if the participant requested to reduce fatigue.

In order to control for the use of different fingers corresponding to different emotions, the emotions were counterbalanced across hands. Thus, to eliminate an effect of laterality, each condition was equally represented by both hands across participants. Presentation order of the explicit and implicit tasks was also counterbalanced across participants.

EEG data acquisition.

Data was collected using high-density EEG. Participants were asked to come to the laboratory with clean, dry hair, and to reduce their caffeine and nicotine intake two to

three hours before the experiment. Participants sat in a sound-attenuated booth, with standardized ambient lighting. The ActiveTwo BioSemi electrode system (BioSemi, Amsterdam, the Netherlands) was used to record continuous EEG activity from 128 sintered Ag/AgCL electrodes embedded in an elastic cap, which provides low noise, low offset voltages and stable DC performance. Electrodes were positioned in a modified 10-20 equiradial layout relative to the vertex, which was measured individually for each participant (centrally located between the nasion—inion and left preauricular—right preauricular points). Water-soluble conductive electrode gel (SignaGel) was used with no additional skin preparation. Eye movement artefacts were monitored through both horizontal electro-oculargram (HEOG) from electrodes placed at the outer canthi, and vertical electro-oculargram (VEOG) from electrodes placed under each eye. Two additional electrodes, the common mode sense (CMS) active electrode and the driven right leg (DRL) passive electrode were also used. These electrodes replace the “ground” electrodes used in conventional systems (http://www.biosemi.com/faq/cms&drl.htm). As the BioSemi system is an active electrode system there is no conventional reference electrode, and a monopolar signal is stored for each active electrode and all re-referencing is done in software after acquisition. DC offset was kept below +/- 25 KΩ. The continuous signal was acquired with an open pass-band from DC to 150Hz and digitized at 512Hz. The signal was bandpass filtered off-line at 0.01Hz and re-referenced to a common mastoid reference.

EEG data processing.

EEG data processing was conducted using Brain Electrical Source Analysis (BESA) software (version 5.2). Trials contaminated by eye movements or muscle activity were rejected from the analyses. This was done using the blink rejection tool, provided by BESA software. Artefacts in the continuous EEG greater than 120mV were rejected, based on movements detected in the forehead EEG channels (FP1, FP2, FPz, by standard EEG nomenclature) and eye channels (left and right HEOG and VEOG electrodes). The artefact rejection epoch ranged from 200ms pre-stimulus onset to 800ms post-stimulus onset. Data were baseline corrected using a 200ms baseline (-200ms – 0ms). Event-related potentials (ERP) were time-locked to stimulus onset and selectively averaged for each of the emotions (with a 500ms pre-stimulus to 1000ms post-stimulus time-window).

Grand-averages for each condition were then computed by averaging the single-participant ERP averages. In order to examine differences between emotions, grand-averaged ERP waveforms and topographical maps for each emotion were inspected and compared for latency and amplitude of peak voltage activity in the main components (the left and right P1, left and right N170, VPP, P150, EAP, and LPP). Sensors of interest (SOIs) were selected based on peak voltages and neighbouring electrodes showing similar voltage amplitudes. A +/-20ms window surrounding the peak of interest was selected, from which BESA extracted the voltage activity of the peak or prolonged mean amplitude (in microvolts) as well as the latency (in ms) for peaks. SOIs for peak values included electrodes, identified in standard electrode nomenclature (NB: prime [‘] signifies a close but not exact match as position match with the international 10/20 system is not precise), over right and left occipito-temporal region (P1: P4, PO6, PO8 over the right hemisphere, and P3, PO5, PO7 over the left; N170: PO8, PO8’, P8, over the right and PO7, P7, P8 over the left;) and those along anterior midline electrode sites (VPP: Cz’, CPz, CPz’; P150: FPz, FPz’, AFz, AFz’). The EAP was recorded broadly from anterior electrodes (F5, F5’, F7, FT7; FT8, F6, F8, AF2, AF3, FP2, Af1, AF3, FP1). The posterior and N4a were recorded, respectively, from posterior central sites (LPP: CPz, CPz’, Pz) and midline sites over frontal cortices (N4a: FPz, FPz’, AFz).

Statistical analyses.

Accuracy and reaction time data were analysed with paired-samples *t*-tests to determine differences between emotions within each condition. To analyse amplitude and latency differences in ERP components of interest, repeated-measures ANOVAs were conducted. The P1 was analyzed using a 2 x 2 x 4 (Condition [Implicit, Explicit] x Topography [Left, Right] x Emotion [Fearful, Happy, Sad, Neutral]) repeated-measures ANOVA. The left and right N170 and the VPP were included in a single 2 x 3 x 4 (Condition x Topography [VPP, Left and Right N170s] x Emotion) repeated-measures ANOVA, due to previous evidence that the VPP is the inverse of the N170 (Schacht & Sommer, 2009) and would thus be predicted to behave similarly. The early frontal positivity (P150) was analyzed using a 2 x 4 (Condition x Emotion) repeated-measures ANOVA. The mean amplitude of the EAP, N4a and LPP were analyzed using three separate 2 x 4 (Condition x Emotion) repeated-measures ANOVAs. A Greenhouse-Geisser correction was employed to correct for sphericity in the data.

Paired-samples *t*-tests were run to examine a priori hypothesized differences between emotions in the implicit and explicit conditions, with an alpha level of .05. Post-hoc *t*-tests were employed in the case of significant main effects or interactions of interest with a Bonferroni correction to control for family-wise error.

Effect size estimates of repeated-measures ANOVA main effects and interactions were computed using partial eta squared. For *t*-tests, estimates of effect size were computed with Cohen's *d*, using the pooled standard deviations. Effect sizes of .3 are considered small, .5 are considered medium, and .8 and above are considered large (Cohen, 1992).

Results

Behavioural

Accuracy.

Paired-samples *t*-tests (α=.05/6=.008) revealed that participants were less accurate when responding to explicitly presented fearful and sad faces compared to explicitly presented neutral faces (t_{15}=-3.96, p=.001, Cohen's d=-1.13; t_{15}=-4.17, p=.001, Cohen's d=-1.32), although participants were highly accurate in responding to all conditions (84% accuracy or greater). There were no significant differences between emotions in the implicit condition.

Reaction time.

A priori paired samples *t*-tests, revealed that in the explicit condition, participants responded slower to fearful (t_{15}=4.93, p<.001, Cohen's d=.85) and sad (t_{15}=3.67, p=.002, Cohen's d=.55) faces than to neutral faces, and faster to happy faces than to neutral faces (t_{15}=-4.36, p=.001, Cohen's d=.63). There were no significant differences between emotions in the implicit condition.

See Table 1.1 for mean reaction times across emotions and conditions.

Electrophysiological Data: Amplitude

P1 (87-127ms).

A repeated-measures ANOVA revealed a significant main effect of hemisphere ($F_{1,14}$=16.52, p=.001, partial η^2=.54). No other main effects or interactions were significant, and a priori paired-samples *t*-tests revealed no significant differences between emotions.

N170-VPP (140-180ms).

The repeated-measures ANOVA revealed a significant main effect of Emotion ($F_{3,42}$=4.25, p<.02, partial η^2=.23). There were also significant Condition x Topography ($F_{2,28}$=6.54, p=.01, partial η^2=.32) and Topography x Emotion interactions ($F_{6,84}$=3.65, p=.01, partial η^2=.21).

Given the a-priori hypothesis of differential effects of emotion for implicit vs. Explicit expressions, we proceeded with paired-samples *t*-tests within each condition. For implicitly presented emotions, fearful faces elicited larger peak negativities than neutral faces over right N170 electrode sites (t_{14}=2.09, p=.05, Cohen's d=.27) Sad faces elicited larger positivities compared to neutral faces in the VPP (t_{14}=2.12, p=.05, Cohen's d=.33). For explicitly presented emotions, no difference was found between emotional and neutral expressions in the N170. The VPP was, however, modulated by explicitly presented emotional expression with fearful (t_{15}=2.16, p=.05, Cohen's d=.23) and sad (t_{15}=2.94, p=.01, Cohen's d=.25) faces eliciting larger peak responses than neutral faces. See Figure 1.2 for topographical maps and Figure 1.3 for representative waveforms.

P150 (130-170 ms).

The repeated-measures ANOVA revealed main effects of Emotion ($F_{3,42}$=3.45, p=.04, partial η^2=.20). No other main effects or interactions approached significance.

Given the a-priori hypothesis of differential effects of emotion for implicit vs. Explicit expressions, we proceeded with paired-samples *t*-tests within each condition. For implicitly presented emotions, sad faces elicited greater positivity over early fronto-central sites than neutral faces (t_{14}=2.70, p=.02, Cohen's d=.28). This effect was also significant in the explicit condition (t_{15}=2.48, p=.03, Cohen's d=.30). No contrasts involving other emotions were significant. See Figure 1.2 for topographical maps and Figure 1.3 for representative waveforms.

EAP (180-280ms).

The repeated-measures ANOVA revealed main effects of Emotion ($F_{3,42}$=3.30, p=.03, partial η^2=.19) and of Condition ($F_{1,14}$=14.84, p=.002, partial η^2=.51).

Given the a-priori hypothesis of differential effects of emotion for implicit vs. Explicit expressions, we proceeded with paired-samples *t*-tests within each condition. For explicitly presented emotions, only sad faces elicited greater amplitude positivity broadly over frontal electrodes than neutral faces (t_{15}=7.49, p<.001, Cohen's d=.67). Implicit presentations of emotional expression did not elicit differences between emotion and neutral expressions. See Figure 1.4.

Furthermore, collapsed across emotion, explicit presentations of emotion elicited significantly greater mean amplitudes than implicit presentations (t_{14}=3.85, p=.002, Cohen's d=.82).

N4a (350-600ms).

The repeated-measures ANOVA revealed a main effect of Condition ($F_{1,13}$=5.35, p=.04, partial η^2=.31).

Given the a-priori hypothesis of differential effects of emotion for implicit vs. Explicit expressions, we proceeded with paired-samples *t*-tests within each condition. For implicitly presented emotions, sad faces elicited greater amplitude negativity over anterior midline electrodes than neutral faces (t_{14}=-2.75, p<.02, Cohen's d=.53). Explicit presentations of emotional expression did not elicit differences between emotion and neutral expressions. See Figure 1.5.

To examine the main effect of Condition, post-hoc paired-samples *t*-tests (α=.05/2=.025) revealed that, collapsed across emotion, explicit presentations of emotion elicited significantly greater mean amplitude than implicit presentations (t_{14}=5.35, p<.001, Cohen's d=.98).

LPP (350-600ms).

The repeated-measures ANOVA revealed a main effect of Condition ($F_{1,14}$=6.15, p=.03, partial η^2=.31).

Given the a-priori hypothesis of differential effects of emotion for implicit vs. Explicit expressions, we proceeded with paired-samples *t*-tests within each condition. A priori paired-samples *t*-tests revealed no significant differences between emotions. However, explicit presentations of emotion elicited significantly greater mean amplitude than implicit presentations when collapsed across emotion (t_{14}=5.11, p<.001, Cohen's d=.66). See Figure 1.5.

Table 1.2 shows means and standard errors of the mean of peak amplitudes to implicit and explicit presentations of emotions for each ERP component.

Electrophysiological Data: Latency

P1 (87-127 ms).

There was a significant main effect of Topography ($F_{1,14}$=6.15, p=.03, partial η^2=.31). No other main effects or interactions were significant. P1 peaked earlier over the right hemisphere than over the left

N170-VPP (140-180 ms).

The repeated-measures ANOVA revealed a main effect of Topography ($F_{1,14}$=4.94, p<.04, partial η^2=.26). This was due to the VPP peaking significantly earlier than the Right N170. No other main effects or interactions were significant.

Given the a-priori hypothesis of differential effects of emotion for implicit vs. Explicit expressions, we proceeded with paired-samples *t*-tests within each condition. In the implicit condition, sad faces peaked later over the left N170 than neutral faces ([M_{Sad}=165.73ms (SD=2.29) and $M_{Neutral}$=161.56ms (SD=2.66)]; t_{15}=2.865, p=.01, Cohen's d=.43). In the explicit condition, sad faces peaked later than neutral faces in the VPP ([M_{Sad}=152.68ms (SD=2.98) and $M_{Neutral}$=150.22ms (SD=2.81)]t_{15}=3.766, p=.002, Cohen's d=.81) and in the right N170 (M_{Sad}=158.98ms (SD=1.93) and $M_{Neutral}$=155.76ms (SD=1.69)]; t_{15}=2.12, p=.05, Cohen's d=.44).

P150 (130-170 ms).

The repeated-measures ANOVA revealed no significant main effects or interactions.

Discussion

The goals of this project were to examine the effect of implicitly and explicitly presented emotional facial expressions on reaction time and ERP components known to be sensitive to facial and/or emotional processing, using a well-controlled paradigm.

Participants' reaction times were fastest to explicitly presented happy faces and slowest to sad and fearful faces. Furthermore, participants were least accurate when responding to explicitly presented sad and fearful faces, suggesting these expressions required more effort and were more difficult to readily identify than neutral and happy faces. This finding is similar to Williams et al's (2009) study, which found participants to respond slowest to fearful faces and most accurately to happy faces in the explicit condition. However, in contrast to their study, emotions in the implicit condition did not elicit different reaction times in the current study. This difference may be in part attributable to the simplicity of the implicit task in this experiment and/or to the very fast exposure time (200ms) of the faces in this study relative to other studies presenting emotional faces.

EEG results from the present study also provided support for the hypothesis that implicit and explicit presentations of emotion are processed within separate neural networks (Williams et al., 2006). Specifically, implicit presentations of fearful faces elicited a greater voltage peak negativity in the N170 whereas explicit presentations did not. Conversely, explicit presentations of fear modulated the VPP but implicit presentations did not. Likewise, implicit presentations of sad faces modulated the frontal N4a whereas this extended negativity was not modulated by explicitly presented sad faces. The inverse was true for the posterior LPP, which was greater for explicit than implicit faces. In summary, the present study succeeded in finding differences between implicit and explicit presentations of emotions in the predicted direction, although some of the effects were subtle. Discrepancies with findings in neuroimaging studies may reflect differences in EEG and fMRI technology. Although EEG is ideally suited for capturing the time course of emotional face processing, primarily recording cortical activity, EEG cannot reliably detect limbic system activation, which is an important part of the implicit

versus explicit distinction. Further investigation into this question could be completed using source analyses to help localize the likely generator of ERP scalp effects.

This study also examined how emotional expressions modulated ERP effects between 100ms and 600ms post-stimulus onset. The present study generally supported the hypothesis that different emotions uniquely modulate ERP effects. Of particular note, fearful and sad faces were differently processed despite both being negatively valenced emotions. This finding is in contrast to earlier research on the LPP which concluded that emotions did not uniquely modulate ERP components. This study showed that early ERPs are differently modulated by fearful vs. sad faces, consistent with evidence from neuroimaging studies.

With regard to fearful faces, implicitly presented fearful faces elicited greater peak right N170 response than neutral faces. This indicates that the N170 is modulated by emotional expressions of fear when fear is implicitly presented, consistent with a plethora of research which has found that the N170 can be modulated by emotional facial expressions (Batty & Taylor, 2003; Caharel et al., 2005; Kolassa & Miltner, 2006; Krombholz et al., 2007; Rossignol et al., 2005; Williams et al., 2006), and also supports the original hypotheses of this study that implicitly presented emotion would influence the N170 whereas explicitly presented emotions would not. The fact that implicit presentations of fearful faces alone modulated the right N170 reflects the importance of posterior brain regions – in particular right ventral temporo-occipital cortex- in the rapid processing of threat. It also indicates that the VPP and N170 are dissociable components as they were differently modulated by implicit and explicit presentations of emotional facial expressions.

Modulation of the N170 by fear supports seemingly supports fMRI research showing connections between the amygdala and posterior brain regions. Given extensive connections between limbic and paralimbic regions of the brain and posterior occipito-temporal regions (Kober et al., 2008), one could hypothesize a direct threat pathway between the amygdala and regions of the brain typically associated with processing faces (such as the fusiform gyrus) for rapid detection of threat from facial expressions. A plethora of evidence, dating back to Darwin (1872/1998), points to the importance of human faces in communicating important information about another person's internal

state and about the environment. It would make biological sense to have extensive connections between these brain regions to facilitate rapid recognition of a fearful face to quickly facilitate an adaptive response.

Furthermore, implicitly presented emotional faces were processed more rapidly over the right hemisphere than over the left at 100ms and 170ms post-stimulus onset. Taken together with modulation of only the right N170 by fearful faces, these findings are consistent with previous results indicating that faces are preferentially processed in the right hemisphere (Kanwisher, McDermott, & Chun, 1997).

It is noteworthy in this study that sad faces elicited a unique pattern of results compared to fearful and happy faces. This pattern started early over frontal cortices, at approximately 150ms, and continued through to the end of the LPP (600ms in this study), suggesting a unique system in the prefrontal cortex that processes sad faces. In the explicit and implicit conditions, sad faces elicited greater peak positivities early in the epoch over frontal cortices (P150, VPP). This continued through the epoch with explicit presentations eliciting a greater sustained positivity in the EAP and implicit presentations eliciting greater negativity in the N4a. Explicit presentations of sad faces also uniquely modulated the LPP.

Unlike an expression of fear, sad faces do not convey threat. A fearful face may be perceived as warning of potential danger in the environment, thus eliciting an automatic response in the brain. By contrast, sad faces convey the internal, emotional state of another person. As a result, they are more socially salient and carry more social significance than fearful faces. Such processing, in which sad faces are rapidly recognized followed by attribution what it is to feel sad, seems to occur fairly rapidly within the frontal cortex, as reflected by ERP modulations over anterior scalp regions. Frontal cortices have been hypothesized to play an important role in the recognition of emotion and in attributing meaning to that emotion (Allison et al., 2001). In this study, these modulations were unique to sad faces, perhaps suggesting that other facial expressions (namely happy and fearful) do not evoke internal reflection or do not carry the same social significance as sad faces. Previous research has suggested a functional distinction between emotional responses to stimuli of immediate survival value, or related to primary motives, such as threat, food, drink or erotic stimuli (Morris & Dolan,

2002; i.e. fearful faces in this study) and social stimuli, not directly related to survival (Eisenberger, Lieberman, & Williams, 2003; i.e. sad faces in this study). Results from the current study suggest a dissociation between these two emotions: Emotional expressions important for primary motives may recruit occipito-temporal cortical brain regions, important for discerning the basic attributes of a faces, whereas socially motivating emotional facial expressions recruit regions of the frontal cortex, implicated in recognizing the emotional significance of the facial expression and forming a conscious representation of emotional facial expression (Adolphs, 2003; Eimer & Holmes, 2007).

Happy faces also reflect an internal emotional state, and are thus also socially motivated and relevant. However, unlikely sadness, happy faces communicate that all is well, no compensatory action is required, and they do not elicit empathy. Furthermore, smiles are often used as conversational markers in the absence of genuine emotion (non-Duchenne smiles), and therefore may have less emotional impact than sad faces, which typically communicate others' sorrow and unhappiness. They may convey that, socially, the observer has made an error and upset another person. Alternatively, a sad face may elicit empathy in the observer and an appreciation of how the person is feeling. By social convention, the observer may feel compelled to make efforts to improve the person's mood. In essence sad faces communicate an internal state of unhappiness, which indicates that something is wrong, and may ultimately implore the observer to respond in a socially appropriate manner. In contrast, happy faces communicate that all is well, which is likely what the observer expects. Sad faces mismatch such expectation by communicating that all is not well.

The dissociation identified in the current study between sad faces (social) and fearful faces (biological) are very similar to those reported by Sakaki, Niki, and Mather (2012) using fMRI. Using socially and biologically emotional pictures, this group found that social pictures induced relatively greater activity in the bilateral medial PFC compared to biological pictures. The dorsal medial PFC has been linked to elaborative processing of affective stimuli. In contrast, biological pictures elicited greater activity in the occipital gyrus and cerebellum than social pictures (Sakaki et al., 2012). Moreover, functional connectivity analyses revealed stronger functional connectivity between the amygdala and occipital lobe/inferior parietal region for biologically relevant emotional pictures, whereas there was a stronger correlation between the amygdala and the left dorsal PFC

for socially relevant emotional pictures (Sakaki et al., 2012), indicating different neural networks for processing biologically versus socially salient affective information.

These findings complement the dissociation between how fearful and sad faces modulated posterior versus anterior ERP components in the current study. Information from the current study, however, provides additional insight into how the brain processes so-called biologically relevant versus socially relevant information from faces. Sad faces were processed as rapidly as fearful faces in the brain, with both expressions modulating ERP components as early as approximately 150ms post-stimulus onset. This suggests a system in the brain designed to rapidly process socially salient emotions, at least from facial expressions, in addition to a system for threat and biologically relevant emotions.

The findings from this study, and those of Sakaki et al (2012), suggest that there may be a system in the brain, with heavy involvement from regions in the prefrontal cortex, that is recruited by socially salient information. What remains to be clarified is whether these regions of the frontal cortex are uniquely sensitive to detecting negative internal states of another person that typically elicit empathy, or if they are sensitive to detecting negative feedback in a social interaction. Studying these ERP effects in response to angry faces in addition to sad faces would help clarify whether or not these brain areas are uniquely primed for empathy or if they are responsive to negative feedback in social situations. A similar response to angry faces would indicate that this system is sensitive to negative feedback in a social interaction rather than being specific to emotional expressions eliciting empathy. Furthermore, examining this system with fMRI would help clarify exactly which areas of the brain, both cortical and sub-cortical, are recruited in recognizing sadness in a face. Future studies could also examine the development of this system across the lifespan. Recognizing the internal emotional state of another person is very important in the context of social interactions and social communication. It would thus be interesting to study this proposal in individuals with conditions affecting social cognition and theory of mind, such as autism, and in individuals who may be hyper-sensitive to erring in social interactions or to negative affect in another person (e.g. social anxiety). One might expect hypo-activation in this system in individuals with autism, given difficulties inferring and appreciating the internal emotional state of others. In contrast, one might expect hyper-activation in response to sad faces in individuals with social anxiety.

Tables

Table 1.1.
Mean (SD) Reaction Times to Implicit and Explicit Emotions in Miliseconds.

	Neutral	Fearful	Sad	Happy
Implicit	724.25[x]	725.70[x]	733.05[x]	715.36
	(102.44)	(110.93)	(103.55)	(96.62)
Explicit	831.88	918.58[*]	884.82[*]	771.78[*]
	(96.75)	(107.35	(96.02)	(93.16)

Note: [*]Reaction time is significantly different from neutral at p=.05 level. [x]Reaction time faster in the implicit than in the explicit condition at p=.013.

Table 1.2.

Peak and Mean Amplitude (SEM) of Implicit and Explicit Presentations Emotions for each Component in Microvolts.

	Implicit				Explicit			
	Neutral	Fearful	Sad	Happy	Neutral	Fearful	Sad	Happy
Right P1	5.35 (.79)	6.03 (.95)	5.96 (.96)	6.24 (.92)	6.53 (.70)	6.42 (.86)	5.95 (.61)	5.96 (.76)
Left P1	4.44 (.92)	4.48 (.93)	4.49 (.89)	3.96 (.77)	4.37 (.66)	4.27 (.65)	4.57 (.81)	3.96 (.69)
Right N170	-3.34 (.71)	**-4.11*** (.74)	-3.05 (.65)	-4.02 (.79)	-3.76 (.74)	-3.62 (.77)	-3.82 (.81)	-4.28 (.76)
Left N170	-2.90 (.38)	-3.48 (.37)	-3.32 (.38)	-3.19 (.37)	-2.94 (.39)	-2.84 (.47)	-2.87 (.41)	-3.04 (.48)
VPP	2.99 (1.33)	3.74 (1.32)	**4.63*** (1.33)	3.40 (1.12)	4.61 (1.19)	**5.82*** (1.42)	**5.85*** **(**1.28)	4.82 (1.32)
P150	1.51 (1.25)	2.12 (1.15)	**2.79*** (1.09)	2.49 (1.15)	2.50 (1.29)	3.13 (1.35)	**4.12*** (1.40)	3.65 (1.33)
EAP	-3.30 (.55)	-3.16 (.54)	-2.79 (.55)	-3.17 (.69)	-2.05 (.60)	-1.39 (.53)	**-0.49* (.56)**	-1.72 (.61)
Anterior LPP	.06 (.87)	-.49 (.79)	**-1.58* (.71)**	-.14 (.77)	2.78 (1.09)	2.59 (1.06)	3.00 (1.18)	2.53 (.92)
Posterior LPP	8.12 (1.54)	8.16 (.96)	7.96 (1.11)	8.30 (.94)	10.85 (1.62)	11.79 (1.18)	11.02 (1.57)	10.72 (.99)

*Emotion is significantly greater than neutral at p=.05 level.

Figures

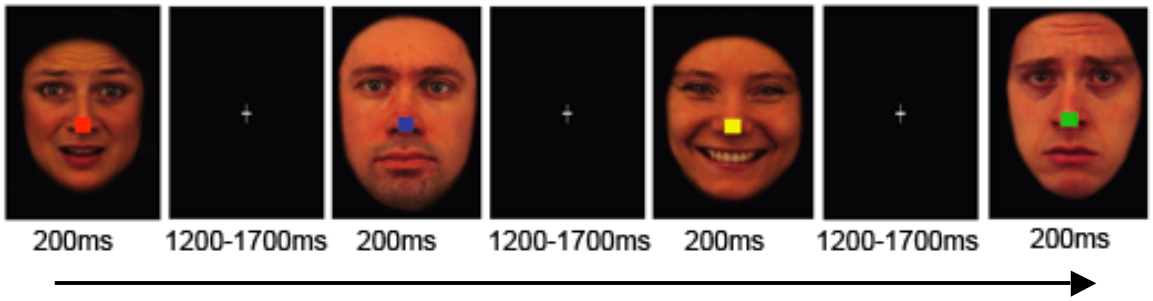


Figure 1.1. The timecourse of stimulus presentation in the implicit and explicit conditions.

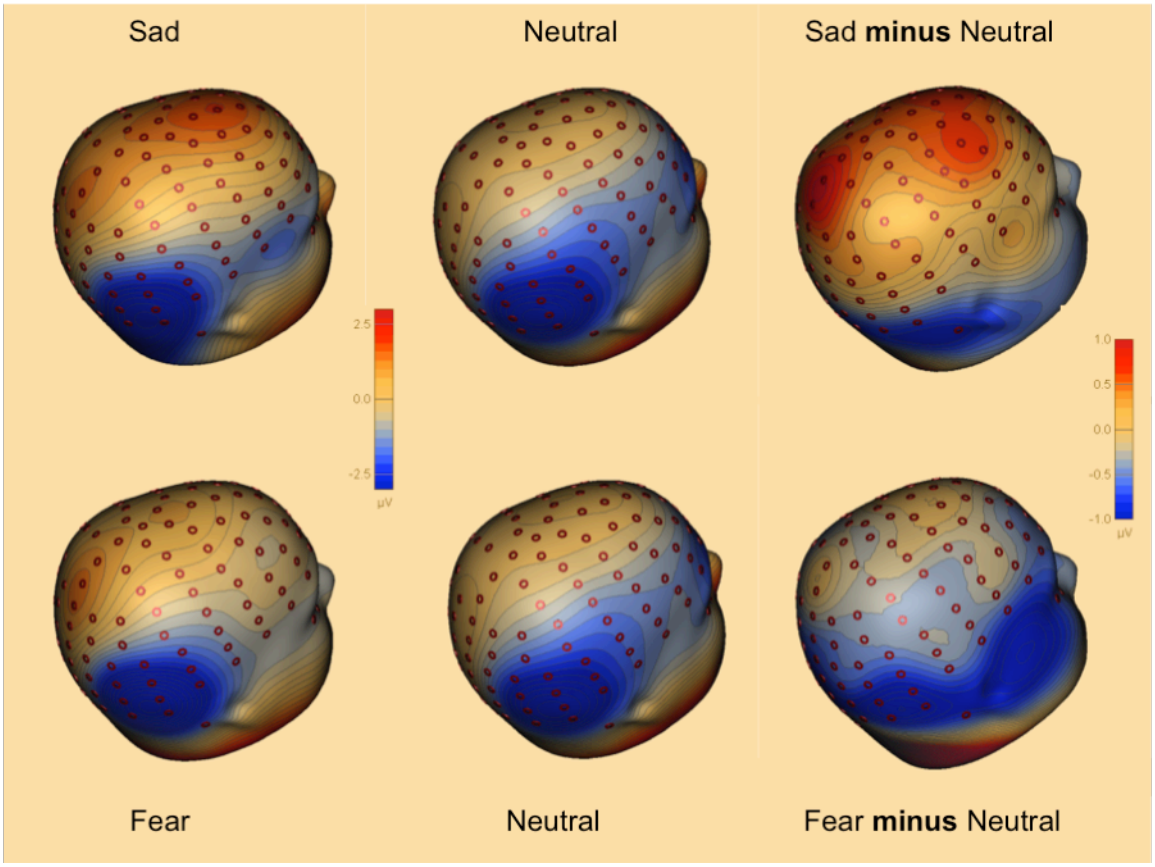


Figure 1.2. Topographical maps showing the distribution of scalp negativity and positivity at time point of the P150, VPP, and N170.

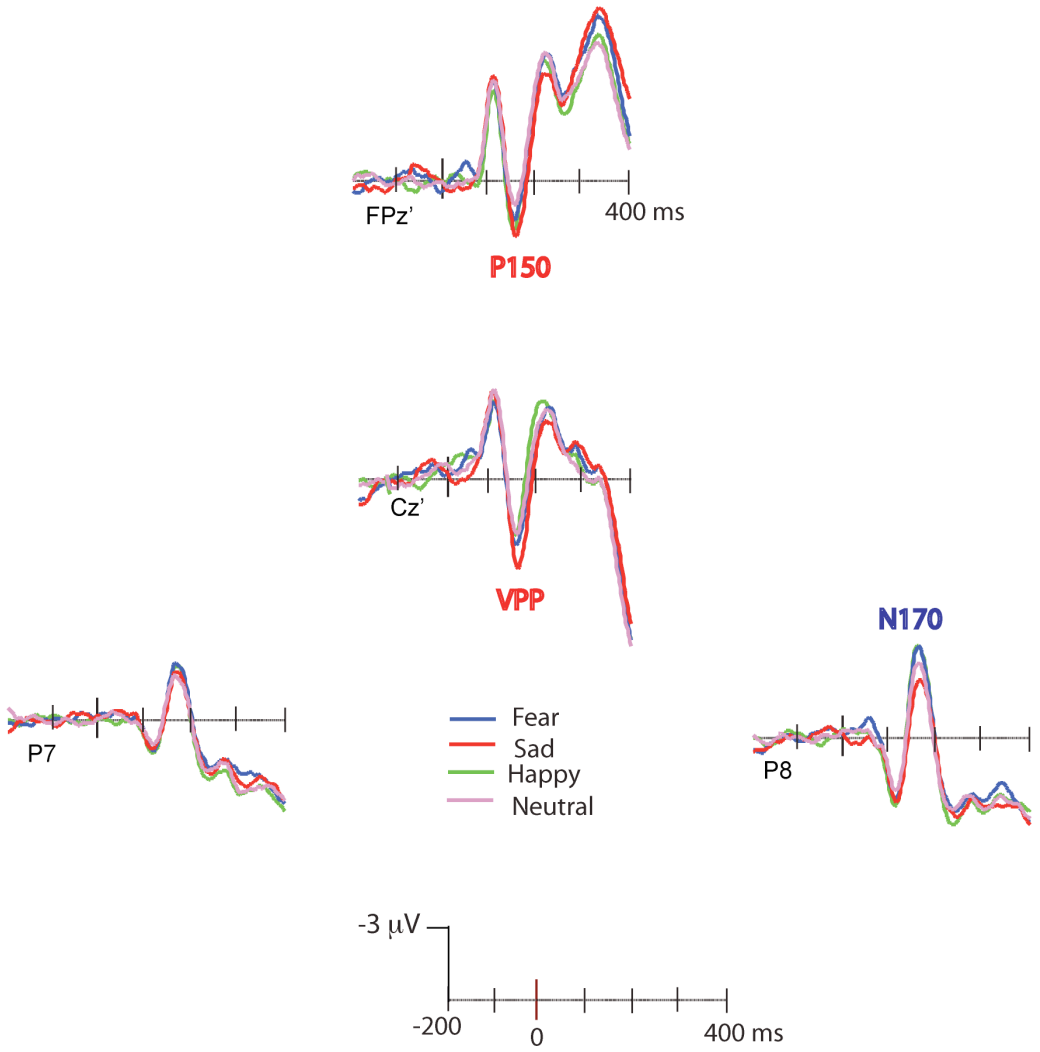


Figure 1.3. Modulations in the P150, VPP, and N170 in response to implicit presentations of fearful, sad, happy, and neutral facial expressions. Y axis in microvolts (uV), X axis in ms.

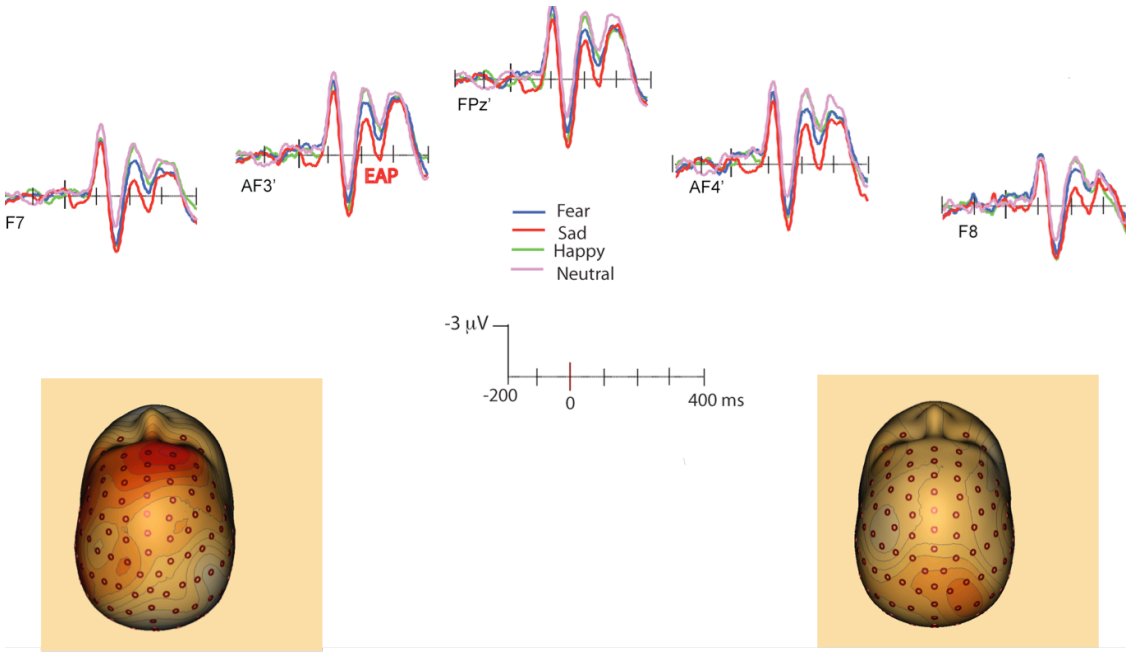
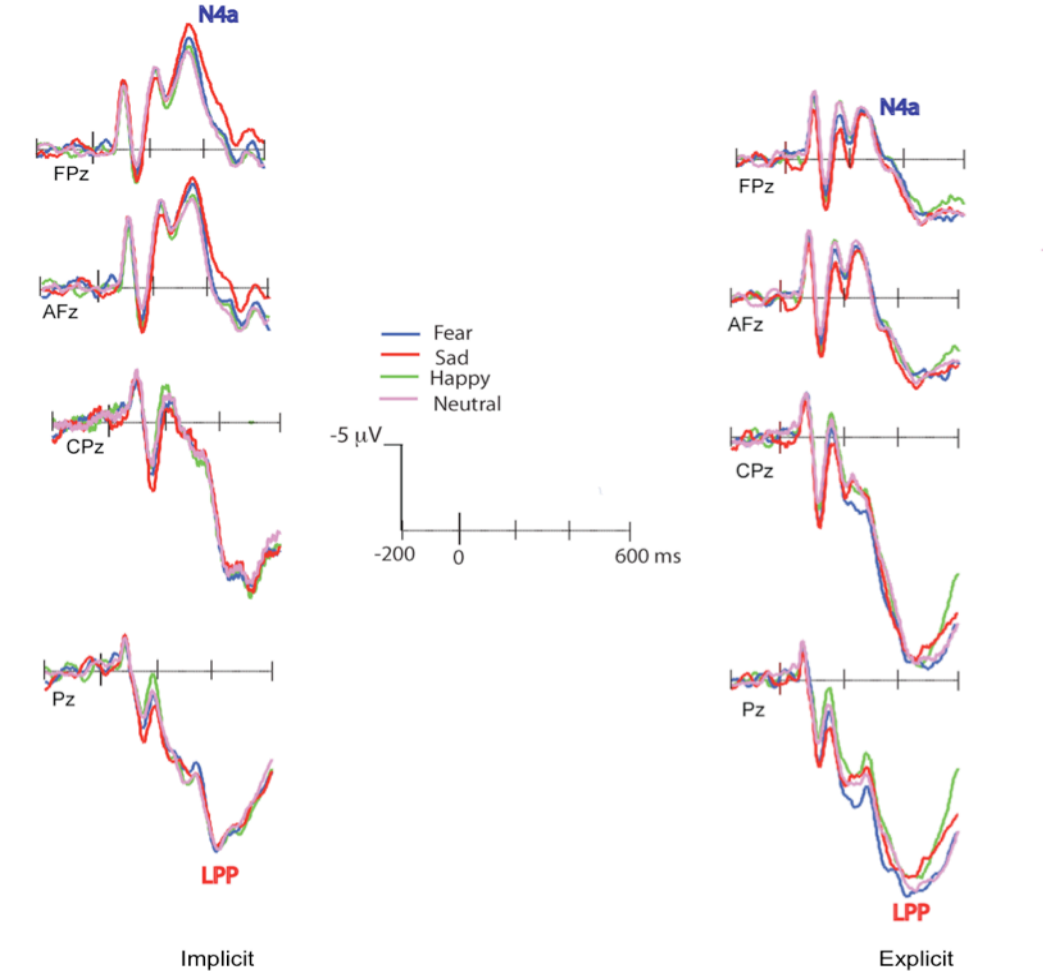
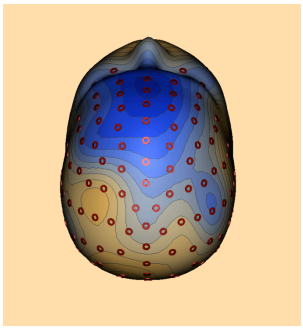
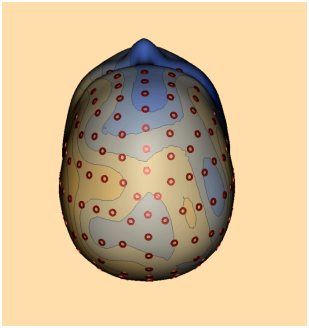


Figure 1.4. Modulations of EAP in response to explicit presentations of fearful, sad and neutral facial expressions. Y axis in microvolts (uV), X axis in ms. Top: Average waveforms for the various emotions in the explicit condition. Bottom: Scalp distribution of the difference waves Sad minus Neutral (left) and Fear minus Neutral at the EAP peak (246 ms).







Sad minus Neutral (394 ms)

Fear minus Neutral (394ms)

Figure 1.5. Modulations of the N4a and LPP implicit and explicit presentations. Top: grandaverage waveforms for fearful, sad, happy and neutral facial expressions. Bottom: Scalp distribution of the difference wave Sad minus Neutral at the N4a peak (394 ms) for Implicit (left) and Explicit (right) condition.

Chapter 2.
The Neural Mechanisms of Emotional Face Processing in Autism

Autism is a pervasive developmental disorder (PDD) characterized by delays in language and communication, repetitive or stereotyped behaviour, and deficits in social and emotional functioning (American Psychiatric Association [APA], 2000). The prevalence of PDD is estimated to be approximately 60-70 per 10,000, whereas autism proper has an estimated prevalence of 22 per 10,000 (Saracino, Noseworthy, Steiman, Reisinger, & Fombonne, 2010), and Asperger's Syndrome, three per 10,000 (Fombonne, 2005).

In both Kanner's (1943) and Asperger's (1944/Frith, 1991) initial descriptions of autism, emotional abnormalities and social difficulties were described in children with autism. Since these original descriptions, a plethora of research has examined the socio-emotional deficits apparent in autism. Difficulties with social and emotional communication are of particular interest to research because these are often very distressing to family members and are very noticeable in the current world that emphasizes a high degree of social relations. Furthermore, although individuals with autism, PDD-NOS, and Asperger's Syndrome (henceforth referred to collectively as autism) may have differences in many aspects of cognition, evidence suggests that social cognition is disproportionately affected (Adolphs, 2001; Baron-Cohen, 1995).

Socio-emotional functioning has been studied in various ways. This study focussed on the time course of underlying neural correlates of basic emotional face processing to implicitly (i.e. not the focus of attention) and explicitly (i.e. the focus of attention) presented emotional faces in high functioning individuals with autism (Abbreviated IQ between 90-110). Of particular interest was how sad faces were processed in autism compared to other emotions and compared to their typically developing (TD) peers. A very brief overview of basic face processing in autism is provided below, prior to reviewing results from studies focusing on emotion recognition and emotional face processing.

Face Processing in Autism

A prominent deficit observed in children with autism is their lack of interest in human faces. While most infants orient towards a human face from birth (Morton & Johnson, 1991), children with autism have retrospectively been shown not to demonstrate this preference (Osterling & Dawson, 1994), nor do they appear to develop a strong preference for faces over the course of their life (Swettenham et al., 1998). Studies using parent reports and retrospective review of home videos have provided evidence of atypical social behaviours in children who were later diagnosed with autism. Specific examples of such behaviours included reduced eye contact, poor gaze following, less time spent looking at faces, fewer instances of social smiling, and fewer instances of imitating other people (Volkmar, Chawarska, & Klin, 2005).

This may have important implications for the development of social relationships and social functioning over time, as understanding the importance of the face and its role in social interactions is important for engaging in successful social exchanges. Faces and facial expressions are an integral part of socio-emotional communication and social understanding as they provide essential information about another person's emotional state and the surrounding environment (Darwin, 1872/1998; Dolan, 2002). Experience with faces is likely necessary for the typical development of emotional expression recognition (Leppanen & Nelson, 2006; Sasson, 2006). Consequently, it is proposed that the lack of facial interest demonstrated by infants and young children with autism is a crucial component of their difficulties with socio-emotional processing and socio-communication observed later in life. It may represent the start of an atypical developmental trajectory that results in differences in social-perception later in life (Leppanen & Nelson, 2006; Sasson, 2006).

Behaviourally, three popular theories that have been proposed to explain differences in social communication in autism include impairments in theory of mind (ToM), impairments in central-coherence (O'Connor, Hamm, & Kirk, 2005), and Hobson's affective theory of autism, which proposes that autism is a disorder of affect and social relations (1989). The first suggests that social deficits in autism result from a general impairment in ToM (i.e. a deficit in interpreting the mental state of others and attributing thoughts and intentions to others; Baron-Cohen, Ring, Bullmore, Wheelwright, Ashwin, & Williams, 2000). Consistent with this theory, deficits in establishing joint attention and in

imitating others—potential precursors to ToM—have been empirically demonstrated in young children with autism (Charman, 2003).

The central-coherence theory of autism posits that social communication difficulties in autism may arise from a difference in the ability to integrate details into a coherent whole and understand the context in which information is presented (Behrmann, Thomas & Humphreys, 2006; Frith, 1989; Frith & Happe, 1994; Happe & Frith, 2006). This is consistent with the commonly reported processing style in autism of being highly attuned to and adept at processing the specific details of a written text, a pictured situation, or a face instead of focussing and processing the overall meaning of the stimulus (Happe, 1997; Joliffe & Baron-Cohen, 1999; Vermeulen, 2001).

Hobson's affective theory proposes that typically developing infants are genetically programmed to be sensitive to and comprehend another person's emotions (1989). Fundamentally, this theory proposes that humans are biologically wired to recognize the internal mental or emotional state of another human being. The affective theory of autism suggests that this biological wiring for comprehending emotional states in other humans is dysfunctional in autism, resulting in difficulties understanding emotional expressions, difficulties with imitation, pretend play, imitation, and role-taking activities in which the individual is required to understand the intentions of another person (Hobson, 1989).

Past research on basic face recognition and processing has found evidence that individuals with autism process faces differently than their typically developing peers, as is posited by the central coherence theory of autism. For example, individuals with autism tend to favour and show a relative facility with a piecemeal, analytical approach to face processing, in opposition to control participants who tend to show a preference for holistic face processing. This is consistent with a general processing style that focuses on visual details (Mottron, Dawson, Soulieres, Hubert, & Burack, 2006). With regard to face processing, individuals with autism tend to show similar performance for recognizing upright and inverted faces (Hobson, Ouston, & Lee, 1988) whereas TD individuals demonstrate a relative deficit when processing inverted faces (Yin, 1969). Similarly, individuals with autism seem to rely on high spatial frequencies to recognize the identity of a face as opposed to TD individuals, who typically rely more strongly on

low spatial frequencies (Deruelle, Rondan, Salle-College, Bastard-Rosset, & DaFonseca, 2008). Past research has also shown that TD individuals fixate more on the eye region of the face whereas individuals with autism spend more time looking at and deriving information from the mouth region (Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Klin, Jones, Schultz, & Volkmar, 2003; Joseph & Tanaka, 2003). Although see Lahaie, Mottron, Arguin, Berthiaume, Jemel, & Saumier, 2006; Pelphrey, Sasson, Reznick, Paul, Goldman, & Piven, 2002; van der Geest, Kemner, Camfferman, Verbaten, and van Engeland, 2002 for contradicting evidence. Taken together, these results suggest that individuals with autism use a piecemeal, part-based approach to analyzing faces as opposed to the holistic approach employed by their TD peers.

Differences in face processing have also been reported at the neural level between TD individuals and individuals with autism. For example, it has often been reported that individuals with autism show hypoactivation of the fusiform gyrus (or Face Fusiform Area, FFA), a region of the brain that is typically activated by very familiar stimuli (or stimuli of expertise), most typically faces in TD populations (e.g. Golarai, Grill-Spector, & Reiss, 2006; Pierce, Muller, Ambrose, Allen & Courchesne, 2001; Sasson, 2006; Schultz et al., 2000). Hypoactivation has also been reported in the superior temporal sulcus (STS; Golarai et al., 2006; Pierce et al., 2001), a region of the brain strongly associated with processing biologically meaningful motion, including eye, mouth, and face movements (Allison, Puce, & McCarthy, 2000). Different explanations have been proposed to explain this hypoactivation, including less time spent looking at the eyes (Dalton et al., 2005), and variations in task demands (Harms, Martin & Wallace, 2010). These findings may also be interpreted as a relative lack of unique expertise with faces in people with autism as a result of under-exposure to faces from a young age or, alternatively, be the consequence of an innate impairment in a specialized neural system (Sasson, 2006). Similarly, certain regions of the brain typically associated with object processing in TD individuals (inferior temporal and lateral occipital regions) have shown *enhanced* activation in response to faces in individuals with autism (Hubl et al., 2003; Schultz et al., 2000), perhaps suggesting that individuals with autism process faces like an everyday object or, alternatively, that they are not perceiving faces as an important communicator of socially relevant information. Together, these findings suggest that different neural networks may be involved in processing faces in autism, although there

is variability in the literature and not all studies have reported hypoactivation in the fusiform gyrus and STS (see Hadjkhani et al., 2004; Hakjikhani, Joseph, Snyder, & Tager-Flusberg, 2007; Pierce, Haist, Sedaghat, & Courchesne, 2004; for a review on face processing in autism see Jemel, Mottron, & Dawson, 2006).

Emotional Face Processing in Autism: Behavioural Results

As very briefly reviewed above, experimental studies have looked at various aspects of face processing in autism, often finding differences in facial recognition abilities (Boucher, Lewis, & Collins, 1998; Gepner et al., 1996), how individuals with autism process faces (Barton et al., 2004; Behrmann et al., 2006; Lahaie et al., 2006), and the brain regions recruited to process faces (Golarai et al., 2006; Hubl et al., 2003; Pierce et al., 2001; Sasson, 2006; Schultz et al., 2000). However, of particular interest to this study are those studies specifically examining emotional face recognition and processing in autism.

With regard to emotion recognition, some studies have shown that individuals with autism have difficulties recognizing basic emotions from facial expressions, such as happy, sad, anger, disgust, surprise and fear, obviously leading to deficits in interpreting and understanding the social implications and meaning of these facial expressions (Ashwin, Wheelwright, & Baron-Cohen, 2006; Corden, Chilvres, & Skuse, 2009; Howard et al., 2000; Wallace, Coleman, & Bailey, 2008). Interestingly, some studies have found that participants with autism have difficulty recognizing certain emotions (typically negative emotions), but not others. For example, some researchers have reported that children with autism are impaired at recognizing surprise, but not happiness or sadness (Baron-Cohen, Spitz, & Cross, 1993), whereas other groups have found that recognizing anger, (O'Connor, Hamm, & Kirk, 2005), disgust (Humphreys, Minshew, Leonard, & Behrmann, 2007), fear (Howard et al., 2000; Humphreys et al., 2007; Pelphrey, Sasson, Reznick, Paul, Goldman, & Piven, 2002), or sadness (O'Connor et al., 2005) were relatively impaired compared to other facial expressions. Such results indicate that individuals with autism may have difficulties recognizing and correctly identifying at least some of the basic emotional expressions from faces, typically negative emotional expressions, suggesting that it is at this basic level that emotional face processing may

break down, contributing at least in part to differences in socio-emotional functioning evident in autism.

Several other studies have reported that individuals with autism do not have deficits identifying basic emotions, but they rather have difficulties recognizing higher-order emotions or mental states from faces, such as trustworthiness, flirtatiousness, arrogance (Adolphs, 1999; Baron-Cohen, Wheelwrite, & Jolliffe, 1997), pride, shame, or guilt (Williams & Happe, 2010). These findings are consistent with a variety of tasks that have suggested that the central difficulty in autism arises from the inability to perceive and make sense of other people's mental states, consistent with the ToM hypothesis of autism (Baron-Cohen, 1995). In a detailed analysis of emotional face processing in autism, Adolphs, Sears and Piven (2001) conducted a series of experiments that provided support for the idea that people with autism are able to form normal perceptual representations of faces, and are able to retrieve knowledge regarding basic emotional expressions, but that they fail to link perception of the face to the social judgments required for successful social interactions.

There are several possible explanations for such discrepancy between studies. Differences in outcome may reflect differences in task demand (Harms et al., 2010). The various studies have employed different stimuli, different facial expressions, and tasks of varying difficulty. Discrepancies may also reflect differences in level of functioning across studies. For example, Loveland et al. (1997) found no difference between high functioning individuals with autism and TD controls on a basic emotion identification task but did find differences between individuals with high and low functioning autism, with low functioning individuals showing a relative deficit, suggesting that perhaps the discrepancy in the literature thus far is explicable by differences in the participants' level of functioning. In contrast to this idea, other studies have not found differences between individuals with autism with Full Scale IQs around 70 and their peers with Learning Disabilities with similar IQs (e.g. Williams & Happe, 2010), although it is important to note that a Learning Disability suggests atypical brain development. Studies have also employed different control groups, matching to the autism group on different variables (Harms et al., 2010). Some studies match at the overall group level whereas others individually match participants in the autism and control groups (Harms et al., 2010). Autism and control groups have been matched on chronological age or mental age

alone, Verbal IQ, Performance (nonverbal) IQ, or Full Scale IQ, on occasion handedness, or some combination of the above variables (for a review see Harms et al., 2010). This may further complicate interpretation of the literature. Specifically, some child studies have found differences in emotion recognition when participants are matched on nonverbal ability but no differences when matched on verbal ability (Braverman, Fein, Lucci, & Waterhouse, 1989; Fein, Lucci, Braverman, & Waterhouse, 1992; Ozonoff, Pennington, & Rogers, 1990), suggesting differences in results across studies may simple reflect differences in matching procedure employed. Another possible explanation, which cannot yet be ruled out and may in fact account for much of the variability among studies is that differences between studies reflect genuine differences in participants with autism. Autism is a heterogeneous spectrum disorder whose diagnostic criteria have changed since its recognition in 1943. The diagnosis and assessment of autism continues to evolve as our understanding changes (Saracino et al., 2010), and differences in diagnostic criteria used for inclusion may also account for variability in the literature.

In high functioning autism, the variability in results may also be explained by variations in task demands. Under standard viewing conditions with prototypical emotional expressions, several studies suggest that many individuals with autism can identify emotions as well as their typically developing peers. Some studies, by contrast, suggest a deficit with negative and so-called higher-order or social emotions (e.g. arrogance, flirtatiousness, shame, pride; Adolphs, 1999; Baron-Cohen, Wheelwrite, & Jolliffe, 1997; Happe & Williams, 2010). However, when facial-emotion processing is made more difficult (e.g., presenting two incongruent emotions simultaneously or shortening the total presentation time), facial emotion recognition deficits emerge in high-functioning individuals with autism (Harms et al., 2010). This may suggest that the process of recognizing emotions is not as automatic, not as natural and easy, for individuals with autism as it is for their TD peers. Alternatively, it may reflect an overall difference in processing style between the two groups. As task demands increase, so typically do extraneous details. As a plethora of previous research has suggested that individuals with autism are better at recognizing and attending more to the details than to the gestalt of an image, difficulties with increasing task demands may rather reflect difficulties

focussing on the face rather than difficulties recognizing the emotional expression per se.

It, nonetheless, remains unclear how individuals with high-functioning autism successfully recognize emotions from faces and at what level the ability to infer socio-emotional meaning from faces and respond in a socially expected manner breaks-down resulting in commonly reported difficulties with socio-emotional functioning. It seems possible, at least in high functioning individuals with autism, that social communication becomes difficult at some higher level of social cognition, perhaps at the point where the emotional expression displayed on a face must be attached to an internal representation of the socio-emotional significance of this expression and ultimately its social meaning.

Emotional Face Processing in Autism: Neuroimaging Results

In recent years, a growing body of research has emerged describing differences in the neural systems possibly underlying social emotional communication difficulties in individuals with autism. After reviewing the literature, results from these studies seem to fit into one of four main hypotheses/theories that are arguably convergent and likely compatible. An early theory, the amygdala theory of autism, identifies a dysfunctional amygdala as the source of socio-emotional and social cognitive difficulties in autism (Baron-Cohen, Ring, Bullmore, Wheelwright, Ashwin, & Williams, 2000). The second proposed theory references early dysfunction in the mirror neuron system as an underlying cause of relative difficulties with socio-emotional communication in autism (Dapretto et al, 2006; Williams, Whiten, Suddendord, & Perret, 2001). Thirdly, specific areas of the brain have been deemed essential in processing socially relevant information, such as emotional faces, and are hypothesized to work in concert to accomplish successful emotional/social processing. These regions, collectively known as the social brain, include the amygdala, the STS, the fusiform gyrus, and regions of the frontal cortices, most notably, the orbito-frontal cortex OFC and the ACC (Allison et al., 2000; Baron-Cohen, 1995; Brothers, 1990). The social brain is hypothesized to work differently in autism (Hakjikhani et al., 2007). Finally, abnormal neural connectivity between regions of the brain, and most notably regions of the frontal cortices (including motor cortices) with more posterior regions of the brain (Just, Cherkassky, Keller, Kana, & Minshew, 2007; Kana, Keller, Cherkassky, Minshew, & Just, 2006; Kleinhans et al.,

2008; Koshino, Kana, Keller, Cherkassky, Minshew, & Just, 2008; Mostofksy et al., 2009; Mostofksy & Ewan, 2011; Villalobos, Mizuno, Dahl, Kemmotsu, & Muller, 2005; Wicker, Fonlupt, Hubert, Tardif, Gepner, & Deruelle, 2008) has been proposed as a possible explanation for social-communication deficits observed in autism.

Amygdala Theory of Autism

The amygdala has long been implicated as being an essential component to processing socially relevant stimuli. Traditionally, the amygdala has been linked to processing fearful or aversive stimuli; however, more recently it has been linked to processing stimuli that are pleasant, as well as aversive (Adolphs, 1999). It has been proposed that this structure may play a more general role in allocating resources to process biologically salient stimuli that require additional processing, regardless of the stimuli's emotional valence (Whalen & Henker, 1999). Alternatively, the amygdala may be involved in attaching emotional salience to sensory input (Adolphs, 1999) or play a role in attaching meaning to socially salient cues (e.g. Allison et al., 2000).

It has been suggested that, at some point during development, children acquire the link between a facial expression and the personal experience of that emotion (Adolphs, Damasio, Tranel, & Damasio, 1996). This would imply that there is a neural structure that can perceive the facial expression of, for example fear, as well as link this expression to the experience of fear (Adolphs et al., 1996; Dawson et al., 2004). Due to substantial evidence implicating the amygdala as playing an important role in processing emotional information from faces (e.g. Morris, Ohman, & Dolan 1998; Zald, 2003), Aggleton and Young (2000) have proposed this to be the role of the amygdala. It has thus been hypothesized that the amygdala may account for many of the differences in processing expressive faces in autism (e.g. Baron-Cohen et al., 2000; Hall, West, & Szatmari, 2007).

Supporting this proposal, structural abnormalities in the amygdala have been reported in individuals with autism. Some structural examinations have found enlarged amygdala volume in children (e.g. Schumann, Bauman, Machado, & Amaral 2006) and adults with autism (Howard et al., 2000) whereas other studies have found reduced amygdala volume (Aylward et al., 1999). In contrast, other authors have not found structural

amygdala differences between healthy controls and individuals with autism (Haznedar et al., 2000).

Experiments conducted with the aim of clarifying and comparing socio-emotional processing in individuals with autism to those with amygdala damage have found similarities between the two groups (e.g. Adolphs et al., 2001; Howard et al., 2000). Specifically, individuals with autism and amygdala damage had more difficulty recognizing fearful faces, using information from faces to make appropriate social judgments, and recognizing faces after a delay. Both groups also tended to attend less to the eye region than TD individuals (Adolphs et al., 2001; Howard et al., 2000).

Some fMRI studies have provided support for the amygdala theory of autism. Specifically, decreased amygdala activation has been observed in response to both emotionally expressive and neutral faces in adults with autism compared to TD adults (e.g. Critchley et al., 2000; Howard et al., 2000; Pierce et al., 2001). However, although the amygdala undoubtedly plays an important role in emotional face processing and socio-emotional functioning more generally, studies have established that it is likely only part of a complex neural network responsible for emotion, face (Golarai et al. 2006), and social processing (Allison et al., 2000; Ashwin, Baron-Cohen, Wheelwright, O'Riordan, & Bullmore, 2007; Brothers, 1990).

The Mirror Neuron System Hypothesis of Autism

It has been proposed that many of the differences in social functioning observed in autism, including in ToM, social communication, and understanding the intentions of others, arise from early dysfunction of the mirror neurons (Williams et al., 2001). Mirror neurons were first discovered in macaques and refer to cells in the ventral premotor cortex that fire while the monkey is performing an action and while the monkey is watching an experimenter perform the same or a similar action (Rizzolatti, Fadiga, Gallese, & Fogassi, 1996). Early studies in humans have reported mirror neuron activity in the inferior frontal gyrus (IFG) during action observation (Johnson-Frey & Grafton, 2003), intention understanding (Iacoboni, Molnar-Szakacs, Gallese, Buccino, Mazziotta, & Rizzolatti, 2005) and imitation (Iacoboni, Woods, Brass, Bekkering, Mazziotta, & Rizzolatti, 1999) in TD samples. Furthermore, and of particular relevance to this paper,

recent investigations have proposed a system involving the frontal MNS and structures of the limbic system that may help humans understand the emotional states of other human beings (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003). Specifically, Carr et al (2003) postulated that the mirror neurons in the IFG work in concert with the insula and the amygdala to process emotionally relevant information, and may constitute a circuit involved in empathy, hypothesized to be hypoactive in individuals with autism.

Studies have revealed abnormal and hypoactivation in the MNS in participants with autism compared to TD participants, as elicited by motor imitation and observation tasks (Martineau, Andersson, Barthelemy, Cottier, & Destrieux, 2010; Nishitani, Avikainen, & Hari, 2004; Oberman, Hubbard, McCleery, Altschuler, Ramachandran, & Pineda, 2005; Theoret, Halligan, Kobayashi, Fregni, Tager-Flusberg, & Pascual-Leone, 2005). Dapretto et al (2006) conducted an fMRI study to compare activity in this proposed system between individuals with autism and TD controls using emotional faces. Participants were instructed to either imitate or observe emotional faces. In both conditions, results revealed that emotional faces elicited strong activations in the so-called frontal MNS, the amygdala and the insula in TD controls, as well as regions in the premotor and motor cortices, face processing regions (including the fusiform gyrus), and occipital cortices. In contrast, imitation and passive viewing of emotional faces did not elicit activations in the insula and frontal MNS in autism (although other brains regions showed similar activation between the two groups). These neurological differences were significant in the presence of equal behavioural performance on the imitation task and no group differences in terms of time spent looking at the face and eye region. These findings were interpreted as indicating that individuals with autism were using a different strategy to imitate and recognize emotional faces. To further support the hypothesis that dysfunction in the MNS may be at the core of social difficulties in autism, this group correlated scores on the social sections of two widely used diagnostic instruments, the Autism Diagnostic Observation Scale (ADOS) and the ADI-R, with amount of activation in the frontal MNS. They found that increased activation in the MNS was correlated with fewer social deficits as measured by the ADI-R and ADOS (Dapretto et al., 2006). Another fMRI examined the MNS in autism across development, using short movies that showed facial expressions, facial movements and an actor experiencing a disgusting taste. This study found that the MNS in the inferior frontal gyrus increased with age in

the autism group but not the TD group (Bastiaansen et al., 2011), suggesting delayed development of the MNS in autism.

These results implicate the importance of mirror neurons in facilitating social communication from emotional faces in TD controls, and further support the broader theory that mirror neurons form a neural system by which the actions and intentions of other people can be understood (Iacoboni et al., 1999; Iacoboni et al., 2005; Johnson-Frey & Grafton, 2003). Although preliminary, Dapretto et al.'s study (2006) also provides support for a role of mirror neurons in processing emotional faces and in autistic symptomology more broadly.

The Social Brain and Autism

In typically developing individuals, faces, eyes, and other social stimuli have consistently elicited activation in key brain regions. These brain areas have been identified across studies using various tasks and different methods of neuroimaging, including fMRI, positron emission tomography (PET), and electroencephalography (EEG). These brain areas are consistently activated by social stimuli and are believed to form a circuit integral to successful processing of social information. Brain regions consistently activated by social stimuli across studies include the amygdala, sometimes in conjunction with other subcortical brain regions including the pulvinar and superior colliculus (Kleinhans, Richards, Johnson, Weaver, Greenson, Dawson, & Aylward, 2010), the insula, areas in the prefrontal cortex and regions of the temporal and occipital cortices, including the fusiform gyrus and the STS. These brain regions form the so-called social brain (Allison et al., 2000; Baron-Cohen, 1995; Brothers, 1990).

Models of how these brain areas interact to process social information have suggested that the various brain areas subserve different functions (Adolphs, 2001; Haxby, Hoffman, & Gobbini, 2000). Subcortical brain regions subserve a rapid, automatic face detection system (Braeutigam, Bailey, & Switherby, 2001). Posterior regions of the brain, in the occipital and temporal cortices, are involved in processing faces or salient parts of the face, such as the eyes and mouth. In conjunction, more anterior regions, including the OFC and the ACC are thought to be involved in appraising the emotional significance of facial expression and guiding subsequent social behaviours (Baron-

Cohen et al., 1994; Damasio, 1994). Further, some authors have proposed that other anterior regions, the anterior insula and ACC, are involved in the recognition of error and the initiation of adaptive responses to error and negative feedback (Dehaene, Posner, & Tucker, 1994; Lamm & Singer, 2010). Evidence for this comes from studies showing that the anterior insula and ACC are activated by situations that involve social error, a mistake in the social exchange in which the individual is participating, or a change in state of one of the participants. For example, the anterior insular cortex has also been activated by negative feedback in the form of frowning faces in decision-making tasks involving a high degree of uncertainty (Baron-Cohen et al., 1999; Ullsperger & von Cramon, 2004). These structures are also activated by deception (Spence, Farrow, Herford, Wilkinson, Zheng, & Woodruff, 2001), social embarrassment (Berthoz, Armony, Blair, & Dolan, 2002), disgust (Jabbi & Keysers, 2008; Sanfey et al., 2003), and guilt (Shin et al., 1999), all of which could be interpreted as social emotions and social error signals. The anterior insula (including both superior and inferior components) was activated when partners in a dilemma game failed to reciprocate co-operative moves made by the subject, which is another type of social error signal (Rilling, King-Casas, & Sanfey, 2008). These brain regions are also activated by feelings of empathy for the suffering of others (Singer, Seymour, O'Doherty, Kaube, Dolan, & Frith, 2004). The anterior insula and ACC are also activated by pro-social signals, such as love and trust (Bartels & Zeki 2004; Singer et al. 2004), which suggests that these structures register both negative and positive aspects of feedback in social interactions. These results are very consistent with those found in Study 1, with regions of the frontal lobe being uniquely recruited by sad faces.

Various neuroimaging studies using emotionally expressive and neutral faces have found activation in regions of the social brain in TD individuals and hypoactivation in these same brain regions in individuals with autism (Ashwin, Baron-Cohen, Wheelwright, O'Riordan, & Bullmore, 2007; Baron-Cohen et al., 1999; Dawson, Webb, Carver, Panagiotides, & McPartland, 2004; Hall, Szechtman, & Nahmias, 2003; Ogai et al., 2003; Pelphrey, Morris, McCarthy, & LaBar, 2007). Additionally, several studies have found that a broader neural network is activated in individuals with autism to support social processing. This network includes the temporal pole, the right thalamus, and the right cuneus (Hall et al., 2003). These results indicate that individuals with autism are not

only recruiting alternative brain regions to support emotional face processing, they are also recruiting additional regions of the brain, including areas of the brain typically associated with attention, sensory gating, perceptual knowledge, and categorization, perhaps suggesting that emotion processing is more effortful in autism (Hall et al., 2003).

Functional Connectivity in Autism

The idea of reduced neural connectivity is also popular in autism research and has garnered support from neuroimaging studies. It has long been noted that individuals with autism often focus on details more so than the whole (see above section on central coherence). This processing style has been linked to macroencephaly in autism and has been hypothesized as a marker of poor neural connectivity (White, O'Reilly, & Uta, 2009). Neuroimaging has found support for weaker connectivity between various brain areas in autism (Just et al., 2007; Kana et al., 2006; Kleinhans et al., 2008; Koshino et al., 2008; Mostofksy et al., 2009; Mostofksy & Ewan, 2011; Villalobos et al., 2005; Wicker et al., 2008), generally long-range connections, often involving connections to regions of the frontal/pre-frontal cortex. Specifically, research has reported an increase in white matter volume present in the outer regions of the brain and a decrease in more medial white matter, suggesting that individuals with autism have a greater number of short- to medium range intra-hemispheric connections and fewer long range inter-hemispheric connections (Herbert et al., 2003, 2004).

Kleinhans et al (2008) examined functional connectivity between the fusiform gyrus and other regions of the limbic system in high-functioning adults with autism and age- and IQ-matched TD adults. This group employed a 1-back design, presenting faces and houses in a blocked design to which participants had to press a button whenever two identical stimuli were presented in succession. By individually identifying the right fusiform face area for each participant and using this as a seed point for functional connectivity (Kleinhans et al., 2008), this group demonstrated significant face-specific connectivity between the FFA, bilateral amygdalae, the right cuneus, posterior cingulate cortex (PCC), superior colliculus, thalamus, and the right middle temporal gyrus, in the control participants. Less functional connectivity was reported in participants with autism. Namely, Kleinhans et al (2008) reported connectivity between the right FFA, the left amygdala, and the right middle temporal gyrus. Comparing the autism to the TD groups,

Kleinhans et al. (2008) found significantly reduced connectivity between the right FFA and the bilateral PCC, left cuneus and left amygdala in the autism group. Furthermore, the social score from the ADI-R was significantly correlated to the strength of the connectivity between the right fusiform gyrus and the left amygdala, with autism individuals with lower social scores showing reduced connectivity between these two brain regions (Kleinhans et al., 2008). These results provide support for reduced connectivity between regions of the social brain in adults with autism.

Likewise, another group conducted a study specifically examining connectivity in the brain of high functioning adults with autism compared to controls (Wicker et al., 2008). Using explicitly presented dynamic emotional faces (happy and angry), in which the eyes on the stimulus face either looked from the side towards the participant or from side-to-side. This group identified regions of the brain showing activation to faces and used these sites as seeds from which to identify regions of connectivity. What this study reported was normal activity in response to emotional faces in more posterior regions of the brain, including in the fusiform, the STS, and even the amygdala in participants with autism, but a lack of connectivity between these posterior regions and frontal regions of the brain, including the dorsal-medial prefrontal cortex and the ventral-lateral prefrontal cortex. These findings suggest that emotional face processing differences in adults with autism may reflect a failure to interpret and associate emotional features of the face correctly with their social value (Wicker et al., 2008). These results are similar to an fMRI study that presented passively viewed, neutral faces and reported activation in posterior regions of the social brain, namely the fusiform gyrus, the STS, and occipital regions, and a lack of activity in the prefrontal cortices in participants with autism (Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007).

Electroencephalography Findings: Emotional Face Processing in Autism

Dawson et al. (2004) examined passively viewed fearful and neutral face processing in three and four year old children with autism and chronological-age and mental-age matched TD controls. Results showed differential ERPs between the two groups at multiple time points, recorded over different scalp locations. Namely, they found that fearful faces elicited a larger positivity at 200ms (P2) over central temporo-occipital sites compared to neutral faces in TD children, but not in children with autism. Similarly, a

greater negative deflection was recorded over fronto-central sites at 300ms post-stimulus onset (N300) to fearful vs. neutral faces in TD controls but not children with autism. Lastly, fearful faces elicited enhanced voltage of a negative slow wave recorded between 810ms and 1170ms over left and right temporal-parietal sites in TD children, but not children with autism (Dawson et al., 2004). Although source analyses were not conducted on these data and it is difficult to determine the location of neural generators of these components due to limited spatial resolution, these results suggest the presence of a posterior and anterior generator, consistent with social brain models, that responds preferentially to fearful faces compared to neutral faces in young TD children that is not present in young children with autism. In contrast, a second ERP study (O'Connor et al., 2005), that required participants to look at emotional and neutral faces and verbally identify the emotion (which was then recorded by an examiner who was sitting in the booth with the participant), did not find differences between the two groups with regard to how emotions modulated the P1 and N170; however, results were consistent with previous findings in neutral faces (Grice et al., 2001; McPartland et al., 2004) such that early event-related potentials were delayed and of smaller amplitude in autism adults compared to TD adults (O'Connor et al., 2005).

Another study compared the P1 and N170 in children with autism with two control groups, one based on verbal (mental) age and one based on chronological age, using an implicit emotional face processing task. Batty, Meaux, Wittemeyer, Roge, & Taylor, 2011 used a paradigm in which children were asked to press a button whenever they saw a non-face object. This group found that the P1 and N170 peaks were delayed and attenuated in the autism group when compared to their chronologically age matched peers but not when compared to verbal age-matched TD controls (Batty et al., 2011), suggesting delays reported in studies using chronological age-matching may reflect differences in verbal or cognitive abilities of the groups.

Wong, Fung, Chua, and McAlonan (2008) used implicitly and explicitly presented emotional and neutral faces to examine emotional face processing in children with autism compared to TD children between the ages of six and 10 years. At the scalp level over posterior and anterior sites, this study found no differences between groups or between emotions, but source analyses (using Brain Electrical Source Analysis (BESA) software), revealed that activation of cortical regions responsible for face and emotion

processing were significantly reduced and/or delayed in the autism group. More specifically, they found weaker and delayed response from bilateral fusiform dipoles during implicit emotion processing, and bilateral frontal dipoles between 120-310ms for implicit and explicit emotion processing. Additionally, in the autism group, greater activation from two dipoles localized to the occipital cortices were reported. These results suggest that individuals with autism employ a unique strategy to recognize emotional faces, relying more greatly on perceptual properties of the face to decipher the emotion rather than processing the emotion automatically (Wong et al., 2008).

Summary

Neuroimaging and electrophysiological research has been essential in conceptualizing and elucidating emotional face processing in autism. In the current review, to simplify the inconsistency in the literature, results from various imaging studies were categorized into one of four prominent theories—the amygdala theory of autism, the social brain hypothesis, the mirror neuron hypothesis of autism, and reduced neural connectivity in autism. However, it appears as though these theories are not mutually exclusive and rather overlap and complement each other, converging to highlight regions of the brain necessary to process and make sense of complex socio-emotional information (e.g., such as the processing of emotionally expressive faces). For example, the amygdala has been implicated in both the MNS and in the social brain, suggesting that this brain region is an essential component to processing emotional faces, but is likely working in conjunction with other brain areas to process complex socio-emotional information.

Furthermore, the insula and the prefrontal cortex have also been implicated in the 'social brain' and in Carr et al's (2003) and Dapretto et al's (2006) socio-emotional MNS. The MNS and the 'social brain' hypotheses of autism and the research to date converge to implicate the amygdala and areas of the prefrontal cortices acting in concert with regions important for perceptual processing, such as the fusiform gyrus, the STS, and regions of the occipital cortex, as being essential to processing emotional faces. Given the overlap in the theories and convergence in the literature, it is likely that the theories are addressing the same underlying system, but are highlighting slightly different brain regions and applying different conceptual frameworks to explain their role.

In typically developing individuals, brain areas believed to be important for socio-emotional functioning presumably work together rapidly to decipher and derive relevant information from complex social stimuli, such as emotional faces. However, in atypically developing populations, such as autism, parts of the circuit work differently, be it at the level of recruiting different brain regions or at the level of white matter tracts connecting various brain regions, resulting in behaviourally observed differences in processing socio-emotional information.

Although there is a great deal of variability in the literature to date, the conclusions from a handful of recent studies point to differences in how regions of the frontal/prefrontal lobes process emotions in individuals with autism, in the presence of typically functioning posterior brain regions important for perceptual face processing (Dapretto et al., 2006; Hadjikhani et al., 2006; O'Connor et al., 2005; Wicker et al., 2008). This is an interesting result as anterior brain regions have typically been associated with interpreting the emotional significance of facial expressions and in using this information to inform subsequent social behaviour (e.g. Allison et al., 2000; Baron-Cohen et al., 1994; Carr et al., 2003; Damasio, 1994). Therefore, the findings from this group of studies suggest that differences in social and emotional communication in autism may arise at the level of higher-order social cognition. Reduced recruitment of the frontal and prefrontal cortices would likely, at least in part, be consistent with the ToM hypotheses of autism (Baron-Cohen et al., 2000).

However, the finding of reduced involvement of the frontal lobes, or weak connectivity between frontal regions and posterior regions of this socio-emotional neural circuit, is thus far inconclusive, given discrepancies in research findings. Comparable activation in the fusiform gyrus and STS in individuals with autism and in TD participants is not consistent with earlier findings that reported hypoactivation in these areas (e.g. Pierce et al., 2001; Schultz et al., 2000), and there are many possible explanations for such inconsistencies including time spent looking at the eyes (Dalton et al., 2005), focus of visual attention (Hadjkhani et al., 2004, 2007), differences in task demands and instruction set, level of functioning of autism participants (Harmes et al., 2010; Loveland et al., 1997), matching procedures employed (Batty et al., 2011), and perhaps even individual variability amongst individuals with autism given the heterogeneous nature of autism spectrum disorders. More research employing well-controlled research designs

that includes homogenous autism samples and carefully matched controls are necessary to elucidate reasons for this variability in the literature.

In conclusion, the research to date suggests that individuals with autism may have difficulty in processing more complex, subtle and cognitively demanding social and emotional information from faces. There are many possible and likely overlapping explanations for apparent deficits in social communication. Behaviourally, it remains unclear whether or not individuals with autism have difficulties recognizing basic emotions, such as fear, happy, sad, and anger, from faces. Many studies have failed to find such basic deficits in high functioning individuals. Results from neuroimaging have also produced mixed results, but a subset of recent studies have reported differences in activation within and over frontal/prefrontal cortices in response to socio-emotional information. Such findings are consistent with a body of behavioural research suggesting that individuals with autism have difficulties interpreting and understanding the emotional information available from faces and using this information to respond to social situations accordingly. At present, there are more questions than there are answers and more research is required to address this complex and fundamental question in autism research. Over time, a uniform theory may emerge that will be able to inform future interventions to target and improve social and emotional skills in autism. Alternatively, variability may remain prominent in this field, perhaps leading one to speculate about the role of individual differences in this heterogeneous population.

There are several potential alternative explanations for differences in emotional face processing in autism that have not been adequately addressed in this introduction. One such alternate explanation is that of a fundamental deficit in central coherence, or a different processing style, as discussed briefly in the introduction (for a thorough review of processing style in autism see, Behrmann et al., 2006; Frith & Happe, 1994; Happe & Frith, 2006). Briefly, research supports the idea that individuals with autism rely more heavily on the localized details of the face or excel at processing parts of a face compared to TD controls who typically rely more on holistic features, or interpret the general meaning of a stimulus (e.g. Behrmann et al., 2006; Deruelle, Rondon, Gepner, & Tardiff, 2004; Deruelle et al., 2008; Frith & Happe, 1994; Golarai et al., 2006; Happe & Frith, 2006; Katsyri, Saalasti, von Wendt, & Sams, 2008; Sasson, 2006; Lahaie et al., 2006; Vermeulen, 2001).

In the past decade, the autism literature has also seen a renewed interest in sensory processing in autism (Ashwin, Ashwin, Rhydderch, Howells, & Baron-Cohen, 2009; Bertone, Mottron, Jelenic, & Faubert, 2005; Mottron & Burack, 2001; Scherf, Luna, Kimchi, Minshew, & Behrmann, 2008). Very briefly, these studies have provided evidence of atypical sensory processing in autism and have suggested that fundamental anomalies in visual processing could account for differences in face processing in individuals with autism. Consistent with the idea of unique basic visual processing in autism compared to their TD peers, differences in the grey and white matter in the occipital cortex in individuals with autism has been reported (Bonilha et al., 2008; Hyde, Samson, Evans, & Mottron, 2010). Furthermore, although relatively little attention has been paid to basic visual ERPs in autism, a recent ERP study found that the P1 (typically believed to reflect processing in the occipital lobe) in children with autism was significantly smaller compared to their same-aged and also their verbal IQ-matched TD peers. This suggests unique early visual processing in children with autism compared to their peers (Batty et al., 2011; Grice et al., 2001; McPartland et al., 2004; O'Connor et al., 2005). Similarly, there is evidence from early visual ERPs (<100ms) that individuals with autism process mid-range spatial frequencies similarly to high-spatial frequencies, which would likely bias them towards focussing on the components of a face (Jemel, Mimeault, Saint-Amour, Hosein, & Mottron, 2010).

Such lower level differences in strategy may, in part, explain difficulties deriving the gestalt of socially relevant information from faces. For example, it is feasible to imagine that if individuals are relying on specific details of a face rather than the whole or 'gist', they may easily become overwhelmed in the details and fail to recognize the socio-emotional significance of the emotional face. Compounding this is the proposition that many subtle details of more complex facial expressions are conveyed primarily by the eyes (e.g. Baron-Cohen, Wheelwright, & Jolliffe, 1997), to which individuals with autism may attend less than TD controls (e.g. Dalton et al., 2005; Pelphrey et al., 2002).

Current Study

The goal of the current study was to examine the time course of emotional face processing in autism using explicitly and implicitly presented faces, using simple behavioural tasks that were employed in *The Electrophysiological Correlates of Implicit*

and Explicit Emotional Face Processing in Adults (henceforth referred to as Study 1) on which one would anticipate individuals with autism to perform as well as their age-, gender-, and IQ-matched TD peers.

Given evidence that groups of high functioning individuals with autism can recognize basic emotions as well as their typically developing peers, it was hypothesized that the autism group in this study would be able to successfully identify the colours and emotions in both the implicit and explicit conditions as quickly and as accurately as their TD peers. Participants in both groups were hypothesized to have faster reaction times to faces in the implicit condition compared to the explicit condition, as was found in Study 1. Individuals with autism were also expected to complete both conditions as accurately as the TD group. Specifically, it was hypothesized that the autism and TD groups would make the same number of errors across emotions in the implicit and explicit condition.

It was further hypothesized that, although the autism group would be able to successfully identify the emotions (explicit task) and colours (implicit task), they would recruit a different neural network to process the emotional faces. In particular, it was hypothesized that there would be within-group differences with regard to specific ERP components in the autism group compared to the TD group.

With regard to anterior ERP components, it was hypothesized that individuals in the autism group would not recruit neural networks within the frontal cortices to process emotions. Based on the findings in Study One (in TD adults) It was thus specifically hypothesized that sad faces would not elicit greater peak and mean positivities in the VPP, P150, EAP, and N4a in the autism group. By contrast, it was hypothesized that sad faces would elicit greater peak and mean positivities compared to neutral faces in the TD group, as was found in Study 1.

Furthermore, it was hypothesized that the autism group would show the same pattern between emotions over posterior sites as the TD group, based on evidence that basic emotional face processing may be intact or less compromised in high functioning autism whereas appraisal and understanding of the emotion is what would be impaired. Specifically, it was predicted that fearful faces would elicit greater peak amplitude than

neutral faces in the right N170, as was found in Study 1. Specific emotions were not expected to differentially modulate the P1.

With regard to between-group differences, it was hypothesized that the peaks of early ERP components over occipital-parietal (i.e. P1 and N170) would be delayed and attenuated in the autism group compared to the TD group, confirming existing evidence from previous research in children with autism.

Method

The Simon Fraser University Research Ethics Board approved this experiment. All participants gave their written informed consent before participating in the study and received monetary compensation for their participation.

Participants

Two groups of young adult males (17-21) participated in this study: 14 with high-functioning autism and 16 typically developing. Two participants in the autism group discontinued the experiment due to fatigue and distress in the EEG portion of the task. One participant with autism blinked too frequently and responded to too few stimuli resulting in too few trials to analyze (<30/condition). Thus, his results were not included in further analyses. Lastly, one participant's IQ did not fall within the average range and he was disqualified from being included in further analyses. Six of the TD participants were excluded for either having excessive ocular-motor movements during EEG recordings or for not being appropriate matches (e.g. IQ not falling within average range) or necessary matches to participants in the autism group. As such 10 participants per group were included in subsequent analyses.

The demographic information for both groups is presented in Table 2.1. Participants were matched one-to-one on gender, biological age, verbal intellectual ability (VIQ), nonverbal intellectual ability (PIQ) and abbreviated full scale intellectual ability (ABIQ). The abbreviated, two-subtest version of the Stanford-Binet Intelligence Scales-5th Edition (SB5) was used to estimate overall intellectual ability. The Abbreviated SB5 has been found to reliably estimate the full-scale IQ of high functioning individuals with autism (Coolican, Bryson, & Zwaigenbaum, 2007). It also has the benefit of having a shorter

administration time (approximately 20-30 minutes), making results less susceptible to attention and concentration effects.

All participants were male and performed within the average to above average range on verbal and nonverbal estimates of IQ (scaled scores between 8-13) with the ABIQ falling within the average range (standard scores between 90-110). There were no significant differences between the groups on age, gender, or IQ variables (p>.05). All participants with autism were right-handed and nine participants in the TD group were right-handed.

All participants were asked to complete a medical questionnaire to rule-out self-reported history of neurological disorders (e.g. epilepsy), serious medical conditions, regular drug use, and mood disorder. Self-report was confirmed with parents on interview. To further rule-out the presence of a mood disorder symptoms, given high rates of anxiety or depression in individuals with autism (Brereton, Tonge, & Einfeld, 2006) all participants were asked to complete the Behaviour Assessment System for Children-2nd Edition (BASC-2), a comprehensive self-report questionnaire asking about mood and behaviour. One participant in the autism group, who was included in the analyses, rated himself as having clinically significant levels of anxiety and depression, which his parents described as stable and long-standing on interview. Four participants with autism did not complete the BASC-2. To rule-out clinically significant levels of anxiety or depression, these participants and their parents were interviewed regarding symptoms of anxiety and depression. These four participants and their parents denied the presence of most mood symptoms.

Participants with autism were previously diagnosed either at Sunny Hill Health Centre or through the Westcoast Child Development Group, two reputable diagnostic centres in Vancouver, British Columbia. A diagnosis of autism was confirmed by completing the ADI-R with a parent. To rule-out autism in the TD control group, participants completed Baron-Cohen's Autism-Spectrum Quotient (AQ) and only participants with scores in the normal range were included (a total scores of less than 32; Baron-Cohen, Wheelwright, Skinner, & Clubley, 2001). Furthermore, all participants in the TD group and their parents, as appropriate, were asked about family history of autism or other PDD. None of the TD participants included in the comparison group reported a family history of autism or other PDD.

Materials

These were the same as those employed in Study 1.

Procedure

These were the same as those employed in Study 1.

EEG data acquisition.

The same procedures that were followed in Study 1 were employed in this study.

EEG data processing.

EEG data processing, artefact rejection, baseline correction, ERP averaging and grand-averaging, and SOI selection were completed using the same procedures as were used in Study 1.

With regard to sensor selection, the same procedure as described in Study One were employed, and the following sensors, according to standard EEG nomenclature, were selected (prime [‘] signifies a close but not exact match as position match with the international 10/20 system is not precise): SOIs for peak values included electrodes in right and left temporo-occipital region (P1: O2, O2’, PO6, PO10, over the right hemisphere, and PO9, PO9’, O9, O1 over the left; N170: PO8, PO10, P8 over the right and PO9, P7, P9, over the left;) along posterior and anterior midline electrode sites (VPP: FCz, Cz, Cz’ and P150: AFz, AFz’). Prolonged positivities were derived from a broadly distributed array of electrodes over frontal cortices (EAP: FF8, AF8, AF6, FPz, FPz’, AFz, AF7’, AF5’, F7), midline frontal electrodes (N4a: FPz, FPz’, AFz) and from posterior midline sites (LPP: Cz’, CPz, CPz’, Pz), by extracting the mean voltage between 170-270ms post-stimulus onset for the EAP and between 450-600ms for the anterior and LPP.

Statistical analyses.

With regard to behavioural results, accuracy data were analyzed using independent samples *t*-tests comparing each of the emotional expressions in the implicit and explicit conditions between the two groups. Reaction times were analysed using a 2 x 4

(Condition [Implicit-Explicit] x Emotion [Fearful-Sad-Happy-Neutral]) repeated-measures analysis of variance (ANOVA), with group membership as the between-subject variable. Specific contrasts of interest were analyzed using paired-samples *t*-tests.

To analyse amplitude and latency differences in ERP components of interest, repeated-measures ANOVAs were conducted, with group membership as the between-subjects variable. Specifically, the P1 and N170 were analysed using separate 2 x 2 x 4 (Condition x Topography [Right-Left] x Emotion) repeated-measures ANOVAs. Peak positivities of the VPP and P150 and mean positivities of the EAP, N4a, and LPP were analyzed using separate 2 x 4 (Condition x Emotion) repeated-measures ANOVAs. Group membership was the between-subjects variable for all repeated-measures ANOVAs. A Greenhouse-Geisser correction was employed to correct for sphericity in the data. Effect size estimates of repeated-measures ANOVA main effects and interactions were computed using partial eta squared.

Paired-samples *t*-tests were run to examine a priori hypothesized differences across emotions and groups in the implicit and explicit conditions, with an alpha level of .05. Post-hoc *t*-tests were employed in the case of significant main effects or interactions of interest with a Bonferroni correction to control for family-wise error. Estimates of effect size were computed using Cohen's *d* with pooled standard deviations. By this measurement, effect sizes of .3 are considered small, .5 are considered medium, and .8 and above are considered large (Cohen, 1992).

Results

Behavioural

Accuracy.

The autism group generally made more errors than the TD group, based on mean number of errors. Independent samples *t*-tests revealed that the autism group made significantly more errors than the TD group in response to implicitly presented sad faces (t_{18}=2.15, p=.05, Cohen's d=0.96) and happy faces (t_{18}=2.08, p=.05, Cohen's d=0.93). The autism group also made more errors in response to explicitly presented happy

(t_{18}=2.12, p=.05, Cohen's d=0.95) and neutral faces (t_{18}=2.59, p=.02, Cohen's d=1.16) than the TD group. See Figure 2.1 for a graph representing these data.

Reaction time.

T-tests between emotions in the autism group revealed that reaction times to explicitly presented happy faces were significantly faster than explicitly presented neutral faces (t_9=-4.27, p=.002, Cohen's d= -0.81). In the TD group, reaction times to explicitly presented happy faces were significantly faster than to explicitly presented neutral faces (t_9=-2.63, p=.03, Cohen's d=-0.96), and explicitly presented fearful faces were processed significantly slower than explicitly presented neutral faces (t_9=4.13, p=.003, Cohen's d=1.13).

Electrophysiological Data: Amplitude

P1 (95-135 ms).

The repeated-measures ANOVA revealed no significant within-group main effects. There was, however, a significant interaction between Condition x Emotion ($F_{3,54}$=6.68, p=.001, partial η^2=.27). There was no significant overall group difference between the autism and TD group.

To further examine the significant Condition x Emotion interaction, Bonferroni corrected post-hoc *t*-tests were run (p=.05/6=.008). Collapsed across Topography and Group, there were no significant contrasts evident.

N170 (140-180ms).

Repeated-measures ANOVA revealed no significant within-group or between-group main effects or interactions.

A priori paired-samples *t*-tests did not reveal any significant contrasts between emotions in either condition, in either group. See Figure 2.3.

VPP (140-180ms).

Repeated-measures ANOVA revealed no significant within-group or between-group main effects or interactions.

A priori paired samples *t*-tests revealed that, in the TD group, implicitly presented sad faces elicited significantly greater peak positivities than implicitly presented neutral faces (t_9=2.48, p=.01, Cohen's d=.40). See Figure 2.3 for a representative electrode showing this effect in the TD group. No contrasts reached significance in the autism group.

P150 (135-175ms).

Repeated-measures ANOVA revealed a significant main effect of Emotion ($F_{3,54}$=3.51, p=.03, partial η^2=.16). There were no other significant within-group or between-group main effects or interactions.

A priori paired samples *t*-tests revealed that, in the autism group, implicitly presented happy faces elicited a significantly greater peak positivity than neutral faces (t_9=2.93, p=.02, Cohen's d=.21). In the TD group, implicitly presented fearful and sad faces elicited a significantly greater peak positivity than neutral faces (t_9=2.29, p=.05, Cohen's d=.32 and t_9=2.66, p=.01, Cohen's d=.39, respectively). See Figure 2.3.

EAP (170-270ms).

There were no significant within- or between-group main effects. There was, however, a significant interaction between Condition x Group ($F_{1,18}$=38.86, p<.001, partial η^2=.68).

A priori paired samples *t*-tests revealed no differences between emotions in the autism group. In the TD group, explicitly presented sad faces elicited a significantly greater mean positivity than explicitly presented neutral faces (t_9=2.80, p=.02, Cohen's d=.34). See Figure 2.7 for a representative electrode showing this effect.

To further examine the significant Condition x Group interaction, Emotions were collapsed across Condition and independent samples *t*-tests comparing implicit and explicit presentations were conducted with a Bonferroni correction (p=.05/2=.025). There was a significant difference between the autism and TD group in the implicit condition

(t_{18}=3.46, p=.003, Cohen's d=1.55), with implicitly presented faces eliciting a significantly larger mean amplitude in the autism group compared to the TD group (M_{autism}= 4.19, SD_{autism}= 3.65; M_{TD}= -.69, SD_{TD}= 2.55). There was no significant difference between groups in the explicit condition (p>.025), although it is interesting to note that the autism group had a smaller mean amplitude than the TD group (M_{autism}= .32, SD_{autism}= 3.51; M_{TD}= 2.65, SD_{TD}= 4.09) which likely accounts for the significant Condition x Group interaction. See Figure 2.4 of a bar graph illustrating this interaction.

N4a (400-650ms).

There were no significant within-group main effects or interactions. There was, however, a significant between-group effect ($F_{1,18}$=5.50, p=.03, partial η^2=.24).

A priori paired-samples t-tests revealed that happy faces in the explicit condition elicited a significantly greater sustained negativity than neutral faces in the autism group (t_9=-3.46, p=.01, Cohen's d=-.50). See Figure 2.5. No contrasts were significant in the TD group.

To examine the between-group main effect, Bonferroni corrected independent paired-samples t-tests were run. Collapsed across Emotion and Condition (implicit-explicit), the contrast revealed a greater overall sustained positivity in the autism group (M_{autism}=4.13, SD_{autism}=4.05) compared to the TD group (M_{TD}=-.62, SD_{TD}=4.70; t_{18}2.36, p=.03, Cohen's d=1.08).

LPP (400-650ms).

There were no significant within or between-group main effects. There were, however, significant interactions between Emotion x Group ($F_{3,54}$=3.18, p=.04, partial η^2=.15) and between Condition x Emotion ($F_{3,54}$=4.78, p=.01, partial η^2=.21).

A priori paired-samples t-tests were run to contrast emotions between the two groups and to clarify the significant Emotion x Group and Condition x Emotion interactions. In the autism group, there were no differences between emotions in the implicit or explicit condition. In the TD group, implicitly presented sad faces elicited a significantly more positive mean amplitude than neutral faces (t_9=3.06, p=.01, Cohen's d=.54). See Figure 2.5.

Electrophysiological Data: Latency

P1 (95-135 ms).

Repeated-measures ANOVA showed a main effect of Condition ($F_{1,18}$=8.850, p=.008, partial η^2=.330). No other within- or between-group main effects or interactions were significant.

A priori independent-samples *t*-test was run to see if, overall, the P1 peaked later in the autism group compared to the TD group. These contrasts were not significant.

N170 (140-180ms).

There was a significant main effect of Condition ($F_{1,18}$=6.65, p=.02, partial η^2=.27). There were no other significant main effects or interactions.

An a priori *t*-test was run to see if, overall, the N170 peaked later in the autism group compared to the TD group. This contrast was not significant.

VPP (140-180ms).

Repeated-measures ANOVA revealed a main effect of Condition ($F_{1,18}$=19.27, p<.001, partial η^2=.52) and of Emotion ($F_{1,18}$=5.14, p=.01, partial η^2=.22). There were no other within- or between-group main effects or interactions.

Post-hoc, Bonferroni corrected paired-samples *t*-tests (p=.05/4=.01) revealed that, when collapsed across Emotion and Group, implicit emotions peaked significantly later than explicit emotions (t_{19}=4.39, p<.001, Cohen's d=.41). Collapsed across Condition and Group, sad faces peaked significantly later than neutral faces (t_{19}=2.98, p=.01, Cohen's d=.16).

P150 (135-175ms).

There were significant main effects of Condition ($F_{1,18}$=10.79, p=.004, partial η^2=.38) and of Emotion ($F_{1,18}$=6.50, p=.003, partial η^2=.27). There were no other within- or between-group main effects or interactions.

Post-hoc paired-samples *t*-tests (.05/2=.025) were conducted to compare Condition (collapsed across Emotion and Group) and Emotion (collapsed across Condition and Group) to clarify the main effects. With regard to significant contrasts, implicitly presented faces peaked significantly later than explicitly presented faces (t_{19}=3.04, *p*=.01, Cohen's *d* =.30) and sad faces peaked significantly later than neutral faces (t_{19}=3.64, *p*=.002, Cohen's *d* =.16).

Discussion

Behavioural Results

The goal of the current study was to examine the time course of emotional face processing in autism compared to well-matched TD peers using explicitly and implicitly presented emotional faces. Behaviourally, on a time-sensitive task, individuals with autism generally made more errors on a very basic emotion recognition task (explicit) and on a colour identification task (implicit) compared to their typically developing peers, reaching significance for sad (implicit condition) and happy faces (implicit and explicit condition). This was not predicted, but it is consistent with various studies that have reported that individuals with autism have difficulties in recognizing at least some emotional expressions (Baron-Cohen et al., 1993; Howard et al., 2000; Humphreys et al., 2007; O'Connor et al., 2005; Pelphrey et al., 2002). Although these data were not available from this study, it is possible that individuals with autism would have been able to correctly identify the emotion/colour with more time. However, results from this study indicate that in the allotted time (200ms presentation time, 1700-2200ms inter-stimulus interval for response), individuals with autism were less accurate than their same aged peers at responding quickly to the stimuli. It is interesting to note that the emotional expressions to which the autism group responded less accurately were social emotions (happy and sad faces) as opposed to fearful facial expressions, which are more biologically relevant.

Another interesting result from the reaction time data was that the TD group was significantly faster to respond to facial expression when they were not the focus of attention (implicit condition). This was expected as the implicit task was, objectively and by participant report, an easier task—there was less information to process to reach a

correct response. By contrast, individuals in the autism group were not faster to respond to implicitly presented faces than explicitly presented faces. This suggests that either identifying colours was not easier for individuals with autism (which seems unlikely), that the response times in this group had reached a ceiling effect, that individuals with autism focused more on all of the details available in the picture rather than focussing only on the necessary information to complete the task, or that the autism group had more difficulty ignoring the background facial information and focussing only on the coloured square on the nose

This last option suggests that, although faces may not automatically capture the interest of individuals with autism, they are perhaps less easily ignored when individuals with autism were instructed to look at them, even when told to focus on the colour of a small square superimposed on the nose rather than to the face itself. This interpretation infers an interference effect from faces that slowed the reaction times in the autism group in the implicit condition. Interestingly, individuals with anxiety have also been found to show a similar interference effect to threatening facial expressions, such as anger (Ladouceur, Silk, Dahl, Ostapenko, Kronhaus, & Phillips, 2009; Williams, Mathews, & MacLeod, 1996), rather than to faces in general. This may, thus, suggest that individuals with autism generally find faces more threatening and harder to ignore than their TD peers. Further support for the idea of an interference effect comes from the finding that response times were fastest to happy faces in both groups, suggesting relative facility with this non-threatening facial expression. In the TD group, fearful faces were processed more slowly than other expressions, perhaps indicating that fearful faces were more threatening to TD participants; however, there were no significant differences in reaction time to fearful, sad, and neutral faces in the autism group. This conceivably implies that these three expressions are perceived as equally threatening to individuals with autism.

Electrophysiological Results

A primary goal of this study was to examine how implicit and explicit presentations of emotions differently modulated ERP components in individuals with autism compared to well-matched TD peers. Based on the results from Study 1, how sad faces modulated ERPs over the frontal cortex was of particular interest. However, it was first important to

determine if there were overall group differences in voltage and latency that would make it more difficult to interpret within-group differences (Luck, 2005).

In this study, there were no overall group differences between the high-functioning autism group and their age-, gender- and IQ-matched typically developing controls, except during the extended positivity recorded over anterior electrode sites, the N4a. Specifically, early posterior ERP components were not delayed or attenuated in the autism group compared to the TD group, as has previously been found in the literature. It is proposed that this reflects developmental changes that occur in the brain. In TD children, the N170 is delayed and attenuated compared to the N170 recorded in adults (Mondloch, Le Grand, & Maurer, 2002). Similarly, in children with autism, the N170 has been found to be delayed compared to age-matched TD controls (Batty et al., 2011; Grice et al., 2001; McPartland et al., 2004; although not when compared to children of matched verbal ability, Batty et al., 2011). In this study, the autism group was composed of high-functioning young adults with verbal and nonverbal IQs falling within the average to above average range, who were relatively free of symptoms of psychopathology (i.e. symptoms of depression and anxiety, with one exception, and externalizing disorders; as assessed by a self-report questionnaire and by parent report on interview). Although developmental trajectory was not directly examined in this study, this finding may suggest that there is a developmental delay in early ERP components recorded over the occipito-temporal cortices in autism that “catch-up” with typically developing peers by late adolescence/early adulthood in high functioning individuals. Future studies could examine the developmental trajectory of the P1 and N170 in autism compared to well-matched TD peers directly.

Sad Faces

Although there were generally no between-group differences, there were notable within-group differences that help elucidate emotional face processing in individuals with autism. Generally replicating results from Study 1, explicitly or implicitly presented sad faces elicited a unique response over frontal cortices in the TD group, depending on the timeframe in which the component analysed. Specifically, implicit presentations of sad faces elicited greater positivities in the P150, VPP, and LPP, while explicit presentations

of sad faces elicited a greater mean positivity in the EAP, replicating results from Study 1.

At the neural level, typically developing young adults showed a unique response to sad faces compared to neutral faces at approximately150ms (P150), 170ms (VPP), between 170-270ms (EAP) and, again, between 400-650ms (LPP). Over anterior sites, starting as early as 150ms post-stimulus onset, typically developing young adults showed an enhanced positivity to sad faces compared to neutral, happy, and fearful faces. This unique response to sadness is consistent with results from Study 1 and indicates a very rapid detection and analysis of sad faces that is different from that of other expressions and neutral faces. This rapid analysis is followed by modulation in the LPP, recorded more posteriorly, which is believed to reflect understanding and appreciation of the emotional expression. These results support previous fMRI research (Sakaki et al., 2012), replicate results from Study 1, and provide further support for the idea that sad faces elicit activation in brain areas within the frontal cortices that are uniquely tuned to social emotions. It is further argued that these brain regions, presumably within the frontal cortex, are sensitive to faces communicating social error or eliciting empathy.

As more thoroughly discussed in Study 1, a sad face communicates the internal emotional state of the individual, rather than a reaction to an event. It could be argued that fearful faces reflect an automatic response, a reaction to a stimulus (usually external), and may elicit an automatic startle response in the observer. Sad faces, like all facial expressions, first require the individual to identify and recognize the expression. However, unlike fearful faces, sad faces do not communicate potential threat to the observer and thus do not trigger a biologically adaptive automatic startle response. Like happy faces, sad faces communicate the internal state of the individual, requiring the observer to recognize the emotional experience of the person. However, unlike happy faces, sad faces may elicit a feeling of empathy in the observer who can recognize and appreciate the feeling of sadness in another human being. Alternatively and additionally, sad faces may communicate an error in a social interaction and implore the observe to make efforts to correct this social mistake.

The frontal lobes are hypothesized to form part of a complex circuit that ins involved in recognizing and internalizing emotionally relevant information (Allison et al., 2001). This

circuit may be involved in empathy, which has been hypothesized to be less well-developed in autism. Similarly, specific regions of the frontal cortex (namely the anterior insula and ACC) have been implicated in recognizing feedback in social situations (Baron-Cohen et al., 1999; Ullsperger & von Cramon, 2004). A sad face could easily be interpreted as communicating a social error signal. Results from this study indicate that ERPs recorded over frontal cortex, likely reflecting underlying cortical activation within the frontal lobes, are different in autism compared to their TD peers. Specifically, sad faces did not elicit greater peak and mean positivities in response to sad faces compared to other emotional expressions the autism group, suggesting sad faces did not uniquely capture the attention of individuals in the autism group. This has important implications for our understanding of social interactions in autism, which will be further discussed below.

Although ERPs recorded over the frontal cortex were not uniquely sensitive to sad faces in autism, they were sensitive to happy faces, another social emotion. Unlike with typically developing young adults, this finding seems to suggest that there is not the same heightened response to negative emotions in autism as in the typically developing group. Results rather indicate that happy faces "stand out" more than fearful, sad, and neutral expressions to individuals with autism. This may reflect that happy faces were less threatening, are more consistent with the observer's expectations, are more recognizable in autism, or that underlying brain areas are more sensitive to positive emotional expressions and less sensitive to negative feedback in social interactions or to emotional expressions requiring empathy.

Fearful Faces

In the TD group, fearful faces did not modulate the N170 as they did in Study 1. Differences between this study and the first study may reflect differences in the samples. Whether or not the N170 is modulated by emotions remains debated in the literature. A notable difference between Study 1 and the current study is the gender distribution of participants. In Study 1, half of the participants were female whereas in the current study, all of the participants were male. There is some evidence that faces differentially modulate mid- (~200ms) and late ERP (>300ms) effects in males and females (Jaworska, Blier, Fusee, & Knott, 2010; Wang, Kitayama, & Han, 2011). Similarly, some

studies have found sex differences in the lateralization of the N170 (Proverbio, Brigone, Matarazzo, Del Zotto, & Zani, 2006) and amplitude of the N170 as modulated by gender vs. orientation discrimination tasks (Sun, Gao, & Han, 2010). Taken together, the results from the current study and Study 1 may suggest that the N170 is more involved in emotion processing in young women than in young men. Alternatively, failure to find an effect may be secondary to having a smaller sample size and not enough power to detect an effect in this study. However, this seems less likely as there was enough power to detect small effect sizes over frontal cortices as significant. Moreover, the comparisons of fearful versus neutral face in the N170 was not approaching significance.

However, fearful faces elicited a greater peak positivity than neutral and happy faces in the P150. This unique response indicates that early ERPs recorded over the frontal cortices may be sensitive to threat in addition to sadness, in typically developing young adult males.

Timing and Overall Amplitude of ERP Components

This study did not find overall attenuation or delays in peak responses to faces in the autism group compared to the TD group. Results from this study rather found within-group differences between how emotions were processed.

Overall, when collapsed across emotion, condition and group, explicitly presented emotional faces peaked earlier than implicitly presented faces, except in the P1 for which the reverse was true. This may suggest a relative facility in processing implicit emotions compared to explicit emotions very early on in the epoch, consistent with reaction time data from the TD group. However, explicit emotions elicited earlier peak responses in the N170, VPP, and P150. This was unexpected and specific hypotheses about differences in the time course between implicit and explicit emotions were not made. It would therefore be of interest for future research to examine differences in the time course of implicit and explicit emotions in autism compared to their TD peers. It could be hypothesized that, as was found for reaction times, individuals with autism would not show a difference between the timing of ERP peaks to emotions in the implicit and explicit conditions whereas their TD peers would demonstrate the above pattern.

Interestingly, overall, implicit emotions elicited a significantly larger EAP than explicit emotions in the autism group, the reverse of what was found in the TD group. This may reflect difficulties down-regulating facial stimuli at this mid-stage of processing. This, again, suggests the frontal cortices are processing emotion differently in the autism and TD group, and specifically that processing implicit emotions is more effortful in the autism group. This is consistent with reaction time data in which implicit emotional expressions were not processed more rapidly than explicit emotions in the autism group, again suggesting that processing implicit emotions is more effortful for individuals with autism. Likewise, the N4a was more positive in the autism group compared to the TD group, perhaps suggesting more effort was exerted by the brain when looking at facial expressions, and a relative difficulty downgrading overall activation at this later stage of processing. Further investigation into this idea is warranted.

Conclusion

To summarize, behavioural results suggest that individuals with autism were generally less accurate at responding to emotional expressions, regardless of whether or not the facial expression was the directed focus of attention. In combination with reaction time data showing that the TD group was faster to respond to implicitly presented faces than explicitly presented faces whereas the autism group was not, these findings suggest a relative interference effect for individuals with autism when told to look a face (even if the face is not the intended focus of attention). Providing further support for this proposal, the TD group was faster to respond to happy faces and slower to respond to fearful faces whereas the autism group was faster to respond to happy faces but had statistically equivalent reaction times to all other expressions, perhaps suggesting that fearful, sad, and neutral faces were all interpreted as equally threatening.

ERP results from this study showed a difference in how sad faces were processed over frontal cortices in individuals with autism compared to their TD peers. In the TD group, results from this study largely replicated those from Study 1, with sad faces eliciting greater peak and mean positivities in anterior ERP components and in the LPP. However, this unique response to sad faces was not found in the autism group. Rather, happy faces uniquely modulated the P150 and N4a in the autism group, an effect that was not present in the TD group. These point to differences in how socially relevant

emotions are processed in the frontal cortex in the two groups, which likely translates to behavioural differences in daily life.

This result has interesting implications for our understanding of emotional face processing in autism. Despite being well-matched on intellectual functioning and psychopathology to their TD peers, sad faces, a social error signal often eliciting empathy, did not modulate ERPs over the frontal cortex in individuals with autism. This may be predictive of difficulties with empathy often reported in autism or may rather reflect an overarching, fundamental deficit in affective processing, as proposed by Hobson's Affective Theory of Autism (1986). Although it is not clear from this study which structures in the brain are being recruited to process sad faces, a question fMRI or PET technology would be better equipped to answer, this suggests generators (likely within the frontal or prefrontal cortex) are not as finely tuned to negative social emotions in autism. The frontal cortex has been linked to understanding such social error signals, connecting the facial expression to internal representation of this emotion, and appreciating the significance of a facial expression. This system appears to operate differently in autism. This finding likely, at least in part, explains some of the social difficulties so commonly reported in autism. It would be interesting to examine how early interventions targeting social skills development affect the outcome of these ERP patterns, as early interventions were not asked about or examined in this study.

Limitations

Notable limitations to the current study include the restricted and small sample size. Only 10 participants with autism completed the EEG portion of the task with an adequate number of correct responses to be included in further analyses (accordingly, only 10 TD participants were included in the control group). This is obviously a smaller sample size that was originally intended and desired. It is difficult in well-controlled studies with low base rate disorders to recruit enough participants to have sufficient power to detect subtle group differences and effects, potentially resulting in Type II errors. This problem of sample selection and low power likely accounts for some of the variability in the autism literature to date, and may account for some of the null results in this study. It is possible with more participants and greater power, more emotions would have differentially modulate ERP effects in the autism group.

Some research also suggests that emotional face processing develops over time, making it easier to miss the optimal age window to examine the effects of interest. For example, the participants in this study may have been too old to detect subtle differences between groups (e.g. at the level of the P1 and N170).

Results from this study also have limited generalizability. Individuals included in this study were screened based on specific inclusion and exclusion criteria (average range Abbreviated Full Scale IQ, Verbal and Performance IQ; right-hand dominance; males between 17-21 years of age). Such strict criteria helped to isolate differences between the two groups to a diagnosis of autism; however, results may not translate or apply to individuals with autism with lower or higher IQs, females with autism, or other age groups.

Another limitation to this study is that four participants in the autism group did not return their self-report questionnaires regarding mood and behaviour. Although this precludes objective comparison of symptoms of depression, anxiety, and behavioural difficulties (e.g. attention) between the two groups, it is unlikely that findings from the current study could be attributed to differences in overall level of anxiety or depression in the autism and TD groups. All participants, including those who failed to complete their questionnaires, completed a medical screener that asked about symptoms of depression and anxiety, and participants and their parents were asked about their current symptoms of mood disorders, and all denied current difficulties with depression or anxiety in the participants.

Future Directions

Replication with a developmental focus to examine the trajectory of this pattern of results will be important in understanding how the brain in individuals with autism develops compared to their same aged-peers. This will help elucidate the developmental pattern of emotional face processing, which sheds light on the development of socio-emotional processing and the neural mechanisms that support these skills more generally.

Another question that arises from this study is how emotional face processing in autism compares to emotional face processing in individuals with anxiety. The results from this study suggest an interference effect with regard to faces even when they are not the

focus of attention, similar to findings that individuals with anxiety are slower to respond to threatening faces. It would be of interest to specifically compare and contrast the behavioural and neural responses to socio-emotional stimuli in these two groups. Likewise, given high rates of co-morbid depression and anxiety in high functioning adolescents/adults with autism, it would be of interest to examine how the brain processes these stimuli in individuals with co-morbid depression or anxiety to individuals relatively free of mood disturbance, as was the case for participants in this study.

Finally, a major focus of future research could be to examine the effect of early intervention and social skills training on the developmental trajectory of socio-emotional and face processing in autism. As researchers and clinicians move towards identifying and diagnosing individuals at-risk for autism at younger ages and interventions continue to target younger age groups (Zwaigenbaum et al., 2009), the importance of examining the long-term effect of this on social and emotional development increases. It will be of interest to examine the effect of early intervention on brain development and basic reaction times, but also, and perhaps more importantly, to examine the functional outcome of interventions. Specifically, studying the broader implications of these interventions and (presumed) improved social interactions on quality of life and overall success of individuals in society will be of great importance in coming years. It will also be interesting to monitor rates of psychopathology (e.g. depression and anxiety) in individuals with autism who have undergone early and rigorous intervention. One might hypothesize that improved social skills and a better ability to navigate the social world may result in decreased risk of developing a mood or anxiety disorder, a hypothesis for which some preliminary research has found support (Hillier, Fish, Siegel, & Beversdorf, 2011) and for which more research is certainly warranted.

Tables

Table 2.1.

One-to-one matching of participant pairs and overall group comparisons on the above variables included at bottom of table.

Participant Pairs	Age	Gender	VIQ	PIQ	ABIQ
1. Autism	17	Male	13	9	106
TD	17	Male	12	9	103
2. Autism	17	Male	10	9	97
TD	17	Male	11	9	100
3. Autism	17	Male	13	9	109
TD	17	Male	12	11	109
4. Autism	19	Male	13	9	106
TD	20	Male	12	9	103
5. Autism	18	Male	9	9	94
TD	18	Male	10	10	100
6. Autism	19	Male	8	9	91
TD	18	Male	10	8	94
7. Autism	18	Male	10	13	109
TD	17	Male	11	12	109
8. Autism	22	Male	9	12	103
TD	21	Male	10	11	103
9. Autism	19	Male	9	13	106
TD	19	Male	11	11	106
10. Autism	19	Male	8	10	94
TD	18	Male	10	10	100
M_{autism} (*SD*)	**18.2 (1.874)**	**100% Male**	**10.20 (2.04)**	**10.20 (1.75)**	**101.50 (6.82)**
M_{TD} (*SD*)	**18.2 (1.398)**	**100% Male**	**10.90 (0.90)**	**10.00 (1.25)**	**102.70 (4.57)**

Figures

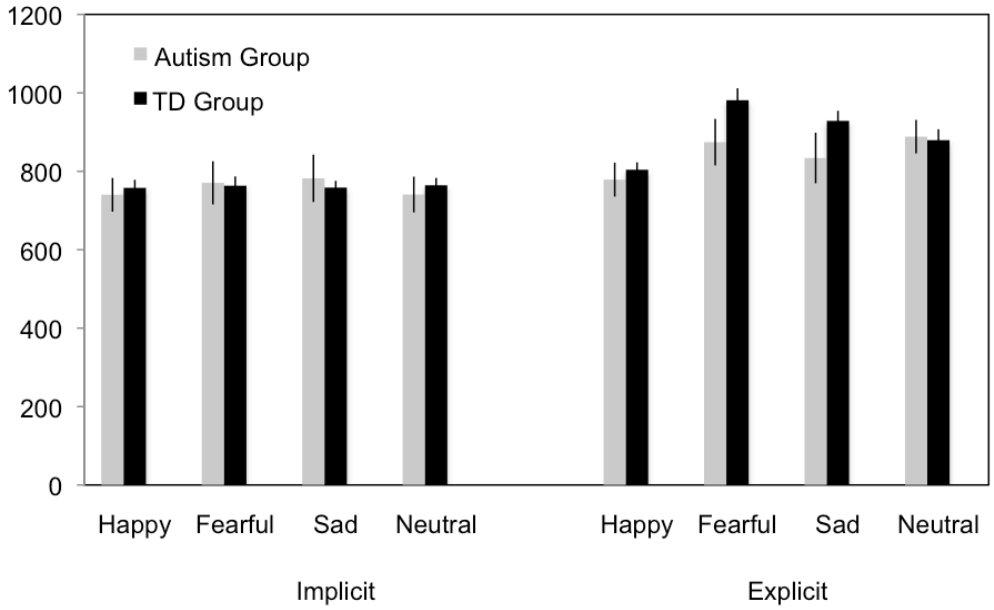


Figure 2.1. Mean (ms) reaction time (SEM) of both groups to emotions across both conditions.

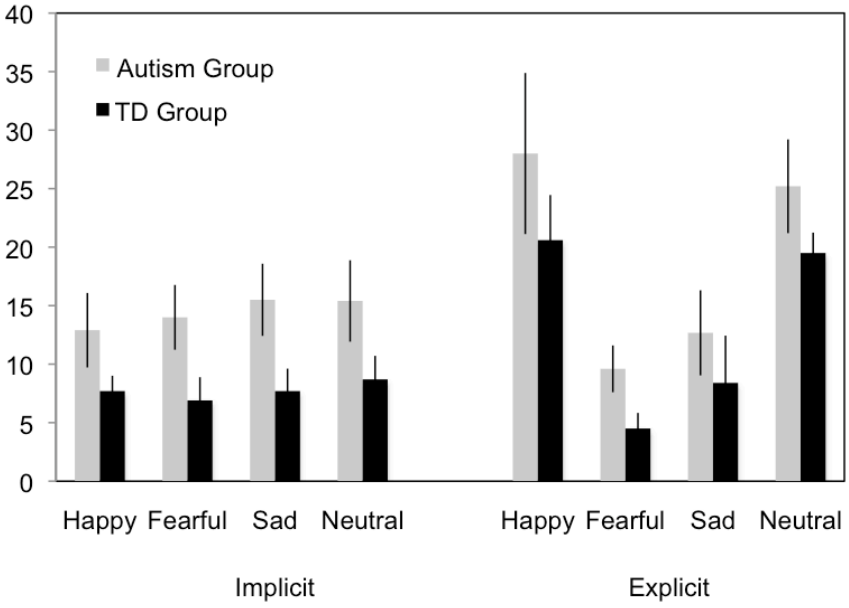


Figure 2.2. Mean number of errors (SEM) made by each group across emotions and conditions.

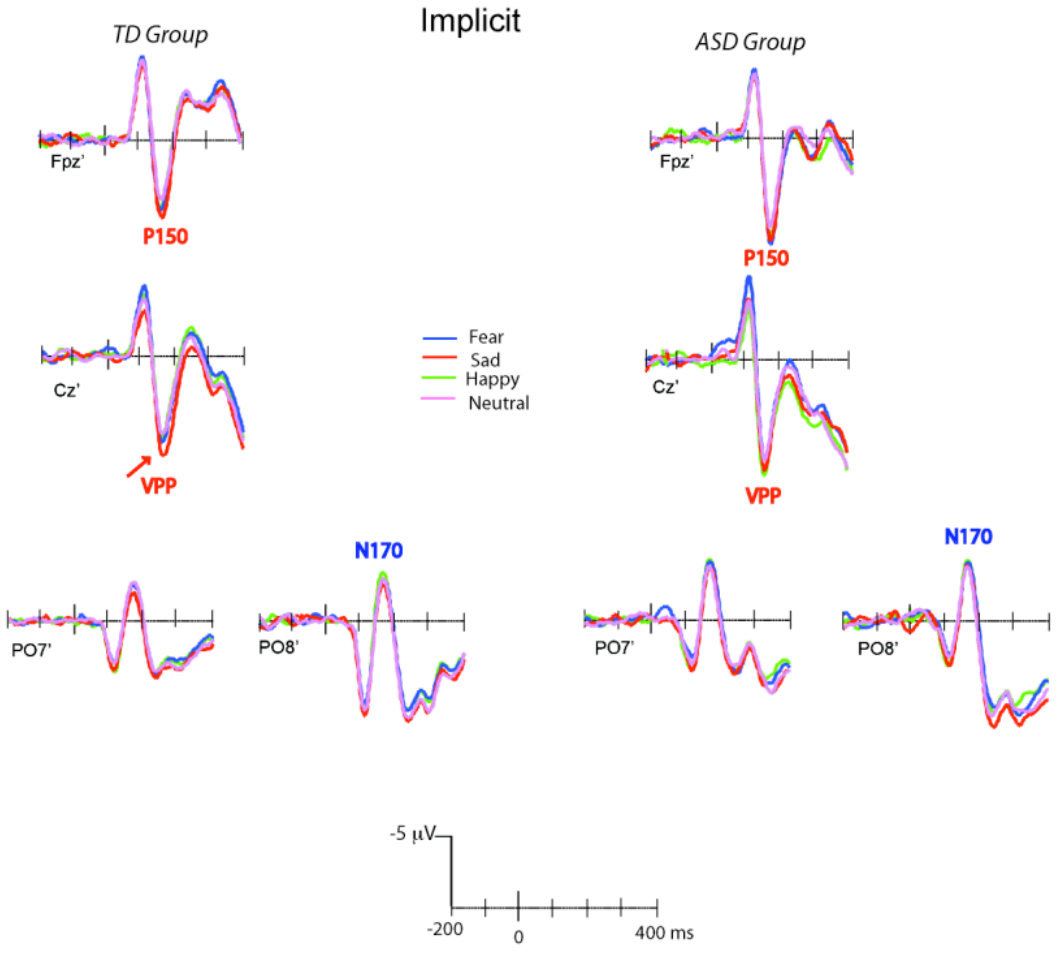


Figure 2.3. Representative electrodes showing the N170, VPP, and P150 in the TD and Autism group, as modulated by implicit presentations of Happy, Sad, Fearful, and Neutral faces. The x-axis shows time (ms) and the y-axis shows voltage (uV).

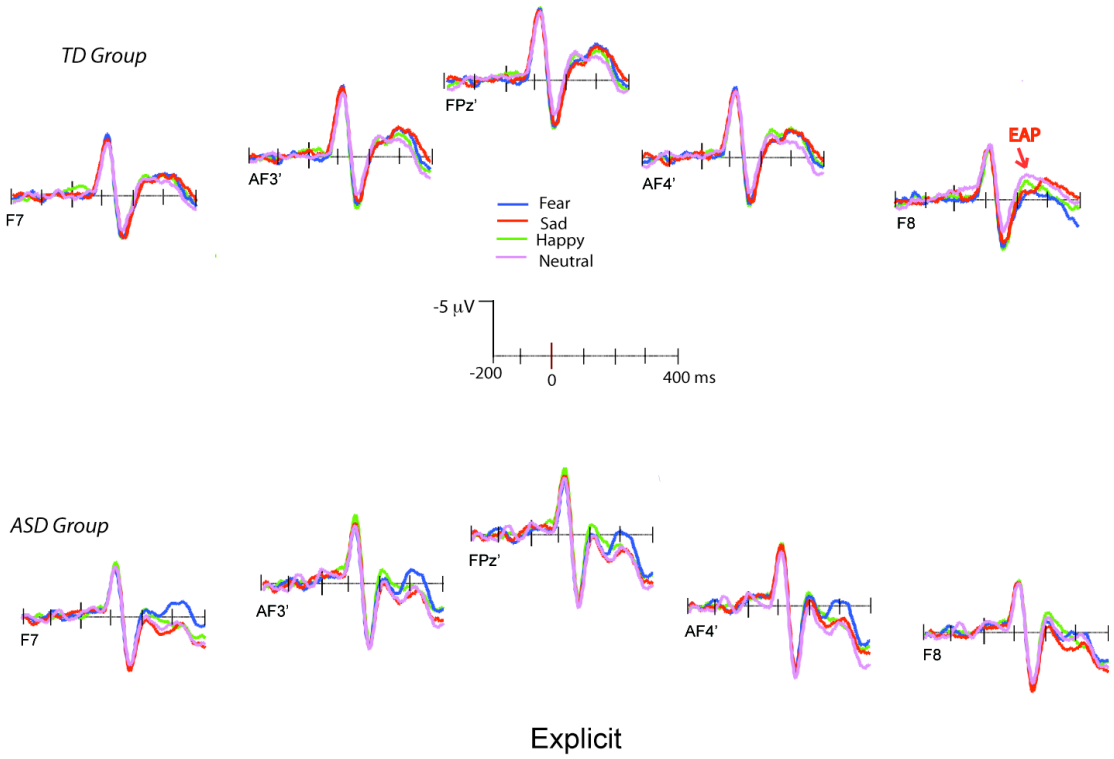


Figure 2.4. Representative electrodes showing the EAP as modulated by explicit presentations of Fearful, Sad, Happy, and Neutral faces. The x-axis shows time (ms) and the y-axis shows voltage (uV).

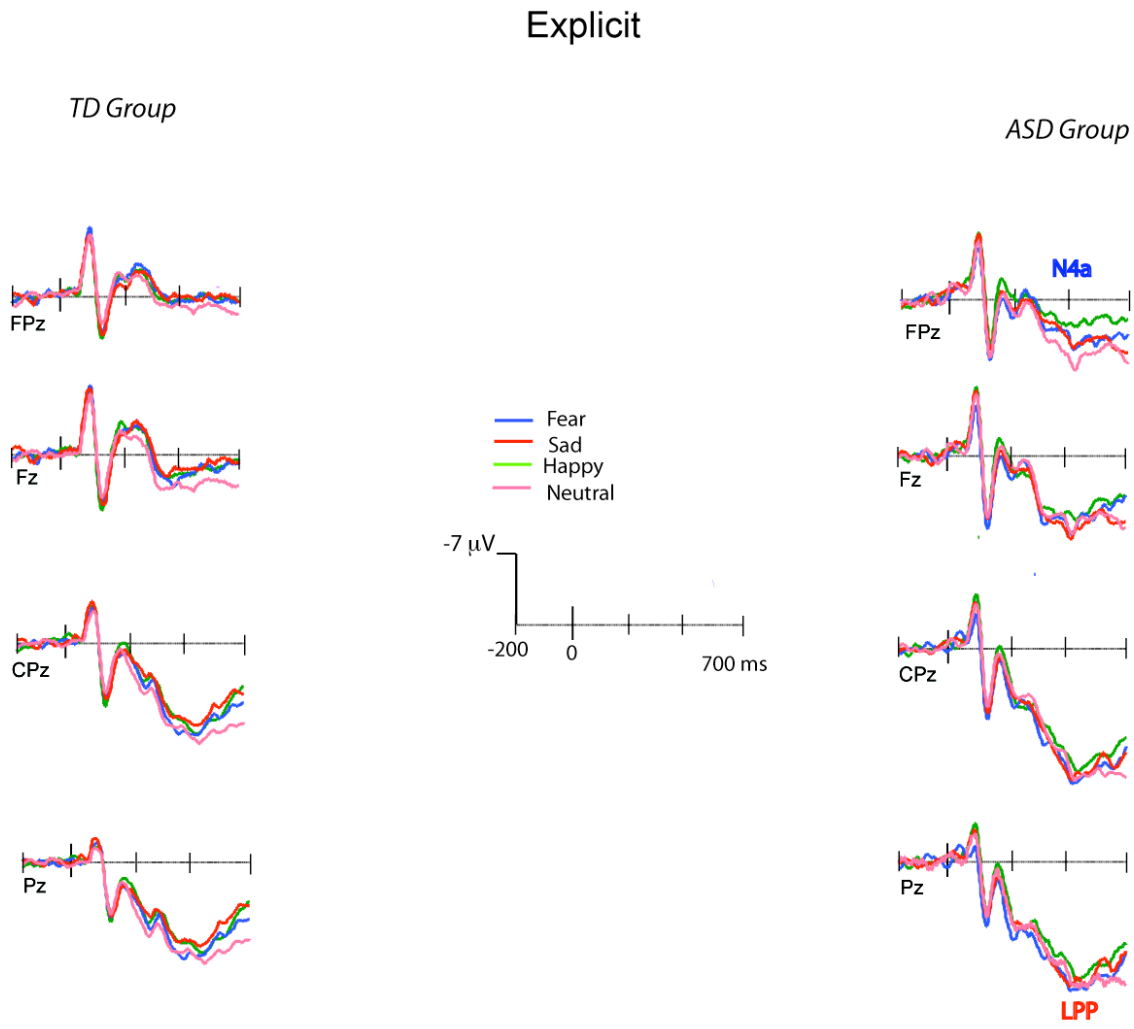


Figure 2.5. Representative electrodes showing the N4a and LPP as modulated by explicit presentations of Fearful, Sad, Happy, and Neutral faces. The x-axis shows time (ms) and the y-axis shows voltage (uV).

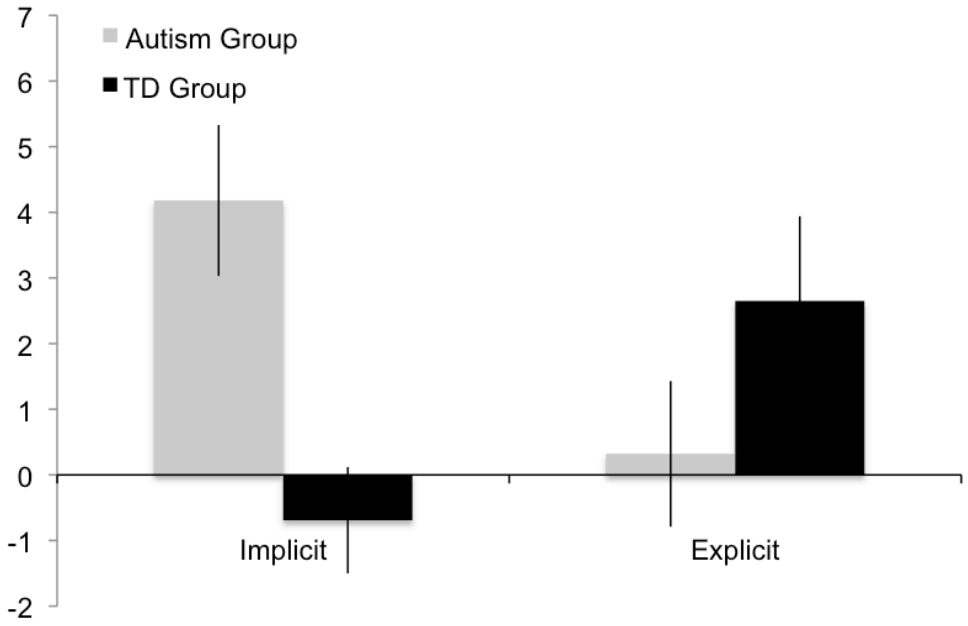


Figure 2.6. Mean amplitude (uV) of EAP (SEM), collapsed across emotion, in the Implicit and Explicit condition for both groups.

General Conclusion

The goals of this project were to, first, develop a paradigm that would successfully determine the effect of emotion type and emotion relevance on ERP components of interest in typically developing young adults. The second goal was then to determine how the time course and modulation of these ERP components by emotional faces differed in autism. Results from both studies revealed that different emotional facial expressions and emotion relevance differently modulated ERPs, starting as early as 150ms onset and extending to 600ms post-stimulus onset. This suggests that the brain rapidly processes different emotions uniquely. Furthermore, it provides further support for research showing that implicit and explicit presentations of emotional facial expressions are processed by partially distinct underlying neural mechanisms.

Perhaps the most interesting finding from this research was evidence that sad faces were distinctively processed in TD adults. ERP effects revealed unique responses at early, mid- and late-time points in the EEG, suggesting an initial rapid detection of the emotional expression from faces followed by more meaningful processing, recognition, and appreciation of the emotional significance of sad faces. These ERP effects may reflect an underlying neural network important in interpreting sadness in another person and understanding this negative and socially relevant emotional response in another human being. Specifically, sad faces may communicate social error and elicit empathy, skills which have long been proposed to be relatively weak in individuals with autism.

Consistent with such proposals, results from the autism group provided preliminary evidence that ERPs recorded over frontal cortices were differently sensitive to social emotions in autism. In particular, rather than sad faces uniquely modulating ERPs recorded over the frontal cortex, happy faces differently modulated early and late anterior ERP components in individuals with autism.

Although preliminary and requiring replication, this unique finding may reflect an underlying network that is less sensitive to negative feedback in social interactions or to feedback that typically elicits empathy in autism. Although not previously reported with ERPs, results from these studies are similar to fMRI research that has found less

activation within the frontal cortex and less connectivity between the frontal cortex and more posterior regions of the brain in individuals with autism (Dapretto et al., 2006; Hadjikhani et al., 2006; O'Connor et al., 2005; Sakaki et al., 2012; Wicker et al., 2008). Increased difficulty appreciating sadness, and perhaps other negative social emotions, and knowing how to respond appropriately would greatly hinder an individual's ability to engage in successful social interactions. This finding may thus have significant implications for our understanding of the development of socio-emotional functioning in autism. As such, further investigation into this system in autism is warranted, as are studies examining the effect of early intervention on underlying brain responses and behavioural outcome.

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