

Does parental bonding and its interaction with child temperament influence facial affect recognition in high-risk offspring for developing anxiety disorders?

By

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### Abstract

**Purpose:** This thesis investigated whether perceived parental care and overprotection predicted accuracy of face emotion recognition in psychiatrically healthy youth. The study also examined whether child gender and having a parent with a history of anxiety moderated the relationship between parental bonding and facial emotion recognition, and whether behavioural inhibition mediated this relationship. **Methods:** The sample comprised 176 males and females aged 7-18 years. Participants completed the Parental Bonding Instrument, Childhood Self-Report of Inhibition, and the Ekman emotion recognition task. **Results:** Child gender and parental history of anxiety moderated the relationship between perceived parenting style and affect recognition. In boys, overprotection by father predicted deficits in recognizing fearful faces; in children with parental anxiety, low paternal care predicted deficits in recognizing angry faces; and in boys with parental anxiety, negative maternal bonding predicted deficits in recognizing expressions of surprise. Also, maternal overprotection predicted intensity of subjective anxiety while viewing expressions of surprise and happiness for all offspring, and behaviour inhibition mediated these relationships. **Implications:** The present study provides preliminary evidence that parental bonding interacts with risk group and gender in predicting accuracy of facial affect recognition in healthy youth. Further research is needed to confirm these findings and determine whether the interaction between gender, risk group and deficits in social cognition increase risk for developing pathological anxiety.

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## **Does parental bonding and its interaction with child temperament influence facial affect recognition in high-risk offspring for developing anxiety disorders?**

Anxiety disorders (AD) are a cluster of mental illnesses that are chronic in nature and are among the most prevalent types of adult and childhood psychopathology (Bernstein, Borchardt, & Perwien, 1996; Esbjørn, Bender, Reinholdt-Dunne, Munck, & Ollendick, 2012; Lindhout, 2008; Turner, Beidel, Roberson-Nay, & Tervo, 2003). There are many subtypes of anxiety disorders but the common features shared by all are excessive fear and anxiety, physiological symptoms of arousal caused by activation of the sympathetic nervous system, and attentional biases toward threat-related information (DSM-5, APA, 2013). The 12 month prevalence for any anxiety disorder is estimated to be over 18.1% (Kessler, Chiu, Demler, & Walters, 2005) and one in four Canadians will be diagnosed with at least one anxiety disorder in their lifetime (Cairney & Steiner, 2010; Offord, Boyle, Campbell, Goering, Lin, Wong, & Racine, 1996).

Anxiety disorders tend to cluster together (Kessler et al., 2006), have high comorbidities with other types of psychopathology (Guyer et al., 2007) and aggregate in families (Merikangas, Lieb, Wittchen, & Avenevoli, 2003). While the exact causes of anxiety disorders are unknown, there is empirical evidence pointing to cognitive factors (e.g., attentional biases) (Gamble & Rapee, 2009; Stirling, Eley, & Clark, 2006), neurobiological and genetic underpinnings (Schumacher et al., 2011), temperamental factors (e.g., behavioural inhibition) (Clauss & Blackford, 2012), and environmental factors (e.g., negative parenting) (Rapee, Schniering, & Hudson, 2009).

This thesis examined the interaction of environmental, temperamental and cognitive factors that are hypothesized to predispose people for developing anxiety disorders. Specifically, I examined the relationships between parental bonding and facial affect recognition in psychiatrically healthy children and adolescents. I also examined whether child gender and a parental history of anxiety moderated the relationship between perceived parental bonding and facial affect recognition, and whether behavioural inhibition mediated this relationship.

### **Facial Affect Recognition and Anxiety Disorders**

Facial affect recognition denotes people's ability to correctly identify emotions in human faces, an ability that is said to provide essential information for social interactions and adaptive

behaviours (Bilodeau, Bradwejn, & Koszycki, 2014; Guyer et al., 2007). In addition to providing important communicatory functions in conveying social cues and signalling acceptable behaviours, accurate facial affect recognition is considered an adaptive skill which helps us to correctly understand the emotional states, motivations, and intentions of others (Fairchild, Van Goozen, Calder, Stollery, & Goodyer, 2009). Detecting and understanding emotions in others is essential for establishing relationships and facilitating social bonding (Bal, Harden, Lamb, Van Hecke, Denver, & Porges, 2010; Simonian, Beidel, Turner, Berkes, & Long, 2001). Based on such information, people can acquire social capital, in addition to developing approach or withdrawal strategies in interpersonal transactions (Izard, Fine, Schultz, Mostow, Ackerman, & Youngstrom, 2001).

Developmental studies have shown that the ability to effectively recognize facial affect begins prior to the first two years of a child's life (Nelson, 1987) and continues to mature throughout childhood (Herba & Phillips, 2004). Happiness and sadness are the first of the basic emotions that children become proficient at recognizing, and by 11 years of age, most children can recognize other complex emotions such as fear, anger, and disgust (Durand, Gally, Seigneuric, Robichon, & Baudouin, 2007). Research has found that the ability to correctly identify and understand emotions in early childhood is positively related to the development of adaptive social behaviour (see Izard et al., 2001 for review).

In child populations, correctly recognizing and labelling emotions has been shown to predict peer acceptance and social competence (Mostow, Izard, & Fine, 2002). Research suggests that early models of emotion-cognition connections help children decode and accurately interpret social cues (Simonian et al., 2001; Vicari, Reilly, Pasqualetti, Vizzotto, & Caltagirone, 2000; Williams et al., 2009). Emotion knowledge is especially important in the early stages of development because it facilitates adaptive social behaviour. For example, a child that correctly recognizes the expression of sadness in another and engages in soothing and supportive behaviours is concurrently expanding their "social capital", which is defined as the sum of the resources that an individual accumulates through establishing relationships with people (Bourdieu & Wacquant, 1992). Emotional knowledge and cognitive processes are involved in these complex social interactions and they influence not only the social environment of the child but also their perceived social competence (Manassis & Young, 2000; O'Toole, Hougaard, & Mennin, 2013; Young, Lennie, & Minnis, 2011).

Accuracy and speed in recognizing facial affect improves as children develop (Herba, Landau, Russell, Ecker, & Phillips, 2006), presumably due to maturation of brain structures associated with facial emotion processing and social experiential factors (Herba, et al., 2006; Vicari et al., 2000; Williams et al., 2009). The brain structures that are involved in the processing of emotional facial expressions include the amygdala, fusiform gyrus, the orbitofrontal cortex, the superior temporal gyrus and somatosensory-related cortices (Adolphs, 2002b; LeDoux, 1993; Morris et al., 1996; Todd & Anderson, 2009). These structures are said to be involved in two types of facial affect recognition; perceptual based processing (i.e., discriminating among different facial stimuli) and affect based processing. For example, when presented with a fearful face, these brain structures might work together by combining the perceptual properties of the face, in addition to decoding the lexical properties of “fear” and the emotional response associated with it based on past experience (Adolphs, 2002b). This suggests that facial affect recognition depends as much on maturation processes in the brain as it does on knowledge-based information and emotional responses from past experience.

### **Facial Affect Recognition in Clinical Populations**

There are considerable individual differences in the ability to accurately detect and recognize emotions in faces, and these differences are more pronounced when we look at individuals with different psychopathological disorders compared to normal controls (Anokhin, Golosheykin, & Heath, 2010; Corden, Critchley, Skuse, & Dolan, 2006; Lau et al., 2009). Deficits in facial affect recognition have been reported in individuals with schizophrenia, (for review see Kohler, Walker, Martin, Healey, & Moberg, 2010), antisocial personality (Gross, 2004; Kohler et al., 2010; Marsh & Blair, 2008), autism spectrum disorders (Wong, Beidel, Sarver, & Sims, 2012), eating disorders (Castro, Davies, Hale, Surguladze, & Tchanturia, 2010; Zonnevijlle-Bendek, Van Goozen, Cohen-Kettenis, Van Elburg, & Van Engeland, 2002), attention deficit hyperactivity disorder (Cadesky, Mota, & Schachar, 2000; Marsh & Williams, 2006), depression (Bourke, Douglas, & Porter, 2010; Mandal & Bhattacharya, 1985; Mikhailova, Vladimirova, Iznak, Tsusulkovskaya, & Sushko, 1996) and bipolar disorder (Brotman et al., 2008; Kucharska-Pietura & David, 2003; Lembke & Ketter, 2002; McClure, Pope, Hoberman, Pine, & Leibenluft, 2003). Because accurate face emotion recognition is an important component of successful interpersonal functioning throughout the life span, deficits in emotion recognition

might explain the high level of impaired social functioning in these diverse psychological disorders (Surcinelli, Codispoti, Montebanocci, Rossi, & Baldaro, 2006).

### **Facial Affect Recognition in Anxious Adults**

Adults with anxiety disorders have been found to display deficits in recognizing and interpreting facial affect (Winton, Clark, & Edelman, 1995; Veljaca & Rapee, 1998). Adults with social anxiety show an increased bias toward negative facial expressions and a lower accuracy in discriminating other emotions (Winton et al., 1995). In the presence of ambiguous emotional expressions, anxious adults misclassify ambiguous faces as fearful compared to less anxious adults (Richards, French, Calder, Webb, Fox, & Young, 2002) and adults with generalized social phobia show enhanced recognition memory for negative emotional faces compared to neutral faces (Foa, Gilboa-Schechtman, Amir, Freshman, 2000). A study by Joormann and Gotlib (2006) found that adults with social phobia were more accurate in identifying low to moderately intense angry faces compared to controls, suggesting that increased accuracy of identifying emotions that denote threat (i.e., anger) might predispose anxious individuals to avoid social situations and judge them as being high conflict, thus reinforcing already established negative appraisals.

Individual differences in anxiety-related traits have also been found to influence face emotion recognition in non-clinical samples. Individuals with high trait anxiety have been found to recognize fearful faces better than those with low trait anxiety (Surcinelli et al., 2006) and other studies have shown significant correlations between fearful face detection sensitivity and trait anxiety and neuroticism (Doty, Japee, Ingvar, & Ungerleider, 2013; Japee, Crocker, Carver, Pessoa, & Ungerleider, 2009). Others have found that non-clinical adults with moderate to high levels of anxiety show increased vigilance for threat faces (Bradley, Mogg, & Millar, 2000; Lagattuta & Kramer, 2017). Many studies have shown that faces displaying threat emotions (e.g., fear) are detected faster and tend to maintain a person's attention for longer durations than positive or neutral faces (Fox, Russo, & Dutton, 2002; Georgiou et al., 2005). Experiments conducted by Davis et al. (2011) have shown that the information conveyed by angry and fearful facial expressions differ, with fearful affect signalling that the expressor has detected something threatening and angry faces signalling that the expressor is a source of threat (Davis et al., 2011).

Hypervigilance to threatening faces is believed to rely on processes that are automatic and evolutionarily adaptive in helping individuals extract important survival information when

needing to quickly recognize friend or foe and as such, has been under strong selection pressure (Grossmann & Johnson, 2007). Threat avoidance on the other hand can be another manifestation of anxiety, which is proposed to indicate strategic avoidance to reduce distress (Mogg & Bradley, 1998). It has been suggested that the higher the threat perception, the stronger the threat avoidance bias (Cisler & Koster, 2010) while in the context of low threat, anxious individuals would prioritize positive and neutral stimuli and ignore ambiguous or low threat stimuli (Mogg & Bradley, 2016).

Impaired facial affect recognition as a manifestation of anxiety related bias could represent a synergistic outcome between hyper-vigilance and deficits in task related processing (Mathews & Mackintosh, 1998). In anxious individuals, hyper-vigilance toward threatening stimuli could arise as an automatic cognitive process (Williams et al., 2009), and second, because this attention bias competes for resources in other types of cognitions (i.e., recognition tasks), performance on affect recognition tasks would be impaired (Mathews & Mackintosh, 1998). This could explain affect recognition biases that are manifested in misrecognition of ambiguous or low threat intensity face emotions (Demenescu, Kortekaas, den Boer, & Aleman, 2010). For example, when an anxious individual is exposed to an ambiguous or low threat face, automatic attentional biases are activated, resulting in hyper-vigilance (Mogg, Bradley, De Bono, & Painter, 1997). The hyper-vigilance would use cognitive resources that would be needed by the recognition task, thus leaving a shortage of cognitive resources needed to make a correct recognition decision (Demenescu et al., 2010; Mathews, 1990; Mogg et al., 1997).

Facial affect recognition deficits could also result from a disengagement of attention toward stimuli that are perceived to indicate high danger, or stimuli that have led to increased anxiety due to past-learned experience (Mogg & Bradley, 1998). The activation of anxiety-related cognitions could lead to strategic automatic avoidance of the presented stimuli in order to reduce distress. This avoidance pattern might lead to impaired face recognition for high threat stimuli as well as generalize for other-valence stimuli (Mogg & Bradley, 1998). It is also possible that these two paths might be influenced by state anxiety, environmental influences or genetic vulnerabilities. Different types of attentional biases toward a variety of stimuli have been found in many anxiety disorders (e.g., social anxiety, panic disorder), and are proposed to represent core components of these disorders. A meta analysis of 172 studies by Bar-Haim, et al. (2007), found similar patterns of threat related biases in both adults and children and these

attentional biases are displayed by both clinical populations and high anxiety non-clinical populations.

### **Children with Anxiety**

Facial affect recognition deficits have been reported in children and adolescents with autism (Harms, Martin, & Wallace, 2010), schizophrenia (Habel, Krasenbrink, Bowi, Ott, & Schneider, 2006), bipolar disorder (Brotman et al., 2008), eating disorders in females (Zonnevillle-Bendek et al., 2002), attention deficit hyperactivity disorder (ADHD) in boys (Boakes, Chapman, Houghton, & West, 2008), and conduct disorder in boys (Fairchild et al., 2009). It has been proposed that anxiety disorders are neurodevelopmental in nature (Biederman et al., 2001) and several studies show that in clinical child populations, deficits in facial affect recognition have an early onset (Collin, Bindra, Raju, Gillberg, & Minnis, 2013; Simonian et al., 2001). Children with anxiety disorders and those who exhibit anxious traits make more errors recognizing both negative (Battaglia et al., 2004; Easter et al., 2005; Jarros et al., 2012; Simonian et al., 2001) and positive valence face emotions (Simonian et al., 2001; Easter et al. 2005) relative to controls.

Results on affect recognition biases are not consistent however, and some studies have failed to find differences between clinically anxious and non-anxious children's ability to recognize facial affect (Beesdo et al., 2009; McClure et al., 2003). A study by Lau et al. (2012) found no difference between clinically anxious and non-anxious children in their ability to recognize negative valence faces, but they did find that anxious children were more likely to correctly identify expressions of disgust and to avoid a conditioned stimulus paired with a masked angry face. Allen, Abbott and Rapee (2006) found that children with anxiety disorders as well as controls, mislabelled faces denoting disgust as anger, and children with OCD showed more accurate recognition of expressions of surprise compared to controls. Other studies have not found higher recognition deficits of children with anxiety compared to other types of psychopathology (Guyer et al., 2007; Manassis & Young, 2000).

While some studies have used the facial recognition task to assess deficits in social cognition, others have used the visual dot probe task (Heim-Dreger, Kohlmann, Eschenbeck, & Burkhardt, 2006; Waters, Lipp, & Spence, 2004) and the facial emotion Stroop task (Heim-Dreger et al., 2006; Richards, Richards, & McGeeney, 2000; Richards, French, Nash, Hadwin, & Donnelly, 2007) to evaluate attentional biases toward face emotions in anxious and non-anxious

youth. During the visual dot probe task the child is in front of a computer and is asked to stare at a fixed point on the center of the screen. The task stimuli involving pictures appear on either side of the screen for a pre-determined length of time, following a dot that appears on either side of the screen. The amount of time it takes to locate the dot indicates attention bias for threatening stimuli (if the dot is more quickly detected appearing after a threatening face) compared to attention toward neutral or positive stimuli (MacLeod, Mathews, & Tata, 1986; Heim-Dreger et al., 2006; MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002; Waters et al., 2004). During the Stroop task, children are presented with valenced faces that have a colored filter superimposed (e.g., red, blue, yellow and green). After each valenced face presentation, participants are instructed to name the color of the filter superimposed on the faces as fast as possible. The time difference in naming the color imposed on the threatening faces compared to neutral and positive faces is interpreted as a marker of anxiety-related interference (Richards et al., 2007). That is, if children take longer in naming the superimposed color on the threatening face, it means that they are hyper-focusing on the threatening stimulus, thus manifesting anxiety related interference.

Studies using the visual dot probe task reveal that clinically anxious children display higher attentional biases (i.e., greater vigilance) for both angry and happy faces compared to non anxious controls (Roy et al., 2008; Telzer et al., 2008; Waters, Mogg, Bradley, & Pine, 2008). However, other studies have shown that clinically anxious adolescents and those with high trait anxiety exhibit greater avoidance of negative valence facial emotions (Monk et al., 2006; Stirling et al., 2006). These attentional biases toward threatening facial affect have been reported in some (Heim-Dreger, Kohlmann, Eschenbeck, & Burkhardt, 2006; Richards, Richards, & McGeeney, 2000) but not all (Kindt, Bogels, & Morren, 2003) studies. Whether displaying hypervigilance toward threatening stimuli, measured as increased attention toward the target emotion or greater avoidance of such stimuli, these findings indicate a relationship between anxiety and selective attention to threat in anxiety-disordered children. The disengagement of attention away from negative faces might be another manifestation of attentional biases in avoiding social threat (Heim-Dreger et al., 2006; Stirling et al., 2006). Conversely, hypervigilance to threatening stimuli might reflect underlying biological vulnerability for attending to relevant threat cues and for developing anxiety disorders (Craske, 2003; Roy et al., 2008).

### **Children at Risk**

While attentional deficits in cognitive biases have been documented in clinical adult and child samples, the research is sparse in children who are at risk for developing anxiety. A growing body of research has pointed toward the link between parental anxiety and offspring anxiety (Beidel & Turner, 1997; Turner et al., 2003) but little is currently known about the mechanisms under which parental anxiety is transmitted to their offspring. One of the earlier studies that looked at vulnerability factors of anxiety in high-risk children was conducted by Merikangas et al. (1999), where diagnostic interviews, developmental measures, family functioning and measures of brain functioning, including memory indices, were administered to offspring of probands with anxiety disorders, substance abuse and unaffected controls. It was found that high-risk offspring performed more poorly on memory tasks and made more errors than controls, which was proposed to constitute a premorbid risk factor for the development of childhood anxiety (Merikangas, Avenevoli, Dierker, & Grillon, 1999). Other studies using the high-risk methodology have found no such impairments between high-risk and control children (Vasa et al., 2007) but their memory indices included non-emotionally evocative word lists. They did however find memory impairments in offspring with a current diagnosis of social phobia as these offspring displayed higher impairment in visual memory compared to controls.

Deficits in the processing of facial affect have been reported in at-risk child populations that have used facial affect recognition tasks. A study by Pine et al. (2005) compared face emotion processing in children with parental panic disorder (PD), parental major depressive disorder (MDD) and no parental history of psychiatric disorders. It was found that children of parents with PD showed more fear when viewing threatening faces and a longer latency to report their fear than children with no parental PD. It was proposed that children with a genetic vulnerability to PD might disproportionately allocate their attentional resources toward fear recognition. This in turn might disrupt their attentional allocation toward other facial emotions and result in greater overall facial affect recognition biases. It should be noted though, that their high-risk sample included children with a current diagnosis of an anxiety disorder, which may have confounded findings.

Facial affect recognition biases in unaffected high-risk offspring of parents with PD with and without agoraphobia were also explored by Bilodeau et al. (2014). They hypothesized that high-risk children would exhibit deficits in recognizing threat related faces, in addition to

reporting a heightened state of anxiety while viewing negative valence faces. Their hypotheses were based on previous findings suggesting that people with PD are more likely to show deficits in recognizing threat related stimuli due to heightened neurological systems proposed to pick up these emotion signals. The authors found that children at high risk to develop PD exhibited deficits in recognizing negative valence emotions but were comparable to low-risk children in recognizing positive and neutral facial affect. The capacity to discriminate sad faces varied by gender, with high risk girls making more mistakes in identifying sad affect compared to low risk girls and mislabelling sadness as fear. This finding might reflect a different processing of emotional stimuli by females and is congruent with epidemiological data showing that females are at greater risk for developing both depressive and anxiety disorders compared to males. The authors also found that high-risk children mislabelled angry and fearful faces as denoting surprise, which might have been due to a negative bias in the interpretation of surprised faces also found in other studies with child and adolescent populations (Tottenham, Phuong, Flannery, Gabard-Durnam, & Goff, 2013).

### **Facial Affect Recognition and Neural Correlates**

Neuroimaging studies have explored the neural correlates that underlie deficits in social cognitions (Corden et al., 2006; Marsh & Blair, 2008). Corticolimbic brain circuits are most often associated with the evaluation of faces and facial expressions (Adolphs, 2002; Todd & Anderson, 2009), and dysregulation of these circuits, especially the amygdala, have been implicated in the pathophysiology of anxiety disorders and may underlie impaired facial expression processing in anxious individuals. Neuroimaging studies have reported that adult patients with diverse anxiety disorders show greater activation of the amygdala and other affective processing regions while viewing negative (e.g., angry and fearful) faces than non-anxious controls (Chechko et al., 2009; Etkin & Wager, 2007; Evans et al., 2008; Hahn et al., 2011; Stein, Goldin, Sareen, Zorrilla, & Brown, 2002). Similar findings have been observed in anxious youth. McClure et al., (2007) and Thomas et al., (2001) noted that youth with anxiety disorders exhibit enhanced amygdala reactivity to faces with fearful and angry expressions, as opposed to neutral and happy faces, during a face-processing task. Similarly, Perez-Edgar et al. (2007) reported that behaviorally inhibited adolescents show exaggerated amygdala activity to fearful and angry facial expressions compared to non-inhibited controls. In a study using events related potential methodology, shy children exhibited smaller N400 amplitudes in response to

hostile and neutral facial expressions relative to non-shy children (Battaglia et al., 2005). In studies using amplitude response specifically measuring structural encoding of emotional faces, studies have shown that the N170 amplitude is sensitive to emotion, such that N170 amplitudes are larger when people are exposed to emotional versus neutral faces (Ashley, Vuilleumier, & Swick, 2004). Overall, it appears that anxiety disordered and anxiety-prone children and adolescents have a heightened sensitivity (i.e., hyperactive amygdala response) in brain systems that may contribute to deficits in processing of threat-related facial stimuli.

There is ample research suggesting that anxiety runs in families (Hirshfeld-Becker, Micco, Simoes, & Henin, 2008; Rapee et al., 2009; Shamir-Essakow, Ungerer, & Rapee, 2005; Siqueland, Kendall, & Steinberg, 1996) and that genetic factors may contribute to individual differences in facial affect recognition. Studies have found associations between specific genes, (i.e., 5-HTTLPR, the serotonin transporter gene) and attentional biases to threatening faces (i.e., avoidance of angry faces) in the presence of environmental stressors (i.e., maternal criticism) (Gibb et al., 2011). Interactive genetic models propose that genetic vulnerabilities might be moderated by environmental factors, and a few studies have provided evidence for the proposed interactions between these two factors in identifying risk for the development of anxiety (Beevers, Wells, Ellis, & McGeary, 2009; Klengel & Binder, 2013; Wilkinson, Trzaskowski, Haworth, & Eley, 2013).

In summary, various studies have shown affect recognition biases in children, adolescents and adults with anxiety disorders as well as in children at genetic risk for anxiety disorders (Lau et al., 2009; Simonian et al., 2001; Vicari et al., 2000; Waters, Lipp, & Spence, 2004). Other studies have shown significant attentional biases toward specific threat emotions, such as fear and anger and others have not (Guyer et al., 2007; Wong et al., 2012). These inconsistent findings might be attributed to differences in study population, methodologies, test design, sample size and sample characteristics. Overall, these findings suggest that anxious children might not process facial affect the same way as non-anxious children, which might put them at a disadvantage for developing appropriate social behaviours. Based on findings from neuroimaging, fMRI and genetic studies, it is possible that the deficits in facial affect recognition and attentional biases toward threat related emotions could represent pre-existing risk factors for the development of anxiety disorders, especially in the presence of environmental risk factors.

One potential environmental risk factor that could moderate facial affect recognition is parenting style.

### **Parental Bonding and Risk for Anxiety Disorders**

The concept of parental bonding is used to describe parental behaviours and attitudes that are perceived by the offspring to indicate care and control (Parker, 1979). Parker proposed the dimensions of *care* and *overprotection* as best capturing parental behaviours and attitudes that indicate care, affection, sensitivity, cooperation, accessibility, indifference, strictness, punitiveness, rejection, interference, control, overprotection, encouragement of autonomy and independence. Parker and colleagues operationalized these two dimensions of parental bonding in the Parental Bonding Instrument (PBI) (Parker et al., 1979), which is one of the most well-established and validated self-report measures of parental bonding as perceived by offspring (Tsaousis, Mascha, & Giovazolias, 2012), with longitudinal studies confirming the scale's stability over a 20-year period (Wilhelm, Niven, Parker, & Hadzi-Pavlovic, 2005). The care and overprotection subscales are completed separately for mothers and fathers. Examples of items on the scale include "She /he invades my privacy "(Overprotection) and "She/he is affectionate to me" (Care). Scores on the PBI capturing perception of care and overprotection result in four quadrants: affectionless control (high overprotection and low care), affectionate constraint (high overprotection and high care), weak bonding (low care and low overprotection) and optimal bonding (high care and low overprotection). With the exception of optimal bonding, the three other parental bonding styles are recognized as environmental risk factors for developing mental health problems, including pathological anxiety (Koszycki, Bilodeau, Zwanzger, Schneider, Flament, & Bradwejn, 2013; Zheng et al., 2011; Parker 1983).

Parental bonding is sometimes mistaken for attachment style but the two are different constructs. Attachment is said to represent the overall relationship between the child and the parent, which in optimal circumstances, makes the child feel safe, secure and protected (Bowlby, 1969). Attachment captures the child's ability to use the parent as a secure base from which to explore and when needed, to use the parent as a provider of safety and comfort (Benoit, 2004). As such, attachment is said to represent the child's first blueprint in understanding their role in the world and their expectations about relationships with people as well as serving as a basis for their self-worth (Ainsworth, 1979; Bowlby, 1969; Colonesi et al., 2011). If the world is perceived as a safe place to explore and caregivers are perceived to be caring and available, the

child will have positive expectations of the world and of other people. According to Bowlby (1973), the expectations of the child regarding the world and other significant people (i.e., primary caregivers) determine the child's internal working models (IWMs). These IWMs are not static models however and it is proposed that they are shaped by experiences and become more complex as the child's interaction with the world becomes more complex throughout development (Ainsworth, 1979; Breinholst, Esbjørn, & Reinholdt-Dunne, 2015). One of the most important influences on the development of a healthy IWM is the parental-child bond. For example, Bowlby (1973) proposed that if an individual feels fear due to a perceived or imaginary threat in the environment, he/she will seek a protective and comforting attachment figure (i.e., the parent), and if the parent is not perceived to be available, the child will experience uncertainty and anxiety. In this way, the parental-child bond can negatively influence the IWM, thus leaving the child prone to experiencing fear, and making the child vulnerable to anxiety (Cassidy & Mohr, 2001; Cassidy, Lichtenstein-Phelps, Sibrava, Thomas, & Borkovec, 2009). Thus, a lack of warmth and over-protectiveness from the parent signals to the child that the environment is not safe. Parental warmth and the encouragement of autonomy provide a sense of security to the child, and contributes to the development of the blueprint in which the child will actively explore the environment (Yap, Pilkington, Ryan, & Jorm, 2014). In summary, attachment can be viewed as the blueprint of the roles and expectations that the child has about the world, and parental bonding is one of the most significant contributors of building that dynamic blueprint.

While sometimes attachment style and parental bonding are used interchangeably in the literature, they represent different constructs. For the purposes of this thesis, parental bonding will be used to refer to the pattern of behaviours that parents display toward their offspring early in life until adulthood (Crouch & Manderson, 1995; Stein et al., 2000). Parental bonding is reported to be a possible environmental risk factor for the development of anxiety disorders. Clinical and community samples of adults with anxiety disorders retrospectively describe their parents as overprotective, low on care, or both, during childhood (Silove, Parker, Hadzi-Pavlovic, Manicavasagar, & Blaszczynski, 1991; Turgeon, O'Connor, Marchand & Freeston, 2002; Gerlsma, Emmelkamp, & Arrindell, 1990) and observational studies of anxiety disordered children have noted similar negative parenting styles (Siqueland, Kendall, & Steinberg, 1996; Hudson & Rapee, 2002; Negreiros & Miller, 2014; Rapee, 1997; Siqueland et al., 1996). A meta-

analysis on 47 studies conducted by McLeod et al. (2007) suggests that parental control might play a significant role in the development and maintenance of childhood anxiety, but the specific processes under which this occurs, whether as an expression of parental anxiety or a parental response to the child's anxiety, still remain to be determined (McLeod, Weisz, & Wood, 2007; Negreiros & Miller, 2014).

Parents with anxiety disorders are more likely to display a rejecting and/or overprotective parenting style compared to parents without anxiety disorders (Challacombe & Salkovskis, 2009; Lindhout et al., 2006; Whaley, Pinto, Sigman, 1999). However, research on parental bonding of anxious parents is inconsistent (Koszycki et al., 2013; McClure, Brennan, Hammen, & Le Brocque, 2001; Merikangas et al., 2003; Woodruff-Borden, Morrow, Bourland, & Cambron, 2002) and the role that negative parental bonding has in the transmission of child anxiety or in factors that increase anxiety risk is unclear. For example, McClure and colleagues (2001) found no evidence that parental bonds mediated the transmission of anxiety disorders in a heterogeneous group of anxious parents and their children (McClure et al., 2001). Merikangas et al. (2003) found no consistent evidence between parental bonding and social anxiety in high-risk children. Also, Ginsburg, Grover and Ialongo (2005) found no differences in parental bonding between mothers with anxiety disorders and controls. However, at the six year follow up, they found that higher levels of criticism and lower levels of autonomy were significantly related to higher anxiety levels in children of anxious mothers (Ginsburg, Grover, & Ialongo, 2005). Although inconsistent, these findings suggest a link between parenting behaviour in anxious parents and increased risk for anxiety in offspring. In order to elucidate the role that parental bonding plays in the development of anxiety, we also need to explore the specific ways that parenting behaviour changes as a result of child temperament.

### **Behavioural Inhibition and Anxiety Disorder Risk**

Research shows that *behavioural inhibition*, an early appearing temperamental predisposition to be shy, fearful and avoiding the unfamiliar, is a significant risk factor for the development of anxiety (Biederman et al., 2001; Rosenbaum et al., 2000; Shamir-Essakow et al., 2005). Childhood temperament has been defined as a set of inherited behaviours that are evident at a very young age (Buss, 1989) and are viewed as a basis for the formation of personality (Degnan, Almas, & Fox, 2010; Katainen & Raikkonen, 1998; Rapee, 2002). Children who are described as behaviourally inhibited show higher physiological signs of arousal, higher cortisol

levels and increased heart rate when exposed to novel auditory and visual stimuli than non inhibited children (Fox, Henderson, Marshall, Nichols, & Ghera, 2005). One of the critical components of behavioural inhibition in children is behavioural withdrawal and avoidance (Rapee, 2002), which has been recorded as avoiding eye contact, reduced verbal utterances and avoidance of threatening stimuli (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984).

It is estimated that 15 % of children display behavioural inhibition (Fox et al., 2005) and several studies show that these children are at a higher risk for developing anxiety disorders later in life (Biederman et al., 1990, 2001; Coplan, Wilson, Frohlick, & Zelenski, 2006; Shamir-Essakow et al., 2005). While not all children who are behaviourally inhibited develop anxiety disorders, behavioural inhibition has been identified as a significant risk factor (Biederman et al., 1993; Chronis-Tuscano et al., 2009; Hirshfeld-Becker et al., 2003) and a recent meta-analysis of 7 studies estimates that almost half of behaviourally inhibited children will develop social anxiety disorder by adolescence (Clauss & Blackford, 2012).

Behavioural inhibition has been shown to be stable and consistent over time, but there is also ample evidence that environment factors interact with childhood temperament (Degnan et al., 2010; Katainen & Raikkonen, 1998; Shiner et al., 2012) and that child temperament influences parenting style (Brumariu & Kerns, 2010; Clauss, Avery, & Blackford, 2015; Hudson, & Rapee, 2002; Manassis & Bradley, 1994; Siqueland et al., 1996). In an observational study, Hudson and Rapee (2001) examined mother-child interactions in children with anxiety disorders and controls. They reported that mothers of anxious children provided less autonomy to their children and were more intrusive compared to mothers of control children. Furthermore, the authors found that the mothers of anxious children exuded less warmth and were more critical when addressing their children, compared to mothers of non-anxious children (Hudson & Rapee, 2001).

It is suggested that behavioural inhibition by itself is not a sufficient factor for the development of anxiety disorders in children and adults; instead, when behavioural inhibition is combined with negative parental bonding, such as lack of care and overprotection, it can become a risk factor for the development of anxiety and other psychopathologies (Chorpita & Barlow, 1998; Manassis & Bradley, 1994; Rapee, 1997). It is proposed that parents of behaviourally inhibited children are overly sensitive to their child's distress, and in an effort to reduce the distress, engage in overprotective parenting behaviours. In turn, this reinforces the child's

withdrawn and fearful behaviour, reduces their sense of self-efficacy in coping with the demands of novel contexts, and ultimately increases their risk for developing pathological anxiety (Hudson, & Rapee, 2002; Rapee, 1997). Inhibited children with parents who lack sensitivity and warmth and who impose socialization demands in an unsupportive and critical way are vulnerable to feelings of guilt and sensitivity to criticism (Longan & Phillips, 2001).

Aktar et al. (2013) explored the link between environmental risk factors for anxiety disorders (i.e., parental bonding) and behavioural inhibition. They hypothesized that the display of anxious behaviours by parents at home could be internalized and modelled by children and those children who tend to be behaviourally inhibited would also model anxious behaviours. They found that behavioural inhibition interacted with parental anxiety and this interaction led to the manifestation of fearful and avoidance behaviours in children. Interestingly, parental anxiety by itself did not predict children's fear and avoidance, highlighting the possibility that the interaction between parental anxiety, manifested through parental style and behavioural inhibition, might explain why anxiety tends to run in families (Aktar, Majdandžić, De Vente, & Bögels, 2013; Last, Hersen, Kazdin, Orvaschel, & Perrin, 1991). These findings suggest that intergenerational transmission of anxiety might work by shaping cognitive processes. That is, parents of anxious children are likely to display their own cognitive biases toward threat, and in behaviourally inhibited children, those biases are likely to be internalized and to be manifested in the child's future behaviours (Hudson & Rapee, 2002).

It has been proposed that parental bonding and behavioural inhibition interact in the development of anxiety disorders, and this interaction might contribute to the maintenance of anxiety symptoms by affecting cognitive processes that are presumed to underlie the onset and maintenance of anxiety (Bourke et al., 2010; Gamble & Rapee, 2009; Miskovic & Schmidt, 2012; Pérez-Edgar et al., 2007; Richards, French, Nash, Hadwin, & Donnelly, 2007). Specifically, individuals who report anxiety symptoms or who are at risk for developing anxiety have been shown to manifest higher attention biases to threatening stimuli (MacLeod et al., 2002; Richards et al., 2007; Waters, Henry, Mogg, Bradley, & Pine, 2010), hypervigilance toward threatening stimuli (Heim-Dreger et al., 2006; Pérez-Edgar et al., 2007), and negative interpretation biases for ambiguous stimuli (Vasey & Macleod, 2001).

This has also been found in behaviourally inhibited children who show hypervigilance to threat signals, which over the course of development may result in social withdrawal and

extreme shyness (Buss, 2011; Hirshfeld-Becker et al., 2008). The relationship between behavioural inhibition in early childhood and attentional biases to threatening stimuli in adolescence was examined by Pérez-Edgar et al. (2010). They hypothesized that adolescents who were classified as behaviourally inhibited as children would display attentional biases to threatening stimuli, especially after being exposed to threatening cues. Furthermore, they predicted that high levels of attentional biases displayed in adolescence would predict the presence of childhood behavioural inhibition, assessed as high reactivity level at 4 months of age. They found that adolescents who were behaviourally inhibited as young children showed high levels of attention bias to threatening faces compared to control children, who displayed a similar bias toward happy faces. In addition, they found that early childhood behavioural inhibition was linked to adolescent social withdrawal but only among adolescents who displayed attentional biases to threatening stimuli (Pérez-Edgar et al., 2010). In the context of understanding the continuity of behavioural inhibition into adolescence, this finding points to the moderating effect of attentional bias toward threatening faces in shaping social behaviour in children high in behavioural inhibition and consequently placing them at risk for anxiety.

Cognitive biases toward ambiguous stimuli have been linked to parental behaviours when parents provide negative information to their children regarding such situations (Fliet, Dibbets, Roelofs, & Muris, 2017). Consequently, this negative bias has been proposed to account for children's avoidance behaviours toward non-threatening stimuli (Hudson, & Rapee, 2002; Negreiros & Miller, 2014). There is evidence linking information-processing bias in children and parental practices (i.e., over-control) as children are said to model adult behavioural practices through direct experience or observation (McLeod et al., 2007; Negreiros & Miller, 2014; Volbrecht & Goldsmith, 2010). While there is evidence showing deficits in attention processing and facial emotion recognition in children at risk for anxiety, the specific pathways of such altered cognitions have yet to be uncovered. Some studies have shown that children with anxiety symptoms or at risk for anxiety, make more errors in facial affect recognition (Heim-Dreger et al., 2006; McClure et al., 2003; Simonian et al., 2001), but other studies have not (Manassis & Young, 2000; McClure et al., 2003). Environmental risk factors, such as parental bonding, have also been recognized as potential contributors to childhood anxiety, with or without the presence of specific facets of child temperament (i.e., behavioural inhibition). However, we do not yet

know the nature of the relationships between parental bonding, behavioural inhibition and facial affect recognition and the development of anxiety disorders.

### **Gender and Anxiety Disorder Risk**

It is well established that female gender is a risk factor for the development of anxiety disorders (Bruce et al., 2005; Weinstock, 1999). Epidemiological research has demonstrated that anxiety disorders are significantly more prevalent in females, with a female to male ratio of about 2:1 (Kessler et al., 2006; Kessler et al., 1994). Although the reasons why females are more susceptible to developing anxiety is not fully understood, psychological, biological and environmental influences are thought to play a role (Jang, Stein, Taylor, & Livesley, 1999; Kendler, Neale, Kessler, Heath, & Eaves, 1992; Pearlstein et al., 1990; Seeman, 1997) Muris, Meesters, & Knoop, 2005; Stevenson-Hinde & Shouldice, 1993). Females score higher than males on measures of personality traits that have been linked with a higher risk for developing pathological anxiety (e.g., neuroticism, negative affectivity) (Costa, Terracciano & McCrae, 2001) and report greater fear responses (Weissman & Merikangas, 1986; Weissman, Leckman, Merikangas, Gammon, & Prusoff, 2009), although in clinical samples results have been mixed (Foot & Koszycki, 2004; Schmidt & Koselka, 2000; Van Beek, Perna, Schruers, Muris, & Griez, 2005). While gender differences in early temperament are negligible in very young children (e.g., Rothbart, 1986), in girls, behavioural inhibition influences the stability of internalizing problems more than in boys (Hastings et al., 2015), perhaps due to differences in socialization (i.e., parenting style, gender roles, societal expectations) (Muris et al., 2005; Stevenson-Hinde & Shouldice, 1993).

The same factors have been proposed to account for gender differences in anxiety disorder risks in children and adolescents (Kendler et al., 1992; Lewinsohn & Gotlib, 1998; Weissman et al., 2009), and psychosocial variables, in combination with biological differences, are presumed to be established during the early formative years (McLean, Carmen P, Asnaani, Anu, Litz & Hofmann, 2012). For example, a study by Pomeranz and Ruble (1998) reported greater maternal control and less autonomy granting toward girls compared to boys. Less autonomy and higher tendency to take responsibility for failure, which is predominantly seen in girls versus boys, reduces self-efficacy and increases helplessness and anxiety vulnerability in girls (Fox & Ferri, 1992; Mathews, Koehn, Abtahi, & Kerns, 2016). Furthermore, the tendency to worry and ruminate is overwhelmingly identified in adult women with anxiety (Tompkins,

Hockett, Abraibesh, & Witt, 2011) as well as in pre-adolescent girls (Campbell & Rapee, 1994) and adolescent girls (Brumariu & Kerns, 2010; Rose, Carlson, & Waller, 2007).

Cognitive factors have also been implicated in the differential anxiety rates between the two genders, with mounting evidence pointing to distinctive attention biases toward threatening stimuli for women versus men (Lee, Herbert, & Manassis, 2014; Wieckowski et al., 2016). Females with high trait anxiety have been shown to display enhanced attention toward aversive faces compared to males (Waters, Nitz, Craske, & Johnson, 2007) and gender differences in attentional biases to threat, in the form of hypervigilance, have also been reported in studies using subliminal affective pictures (Tan, Ma, Gao, Wu, & Fang, 2011) and the dot probe task, with anxious women displaying higher attentional biases toward angry faces compared to men (Tran, Lamplmayr, Pintzinger, & Pfabigan, 2013). It has been suggested that the cognitive biases that females manifest in the processing of emotional stimuli might interact with specific social patterns of reinforcement that predispose them to rely more on avoidant coping and withdrawal, and consequently render them at a higher risk for developing anxiety (Kendler, Myers, & Prescott, 2000; McLean & Anderson, 2009).

In sum, the findings from the parental bonding literature suggest that negative parental bonding is a risk factor for the development of anxiety disorders. It has also been proposed that parental bonding might interact with child temperament and behavioural inhibition in particular, in further increasing anxiety vulnerability. While cognitive biases have been reported in individuals with anxiety disorders, it is not clear whether these cognitive biases are pre-morbid markers of anxiety, or they are part of the pathways in which anxiety is manifested. The present research aims to provide clarity in the ways that these factors might interact.

### **Present Study**

The present study aims to elucidate the pathways in which environmental factors (i.e., parental bonding), temperamental factors (i.e., behavioural inhibition) and cognitive factors (i.e., facial affect recognition) may contribute to the development of anxiety. The data for this study were collected as part of two larger studies funded by the Canadian Institutes of Health Research (Dr. Diana Koszycki, Principal Investigator) that examined biological, psychological, and environmental markers in healthy children with a parental history of anxiety disorders: generalized anxiety disorder (GAD), social anxiety disorder (SAD) and panic disorder (PD) with

or without agoraphobia, and those with no parental history of psychopathology. The studies were approved by the Research Ethics Board of the Royal Ottawa Mental Health Center (Study 1) and the University of Ottawa (Study 2).

### **Objectives and Hypotheses**

1. To determine whether parental bonding influences facial affect recognition in children and adolescents with no psychiatric history. The working hypothesis is that a negative parental bonding will predict impaired facial affect recognition.
2. To determine whether child gender and having a parent with a history of anxiety disorders moderates the relationship between parental bonding on facial affect recognition. The working hypothesis is that the interaction between negative parental bonding and deficits in facial affect recognition will be stronger for children with a parental history of anxiety versus children with no parental psychopathology and for girls versus boys. Although numerous studies and epidemiological data show that females are more likely to develop anxiety disorders than males, this research will explore the hypothesis that these differences might reveal vulnerability factors that are related to parental bonding practices that might be experienced differently by the two genders.
3. To determine if child temperament mediates the relationship between parental bonding and facial affect recognition. The working hypothesis is that elevated levels of behavioural inhibition will mediate the relationship between negative parental bonding and impaired facial affect recognition and this will be more evident in high-risk children.

## **Method**

### **Participants and recruitment**

Participants were psychiatrically healthy children and adolescents between 7 to 18 years of age who participated in two larger studies involving offspring at high and low familial risk for anxiety. Families were primarily recruited via advertisements placed in local newspapers and on the Internet, as well as flyers placed on public bulletin boards. In addition to these recruitment strategies, some participants were recruited via their parents who had participated in a genetic study for PD. Participants were given monetary compensation for their participation. Whenever the child was below the age of consent, written informed consent was obtained from the child's

legal guardian as well as written assent from the child. Participants aged 16 years and over provided their own written consent.

### **Assessment of Parents**

Parents who expressed an interest in the study were initially pre-screened via a telephone interview conducted by a research assistant, who explained the purpose of the study and obtained information about the parents' history of psychopathology and their child's psychiatric and medical history and medication use. If the pre-screen interview deemed the family eligible, a second telephone interview was scheduled to formally evaluate the diagnostic status of the parent. Current or lifetime diagnosis of primary PD with or without agoraphobia, SAD or GAD in the parent(s) of high risk offspring and the absence of lifetime psychopathology in both parents of low risk offspring was confirmed with the Structured Clinical Interview for DSM (SCID, First, Spitzer, Gibbon, & Williams, 1996). SCID interviews with the affected parent were conducted by a licensed psychologist with extensive experience using this diagnostic instrument. Interviews with both parents of low risk offspring were conducted by trained research assistants who were supervised by a psychologist throughout the study and who were trained to high levels of inter-rater reliability.

### **Assessment of Offspring**

Potentially eligible offspring came to the laboratory for an interview in order to confirm the absence of a current or past history of any psychological disorder. Offspring were assessed with the child version of the SCID (Matzner, First, Spitzer, Williams & Gibbon, 1998) (Study 1) or the Anxiety Disorders Interview Schedule-Child Version (ADIS-C, Silverman & Albano, 1996) (Study 2). The interviewers were clinicians and research assistants who were unaware of parental diagnosis. Offspring were excluded if they had a current or past history of any threshold (i.e., meeting full criteria for a diagnosis) or subthreshold (i.e., has symptoms of the disorder but does not meet full criteria for a diagnosis) of psychiatric disorders, reported any significant medical conditions or were currently using medications with peripheral and central nervous system effects. Eligible offspring completed self-report and other study measures and were scheduled for a second visit to complete the facial affect recognition task.

## Measures

**Childhood Self-Report of Inhibition-Version 2.** (*CSRI*) is a 30-item scale developed by Reznick, at the University of North Carolina, to assess childhood behavioural inhibition and is based on the adult self-report Retrospective Childhood Inhibition Scale (RCIS) (Reznick, Hegeman, Kaufman, Woods, 1992). The child-appropriate version stays true to the contents of the CSRI but it is written in a language that could be understood by children as young as 7 years old. Questions ask about a wide range of childhood behaviours that are associated with behavioural inhibition, such as separation anxiety, withdrawal from social situations, fears as well as complaints related to illness. Psychometric properties of the CSRI are unavailable, however, research on the RCIS reveal high internal consistency coefficients ranging from 0.79 to 0.91 (Bilodeau et al., 2014).

**Parental Bonding Instrument** (*PBI*; Parker, 1983) is a 25-item self-report questionnaire designed to measure the parenting style of the mother and father as perceived by the offspring. As initially described by Parker et al (1979) the PBI measures two broad parenting styles: *overprotection* and *care*. These dimensions can be further divided into four separate categories; high care and high protection (affectionate constraint), high protection and low care (affectionless control), high care and low protection (optimal parenting), and low care and low protection (neglectful parenting; Parker et al., (1979). The PBI has satisfactory psychometric properties and it has been shown to be stable over a 20-year period (Wilhelm et al., 2004).

**Pictures of Facial Affect** (*POFA*; Ekman & Friesen, 1976). The POFA was used for facial emotions identification. The POFA includes 110 black and white photographs of 8 females and 6 males expressing either an emotion (happiness, sadness, fear, anger, or surprise) or a neutral facial expression. In the present study, participants were presented with pictures displaying 5 emotions (happiness, sadness, anger, fear, and surprise) or a neutral expression. Disgust affect was not included because it is the least accurately identified affect in children, with rates of accuracy averaging between 30% and 40% in healthy children (Williams et al., 2011). The task included 36 trials divided into 6 blocks of 6 pictures each (3 males, 3 females). Each block included pictures of all 5 facial affect and a neutral expression. Pictures were chosen at random and the presentation order was counterbalanced for each block. Pictures were presented sequentially on a computer screen using PowerPoint-presentation. Children were provided with a list of all possible answers and after each picture had been displayed for 10

seconds, they were instructed to enter the correct choice by identifying the displayed emotion. The last block of the POFA was presented in the same way as the others, however, participants were instructed to rate how anxious they felt while viewing each picture on a scale of 1 (not at all) to 5 (extremely).

### **Statistical Analyses**

Data were analyzed with SPSS Version 23. I used multiple regression analyses to assess the relationship between parental bonding (separately for maternal and paternal bonding) and facial affect recognition. I used the bootstrapping resampling procedure included in the SPSS software, due to its higher precision in parameter estimation when the number of predictors increases (i.e., interaction terms) and the assumptions of the procedures (i.e., multicollinearity) are not as robust (Varian, 2005). The bootstrapping procedure computes parameter estimates by drawing or resampling 1000 random samples from the current sample data and calculates the parameters as specified in the analysis. No modification of data is needed for this procedure and all parameter estimates reported below have been computed with this procedure.

Associations between behavioural inhibition, PBI scales denoting care and overprotection, accuracy in facial affect recognition, and anxiety ratings when viewing affective faces, were assessed using bivariate correlations. I assessed the moderating effects of gender and risk group by computing interaction terms between the predictor variables and the moderators and entered them sequentially in the multiple regression models.

The outcome variables for the facial emotions, were computed as averages of the participant responses, coded as 0= match and 1= error. Thus, greater numbers indicate greater recognition errors for each facial emotion. The scales of care and overprotection were computed based on the instructions by Parker (1983), and were used as predictors in the regression analyses.

### **Missing data**

Due to a problem with the computer keyboard, data from 2.08 % of the facial affect recognition trials were missing. I used the process of Multiple Imputation for categorical variables to impute the missing data in the facial recognition task for participants that had fewer than 2 missing data points for each facial affect. Due to the fact that each face denoting a specific

emotion was presented 5 times, I excluded from the analyses participants ( $n=2$ ) who had 3 or more missing data for the same emotion. That was the case only for fear. For the other face emotions, Little's Missing Completely at Random (MCAR) procedure did not reveal systematic patterns of missing data. Even though the literature proposes no cause for concern when the percentage of missing data is less than 5 %, I imputed missing data for the facial affect recognition task and anxiety ratings by using the Multiple Imputation (MI) method as it is advantageous over other imputation methods (Graham, 2009). It is aimed to simulate multiple random samples when imputing the missing values and testing the parameters with multiple regression analyses, which results in better parameter estimates and more precise confidence intervals (Graham, 2009).

## Results

### Participant characteristics

Combining the databases from Cohort 1 and Cohort 2 resulted in a final database with 179 children. Children who were missing all data from the facial recognition task ( $n=1$ ), or children who were missing parental bonding data for both parents ( $n=2$ ), were excluded from the analysis. The participants ( $n=2$ ) who were missing all data for maternal bonding and those ( $n=2$ ) who were missing all data for paternal bonding were excluded from analyses when testing maternal and paternal bonding respectively. The final dataset had facial recognition data for 176 children. The high-risk (HR) group ( $n=73$ ) had 35 females and 38 males with a mean age of 11.73 ( $\pm 3.23$ ) and the low risk (LR) group ( $n=103$ ) had 50 females and 53 males with a mean age of 11.00 ( $\pm 3.16$ ). From the children with same ethnicity parents ( $n=157$ ), one hundred twenty-four were Caucasian, twelve were black, nine were Hispanic, ten were Asian and the rest had mixed ethnicity parents ( $n=19$ ).

### Correlations between predictors, mediators and outcome variables

The parental bonding scales denoting mother and father care were negatively associated with childhood behavioural inhibition whereas the scales measuring mother and father overprotection were positively associated with behavioural inhibition (see Table 1). As expected, behavioural inhibition scores (BI) were negatively correlated with maternal and paternal care and positively correlated with maternal and paternal overprotection. Correlations between facial

affect recognition errors revealed that deficits in recognizing sadness were positively associated with deficits in recognizing fear and anger, whereas deficits in recognizing happy faces were negatively associated with deficits in recognizing faces expressing fear.

Table 1

*Bivariate Correlations Between Parental Bonding, Behavioural Inhibition (BI) and Facial Recognition Errors*

	BI	Happy	Sad	Surprise	Fear	Angry	Neutral
Mother care	-.21**	-.03	.09	-.02	.09	.05	.07
Mother overprotection	.19*	.05	-.03	.04	-.03	-.06	-.05
Father care	-.19**	-.01	.01	-.05	.05	.05	.04
Father overprotection	.22**	-.01	.01	.03	.06	-.11	-.12
Happy	.11	1	.09	.04	-.17*	.14	-.03
Sad	.12		1	-.01	.19**	.20**	-.02
Surprised	.08			1	-.07	.08	.11
Fear	.04				1	.11	.07
Angry	-.08					1	.01
Neutral	.14						1

$p < .05$ . \*  $p < .01$ . \*\*.  $N = 176$ .

### **Does parental bonding influence facial affect recognition (Hypothesis 1)**

The results of the multiple regression analyses were not significant when the paternal scales of care and over protection were used to predict facial affect recognition errors. Moreover, risk group was not found to be a moderator in this relationship for any of the faces denoting facial affect.

Also, the results of the multiple regression analyses revealed that maternal care and overprotection were not significant predictors of the ability to accurately recognize facial affect for any of the facial emotions. When I tested for the moderating effect of risk group, results were not significant for any of the faces denoting emotion, with the exception of the neutral face. Specifically, the results showed that risk group moderated the relationship between mother care and the ability of offspring to recognize neutral faces, as indicated by a significant increase in the

proportion of variance explained and a significant interaction,  $R^2 = .03$ ,  $F(1, 167) = 4.06$ ,  $p = .045$ ,  $b = -.89$ ,  $t = -2.01$ ,  $p = .049$ . Examination of the interaction plot (see Figure 1) showed that low risk offspring who perceived their mothers to display higher levels of care, made more errors when recognizing the neutral face than offspring who perceived their mothers to be less caring.

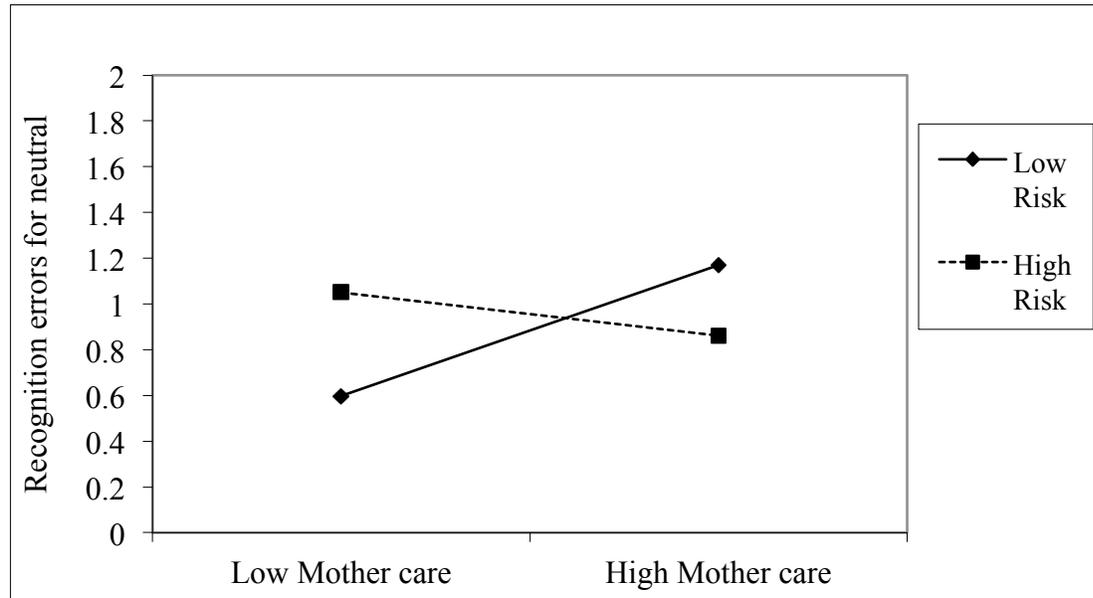


Figure 1. Risk group as a moderator between mother care and recognition errors for the neutral face.

### Does gender moderate the relationship between parental bonding and facial affect recognition? (Hypothesis 2)

To test the hypothesis that child gender moderates the relationship between parental bonding and facial affect recognition, I conducted separate multiple regression analyses with the maternal and paternal PBI subscales as predictor variables. The working hypothesis was that negative parental bonding will result in higher facial affect recognition errors for girls compared to boys and this will be more evident in high-risk offspring. After inserting gender as a moderator in the regression model, I also tested for the interaction between gender and risk group in the relationship between parental bonding and facial affect recognition.

#### *Maternal bonding*

The results of the multiple regression analyses did not support the hypothesis that child gender moderates the relationship between maternal bonding and facial affect recognition for any of the facial emotions. However, it was found that irrespective of perceived maternal care and overprotection, gender interacted with risk group in predicting facial recognition errors for the face denoting sadness,  $R^2 = .07$ ,  $F(9, 161) = 1.49$ ,  $p = .15$ ,  $b = -.94$ ,  $t = -2.63$ ,  $p = .009$ . The interaction plot shows that low-risk males made more errors in recognizing sad affect than high-risk males, whereas high-risk females made more errors recognizing sad affect than low-risk females (see Figure 2).

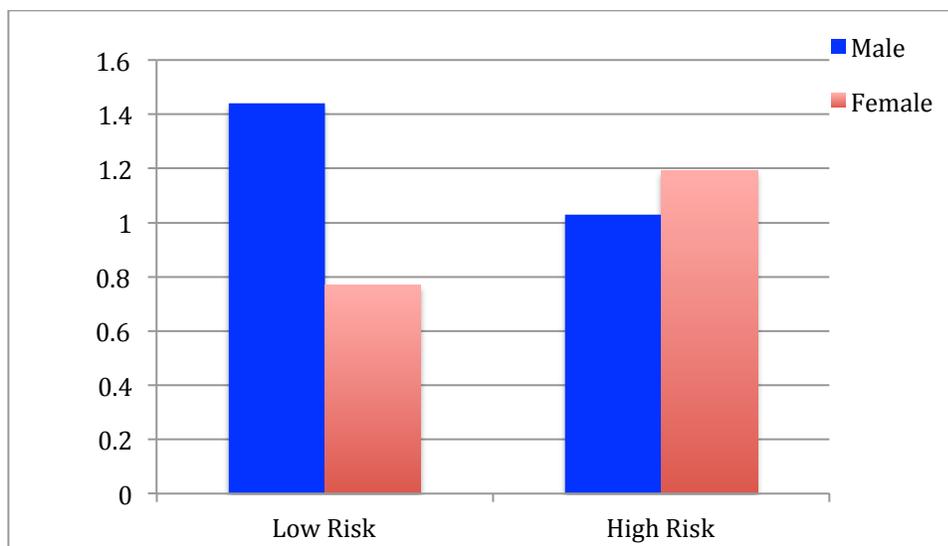


Figure 2. Recognition errors for sad by gender and risk group

While gender did not moderate the relationship between maternal bonding and recognition errors for the face denoting anger, the interaction between gender and risk group was significant for the face denoting anger,  $R^2 = .06$ ,  $F(9, 161) = 1.24$ ,  $p = .29$ ,  $b = -.66$ ,  $t = -.366$ ,  $p = .006$ . When I plotted the interaction (see Figure 3), it was shown that low-risk males made more errors recognizing angry faces than high-risk males, and this was reversed for females, with high-risk females making more errors recognizing anger than low-risk females.

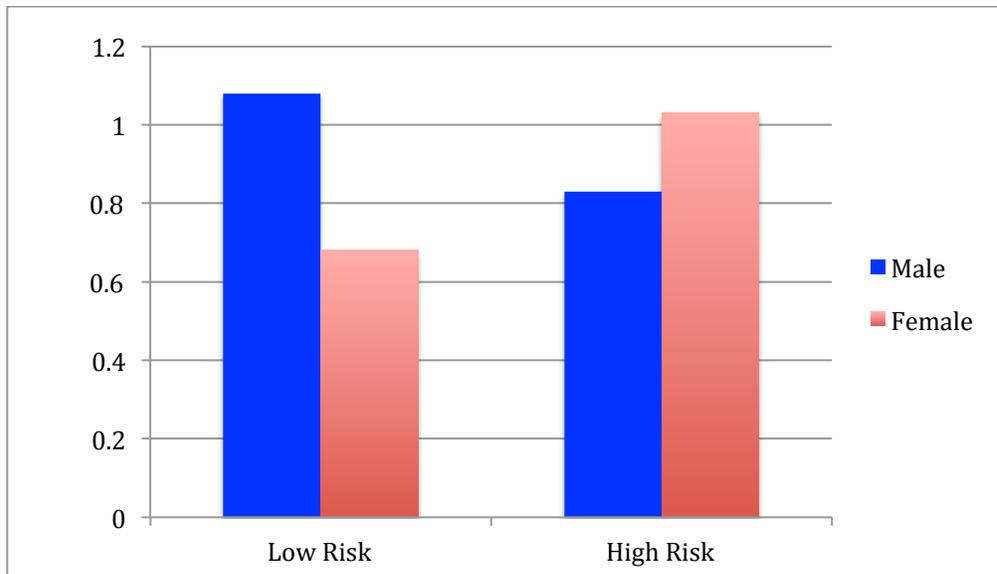


Figure 3. Recognition errors for anger by gender and risk group

#### *Paternal bonding*

The multiple regression analyses did not reveal gender differences for the faces denoting sadness, happiness, surprise and neutral. However, for the fearful face, gender moderated the relationship between father overprotection and recognition errors for the face denoting fear,  $R^2 = .05$ ,  $F(9, 161) = 2.06$ ,  $p = .036$ ,  $b = 0.078$ ,  $t = 1.98$ ,  $p = .028$ . As shown in Figure 4, males who perceived their fathers to be overprotective made more recognition errors for fearful faces compared to males who perceived their fathers not to be overprotective.

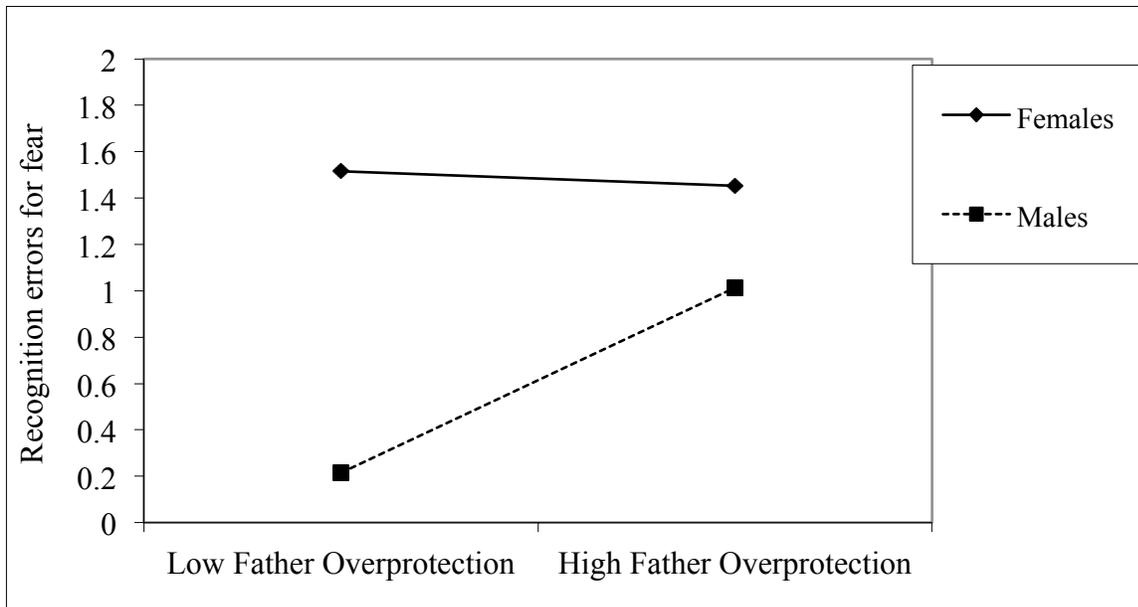


Figure 4. Interaction between father overprotection and gender for fear

For the angry face, gender did not moderate the relationship between paternal bonding and recognition errors. However, risk group was found to interact with father care in predicting recognition errors for the angry face,  $R^2 = .03$ ,  $F(9, 161) = 1.85$ ,  $p = .063$ ,  $b = -.039$ ,  $t = -2.00$ ,  $p = .046$ . As shown in Figure 5, high-risk offspring who perceived their fathers being low in care made more errors when recognizing anger compared to low risk offspring who perceived their fathers to be low in care.

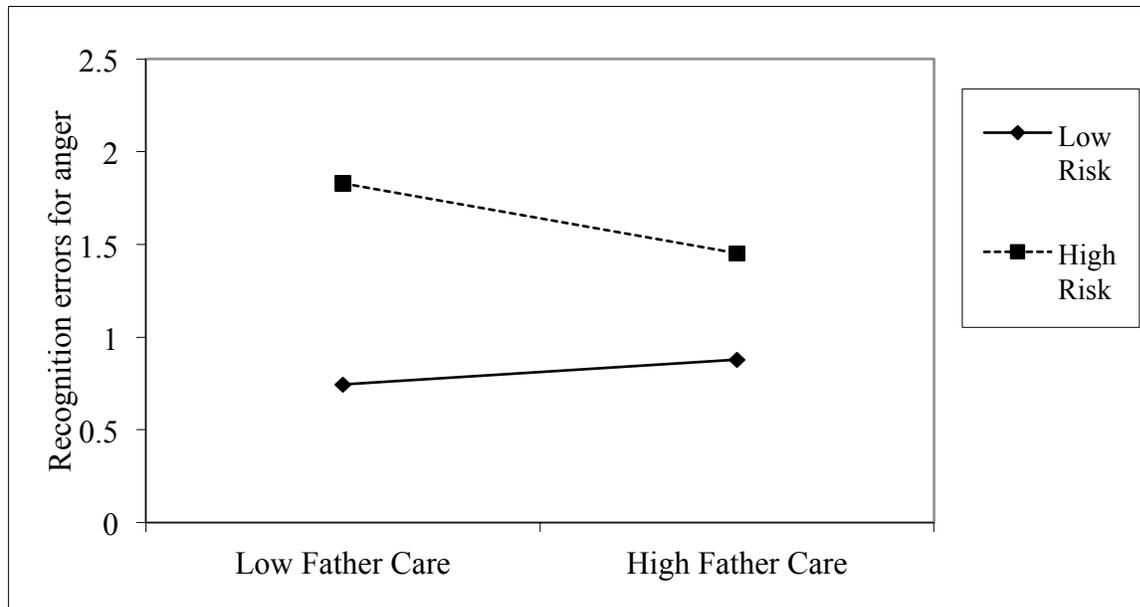


Figure 5. Interaction between father care and risk group for the angry face

In summary, the second hypothesis was partially supported, as results revealed that gender moderated the relationship between father overprotection and facial recognition errors for fear. Specifically, males who perceived their fathers being more overprotective made more errors recognizing fear than males who perceived their fathers being less overprotective. Father care also interacted with risk group when predicting facial recognition errors for anger. Specifically, high-risk offspring who perceived their fathers to be low in care made more errors recognizing anger compared to low risk offspring who perceived their fathers to be low in care. With respect to the proposed moderating effect of gender in the relationship between maternal bonding and gender, the results were not significant. However, irrespective of maternal bonding, gender interacted with risk group in predicting recognition errors for sad and anger. Specifically, low risk males made more recognition errors for sad and anger compared to high-risk males, whereas high-risk females made more recognition errors for sad and anger compared to low-risk females.

### **Does child temperament mediate the relationship between parental bonding and facial affect recognition? (Hypothesis 3)**

The third hypothesis predicted a mediating effect of child behavioural inhibition in the relationship between parental bonding and facial affect recognition. As specified by the three-step mediation procedure by Baron and Kenny (1983), I tested in the first hypothesis the direct

effect between the PBI scales of care and overprotection for mother and father separately, with the facial affect recognition errors scores as the outcome variables. Since none of the regression analyses were significant for any of the facial affect, mediation analyses were not conducted.

### **Exploratory analyses: Parental bonding categories and facial affect recognition**

Exploratory analyses were conducted to examine the effect of parental bonding categories on facial affect recognition. As outlined by Parker et al. (1979), the scales of care and overprotection can be combined into four bonding categories: high care and low overprotection (optimal bonding), low care and low overprotection (absent or weak bonding), high care and high overprotection (affectionate constraint) and low care and high overprotection (affectionless control). The three groups denoting sub-optimal parental bonding (weak bonding, affectionate constraint and affectionless control) and the optimal bonding group were used as predictor variables in the regression analyses. According to the cut off scores for each bonding category specified by Parker et al (1979), ninety-six children (54 %) reported optimal maternal bonding, and eighty children (45.5 %) reported sub-optimal mother bonding (i.e., affectionate constraint (n=32), affectionless control (n=28) and absent bonding (n=20). For paternal bonding, ninety-two children (52.9 %) reported optimal paternal bonding and 82 (47.1 %) of children reported sub-optimal paternal bonding (i.e., affectionate constraint, n=37, affectionless control, n=33 and absent bonding, n=12). The “dummy coding” procedure outlined in Field (2009) was used to represent the four bonding categories as predictors in the regression analyses. Simply put, dummy coding is a way of representing the offspring in the four bonding categories in a decision response matrix by using only 0s and 1s.

### **Does sub-optimal bonding effect facial affect recognition?**

When the overall sample was considered, sub-optimal maternal bonding did not predict facial affect recognition errors for any of the facial emotions. Similarly, sub-optimal paternal bonding did not predict facial affect recognition errors for any of the facial emotions.

### **Does risk group moderate the relationship between sub-optimal parental bonding and facial affect recognition?**

Risk group was entered as a moderator in the multiple regression analysis between the maternal and paternal bonding categories and facial affect recognition. Risk group was not a

significant moderator in the relationship between maternal or paternal bonding categories and facial affect recognition errors for any of the face emotions.

### **Does gender interact with risk group to moderate the relationship between sub-optimal parental bonding and facial affect recognition?**

Similar to the third hypothesis of this study, I explored whether negative parental bonding would result in higher facial affect recognition errors for girls versus boys and that this would be more evident in high risk offspring, I looked at the parental bonding categories as predictors in the regression model. Child gender and risk group were entered in the regression model as moderators and the three-way interaction term between parental bonding categories, risk group and gender, was entered in the third step of the regression analysis.

The interaction between child gender and risk group was not found to moderate the relationship between maternal bonding categories and recognition of faces denoting happiness, anger, fear and neutral. For the face denoting sadness, the results were similar to the ones reported when testing hypothesis 3. Specifically, it was found that irrespective of perceived maternal bonding categories, gender interacted with risk group for the sad face,  $R^2 = .08$ ,  $F(6, 169) = 2.46$ ,  $p = .026$ ,  $b = -.88$ ,  $t = -2.55$ ,  $p = .012$ . That is, low-risk males made more errors in recognizing sad affect than high-risk males, whereas high-risk females made more errors recognizing sad affect than low-risk females.

Most interestingly, when maternal bonding categories were used to predict recognition error rates for surprise, a significant three-way interaction (maternal PBI categories by gender by risk group) emerged. The F test for the overall model was not significant  $R^2 = .059$ ,  $F(7, 169) = 1.51$ ,  $p = .164$ , but when gender and risk group were included as moderators, the interaction was significant,  $b = 1.26$ ,  $t = 2.83$ ,  $p = .005$ . The interaction plot shows that high-risk males who reported negative maternal bonding, made more recognition errors for surprise compared to high risk males who reported optimal maternal bonding (see Figure 6).

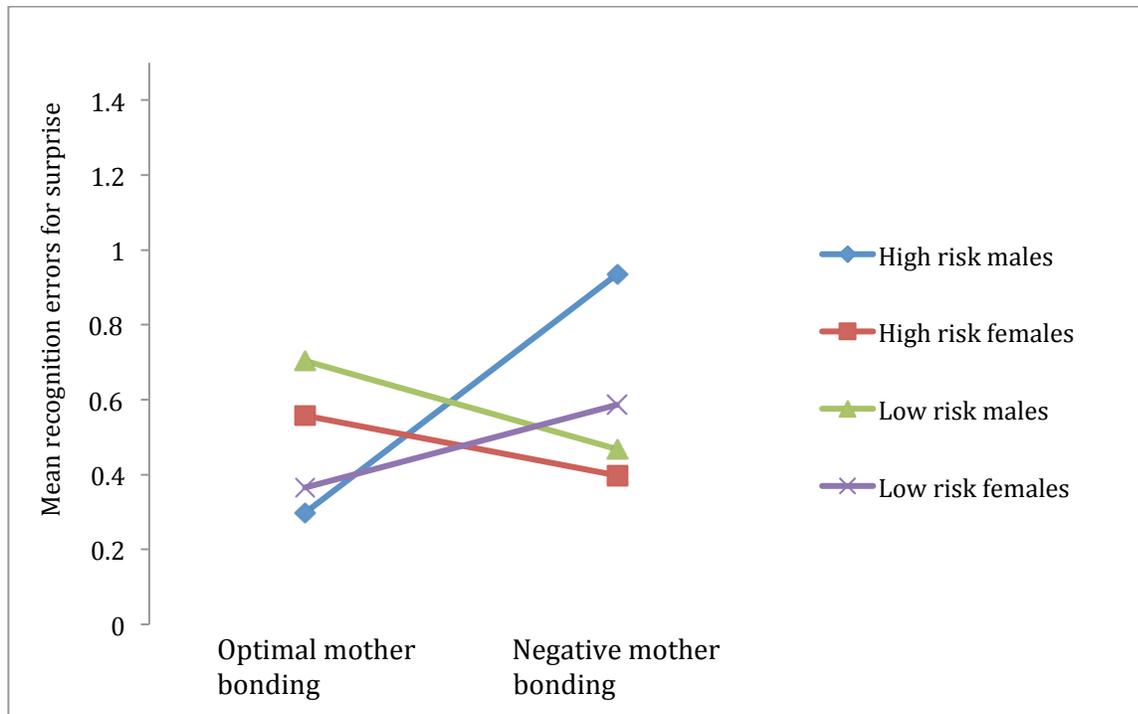
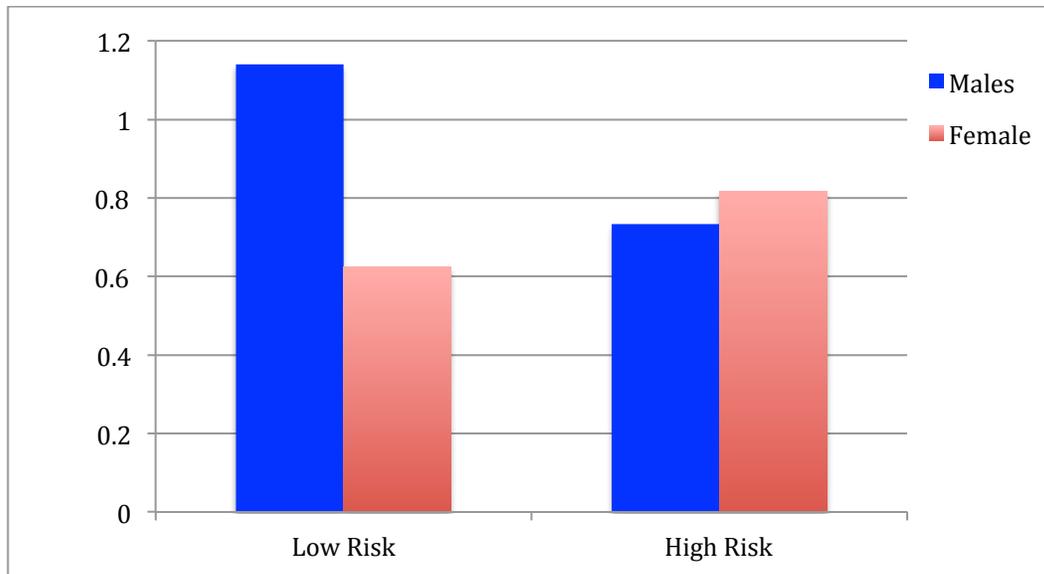


Figure 6. Three-way interaction between maternal bonding categories, risk group and gender for the face denoting surprise.

When paternal bonding categories were entered as predictors in the model, the interaction between child gender and risk group was not significant for the faces denoting happiness, sadness, surprise, fear, and neutral. For the angry face, gender interacted with risk group regardless of perceived paternal bonding. The F test for the overall model was not significant  $R^2 = .049$ ,  $F(7, 169) = 1.38$ ,  $p = .226$ , but the t-test of the interaction between gender and risk group was significant,  $b = -.526$ ,  $t = -2.30$ ,  $p = .023$  (see Figure 7). This finding replicates the result obtained when testing the second hypothesis, revealing that low risk males made more errors when recognizing anger, compared to low risk females and overall higher errors compared to high-risk males and females.



*Figure 7.* Recognition errors for the face denoting anger, as predicted by gender and risk group

To summarize, these exploratory analyses revealed that high-risk males who reported negative maternal bonding made more recognition errors for surprise compared to high risk males that reported optimal maternal bonding. This three-way interaction between maternal bonding categories, gender and risk group represents a unique finding in the literature as it is the first time that sub-optimal maternal bonding as a risk factor for anxiety is shown to interact with gender and risk group in predicting deficits in facial affect recognition. These exploratory analyses also replicated the findings when testing the second hypothesis of the study, with low risk males making more errors when recognizing sad affect compared to high-risk males and high-risk females making more errors compared to low-risk females.

### **Exploratory analyses: Parental bonding and anxiety ratings for facial affect**

A series of exploratory regression analyses were conducted to examine whether parental bonding predicted state anxiety while viewing facial affect. State anxiety was rated on a 1 (not at all) to 5 (extremely) scale, and was measured during the last block of face presentations. In the case of significant associations, mediation analysis was performed to examine whether child temperament mediated the relationship between indices of parental bonding and state anxiety. Additionally, moderation analysis was conducted to examine whether child gender moderated the relationship between parental bonding and anxiety while viewing facial affect.

**Does parental bonding influence anxiety ratings for facial affect?**

The results of the multiple regression analyses revealed that maternal care and maternal overprotection were not significant predictors of anxiety ratings for anger, sad, fear, and neutral. However, maternal overprotection predicted anxiety ratings for surprise and happy, in that children who perceived their mothers to be overprotective reported higher levels of anxiety when seeing the surprised face,  $R^2 = .046$ ,  $F(1, 168) = 1.63$ ,  $p = .15$ ,  $b = 0.18$ ,  $t = 2.48$ ,  $p = .014$  and the happy face,  $R^2 = .056$ ,  $F(1, 168) = 1.99$ ,  $p = .08$ ,  $b = 0.18$ ,  $t = 2.38$ ,  $p = .018$ . Risk group did not moderate the relationship between maternal bonding and anxiety ratings. Paternal care and overprotection were not significant predictors of anxiety ratings for any of the facial emotions.

**Does child temperament mediate the relationship between parental bonding and anxiety ratings for facial affect?**

The three-step mediation procedure by Baron and Kenny (1983) was used to examine whether child temperament mediated the relationship between maternal overprotection and level of anxiety while viewing the facial emotions denoting surprise and happy. A visual representation of the mediation models for each of these emotions is provided in Figure 8 and Figure 9. A bootstrapping resampling procedure that is recommended for mediation analyses was used (Preacher & Hayes, 2014). The bootstrapping procedure was conducted with an SPSS macro (PROCESS, Preacher & Hays, 2008), which is proposed to overcome violations of normality of the sampling distribution of the indirect effects that are tested by mediation. The bootstrapping procedure treats the study sample as if it were a population, by drawing 1000 random samples and computing the indirect effect ( $ab$ ) for each sample. After doing this for 1000 samples, the mean  $ab$  effect over 1000 randomly drawn samples is given as the point estimate for our mediation model. The confidence intervals for a 95 % significance range are computed for each path estimation and if they do not contain zero, the effect is significant.

The linear regressions revealed that maternal overprotection predicted child behavioural inhibition (paths a). Moreover, behavioural inhibition predicted increased anxiety ratings when viewing the faces denoting surprise and happy (paths b). Maternal over-protectiveness predicted increased anxiety ratings when viewing the surprised and happy faces, even when behavioural inhibition was entered in the model (paths c'). Results revealed a partial mediation effect of behavioural inhibition in the relationship between maternal overprotection and anxiety ratings for surprise, which suggests that increased anxiety ratings while viewing the face denoting

surprise are partially due to higher scores on behavioural inhibition. The results of the second mediation revealed a full mediation effect for anxiety ratings for the happy face. This suggests that higher anxiety ratings while viewing the happy face are fully accounted for by the increased behavioural inhibition scores of offspring. Behavioural inhibition as a mediator could account for roughly 20 % of the total effect for anxiety ratings for surprise ( $P_m=.20$ ) and 22 % of the total effect for the anxiety ratings for happy ( $P_m=.22$ ).

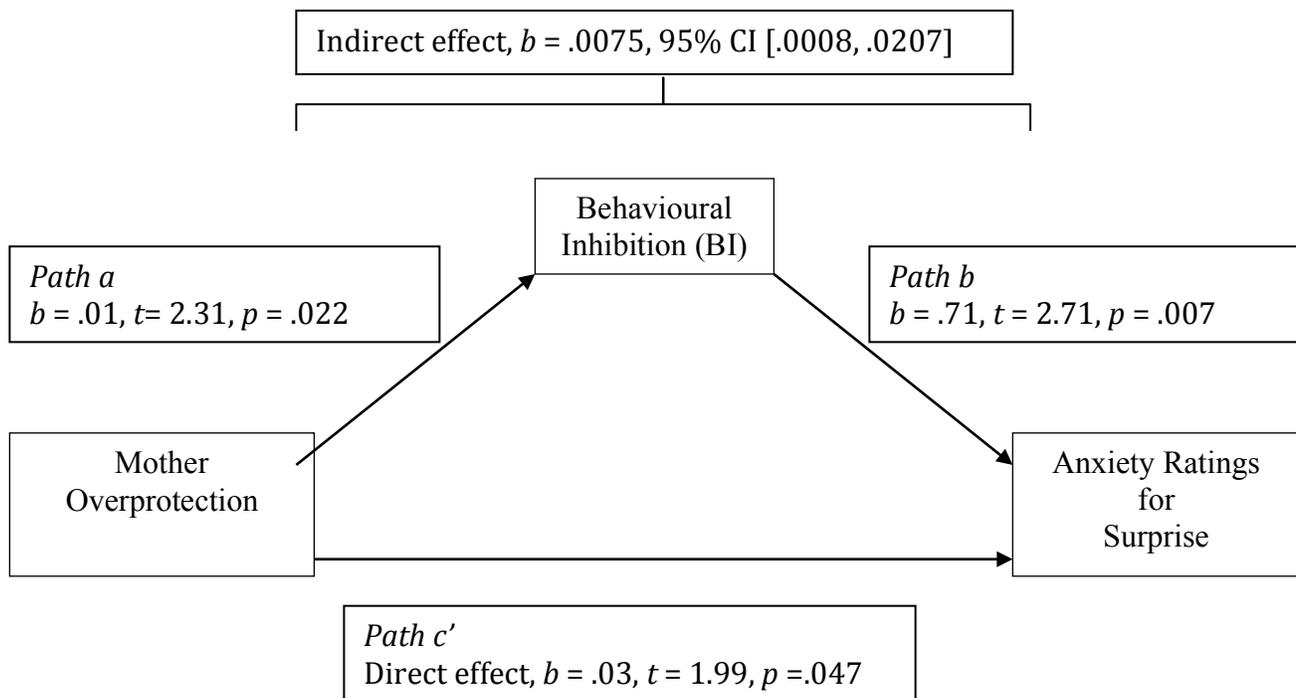


Figure 8. Mediation model of mother overprotection as a predictor of anxiety ratings for the face denoting surprise, mediated by behavioural inhibition

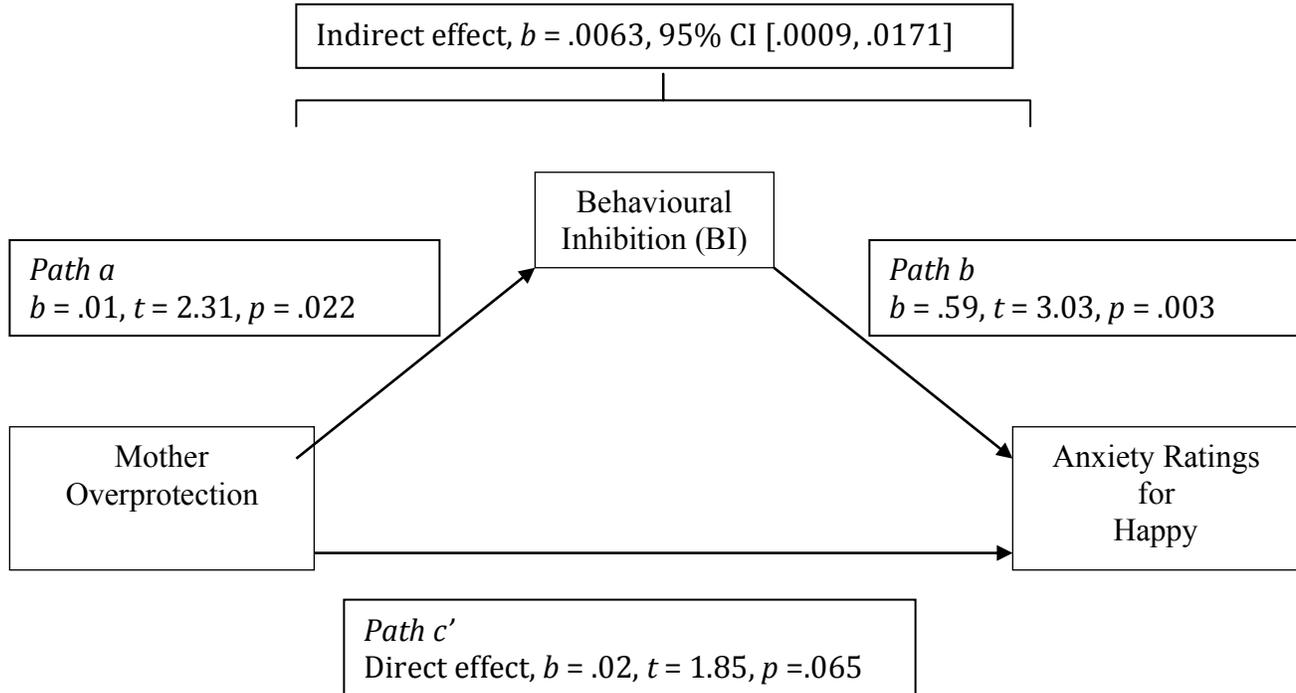


Figure 9. Mediation model of mother overprotection as a predictor of anxiety ratings for the face denoting happy, mediated by behavioural inhibition

### Does gender moderate the relationship between parental bonding and anxiety ratings?

To test the hypothesis that child gender moderates the relationship between parental bonding and anxiety ratings, separate multiple regression analyses were conducted with the maternal and paternal PBI scales as predictor variables. The exploratory hypothesis predicted that perceived parental lack of care and overprotection would result in higher anxiety ratings for girls versus boys and this would be more evident in high-risk offspring. After inserting gender as a moderator in the regression model, I also looked at the interaction between gender and risk group in the relationship between parental bonding and anxiety ratings. Results showed that child gender did not moderate the relationship between maternal bonding and paternal bonding and anxiety ratings for any of the facial affect.

## Discussion

Deficits in recognizing face emotions have been identified as possible social cognitive risk markers for the development of pathological anxiety (Bilodeau et al., 2014; Collin et al., 2013). However, little is known about the factors that might contribute to impaired facial affect recognition. The primary objective of this study was to examine whether perceived parental bonding influences the ability of psychiatrically healthy youth to identify facial affect. Specifically, the study sought to determine whether negative parenting predicted deficits in recognizing faces with emotional expressions and neutral faces. The study also examined whether child gender and parental history of anxiety moderated the relationship between perceived parental bonding and facial affect recognition, and whether behavioural inhibition mediated this relationship.

### **Does parental bonding influence facial affect recognition?**

The hypothesis that perceived negative parenting would predict deficits in facial affect recognition in psychiatrically healthy children and adolescents was not supported. When the overall sample was considered, perceived maternal and paternal care and overprotection did not emerge as significant predictors for any of the face emotions or the neutral face. Additionally, exploratory analyses of parental bonding categories failed to detect differences in facial affect recognition between optimal parental bonding and sub-optimal bonding for any of the faces. The lack of predictive value of parental bonding on face emotion processing in our sample of offspring is surprising considering the proposed importance of family factors, especially parent-child bonds, in the development of social cognition (Vrticka & Vuilleumier, 2012).

Our results are not in concordance with other studies that have found an association between parental bonding and the ability to recognize facial affect. Zheng et al. (2011) found that maternal denial of autonomy predicted low accuracy of recognizing fearful faces whereas maternal care predicted higher accuracy in recognizing sad faces. It should be noted that unlike the present study, Zheng et al. (2011) studied adults who retrospectively assessed parental bonding during childhood, which is known to be prone to bias. Procedural differences in the affect recognition task between their study and the current study might also account for the different results.

Pollak, Cicchetti, Hornug, and Reed (2000) studied the effects of parental neglect or abuse on face emotion processing in children. They found that neglected children displayed higher inaccuracies when discriminating between different facial emotions compared to controls or physically abused children. Furthermore, they reported that neglected children made fewer distinctions between angry, sad, and fearful facial expressions, whereas physically abused children made more errors recognizing facial expressions of sadness and disgust, but not anger. While this study provides support for the effect of aberrant parental emotional environments and their effect on children's social cognitive development, it looked at child populations that are subjected to extremely negative familial environments. Furthermore, they used a mood induction technique before the face recognition task, and they matched the gender of the presented face with the child's gender. Given the methodological differences between their study and the current study, in addition to the different age cohorts and participant characteristics, it remains to be seen whether their findings would generalize in non-abusive family environments where children might be subjected to varying degrees of negative parental bonding.

Negative parenting and maternal depression have also been found to predict emotion recognition skills in a sample of psychiatrically healthy pre-school children (Kujawa, Dougherty, Durbin, Laptok, Torpey, & Klein, 2014). Results indicated that children were most accurate in identifying happy facial expressions and least accurate when identifying neutral facial expressions. Furthermore, parents' hostility and intrusiveness toward their children impacted their children's performance on emotion listening and emotion labelling tasks. While maternal depression by itself did not predict children's emotional understanding, the interaction between maternal depression and negative parenting led to lower emotion recognition composite scores. While Kujawa et al.'s (2014) study sheds light in understanding the development of emotion knowledge in childhood, their cohort of pre-school children with maternal depression and use of emotion knowledge tasks (i.e., auditory and visual) make it difficult to generalize their findings in helping explain the non-significant results of the current study. Future research is needed to determine whether similar impaired recognition knowledge exists in a cohort of pre-school children with parental anxiety.

Positive parenting has been linked to children's emotional knowledge, (Bennett, Bendersky, & Lewis, 2005) and emotion understanding (Alegre & Benson, 2010; Denham, Zoller, & Couchoud, 1994; Denham et al., 2003; Dunn & Brown, 1994). Bennett et al. (2005)

looked at the relationship between maternal characteristics (i.e., parenting style, verbal knowledge and depressive symptoms) and children's emotion knowledge. They found that maternal intelligence was positively correlated with maternal warmth and both of these features predicted children's emotional knowledge. It was proposed that mothers higher in care and verbal intelligence are also more likely to engage in more responsive parenting and as a result, provide greater emotion learning opportunities for their children. Furthermore, emotional knowledge and emotion recognition skills have been found to mediate the relationship between children's cognitive abilities, academic competence and social well-adjustment (Izard et al., 2001). While these studies provide support for the relationship between parental practices and emotional development and enhanced cognitions in children, some of these studies have used facial affect labelling (Bennett et al., 2005) in specific child cohorts (e.g., 4-year old African American children), while others have used self-report emotional competence in adolescents (Alegre & Benson, 2010), observational indices of emotion knowledge related to social competence (Denham et al., 2003) and observations of emotional knowledge at home in longitudinal studies (Dunn & Brown, 1994). Although the present study did not find associations between parental bonding and facial affect recognition, future examinations of this relationship are warranted in light of the reported links between parenting and other facets of emotion knowledge and understanding.

Attachment patterns and how they influence emotional and cognitive development in healthy populations provide insights into the importance of positive models of emotional balance provided by parents and how they might shape emotional knowledge and cognitive development in children (Vrticka & Vuilleumier, 2012). Mother-infant attachment assessed when the child was one year old significantly predicted the child's performance in a task assessing emotion understanding in various social and emotional dilemmas (Steele, Steele, Croft, & Fonagy, 1999). In healthy adults, anxious attachment predicted a heightened state of vigilance for facial affect and this led to poorer accuracy in making judgments about the different facial affect (Fraleigh, Niedenthal, Marks, Brumbaugh, & Vicary, 2006). Anxiously attached adults have also been found to be quicker in recognizing sad and happy faces compared to securely attached adults (Niedenthal, Brauer, Robin, & Innes-Ker, 2002). Other studies have found support for the relationship between attachment patterns, emotional processing and recognition biases in healthy adults (Suslow, Dannlowski, Arolt, & Ohrmann, 2010) and adolescents (Escobar et al., 2013).

**Does child gender moderate the relationship between parental bonding and facial affect recognition?**

The hypothesis that gender would moderate the relationship between parental bonding and facial affect recognition was partially supported. We found that boys who perceived their fathers to be high in overprotection made more errors when recognizing the fearful face compared to boys who perceived their fathers to be low in overprotection. This significant finding goes counter to the working hypothesis, which was based on previous research showing that females show more attentional biases toward threatening stimuli (Lee et al., 2014; Wieckowski et al., 2016) and are at greater risks for developing anxiety disorders compared to males (Kessler et al., 1994; Seeman, 1997). Despite our findings being in the opposite predicted direction, these results are, to our knowledge, the first in the literature that suggest a pathway in which negative bonding in the form of high paternal overprotection predicts facial affect recognition in males, thus pointing to a possible cognitive marker of developing anxiety disorders in males.

Parental overprotection is considered to be the more salient component of parental bonding as it is quantified by restrictive behaviours from the parent toward the child and intrusive actions from the parents, both of which can result in dependency of the child on the parent (Parker, et al. 1979). Parental overprotection signals to the child that the environment is unsafe, new situations could harbour dangers and ultimately, these learned expectations might generalize to situations that realistically would not pose any danger to the child. This can result in loss of self-efficacy and restrictive freedom (Rubin, Burgess, & Hastings, 2002). With time, the cumulative effects of overprotectiveness have been linked to poor adjustment in adulthood (Silove, Parker, Hadzi-Pavlovic, Manicavasagar, & Blaszczynski, 1991), as well as negative psychosocial and psychopathological outcomes (Parker, 1983). Our results show that father overprotection could indicate specific gender based vulnerabilities for boys compared to girls.

Fathers might be seen as more appropriate behavioural models for boys and our results suggest that paternal overprotection might differentially shape cognitive structures involved in facial affect recognition in boys. Research supports the assertion that boys craft their identity and model their behaviour after their fathers (Ellestad & Stets, 1998). It is possible that males are likely to display behavioural processes, such as role-taking, and engage in behaviours that the father directly or indirectly might portray (Burke, 1991). Paternal overprotection could

differentially impair cognitive mechanisms that process fear for boys and not girls, and in the current study they were manifested in recognition errors for fear.

The fearful response in particular, has been proposed to underlie automatic processes that involve neural structures sensitive to threat signals in the environment, and these threat relevant stimuli can trigger anxiety responses subconsciously at first, and with repeated exposures, as part of conscious perception (LeDoux, Ciocchetti, Xagoraris, & Romanski, 1990). Also, vicarious learning has been found to underlie the fear response in children, as a study by Askew and Field (2007) showed that children who viewed pairing of pictures of animals with fearful or happy faces were more likely to self-report fearful or happy beliefs depending on the exposure pairing of the animals with the emotive stimuli. Furthermore, children who had previously seen an animal picture being paired with a fearful face were also slower to approach the same animal. The authors argue that their findings support previous models of fear acquired through observational or vicarious learning (Bandura, 1969) and not conditioning. The vicarious learning of fear was demonstrated in self-report measures, affective priming tasks as well as in forms of behavioural avoidance toward the previously primed fearful stimulus and the fearful response persisted up to three months post exposure (Askew & Field, 2007).

The current study also explored whether the four parental bonding categories proposed by Parker et al. (1979) interacted with behavioural inhibition, risk group and gender in predicting facial affect recognition errors. Results revealed a three-way interaction between maternal bonding, risk group and gender in predicting recognition errors for the face denoting surprise. Specifically, high-risk males who reported negative maternal bonding made more errors when recognizing expressions of surprise compared to high-risk males who reported optimal maternal bonding. These results are the first in the literature that suggest a pathway in which negative maternal bonding might predispose high-risk male offspring toward greater vulnerability in developing anxiety, as manifested in cognitive impairment for affective faces.

Surprise is the only primary emotion to have an ambiguous valence (i.e., one can be positively or negatively surprised). Tottenham et al. (2013) proposed that there are individual differences in how ambiguous stimuli are processed and children in particular, tend to engage in negative interpretations of ambiguous stimuli more than adults. They examined these biases by looking at interpretations of facial expressions conveying surprise and a neutral expression, and they found that surprised faces were perceived as conveying more negative affect. Neta, Davis

and Whalen (2011) also report a negativity bias for surprised faces in healthy adults, and this effect was much more pronounced when the surprised face was shown within a negative context. We cannot know whether our high-risk males who reported suboptimal maternal bonding processed the surprised face through a negativity bias and in light of past research findings (Neta, Davis, & Whalen, 2011; Tottenham et al., 2013) future research could incorporate the effect of parental psychopathology and gender when looking at the cognitive interpretation of ambiguous facial emotions.

### **Does child temperament mediate the relationship between parental bonding and facial affect recognition?**

Our results did not support the hypothesis that behavioural inhibition mediates the relationship between parental bonding and facial affect recognition. These results are surprising considering that many neuroimaging studies report differences in activation levels in the brain in response to facial stimuli as a result of different levels of behavioural inhibition. Differences in amygdala activation in response to fearful facial expressions were found by Clauss and Blackford (2012) in healthy adults with high and low levels of behavioural inhibition. Participants viewed fearful faces that were either novel or new, and activation patterns of the amygdala were recorded and compared when the fearful face was expected compared to when it was not. They found that participants with inhibited temperament showed greater amygdala response when they expected to see the fearful face compared to those with uninhibited temperament whose amygdala activation was stronger when the fearful face was not expected. Other studies (Lang, Bradley, & Cuthbert, 1990) have also found that expectancies in the environment might modulate the physiological reaction that behaviourally inhibited people exhibit following exposure to aversive stimuli. That is, if a behaviourally inhibited person expects an aversive stimulus (i.e., fearful face), they will show hypervigilance and an increased ability to detect such stimulus. This might point to a possible pathway in which an emotionally aversive environment (i.e., suboptimal parental bonding) might interact with behavioural inhibition in shaping social behaviour and increasing the risk for developing anxiety.

Blackford, Avery, Shelton and Zald, (2009) looked at reaction times and amygdala responses to facial pictures in participants with very high and very low levels of behavioural inhibition. They found that compared to uninhibited participants, those with inhibited temperament had faster reaction times and greater amygdala responses toward novel faces

compared to neutral familiar ones, and they attributed this to behavioural avoidance and wariness to novel facial stimuli of those with behavioural inhibition. Others have found increased attention biases toward angry facial displays in behaviourally inhibited adolescents (Pérez-Edgar et al., 2010) and behaviourally inhibited children (Pérez-Edgar et al., 2011). Pérez-Edgar et al. (2010) found that adolescents who were behaviourally inhibited as young children showed high levels of attention bias to threatening faces compared to control children, who displayed a similar bias toward happy faces. In their study, early childhood behavioural inhibition was linked to adolescent social withdrawal but only among adolescents who displayed attentional biases to threatening stimuli. Schwartz, Wright, Shin, Kagan and Rauch (2003) looked at amygdala response to novel faces in healthy adults who had been described as being inhibited at 2 years of age. Compared to uninhibited controls, the inhibited temperamental group showed significant amygdala reactivity when exposed to novel facial stimuli (Schwartz, Wright, Shin, Kagan, & Rauch, 2003).

Taken together, these findings point to neural circuits that are specific to behavioural inhibition and could account for the higher anxiety risk in behaviourally inhibited individuals. While the current study did not find that behavioural inhibition mediates the relationship between parental bonding and facial affect recognition, the neuroimaging studies that have found implicit or automatic face processing biases in these populations point to a possible mediating effect of inhibited temperament between parental bonding and implicit face processing biases (i.e., hypervigilance to threatening faces). That is, it is possible that behavioural inhibition as a temperamental risk factor for anxiety development might not be manifested in affect recognition biases per se, but possibly in attentional biases to threat or in other types of anxiety-related interference (Pérez-Edgar et al., 2010).

Another difference between the current study and neuroimaging research are the study populations and the outcome measures of behavioural inhibition. In the current study we used a measure which captured current self-report behavioural inhibition in children, whereas most neuroimaging studies have either used retrospective reports of behavioural inhibition in adults (Clauss & Blackford, 2012) or have used samples of adolescents that have included those with concurrent psychiatric disorders (Pérez-Edgar et al., 2010). Measuring behavioural inhibition in adult populations not only captures memory biases, but might also indicate a self-selection of adults whose behavioural inhibition levels have shown continuity since childhood. Furthermore,

examining the continuity of behavioural inhibition in participants with diagnosed psychopathology might confound the results in ways that make it challenging for us to generalize in psychiatrically healthy populations.

Research shows that there is a discontinuity of behavioural inhibition, with some studies reporting that as high as one third of behaviourally inhibited toddlers were less inhibited in childhood (Kagan, Reznick, & Snidman, 1988; Kagan, Reznick, Snidman, Gibbons, & Johnson, 1988) and almost half of inhibited children show discontinuity in behavioural inhibition across childhood and adolescence (Kerr, Lambert, Stattin, & Klackenberg-Larsson, 1994; Sanson, Pedlow, & Cann, 1996). It is possible that the discontinuity of behavioural inhibition as a temperamental risk factor for anxiety development might pose a methodological challenge in synthesizing findings from behaviourally inhibited adults and comparing them to findings from behaviourally inhibited children.

### **Does risk group moderate the relationship between parental bonding and facial affect recognition?**

This study found some evidence that risk group moderates the relationship between parental bonding and facial affect recognition. In particular, paternal care interacted with risk group in predicting recognition errors for the angry face. Specifically, high-risk offspring who reported low paternal care made more errors recognizing angry faces compared to low risk offspring who perceived their fathers to be low in care. Research indicates that the lack of paternal care in a high-risk environment places children at a disadvantage for emotional competence (Allen & Daly, 2007; Rohner & Veneziano, 2001). We cannot know whether the deficits in recognizing angry faces in the current study represent a social cognitive system that is hypersensitive to threat stimuli, or whether it is a manifestation of learned patterns of avoidance toward threatening stimuli as part of coping strategies. In either case, our findings are the first in the literature that show such biases in high-risk children as a variation of perceived paternal behaviour.

Neuroimaging research might provide another explanation for the high error rates for recognizing anger displayed by high-risk children who perceived their fathers to be low in care. It is possible that these recognition deficits might point to similar dysregulation patterns of neural circuits that have already been implicated in the pathophysiology of anxiety (Adolphs, 2002b). These neural pathways, which include the amygdala and other affective processing regions, have

been shown to be highly activated when adult patients with anxiety view negative facial expressions, such as anger and fear (Chechko et al., 2009; Evans et al., 2008). Also, a study by O'Toole et al. (2014) found larger N170 amplitudes in anxious children. Specifically, they reported that high anxiety at age 5 was associated with high anxiety at age 7, but only in those children whose neurological indices of anxiety, in the form of N170 amplitudes, were larger when exposed to angry compared to happy faces (O'Toole, Decicco, Berthod, & Dennis, 2013). Similar patterns have been reported in neuroimaging studies with anxious youth (Thomas, Drevets, Dahl, Ryan, Birmaher, et al., 2001) and the current findings might indicate that this neural dysregulation might start earlier in high-risk populations that are exposed to negative parental practices (i.e., low care).

Genetic studies that have looked at perceived parenting and its interaction with the serotonin transporter gene (5-HTTLPR) in predicting facial affect recognition report that perceived parenting may directly affect neural imaging as indicated by either right or left hemispherical activation (Nishikawa, Toshima, & Kobayashi, 2015). Studies indicate that individuals with the short allele of the 5-HTTLPR gene are at higher risk for depression (Caspi et al., 2003), show higher amygdala reactivity when exposed to negative stimuli (Schardt et al., 2010), and demonstrate lower coping potential (Szily, Bowen, Unoka, Simon, & Kéri, 2008), all of which might contribute to higher social anxiety symptoms. Nishikawa et al. (2015) proposed that the 5-HTTLPR gene directly influences the neural systems implicated in facial recognition and that this effect would be mediated by the perceived family environment. Findings showed that those with the long allele of the 5-HTTLPR gene who reported early negative parenting showed different neural activation patterns when evaluating ambiguous stimuli compared to those with the short allele. They also found that maternal rejection predicted right frontal activation during an ambiguous task, such that those who reported greater maternal rejection showed less right frontal activation compared to those who reported lower maternal rejection. The authors concluded that these preliminary findings add to the literature by showing that early perceived parenting style influences neural activity for individuals with certain genotypes when rating ambiguous faces (Nishikawa et al., 2015). In light of neuroimaging and genetic studies pointing to the combined effects of genes and environment on neural functioning, future research could explore the possibility of heightened neural sensitivity toward threat related faces in high-

risk children and whether these neural sensitivities are ameliorated or worsened by different environmental factors, such as parental bonding.

In addition to angry faces, this study revealed that low risk offspring who perceived their mothers to be high in care made more errors recognizing the neutral face than low risk offspring who perceived their mothers to be low in care. Although unexpected and not predicted by the hypothesis, this is the first finding in the literature that has found an interaction between an optimal parental bonding facet (i.e., high care) and risk group in predicting facial recognition errors for neutral faces. It is important to note that the present study examined face recognition accuracy and not face recognition bias. Thus, it is unknown if the neutral face was misrecognized as a negative (e.g., fear) or a positive (e.g., happy) valenced emotion. This is an important question to address, as a tendency to misattribute neutral faces as negative or positive will have different effects on social functioning (Bordon, Rourke, & Hutton, 2017). Findings from the literature suggest that anxious adults and children tend to show a negative bias when identifying neutral facial affect (Bell et al., 2011; Demenescu et al., 2010; Melfsen & Florin, 2002). We could extrapolate, that in the absence of a genetic risk for anxiety (i.e., parental anxiety) and in the presence of an optimal bonding environment (i.e., high maternal care), our low risk and high maternal care offspring might have shown a positive bias when misrecognizing neutral faces. Admittedly, this is extrapolation needs to be confirmed by future research by examining not only recognition accuracy in facial affect, but recognition bias as well.

### **Parental bonding, behavioural inhibition and anxiety ratings**

The current study explored whether differences in participants' ratings of anxiety while viewing the face emotions was influenced by parental bonding and behaviour inhibition. Results revealed that maternal overprotection predicted higher levels of anxiety while viewing faces denoting surprise and happiness. Furthermore, it was found that behavioural inhibition mediated this relationship. Among the children who reported high maternal overprotection, high anxiety ratings while viewing the surprised face were partially mediated by high scores on behaviour inhibition. To our knowledge, this is the first finding in the literature to reveal a pathway in which behaviour inhibition interacts with maternal overprotection in predicting anxiety ratings after a face recognition task in healthy offspring. Observational studies of mother-child interactions show that child temperament influences parenting style in children with anxiety

disorders (Chorpita & Barlow, 1998; Hudson, & Rapee, 2002; Rapee, 1997), and the present findings might elucidate a possible mechanism for the development and maintenance of anxiety.

It follows that increased levels of anxiety of offspring reporting high maternal overprotection might come as a result of mothers reacting to the temperament of their children. Weisz and Stipeck (1982) propose that parental overprotection can lead to the perception of an unsafe environment and the expectation that the perceived negative outcome in the environment (i.e., probability of danger) can be reduced by altering one's own behaviour (i.e., avoidance). A history of lack of control contributes to psychological vulnerability (Overbeek, ren Have, Vollebergh, & de Graaf, 2007), low self esteem (Demo, Small, & Savin-Williams, 1987) and may increase anxiety risk. Studies show that low self-efficacy in adolescent populations was directly related to high levels of parental control and increased anxiety symptoms (Bouton, Mineka, & Barlow, 2001).

Behavioural inhibition was found to fully mediate the relationship between maternal overprotection and offspring's anxiety ratings after watching the happy face. At first glance, this finding is counterintuitive, as positive affect stimuli are not predicted to elicit heightened anxiety responses as a result of temperamental and environmental risk factors. However, a few studies report significant recognition advantages for happy faces in people with low versus high social anxiety (Silvia, Allan, Beauchamp, Maschauer, & Workman, 2006). Among individuals with social anxiety, successful dyadic social interactions were found to produce negative responses (Wallace & Alden, 1997) and approachability ratings of emotion faces were lower for happy faces (Campbell et al. 2009). Prospective studies with adolescents (Oldehinkel, Hartman, Van Oort, & Nederhof, 2015) report that perceived parental rejection predicted high recognition specialization for positive emotions only in adolescents who also had a high probability of developing an anxiety disorder. They suggested that the relationship between parental styles and probability in developing anxiety disorders might be mediated by facial emotion specialization (Oldehinkel et al., 2015).

In the current sample, the higher anxiety ratings following exposure to the happy face might be explained by a negative interpretation bias of positive social events that have been reported by socially anxious individuals (Vassilopoulos, 2006). In their study, they presented individuals high and low in social anxiety with either positive or mildly negative social events and assessed their interpretations of such events. It was found that high socially anxious

individuals were more likely to negatively interpret the positive social events and to catastrophize the mildly negative events. Furthermore, socially anxious individuals underestimated the probability that positive social events could happen to them, compared to those low in social anxiety (Vassilopoulos, 2006). It is possible that the behaviourally inhibited offspring in our study who reported higher maternal overprotection might have engaged in similar interpretation biases of the happy faces. It has been suggested that happy faces might signal social dominance (Heuer, Rinck, & Becker, 2007) and expectations of reciprocity (Schofield, Coles, & Gibb, 2007) both of which potentially increasing avoidant behaviour in order to avoid potential rejection and expected negative evaluations.

### **Methodological limitations**

The present study has a few limitations, which merit the consideration and attention of future research. First, the outcome variables in the present study only looked at recognition accuracy of facial affect as a cognitive risk factor. While this methodological choice was based on previous studies pointing to recognition rates as reliable cognitive markers, such variables (i.e., correct vs. error) are not as sensitive in capturing subtle cognitive biases as are reaction time variables. It is possible that children's biases when recognizing facial affect might also be manifested in time spent deliberating on a decision as well as the decision itself. Future research could expand on our methodology and combine recognition accuracy measures in addition to measuring reaction times.

Second, while our face recognition task has been reliably used and validated in the literature, the face emotions did not convey variability of emotion intensity, which could better capture affect recognition biases. Future research might consider using facial affect of differing affect intensity and morphed facial expressions in order to capture not only differences in recognition error rates but also capture recognition sensitivity thresholds within subjects.

Third, we did not assess children's state anxiety in our study. As previous research has found that state emotions influence attention in mood-congruent ways, it would be useful to assess whether state anxiety had any impact on children's recognition biases either by itself or by interacting with perceived parental bonding, behavioural inhibition or both. Future research should incorporate state measures of anxiety and assess whether they influence cognitive biases directly or through interacting with other risk factors.

Because we measured parental bonding from the offspring's perspective only, future studies should also measure this construct from other perspectives (e.g., parental reports or direct observation). The triangulation of parental bonding measures will provide a comparison framework in evaluating children's perceptions of parental behaviours and whether they are similar to those reported by parents or observed by others. We would then be able to assess other pathways in which parental bonding might affect facial affect recognition and other cognitive markers of anxiety.

### **Implications and future directions**

Although our findings need to be replicated, they have theoretical, pedagogical, and clinical implications, the combination of which would not only increase our knowledge of risk markers for anxiety, but hopefully expand on the psycho-educational programs, interventions and treatment options that are currently used. Specifically, as our understanding of risk markers for anxiety disorders increases by studying at-risk populations, researchers can transfer that knowledge in psycho-educational and pedagogical settings. This knowledge would be especially important today, where the current societal and parenting norms promote overprotective parenting, colloquially known as "helicopter parenting". This type of parenting, described as over-involvement, over-control and comprised of an intensive set of parental behaviours, has been associated with high rates of anxiety and depression in children, perhaps because it is perceived to violate the child's psychological needs of autonomy and competence (Giangreco, Edelman, Luiselli, & MacFarland, 1997). While the negative psychological effects of parental overcontrol in children have been reported in the literature, the present study provides evidence that parental bonding interacts with gender and risk group in predicting deficits in social cognitions. It is possible that parental behaviours in combination with the presence of parental anxiety are initially manifested as subtle cognitive biases and as children further develop, the risk for developing anxiety also increases. Raising awareness, promoting positive parental responding and educating parents on the importance of optimal parental bonding is likely to reduce these cognitive biases.

As our understanding on the risk factors and prognosis of anxiety disorders improves, it is becoming clear that clinicians need to incorporate combined insights from biological, cognitive and environmental factors when developing interventions and treatment options. Specifically, the current research found evidence that the presence of parental anxiety in

combination with environmental factors (i.e., parental control) interacts with unique individual characteristics of the child, such as gender and personality, in the expression of anxiety vulnerability (i.e., impaired facial affect recognition). In clinical settings, this could translate into enhancing the current Family Based Cognitive Behavioural Therapy (FCBT) protocols by incorporating parent training and parental involvement, taking into consideration the child's personality, gender and the presence of parental psychopathology. Currently, these FCBT programs focus on dysfunctional cognitions and how they affect and interact with children's emotions and behaviour, in addition to providing psycho-education, anxiety management and communication skills training for parents (Negreiros & Miller, 2014). Parental control and protection might represent significant roadblocks in the treatment of childhood anxiety as they may result in children's beliefs that they are unable to cope with anxiety-provoking situations. Future enhancements of such programs might need to also focus on specific parental behaviours that enhance child autonomy, foster independence and instil a sense of environmental mastery in the child.

Another area for improved clinical interventions is computerized treatments for children that are already receiving social skills training. Attention Modification Bias programs (Mogg & Bradley, 1998) have been shown to be effective in children with face recognition biases, but these treatments do not deliver personalized interventions based on children's specific impairments. Future research should explore the specific cognitive biases that children at risk for psychological disorders might manifest. The current training programs (e.g., social effectiveness therapy for children, SET-C, Simonian et al., 2001) that target facial affect recognition deficits do not discriminate between specific skill deficits, perhaps because research on identifying such specific facial affect recognition deficits is sparse. In creating such personalized interventions, clinicians need to enhance their assessment of family factors, address specific parental behaviours, and based on the clinical profile of the child, develop the appropriate treatment interventions.

### **Summary**

The main finding of the present study is that child gender moderated the relationship between paternal overprotection and recognition errors for fear, with males who reported higher paternal overprotection making more errors when recognizing fearful facial affect compared to

males who reported low paternal overprotection. Although unexpected and contrary to the hypothesis, this finding is the first in the literature to point to a possible pathway in which parental bonding predicts facial affect recognition as a possible cognitive marker for developing anxiety disorders in males. Another unexpected finding was that high-risk males who reported negative maternal bonding made more recognition errors for faces denoting surprise compared to high-risk males who reported optimal maternal bonding. Considering that surprise is an emotion with ambiguous valence (Tottenham, et al., 2013), we cannot know whether this finding indicates a negative or a positive recognition bias. Nonetheless, our findings point to a particular vulnerability of males with a parental history of anxiety in the form of impaired facial affect recognition. We also found that high-risk offspring who reported low paternal care made more recognition errors for the angry face compared to low-risk offspring who reported low paternal care. This finding is in line with previous studies reporting that children in high risk environments who also report low paternal care are at a disadvantage for developing emotional competence (Rohner & Veneziano, 2001). Finally, we also found that children who were behaviourally inhibited and who reported higher maternal overprotection experienced more anxiety when viewing the surprised and happy faces. Child temperament interacting with parental bonding in predicting anxiety ratings in psychiatrically healthy children is also a novel finding. Taken together, the present research points to novel avenues in exploring the risk factors for developing anxiety disorders. As our findings add to the existing literature, we are hopeful that additional research in combination with clinical interventions will culminate in the creation of better theoretical frameworks in understanding anxiety disorders and the creation of more effective treatments.

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