Attention bias to threat in mothers with emotional disorders predicts increased offspring
anxiety symptoms: A joint cross-sectional and longitudinal analysis

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A report submitted as a partial requirement for the Masters of Clinical Psychology degree,
Griffith University, Mount Gravatt Campus

October, 2015

Word Count: 13, 336

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Thesis Abstract

Depression and anxiety are highly prevalent and disabling emotional disorders. While a number of risk factors have been identified, the exposure of children to their parent's cognitive biases is a possible mechanism through which risk can be conferred. Attentional biases towards negative information are one such cognitive mechanism which has been well-established as distinct in individuals with mood and anxiety disorders. Perhaps then, the increased risk of experiencing anxiety and depression in the offspring of parents with a history of emotional disorders may be attributed to the transmission of such dysfunctional attentional biases from parents to their children. As such, children of parents with emotional disorders may be "cognitive vulnerable" to developing mood and anxiety disorders throughout their lifetime.

This study aims to investigate the relationship between the attentional biases of mothers with and without a history of emotional disorders, and the attentional biases of their non-disordered children, both across time and between the two distinct groups. Further, the study aims to investigate the relationship between both maternal and child attentional biases, and the development of anxious symptomology across 12 months in children at "high risk" or "low risk" for developing anxiety or mood disorders, based on their mother's diagnostic status.

The first chapter of this paper provides a summary of the major literature in this area. The research is reviewed firstly with respect to general aspects of emotional disorders and their risk factors as background and context for the current study. Then literature is reviewed that relates specifically to the hypotheses and methods used in the current study on the relationship between maternal attentional biases, their offspring's attentional biases, and offspring anxious symptomology for a sample of mothers with emotional disorders and their

children (high risk) in comparison to those of never-disordered mothers and their children (low risk).

The second chapter of this study is a complete journal article manuscript, including methodology, results and interpretations of the research findings. The following manuscript's style and formatting is aimed for submission to the *Depression and Anxiety* journal.

Chapter One. A literature review of the possible risk pathways in the children of parents with emotional disorders: Cognitive models of risk transmission in high-risk offspring.

Introduction

Depression and anxiety are highly prevalent and disabling emotional disorders. There are a number of risk factors that have been identified over the years which increase an individual's vulnerability to developing an emotional disorder. In particular, the intergenerational transmission of emotional disorders is well-evidenced, with the children of parents with mood and anxiety disorders at increased risk for psychopathology throughout their lifespan. Although such increased risk in the offspring of parents with emotional disorders is likely to be a complex interplay of genetic, neurobiological, and social learning factors, another mechanism through which risk may be conferred is through the exposure of children to their parent's cognitive biases. Attentional biases towards negative information are one such cognitive mechanism which has been well-established as distinct in individuals with mood and anxiety disorders. Furthermore, there is a small but growing body of evidence to suggest a "cognitive vulnerability" in the offspring of parents with emotional disorders. That is, the children of parents with a history of anxiety or depression appear to have distinct ways of processing and attending to emotionally-salient information. The literature investigating the relationship between maternal patterns of attention and their children's attentional biases are sparse. The current study therefore aims to investigate further how maternal attentional biases relate to their offspring's attentional biases and the subsequent development of anxious symptomatology in a sample of mothers with emotional disorders and their children in comparison to those never-disordered mothers and their children. The following literature review aims: 1) summarise research relating to the more general aspects of emotional disorders and their risk factors, and 2) review the research studies which are most relevant to the current study focusing on attentional biases.

Emotional Disorders- Prevalence and Impact

Depression and anxiety are two of the most common emotional disorders. Depression is reported by the World Health Organisation (WHO) to be the leading cause of disability, with an estimated 350-million people living with depression worldwide (WHO, 2010). In Australia, the National Survey of Mental Health and Wellbeing (SMHWB) (Australian Bureau of Statistics, 2007) reported that one in twenty-five, or 4.1% of the population had experienced a depressive episode in the 12 months prior to the survey. Anxiety is even more prevalent in Australia with as many as one in seven, or 14.4 percent of Australians aged between 18 and 85 having experienced an anxiety disorder.

Thus, emotional disorders are highly prevalent with a large societal, economic and personal burden. Most recent estimates suggest that unipolar depressive disorders are the third leading cause of disease burden throughout the world, with predictions that by the year 2030 depression will be the leading cause of disease burden globally (World Health Organization, 2002). Similarly, in the year 2010, anxiety disorders were found to be sixth leading cause of disability in both high and middle-income countries (Baxter et al., 2014).

Emotional disorders can impact negatively on all aspects of an individual's life, including financial, relationship and psychological wellbeing with estimates suggesting that Australians were not able to carry out their normal activities 6.4 and 4.4 days out of a month due to anxiety and depression, respectively (SMHWB, 2007). Given that emotional disorders are highly prevalent and cause substantial disability across the lifespan, it is important to increase our understanding of the many factors which may contribute to the risk of developing anxiety and depression to ultimately deliver targeted prevention and intervention strategies.

Familial Risk

It is well-documented that emotional disorders run in families, with the children of parents with mood and anxiety disorders at increased risk for psychopathology throughout their lifespan. One longitudinal study conducted across 20-years found that the children of parents with a history of depression were three times more likely to develop an anxiety, depressive and substance disorder in comparison to the offspring of non-depressed parents (Weissman et al., 2006). Similarly, the offspring of parents who have experienced Major Depressive Disorder (MDD) have a threefold increase in the risk for developing depression during adolescence (Hammen, 2008; Williamson et al., 2004). A maternal history of depression has also been associated with an earlier onset and a more severe course of depression in offspring (Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002). Thus, the children of parents with a mood disorder appear to be at increased risk for depression and other psychopathology throughout their lifetime.

There is a similar picture for the offspring of parents with anxiety disorders whom are at 3.5 times greater risk for developing an anxiety disorder than the offspring of non-anxious parents (Merikangas, Avenevoli, Dierker & Grillon, 1999) whilst the children of parents with Panic Disorder (PD), for example, are at increased risk for developing any anxiety disorder themselves (Beiderman et al., 2001; 2006). Similarly, a meta-analysis of sixteen papers looking at anxiety and depression in high-risk offspring found that a parental history of anxiety disorders conferred significantly greater risk for developing either an anxiety or a mood disorders in their children, in comparison to matched controls, with a demonstrated odds ratio of 3.91 and 2.67, respectively (Micco et al., 2009).

Thus, the intergenerational transmission of emotional disorders is well-evidenced. However, relatively little is understood as to how this risk may be passed on from parents to their children (Rapee, 2002).

Mechanisms of Risk

Genetic, neurobiological and social learning factors, such as parenting behaviours, have all been implicated in contributing to risk for offspring of parents with an emotional disorder¹

Neurobiological Factors. Depression and anxiety are characterised by distinct neurobiological differences and similar differences may be present in the children of depressed and anxious populations. For example, dysregulation of the hypothalamus–pituitary–adrenal (HPA) axis has been found consistently to be related to elevated depressive symptoms with a growing evidence indicating that atypical HPA axis functioning may precede the emergence of depression in the children of mothers with a history of depression. Therefore, differences in HPA axis functioning may be a mechanism underlying the risk for depression in high-risk offspring (see Guerry & Hastings, 2011; Lopez-Duran, Kovacs, & George, 2009, for reviews).

The production of stress-hormones, such as cortisol, may also contribute to vulnerability for mood problems in the offspring of depressed parents. For example, by sampling salivary cortisol across two days in mothers with a history of, but not current diagnosis of depression and their children, it was found that mothers with remitted depression and their daughters exhibited higher cortisol production, which was directly related between dyads, in comparison to their low-risk counterparts (LeMoult et al., 2015).

Neurobiological mechanisms may also increase vulnerability for anxiety disorders. Utilising Functional Magnetic Resonance Imaging (fMRI) it has been found that children at high risk for anxiety disorders due to parental history of anxiety exhibited greater activation

¹ This section is not intended to be an exhaustive review of the literature in these areas, but rather a succinct overview of neurobiological, genetic and social learning factors as important possible contributors to the intergenerational transmission of emotional disorders (see Rapee, Schniering & Hudson, 2009's integrative review of multiple risk factors in anxious populations).

of the frontal, temporal and limbic neural regions during the presentation of angry and happy facial stimuli (Christensen, Michael, Van Ameringen, & Geoffrey-Hall, 2015). With a review of the major findings in high-risk populations concluding that children at risk for anxiety disorders due to parental pathology have increased startle reflex, autonomic reactivity, and stress reactivity (Merikangas, Avenevoli, Dierker, & Grillon, 1999).

Genetic Factors. The genetic contribution of familial risk cannot be overlooked. A recent meta-analysis on research in the genetic epidemiology of MDD estimated heritability at 37 percent (Sullivan, Neale, & Kendler, 2000). Similarly, twin studies have revealed mild to moderate genetic influences on child and adolescent measures of anxiety disorders (Bolton, Eley, & O'Connor, 2006; Scaini, Ogliari, & Eley, 2012). A meta-analysis of the genetic epidemiology of anxiety disorders estimated heritability for a range of anxiety disorders to fall between 30 to 40 percent (summary odds ratio ranging from 4 to 6). Such epidemiological findings suggest that the transmission of anxiety and depression from parents to children may be, at least partially, explained by genetic influences. However, there is still a large variance in the heritability estimates of emotional disorders which is not explained by genetic factors. In fact, in the first study of children-of-twins to investigate the relative contributions of genetic and environmental factors in the transmission of anxiety across generations, Eley and colleagues (2015) found greater support for environmentally-mediated transmission of anxiety, over and above any genetic contribution. Whilst, this may appear surprising, given the hereditary estimates reported above, it is consistent with other findings which support the environmental transmission of internalising problems, including depression (Silberg, Maes & Eaves, 2010; Singh et al., 2011, McAdams et al., 2015).

Social Learning Factors. Parental psychopathology may foster an environment that is conducive to the development of emotional disorders in offspring. Children learn how to perceive and interact with the world from observing their parents and how they perceive

interact with the world, a process called vicarious learning (Goodman & Gotlib, 1999; Askew & Field, 2008). Anxious parents may model anxiety by displaying overt signs of fear to the child, such as hyperventilation and shaking, may verbally communicate greater fear-information to the child or may even model maladaptive coping strategies such as avoidance (Field, 2006). There are a number of studies to support the influential role of parental modelling in clinically and non-clinically anxious children (see Fisak & Grills-Taquechel, 2007 for a review).

Parents with emotional disorders may also be more likely to engage in negative parenting behaviours that promote anxiety or depression in offspring. For example, anxious parents have been found to display greater over-controlling behaviour with their children (Eley et al., 2010) and tend to be more negative and critical of their offspring (Creswell et al., 2013) whilst less positive engagement from mothers tends to be more consistently associated with maternal- and self-reported depressive symptoms in children (Olino et al., 2015).

In summary, the role of environmental factors, such as modelling of anxious and depressive behaviours and parenting style, may play an important interactive role in the development of emotional disorders in children at risk due to parental history of psychopathology.

Emotional Disorders: Cognitive Theories

Although risk is likely to be a complex interplay of genetic, neurobiological, and social learning factors, another mechanism through which risk may be conferred is through the exposure of children to their parent's cognitive biases (Goodman & Gotlib, 1999).

Cognitive theories propose that individuals with anxiety and depression, or at risk for their development, process information differently to emotionally healthy individuals (Beck, 1967; Eysenck, 1997; Mogg & Bradley, 1998; Teasdale, 1988; Williams, Watts, MacLeod, &

Mathews, 1997). Specifically, it is thought that certain cognitive processes, such as attention, interpretation, memory and judgement may all be influenced by maladaptive schemas, which predispose an individual to, and maintain psychopathology (Beck, 1976).

The attentional system of individuals with emotional disorders is one aspect of information processing that has been the focus of more than 30-years of research. Specifically, early automatic stages attentional processing in individuals with anxiety and depression have been theorised to be sensitive to, or biased towards, salient negative or threat-related information. An “attentional bias” refers to the preference of attentional allocation resources of an individual in favour of emotionally salient stimuli relative to neutral stimuli (Williams et al., 1997; 1988) and may be a mechanism through which emotional problems develop. As Hadwin and Field (2010) explain: “An attentional system that preferentially filters and selects negative, threatening, anxiogenic information is more likely to potentiate perceptions of threat and danger; activate a range of dysfunctional thoughts, feelings and beliefs about the present and future; and, ultimately, increase an individual’s vulnerability” (p.77) to developing an emotional disorder.

Measuring Attentional Biases: Experimental Paradigms

Experimental studies that have attempted to accurately capture attentional biases have led to the development of a number of attention-bias paradigms. The Emotional Stroop task and the Dot-Probe task are two of the most commonly studied measures and will be discussed briefly in turn.

The *Emotional Stroop Task* is a modified version of the classic colour-naming Stroop paradigm (Stroop, 1935) and was initially the most widely used tool to capture attentional biases. In this paradigm, an individual’s attentional bias is captured by response latency, or reaction time, in responding to the colour of an emotionally salient stimuli such as threat-

related words (e.g. “cancer”) or schematic face signifying threat (e.g. “angry face”) relative to neutral words or facial stimuli (e.g. “plate”, “neutral face”). A threat-related bias is inferred when colour naming takes longer with a threat-stimulus relative to a neutral-stimulus, suggesting the emotionally salient stimuli has disrupted, or slowed attentional processing (MacLeod, 1991). Research since the 1980’s has demonstrated that the colour naming of negatively-valenced words is typically slower in individuals who have emotional disorders, relative to neutral words and that this discrepancy is typically not observed in non-clinical populations (Williams, Matthews & MacLeod, 1996).

The emotional Stroop paradigm has been criticised on the basis that delayed response to emotionally-salient stimuli, such as an angry face, may be confounded by the priming of a negative affective state in anxious or depressed individuals, thus contributing to delayed reaction times. Therefore, it remains unclear if the emotional Stroop task captures the automatic attentional processing of threat cues, or the effortful avoidance of threat-stimuli due to increased anxious arousal (MacLeod, Mathews, & Tata, 1986).

In attempts to address the possible affective biases of the emotional Stroop task, MacLeod and colleagues (1986) developed the *Dot-Probe Paradigm*, which is now one of the most widely researched paradigms of attentional biases. The dot-probe task shows one threat-related (e.g. angry face) and one neutral stimulus (e.g. “happy face”) briefly for either 500ms or 1250ms and then replaces this with a small probe in the location occupied by either the threatening or neutral stimuli. Unlike the emotional Stroop, participants respond as quickly as possible to the probe location, rather than the stimulus itself. The rationale underlying visual probe tasks is that individuals are faster to respond to probe stimuli presented in an attended (rather than unattended) region of the visual display. A faster response to the probe that replaces the threat-related stimulus, such as an angry face, suggests an attentional bias towards negative-valenced stimuli. In adult populations,

individuals with both clinical anxiety and subclinical levels of trait anxiety have been shown to respond faster to probes following threat-related stimuli using the dot-probe paradigm, suggesting vigilance in attention for threat (Broadbent and Broadbent, 1988; MacLeod, Mathews and Tata, 1986; Mogg, Bradley and Williams, 1995).

The dot-probe paradigm is well-evidenced in clinical populations. However, some questions have been raised in more recent times regarding the reliability of the dot-probe task, particularly in non-clinical populations. For example, Schmukle (2005) administered the dot-probe task, using both word and situational-pictorial stimuli, to eighty non-clinical university students at two time points across one week. Estimates of both internal consistency and retest reliability led the author to conclude that both versions of the dot-probe task were “unreliable measures of attentional allocation in non-clinical samples” (p. 595). Similarly, Staugaard’s (2009) follow-up study utilising photographic facial stimuli again did not find sufficient evidence of test-retest reliability of internal consistency of the dot-probe task at either the 100ms or 500ms stimulus presentations. Although the non-anxious participants, as a group, were seen to have stable and consistent vigilance scores for angry faces presented at 500ms.

As sufficient reliability is a pre-condition for research that is concerned with inter-individual differences, the appropriateness of the dot-probe paradigm for measuring within-individual differences is questionable. However, both Staugaard (2009) and Schmukle (2005) concluded that the measure was still useful for capturing between groups differences (i.e. clinical and non-clinical comparisons). As such, the robust cross-sectional association of attentional biases with emotional disorders has been repeatedly evidenced as a pattern of attentional allocation that is distinct from non-clinical populations.

Attentional Biases in Emotionally-Disordered Populations

A wealth of research demonstrates that anxious adults and children display an attentional bias towards threatening sources of information, and this effect is less consistent or typically not observed in non-anxious individuals (see Bar-Haim et al., 2007 for a review). The seminal meta-analysis of Bar-Haim and colleagues which analysed the results of 172 studies of attentional bias in clinical, non-clinical and trait-anxious populations, found strong confirming evidence that individuals with anxiety display a bias towards threatening information ($d = 0.45$), a relationship that is not seen in non-anxious individuals.

Although there is a smaller body of literature, this association has also been demonstrated in anxious children. Dudeney, Sharpe and Hunt (2015) concluded in their meta-analysis of 38 studies of attentional biases in children with anxiety show a similar, although weaker selective bias towards threatening stimuli as seen in adult populations and that is significantly different from non-anxious children ($d = 0.21$). Interestingly, it was also found that the relationship between attentional biases and anxiety in children was significantly moderated by the child's age, with a stronger association found in older children suggesting that attentional biases towards threat in anxious children may be influenced by developmental factors.

Although well-established in anxious populations, the relationship between mood and attentional biases is less clear. Whilst some studies have found evidence for an association of greater attentional allocation for negative-valanced stimuli (Gotlib et al. 2004a; Mathews et al. 1996; Rinck and Becker 2005), others have failed to find such attentional biases in depressed populations (Dagleish, et al. 2003; MacLeod, et al. 1986; Mogg et al. 2000; Neshat-Doost et al. 2000). However, methodological differences in the duration of stimulus presentation, the nature of the stimuli and mood priming have been theorised to contribute to these inconsistent findings (Matthews & MacLeod, 1996). The results of a recent meta-

analysis reviewing 29 methodologically-diverse studies of attentional biases in depression, found that there was evidence of a significantly greater attentional bias to negative information in depressed versus non-depressed populations. Specifically, there was found to be a moderate effect-size in studies utilising the dot-probe task ($d = 0.52$) whilst there was only a marginally significant difference between depressed and non-depressed samples utilising the emotional Stroop task (Peckham, McHugh & Otto, 2010).

In summary, individuals with a history of anxiety and mood problems appear to have attentional biases that are different from those without emotional disorders. However, cognitive models assume that attentional biases are not merely the by-product of an emotional disorder, but play an important causal role in the development of anxiety and depression. It is difficult to determine from the cross-sectional literature summarized above whether maladaptive attentional biases are a symptom of, or a precipitant to emotional disorders, or both. In Van Bockstaele and colleagues (2013) major review of the evidence for the causal impact of attentional bias and fear, previous studies were examined based on Hill's (1965) comprehensive criteria for causation. They concluded, based on the evidence in the literature to date, that attentional bias toward threat is most likely more than a epiphenomenon of fear and anxiety, with attentional biases likely to play a causal role in the development of anxiety disorders. However, they suggest that attentional biases are unlikely to lead to anxiety in a strictly unidirectional manner, but rather, that the relationship is bidirectional with attentional biases maintaining or exacerbating fear and anxiety, and vice versa (see VonBockstaele et al.'s review). The authors close stating that: "given this causal influence, a fine-grained understanding of the effects of attentional factors seems fruitful for both understanding and modifying anxiety." (p. 29).

One population in which this “chicken and egg” problem of causality could be investigated further is in non-clinical children that are known to be at risk for developing depression and anxiety due to a parental history of emotional problems.

Cognitive Vulnerability in High Risk Offspring

There is a growing body of evidence to suggest a “cognitive vulnerability” in the offspring of parents with emotional disorders. That is, the children of parents with a history of anxiety or depression appear to have distinct ways of processing and attending to emotionally-salient information.

Some research has looked at how the children of depressed parents process emotional information in comparison to children of never-disordered parents. In such studies, children are asked to identify ambiguous or low intensity facial expressions signifying different emotional states. Mannie, Bristow, Harmer and Cowen (2007) found that the high risk daughters of mothers with depression demonstrated a significant attentional bias towards negative facial stimuli in comparison to their low-risk peers whilst there was found to be no significant difference in emotional identification abilities between high-risk offspring in comparison to controls. However, in a similar study which required children to identify emotions from a sequence of intensifying facial expressions following negative mood induction, high-risk daughters (i.e. with mothers who had experienced recurrent episodes of depression during their daughters' lifetime) required greater emotion intensity of facial expression than daughters of control-group mothers to accurately identify sad expressions and made significantly more errors identifying angry expressions (Joorman, Gilbert & Gotlib, 2010). Similarly, Dearing and Gotlib (2009) found that after negative mood induction, the young daughters of depressed mothers interpreted ambiguous words more negatively and less

positively, and ambiguous stories more negatively, than did daughters of never-disordered mothers.

Research on emotional processing biases in the offspring of parents with anxiety disorders is relatively sparse. In one study, Pine and colleagues (2005) investigated whether children of parents with a history of PD or MDD would show heightened sensitivity and greater attention allocation to negatively valenced facial stimuli in comparison to the children of non-disordered parents. It was found that the offspring of parents with PD reported significantly more fear and had slower reaction times to rate fear during presentations of angry/fearful facial expressions in comparison to controls, even after controlling for the presence of anxiety disorders in the high risk children. However, due to the presence of anxious symptomology in the high-risk children included in this sample, there is some question as to whether these findings represent a cognitive vulnerability or rather state-sensitivity in anxious high-risk children. More recently, Bilodeau, Bradwejn and Koszycki (2015) clarified these findings in the non-symptomatic offspring of parents with PD. Children completed a standard facial recognition task in which the high-risk children were found to make more errors recognising fearful faces than controls and were more likely to misattribute fearful and angry facial expressions to surprised affect.

Collectively, these studies provide evidence to suggest that children at risk for emotional disorders, even before the onset of symptoms, exhibit biases in the processing of emotionally salient information. This cognitive bias may leave them vulnerable to developing anxious and depressive disorders throughout their life.

Attentional Biases as a Cognitive Risk Marker in High Risk Offspring

Dysfunctional attentional biases, which have been found to characterise anxiety and depression in adult and child populations, may be a specific cognitive marker of risk in non-

symptomatic high-risk offspring. Joorman, Talbot and Gotlib (2010) were the first to assess biases in attentional processing in the high-risk daughters of depressed mothers in late childhood to early adolescence (aged between 9 and 14 years) using the dot-probe paradigm. It was found that, following negative mood-induction, the never-disordered daughters of depressed mothers selectively attended to sad facial expressions, whilst the daughters of never-disordered mothers did not. Rather, these low-risk daughters were seen to selectively attend only to positive facial expressions, and this positive bias was not observed in the high-risk children. The authors speculated that this cognitive vulnerability may be marked not only by an attentional bias towards threat, but also the inability to shift attention away from negative stimuli and towards positive stimuli. Failure to acquire adequate control over attention by late childhood/early adolescence has also been shown to relate to greater threat attention bias (Derryberry & Reed, 2002; Lonigan et al., 2004) whilst well-developed attentional control has been shown to be related to better regulation of distress and anger (Posner & Rothbart, 2000). Thus the development of attentional control mechanisms may be an important factor in determining the vulnerability of children at risk for depression and anxiety to be considered.

Interestingly, Kujawa and colleagues (2011), using a similar design in high-risk children in early childhood (aged between 5 and 7), did not find any significant positive-bias in the offspring of mothers without a history of depression as was seen in Joorman, Talbot and Gilbert (2010). However, they did find that the daughters of depressed mothers demonstrated an attentional bias towards sad facial stimuli, whilst the control-group daughters and interestingly, the sons of depressed mothers, did not exhibit any attentional bias towards such negative-stimuli. These findings were the first to lend support for the development of cognitive biases in children at risk for depression in early childhood, which may be a vulnerability marker for depression in later development. However, they also

suggest that sex differences in these cognitive biases may precede the sex differences in rates of depression that has been seen to emerge throughout later development (Goodman, 2007). Although attentional bias towards negative information has been associated with depression, interestingly, without mood-induction techniques to activate depressive schemas prior to the assessment of attentional processing, Gibb, Benas, Grassia, and McGeary (2009) found that the offspring of mothers with a lifetime history of depression showed a greater avoidance of, rather than selective attention towards, sad faces. Furthermore, high-risk offspring were found to have no bias towards angry or happy faces. Although these findings taken together suggest that there are distinct biases in attentional processing in the high-risk offspring of depressed parents, it appears that some form of stressor, or a negative mood primer is a necessary state-related condition for such selective attentional biases towards threat to become apparent (Gotlib, Joorman & Foland-Ross, 2015).

Although studies in high-risk anxious populations are sparse, one recent study also suggests a distinct pattern of attentional processing in the offspring of anxious parents. Mogg, Wilson, Hayward, Cunning and Bradley (2012) examined attentional biases in 60 mothers with lifetime PD and their daughters, aged between 9 and 14 years, using a visual dot-probe task with pictorial and word stimuli related to physical-health threat at two stimulus presentation durations: 500ms and 1250ms. It was found that the non-anxious daughters of mothers with Panic Disorder demonstrated a significant attentional bias towards stimuli representing a physical health threat (e.g. “racing heart”) in comparison to controls, although this was only at the longer stimulus duration of 1250ms. The authors concluded that, as the bias was not present at the earlier 500ms presentation, the high-risk daughters had developed strategic attentional processes to seek and maintain attention on threatening cues, rather than exhibiting a more automatic early-orienting bias. Other research suggests that intervals of less

than 250ms involves automatic processes and intervals greater than 250ms utilise controlled or strategic processes (Derryberry & Reed, 2002).

In summary, despite some inconsistent findings regarding the specific pattern of attentional allocation, high-risk offspring of parents with emotional disorders appear to have attentional biases that are distinct from their low-risk counterparts.

The Relationship between Maternal and Children's Attentional Biases

Most research in the cognitive literature to date has focused on the offspring's attentional biases as the risk marker of interest. In what manner parental attentional biases may relate to their offspring's biases and later symptomology has rarely been explored. In Mogg and colleagues (2012) study described above, although there was found to be a significant attentional bias for physical health-cues in high risk daughters, there was no relationship between mother and daughters attentional biases in either the high-risk or low-risk dyads indicating that mothers with lifetime PD and their daughters showed different patterns of attentional biases to threat. In fact, the mothers with a lifetime PD did not demonstrated an attentional bias towards threat that was significantly greater than never-disorder mothers. The authors concluded that attentional biases were not a fixed vulnerability factor for PD, as if this was the case, mothers and daughters would be expected to have a similar threat-bias. Thus, the transmission of identical cognitive biases across generations may not fully explain the pattern of attentional biases seen in high-risk children.

However, a recent study investigating attentional biases in mothers with and without emotional disorders and their children, found that high-risk offspring's attentional bias were related to the pattern of attention bias demonstrated by their mothers (Waters, Forest, Peters, Bradley & Mogg, 2015). Specifically, in a sample of 67 high and low-risk mother and daughter dyads, the high risk offspring demonstrated a greater attentional bias towards threat

when their mothers had a reduced bias for positive information, whilst no significant association was observed in the low risk dyads.

Clearly, further investigation of the relationship between maternal and offspring attentional biases is warranted to clarify these findings. Specifically, it would be of interest to determine if mother's attentional biases significantly relate to, and predict the development of their offspring's patterns of attentional biases across time. This would suggest a direct link between the cognitive biases of mothers and the subsequent attentional biases in their children and would have clear implications for prevention and intervention.

Longitudinal Associations of Attentional Bias and Anxious Symptomology

Although information processing biases have been evidenced in children vulnerable for developing depression and anxiety at the cross-sectional level, there is still little evidence to suggest that such attentional biases play a role in the etiology of emotional disorders. In their review of 25-years of research in familial depression, Gotlib, Joorman and Folland-Ross (2015) suggest that "longitudinal studies are urgently needed to investigate whether these biases in cognitive functioning in childhood predict the subsequent onset of MDD" (p. 99). Therefore, an important research goal would be to investigate whether attentional biases predict later symptom expression in high-risk children.

A few longitudinal studies have examined the role of attentional biases as a mechanism through which early markers of vulnerability, such as fearful temperament, may relate to the expression of internalising and externalising symptoms across time. One such study investigating the relationship between the temperamental feature of Behavioural Inhibition (BI) in young children and later social withdrawal in adolescence, considered children's attentional biases towards threat as a possible cognitive mechanism of conduit (Perez-Edgar et al., 2010). A sample of 153 mid-adolescents were followed up and assessed for attentional biases and social withdrawal after being screened for levels of reactivity at the

age of four months. It was found that adolescents with a history of BI showed significantly heightened levels of attentional bias towards threatening facial stimuli in comparison to controls, whilst only their non-inhibited counterparts displayed an attentional bias toward positive facial stimuli. Furthermore, early-childhood temperament and subsequent social withdrawal was found to be moderated by attentional bias towards threat. Thus, only in behaviourally inhibited children was this pattern of attentional bias towards threat seen to be positively related to social withdrawal in adolescence.

A similar longitudinal design conducted by Perez-Edgar and colleagues (2011) examined relationship between, attentional biases to threat, and social withdrawal in early childhood. As was seen in the previous study with adolescents, behavioural inhibition in toddlerhood predicted higher levels of social withdrawal at age five, but only for those children who exhibited a significant attentional bias towards threat. Following this, Morales, Perez-Edgar and Buss (2014) looked at the role of attentional bias in moderating the relationship between a distinct type of behavioural inhibition known as “dysregulated fear” in toddler years and later social withdrawal in kindergarten. Interestingly, it was found that children classified as high in dysregulated fear showed a significant attentional bias away from threat in later development. This was contrary to the findings of Perez-Edgar et al. (2010, 2011) who found that children and adolescents high in behavioural inhibition displayed a bias towards threat, which the authors suggest “may be a distinction of the subgroup of dysregulated fear within the more general classification of behavioural inhibition”. However, Perez-Edgar and colleagues (2011) found that a pattern of attentional bias towards threat predicted later social withdrawal at age six for children classified as high in dysregulated fear at age two.

Collectively, these longitudinal studies suggest that attentional bias towards threat appears to play an important moderating role in the etiology of internalising symptoms when

considering early child-related risk markers. To date, the relationship between risk as determined by parental history of emotional disorder and the progression of offspring's symptomology, through the function of either parent or child patterns of attentional bias has yet to be explored. Furthermore, to our knowledge, studies exploring the relationship of attentional bias in the development of psychopathology have only considered one time-point of assessment of dot-probe data. For example, Wald and colleagues (2011) examined the longitudinal association of attentional bias to threat with the development of PTSD in people exposed to rocket fire in the Gaza strip and non-exposed individuals out of rocket range. Although two time-point measures of attentional bias were obtained across 12 months (during the war and post war), the initial measures of dot-probe task were utilised to predict the later development of PTSD, in a sense assuming a "lag effect" of attentional bias during the war affecting trauma symptomology post-war (cf. Wald et al., 2011). Whilst this study, and other studies (Perez-Edgar et al., 2010; 2011; Morales et al., 2014) are longitudinal in design, they are not true repeated measures. Hence, the literature to date has been limited in its ability to identify within-individual change in attentional bias and its relationship with the change in symptom expression for the individual. As attentional biases are theorised to play an important casual role in the development of anxiety and depression in high-risk individuals, research which can distinguish the true within-subjects (longitudinal) change in attentional bias with subsequent symptom expression, from the overall between group (target vs control population) difference, would be of interest. This being said, the reliability of the dot-probe task for capturing within-subjects differences in attentional biases across time may be a notable limitation of the current study and will be investigated utilising a comprehensive method of analysing longitudinal data.

The Current Study: The aim of the current study was to follow the cohort previously reported in Waters, Forest, Peters, Bradley & Mogg (2015) to investigate the cross-sectional and longitudinal relationship between both maternal and offspring attention bias (AB) in the development of anxious symptomology across 12 months as a function of the child's risk status. To the authors knowledge, this is the first longitudinal study exploring both maternal and child risk markers, utilising a repeated measures design supporting a comprehensive method of analysing longitudinal data as proposed by Diggle, Heagerty, Liang and Zeger (2002).

The current study's hypotheses are as follows: for the high risk but not low risk offspring there would be:

- i) a significant increase in threat attentional bias and anxious symptomology across 12 months
- ii) a significant relationship between maternal threat attentional bias and offspring threat attentional bias, across and within dyads,
- iii) an increase in offspring threat attentional bias would significantly predict an increase in anxious symptomology and
- iv) an increase in maternal threat attention bias would predict an increase in offspring anxious symptomology.

Although this study focused primarily on threat-AB relationships, as Waters et al. (2015) found a significant negative correlation between offspring threat-AB and maternal positive-AB the equivalent to hypothesis ii) in the context of maternal positive-AB was also tested.

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Chapter Two: Manuscript to be submitted for publication in *Depression & Anxiety*

Attention bias to threat in mothers with emotional disorders predicts increased offspring
anxiety symptoms: A joint cross-sectional and longitudinal analysis

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Keywords: Anxiety; depression; attention bias; mothers; offspring

Word Count: 3, 580

Abstract

Background: The intergenerational transmission of anxiety and depression from parents to children is well evidenced; however, the mechanisms through which this vulnerability is passed on are not well understood. Cognitive models propose that the exposure of children to the attention biases of their parents may be one pathway through which risk is passed and is supported by a small body of cross-sectional research. However, longitudinal studies exploring the association of maternal and offspring attention biases with symptom expression across time are lacking.

Methods: With a sample of 43 mothers, classified into high (n=24) and low (n=19) risk groups with the former having a lifetime emotional disorder (DSM-IV lifetime anxiety or major depressive disorder), and their non-symptomatic offspring (aged 7 – 12 years), this study examined the cross-sectional and longitudinal associations of maternal and child attention bias towards threat and child anxiety symptomology based on initial and 12 month follow-up assessments. Mother-child dyad attention biases measures were assessed using a visual probe task with photographic faces presented for 500ms and mothers-report of anxiety.

Results: The major finding was that there was a significant cross-sectional, but not longitudinal association of increased child anxiety symptoms with increased maternal attention threat bias for the High Risk but not Low Risk dyads.

Conclusions: At the cross-sectional level, increases in High Risk offspring anxiety symptomology were associated with maternal threat attention bias in a way that was not seen in the Low Risk offspring. The lack of any longitudinal association may suggest that task-related and situational factors need greater consideration in repeated-measures research utilising the dot-probe paradigm.

Introduction

It is well-documented that emotional disorders run in families. Offspring of parents with anxiety and depression are at increased risk for developing such disorders throughout their lifetime^[1,2]. For example, the offspring of parents with Major Depressive Disorder (MDD) risk for developing depression during adolescence increases threefold^[3,4] with maternal depression associated with an earlier onset and a more severe course of depression in their children^[5]. There is a similar picture in offspring of parents with anxiety disorders, whom are at 3.5 times greater risk for developing an anxiety disorder than the offspring of non-anxious parents^[6,7,8]. Thus although the intergenerational transmission of emotional disorders is well-evidenced, relatively little is understood as to how this risk may be passed from parents to their children^[9].

One mechanism through which risk may be conferred is through the exposure of children to their parents' cognitive biases^[10]. Cognitive models suggest that individuals with emotional disorders, or at risk for their development, preferentially attend to negative stimuli during early stages of stimulus processing^[11-14]. Evidence supports the robust association of emotional disorders with attention biases (AB) in both adults and children^[15,16]. Increased AB towards negative information has been associated with a range of anxiety disorders and non-clinical trait anxiety^[15], whereas depression is associated with increased AB for negative information and reduced AB for positive information^[16,17].

There is also growing evidence to suggest a "cognitive vulnerability" in the offspring of parents with emotional disorders which may contribute to increased risk. Dysfunctional AB may be a specific cognitive risk marker in the offspring of parents with a history of emotional disorders. Following mood-induction, daughters of depressed mothers have been found to preferentially attend to sad faces in comparison to daughters of never-depressed mothers^[19, 20]. However, in other studies without mood induction techniques, children of

mothers with a lifetime history of depression showed greater avoidance of sad faces and no bias for angry or happy faces^[21]. Likewise, the non-anxious daughters of mothers with Panic Disorder (PD) have significant AB towards physical health threat stimuli in comparison to controls^[22]. Despite some inconsistency regarding the specific pattern, at the cross-sectional level, high-risk offspring of parents with an emotional disorder appear to manifest ABs that are distinct from their low-risk counterparts.

Yet, these studies have primarily focused on offspring's AB as the marker of risk and have not considered how maternal AB relates to their child's bias and later symptomology. In our recent cross-sectional study, the threat AB in high-risk offspring was influenced by the pattern of AB of their mothers. Specifically, high risk offspring demonstrated a greater AB towards threat when their mothers had a reduced bias for positive information, relative to no significant association in low risk controls^[23].

The few studies that have examined the longitudinal association between AB and symptom expression in high risk children have focused on AB as a mediating factor between child-related risk factors in early development, such as temperament, in predicting the later development of internalising and externalising symptoms,^[24-26] or have included only one time-point of AB assessment^[27].

The aim of this study was to follow the cohort reported in Waters et al.^[23] to investigate the cross-sectional and longitudinal relationship between both maternal and offspring AB in the development of anxious symptomology across 12 months as a function of risk status. To our knowledge, this is the first longitudinal study exploring both maternal and child risk markers, utilising a repeated measures design supporting a comprehensive method of analysing longitudinal data^[28].

The hypotheses were as follows: for the high risk but not low risk offspring, there would be i) a significant increase in threat-AB and anxious symptomology across 12 months

in offspring, ii) a significant relationship between maternal threat-AB and offspring threat-AB, across and within dyads, iii) an increase in anxious symptomology predicted by an increase in offspring threat-AB, and iv) an increase in offspring anxious symptomology predicted by an increase in maternal threat-AB predicting. Although this study focused primarily on theory-consistent threat-AB relationships, as Waters et al.^[23] found a significant negative correlation between offspring threat-AB and maternal positive-AB, the equivalent hypothesis to that in hypothesis ii) was tested in relation to maternal positive-AB.

Methods and Materials

Participants

One hundred and six parent-child dyads were initially assessed to participate in this study. Participants were recruited through community advertisements, primary school and university notices and newsletters, local newspapers, GPs, and community mental health clinics. All participants provided written informed consent. Initial exclusion criteria for the present study included (a) the child having a psychiatric disorder, including an anxiety or mood disorder; chronic medical condition, intellectual impairment; pervasive developmental disorder; bipolar disorder, oppositional defiant disorder or psychosis, (b) the mother having a past or current chronic medical condition, intellectual impairment, bipolar disorder, psychosis or any psychiatric disorder other than anxiety and unipolar depression, and (c) if the participating parent was not the child's biological mother.

There were 67 dyads in the initial sample^[23], while for the follow-up study 13 of these were uncontactable, and 11 were contactable but either declined to attend the laboratory assessment due to scheduling difficulties or relocating too far from the University. Therefore forty-three dyads were included in the present study with 19 dyads specified as Low Risk (LR) and 24 as High Risk (HR) based on diagnostic measures described below. Children

ranged in age from 7 to 12 years, 11 months ($M = 9.69$ $SD = 1.32$). Of the 24 HR mothers, 10 had a principal lifetime anxiety disorder (PD = 1; Specific Phobia = 2; Generalised Anxiety Disorder = 3; Post Traumatic Stress Disorder = 2; Separation Anxiety Disorder = 1; Social Phobia = 1) and 14 had MDD. Of the 10 mothers with a principal lifetime anxiety disorder, 4 had comorbid anxiety disorders, 2 had comorbid depressive disorders, and 4 had no comorbidity. Of the 14 mothers with a principal lifetime depressive disorder, 1 had a comorbid depressive disorder, 5 had comorbid anxiety disorders, and 8 had no comorbidity.

Diagnostic and Symptom Measures. The Anxiety Disorders Interview Schedule for DSM-IV-Lifetime Version (ADIS-IV-L)^[29] was used to assess current and previous history of emotional disorders for mothers at Time 1 assessment. The Anxiety Disorders Interview Schedule for DSM-IV- Parent Version (ADIS-C-IV)^[30] assessed children's diagnostic status at initial and follow-up assessments. Mothers and children were considered to have a clinically significant disorder if the Clinician Severity Rating (CSR) was four or greater for at least their principal diagnosis (i.e. most severe)^[31]. All ADIS-IV-L diagnoses were reviewed in supervision and 20 percent were audio-taped and coded by an independent rater blind to diagnostic status for reliability purposes. Inter-rater reliability was excellent. For mothers and children the principal diagnosis was $k = .89$ and $.84$ respectively, whilst second diagnosis was $k = .82$ and $.83$.

The Spence Children's Anxiety Scale Parent Version (SCAS-P)^[32] was utilised to assess anxiety symptom severity in children at Time 1 (T1) and Time 2 (T2) assessments. The SCAS-P has sound psychometric properties^[33]. Mean SCAS-P total scores of 14.2 and 31.8 have been reported for non-clinical and clinically-anxious children, respectively^[34, 35].

Attention Bias. The visual-probe task was programmed using E-prime v1.1 (Psychology Software Tools, Inc.) and presented on a Dell Optiplex computer housed in a University research laboratory. The stimuli were photographs of face-pairs from 64 different

actors (half male, half female), each presenting a neutral and either an angry or happy expression^[34]. The experimental stimuli consisted of 32 angry-neutral, 32 happy-neutral and 16 neutral/neutral face-pairs used on filler trials. Each trial began with a 500-ms fixation point followed by two faces presented side-by-side for 500 ms. The face-pair was then replaced with an asterisk (probe) for 1100 ms in the spatial location previously occupied by one of the faces. Participants pressed one of two keys labelled 'left' and 'right' as quickly as possible while avoiding mistakes, to indicate the location of the asterisk with the time taken to respond denoted "response time" (RT). The task began with 10 random practice trials, followed by one block of 80 trials. On angry-neutral face pair trials, the probe appeared in the same spatial location as the angry face in half the trials (congruent trials), and on the opposite location to the angry face in the other half (incongruent trials). The same arrangement applied to trials with happy-neutral face pairs.

Procedure

See Waters et al.^[23] for a summary of experimental procedures for the T1 assessment. Participants were re-contacted via telephone, email or post for T2 assessment. Of those participants who agreed to participate, the ADIS-C-IV was readministered to the mother via telephone or a face-to-face interview. Those dyads then attended the laboratory assessment to complete questionnaires and the dot-probe task.

Data preparation

RTs were excluded from trials with errors, and if RTs were <200 ms, >1100 ms, and then >3 SD above the participant's mean RT. On average, RTs were missing from 7% of trials at T1 and 3% of trials at T2 for children, and 2% of trials at T1 and 3% of trials at T2 for mothers. Overall the mean RT was 584 (SD=96) ms for children, and 481 (SD=63) ms for mothers at T1 and 515 (SD=78) ms and 510 (SD=72) ms at T2, respectively. High- and low-

risk children did not differ significantly in missing RT data at T1: Mann-Whitney $U = 496.5$, $p = .49$, or T2: Mann-Whitney $U = 204.5$, $p = .564$, or in overall mean RT at T1: $t(65) = 0.10$, $p = .92$, or at follow-up: $t(41) = 1.18$, $p = .253$ (T2). Mothers with versus without lifetime emotional disorders, also did not differ in missing data at T1: Mann-Whitney $U = 456.5$, $p = .22$, or T2: Mann-Whitney $U = 206$, $p = .57$, or in overall mean RT, $t(65) = 1.37$, $p = .18$ (T1), $t(41) = -1.67$, $p = .104$ (T2). Consistent with previous studies^[34,35] AB scores at T1 and T2 were calculated separately for angry-neutral and happy-neutral face pairs by subtracting the average RT on congruent trials from the average RT on incongruent trials.

Statistical Analyses

Preliminary analyses and screening ensured the data met assumptions for multiple regression analysis. One HR dyad was missing the child's SCAS-P total data, whilst one HR dyad was missing maternal threat-AB data. Inspection of AB distributions revealed one significant outlier in maternal threat-AB scores (> 100 ms relative to the remainder of the sample with AB scores < 60 ms). This dyad was excluded from all analyses involving maternal threat-AB.

To investigate hypothesis i) 2×2 mixed-factorial repeated measures ANOVAs were conducted with a Bonferroni correction whilst hypothesis ii) was investigated using Pearson's correlation coefficient. In addition, the Linear Mixed Model (LMM) described below was fitted with maternal threat-AB, cross-sectional and longitudinal variables as independent variables, combined with child threat-AB used as the dependent variable.

Hypotheses iii) and iv) were tested separately with the same analytical approach. In each analyses, SCAS-P total score was the dependent variable, as predicted by child (hypothesis iii) or maternal (hypothesis iv) threat-AB. To estimate the HR vs LR effects for T1 as a moderator of the threat-AB scores when these scores were used as a predictor of

SCAS-P total score, a separate intercepts and separate slopes linear regression was fit. This regression can only model between-dyad variation since only T1 scores were employed. Next, the within-dyads difference (i.e. change) in threat AB and symptoms scores for the T1 and T2 data were each calculated, and a corresponding separate intercepts and separate slopes linear regression was fit utilising symptom change scores as the dependent variable as recommended by Staugaard^[36] (i.e. a “change analysis”). To take into account the correlation between T1 and T2 regression residuals due to the shared random dyad effects, a single LMM was fit by jointly estimating both regressions based on the approach devised by Diggle, Heagerty, Liang and Zeger^[28].

To fit this LMM a dataset was constructed by i) appending SCAS-P total scores at T2 to those at T1 as the dependent variable and ordering this data by dyad identifier within time, ii) appending to T1 threat-AB the same values for T2 (i.e. repeating the T1 values) as the cross-sectional independent variable², and iii) appending the T2 minus T1 threat-AB at T2 to the set of zero values for T1 as the longitudinal independent variable³. Both these independent variables were modified by Risk Group. Separate residual variances were estimated for T1 and T2 along with the covariance between times, corresponding to the random dyad variance, using residual maximum likelihood. All analyses were carried out using SPSS (ver.22).

² The corresponding cross-sectional regression coefficient has the interpretation that for every unit change in attentional threat bias across subject dyads (i.e. comparing different dyads) there is a corresponding expected change in the SCAS-P total score equal to the (unstandardized) coefficient.

³ The corresponding longitudinal coefficient has the interpretation that for every unit change in the attentional threat bias between times T1 and T2 there is a corresponding expected change within dyads in SCAS-P total scores equal to the (unstandardized) coefficient. This within-dyad change is that expected on average across the sample dyads.

Results

Comparisons between dyads who continued versus dropped out.

There was no statistically significant difference in dropout rates between HR ($n = 14$) and LR groups ($n = 10$): Fisher's exact test ($p > .09$). The odds ratio was 0.90 (95% CI: 0.29, 2.77). Across groups, there was no significant difference in T1 SCAS-P total scores: $t(65) = -0.62, p = .54$ or T1 threat-AB scores: $t(65) = 0.66, p = .51$ between children who did ($n = 43$) and those who did not ($n = 24$) complete T2 measures. Similarly, there was no significant between-group differences in any maternal socio-demographic variables (see Table 1) or maternal T1 threat-AB scores: $t(65) = -0.78, p = .43$.

Clinically significant diagnoses between risk groups

Results of a chi-square analysis found no significant difference between risk groups in the proportions of children who did versus did not develop a clinically significant ($\text{CRS} > 4$) diagnosis at T2, $\chi^2(1) = .04, p = .80$.

Comparison of children's anxiety symptomology and threat bias data across time and between risk groups.

There was no significant Time: $F(1, 40) = 1.59, p = .22$ or Risk: $F(1, 40) = 0.13, p = .75$ main effects, nor significant interaction between Time and Risk: $F(1, 40) = 1.81, p = .19$ on SCAS-P total scores. Similarly, there was no significant Time: $F(1, 41) = 0.12, p = .73$ or Risk: $F(1, 41) = 0.28, p = .56$ main effect or significant interaction between Time and Risk on child threat-AB: $F(1, 41) = 1.6, p = .21$.

Comparison of maternal threat-AB across time and between risk groups.

A mixed repeated measures ANOVA revealed there was no significant Time: $F(1, 40) = 1.13, p = .30$ or Risk: $F(1, 40) = 0.18, p = .67$ main effects and no significant interaction between Time and Risk: $F(1, 40) = 0.02, p = .89$ on maternal threat-AB. One-sample t-tests revealed that for the sample of mothers overall, for T1 there was a significant positive bias towards threat: $t(42) = 2.17, p = .04$, with a mean difference of 8.88 units from zero, whilst at T2 there was no significant bias towards or away from threat: $t(41) = 0.53, p = .60$, mean difference of 2.92 from zero.

Relationship between child and maternal threat-AB.

Pearson correlation coefficients (see Table 2) demonstrated that no significant correlation was detected between maternal or child threat-AB for either T1 or T2 for the total sample or Risk categories. The only significant correlation was between child threat-AB at T1 and maternal threat-AB at T2 for HR ($r = .50, p < .05$). The LMM gave no significant cross-sectional coefficient for either LR group, $F(1, 36.19) = 0.01, p = .93$, or HR group, $F(1, 37.34) = 0.15, p = .70$ and similarly for the longitudinal coefficient for LR, $F(1, 45.13) = 0.13, p = .72$, and HR group, $F(1, 46.06) = 0.16, p = .69$. Children demonstrated no significant bias towards threat at either T1: $t(42) = 0.02, p = .99$ or T2: $t(42) = 0.81, p = .42$. When these analyses were repeated with maternal positive-AB replacing maternal threat-AB, again no significant relationships were detected including the cross-sectional and repeated measures coefficients for either Risk group⁴.

⁴ The cross-sectional coefficient for the HR group was $-0.32 (SE = 0.31, p = 0.32)$. The repeated measures coefficient for maternal positive-AB for this group was close to significant at $-0.72 (SE = 0.37, p = 0.06)$ for T1, giving support to the finding in Waters et al.^[23], however, for T2 the coefficient was much weaker with estimate $-0.15 (SE = 0.30, p = 0.62)$.

Children's anxiety symptoms as a function of child threat-AB.

For preliminary investigation, a correlation matrix and summary statistics were obtained for the sample overall and when split into HR and LR groups (Table 2). No significant correlation was observed between child threat-AB and SCAS-P total at either T1 or T2. Similarly, there was no significant correlation between T1 child threat-AB and SCAS-P total at T2, for either Risk group. As expected, there was a significant, positive correlation between T1 and T2 SCAS-P total scores. There also was a significant negative correlation between child threat-AB for the sample overall between T1 and T2.

The LMM gave no significant cross-sectional coefficient for either LR group, $F(1, 41.32) = 0.01, p = .91$, or HR group, $F(1, 41.32) = 0.03, p = .86$ and similarly for the longitudinal coefficient for LR, $F(1, 42.44) = 0.28, p = .60$, and HR group, $F(1, 42.44) = 0.11, p = .74$. Graphical analysis of random dyad effect estimates and residuals showed no obvious outliers or non-normality.

Children's anxiety symptoms as a function of maternal threat-AB.

A preliminary correlation matrix revealed a significant correlation between maternal threat-AB at T1 and SCAS-P total scores at T2 for the HR group but not the LR group. Interestingly, there was no significant correlation between maternal threat-AB between T1 and T2. As can be seen in Table 3, the linear coefficient for cross-sectional maternal threat-AB score as a regressor was positive and significantly different from zero for only the HR group $F(1, 43.44) = 4.67, p = .036$. There was no significant positive relationship between SCAS-P total scores and longitudinal maternal threat-AB for either group. Table 3 shows that the estimated covariance in the residuals was positive, substantial ($r = .54$) and reasonably precise ($CV = 0.34$). Graphical analysis of random dyad effect estimates and residuals showed no obvious outliers or non-normality. These results suggest that for the HR but not LR

children, the greater the AB towards threat in their mothers, the greater the anxiety symptoms in children at a cross-sectional but not the longitudinal level.

Discussion

This study had several major findings. The most notable finding was that when utilising a comprehensive method of analysing longitudinal data with two time-points (cf. Diggle et al.^[28]), there was a significant cross-sectional but not longitudinal relationship between maternal threat-AB and child anxiety symptoms for high risk but not low risk dyads⁵. The relationship for high risk dyads was established despite there being no differences in threat-AB between the mothers in high and low risk dyads and no relationship between maternal and offspring's threat-AB within each group. Thus, the threat-AB of mothers with emotional disorders was associated with an increase in offspring's anxiety symptoms in a way that was not observed within the low risk dyads.

The absence of significant differences in maternal threat-AB between the HR and LR mothers and the lack of any theoretically logical association between maternal and child threat-AB calls into question what might explain the association between the threat-AB of mothers with emotional disorders and child anxiety symptoms. By virtue of greater emotional distress and symptom expression, one possibility is that mothers with emotional disorders might verbalise more threat information, model more distress, or engage in more distress-enhancing parenting behaviours in response to threat stimuli to which their attention is

⁵ This results confirms Staugaard's (2009) suggestion that the dot probe task is not recommended for "individual differences research", defined here by longitudinal regression coefficients, but was considered useful for research "between groups", defined here by cross-sectional regression coefficients. This regression approach allows this distinction to be investigated since it does not assume that the cross-sectional coefficient is identical to the corresponding longitudinal coefficient (Diggle et al. 2002). Also, it provides a more rigorous estimation procedure by combining the "T1 only" analysis and the "change analysis", incorporating the T2 symptom score on the T1 threat bias regression (Wald et al., 2011), while accounting for within-dyad correlation.

deployed, which in turn, influences offspring anxiety^[37]. Therefore, the likelihood of emotional disorders developing in at-risk children may be influenced by a complex interplay of child and parent variables^[37, 38]. Moreover, in terms of child threat-AB, the neural circuitry underlying threat-AB undergoes significant maturational change during adolescence^[39,40] and failure to acquire adequate control over attention by late childhood/early adolescence also relates to greater threat AB^[41-43]. Offspring in the present study, who spanned an age range of mid- to late-childhood, did not manifest a significant increase in threat-AB themselves over this 12-month interval. Perhaps the risk for child-related threat-AB and their association with anxiety symptoms is conferred via a complex interplay of parent-related risk variables and child-related risk variables that change across development and into adolescence. Further studies are required that explore cross-sectional and longitudinal interactions between multiple parent and child variables over a wider age range in youth.

Some notable limitations include the age range of offspring in the current sample, restricted to mid-late childhood in addition to the small sample size and attrition from T1. As some effects were theory consistent, future studies with larger samples might reveal significant results. Future studies should additionally assess attention control which prior work suggests might moderate links between risk factors and threat-AB across development^[42-44]. Given that the mothers of high risk offspring had high levels of comorbid anxiety and depressive disorders, inclusion of sad as well as angry face stimuli and short and long exposure durations might have strengthened results^[16, 22]. Furthermore, although the inclusion of two time-points of AB assessment extends prior studies^[24-27], some unexpected associations were found within and between child- and maternal- T1 and T2 threat-AB. In particular, the result that a significant negative correlation was estimated between T1 and T2 child threat-AB for the sample overall with this correlation negative and of similar magnitude for both Risk groups. The correlation between T1 and T2 values of AB are in essence a

longitudinal comparison (i.e. within subjects) that is averaged cross-sectionally (i.e. across subjects). It has been noted above that the reliability of ABs obtained by the dot-probe task is adequate for between-group comparisons (i.e. cross-sectionally) whereas it is not recommended for within-subject estimation or comparisons. Therefore, the negative correlation could be an artefact related to greater unreliability of ABs when measured for children^[45]. Other studies have also questioned the reliability of the dot-probe paradigm^[46]. However, the present findings suggest that greater attention to task-related factors (e.g., exposure duration; number of AB assessments)^[16, 47] and contextual factors (e.g., mood induction techniques;)^[19] in future studies might improve reliability.

Conclusion

This study adds to the previous small body of literature on cognitive risk pathways, utilising a novel method of analysing longitudinal data, with the finding of a relatively robust between-subjects relationship between maternal threat-AB and anxiety symptomology in offspring of mothers with a lifetime emotional disorders. These findings stimulate further research into multiple maternal and child variables that influence both maternal and child threat-ABs throughout development and their contribution to the development of emotional disorders in at-risk youth.

Table 1. Demographic information for both Low-Risk and High-Risk groups.

Demographic		High Risk <i>n</i> =24	Low Risk <i>n</i> = 19
Child	Age (M, SD)	9.68 (1.32)	9.66 (1.36)
	Gender Ratio (M:F)	10:14	8:11
	Country of Birth (N, %)		
	Australia	18 (75)	16 (84.2)
	Other	4 (16.6)	3 (15.9)
Mother	Age (M, SD)	42.96 (4.92)	40.79 (11.41)
	Occupational Prestige (M, SD)	4.59 (1.08)	4.02 (1.23)
	Highest Level of Education (N, %)		
	Tertiary	13 (68.4)	19 (79.2)
	Other	6 (31.6)	3 (20.8)
	Employment Status (N, %)		
	Employed	17 (70.8)	14 (73.7)
	Unemployed	7 (29.2)	5 (26.3)
	Marital Status (N, %)		
	Married	17 (70.8)	18 (94.7)
	Not Married	7 (29.2)	1 (5.3)

Table 2. Correlation matrix and summary statistics for maternal and child threat biases and children's anxiety symptoms and initial and follow-up

Time 1				Time 2		
	Maternal Threat Bias	Child Threat Bias	SCAS-P Total	Maternal Threat Bias ^{NB}	Child Threat Bias	SCAS-P Total ^{NB}
Total Sample	1	1	.03	.04	.00	.13
High Risk		-.08	.40	-.07	-.05	.50*
Low Risk		-.03	-.24	.15	.081	-.22
	2	1	.04	.37*	-.35*	-.10
			.40	-.06	-.38	-.16
			-.04	.50*	-.31	-.08
	3		1	.01	-.11	.60**
				.00	-.14	.66*
				.03	-.11	.57*
	4			1	-.14	-.05
					-.04	-.08
					-.25	-.04
	5				1	-.05
						-.01
						-.04
Mean (SD, SE)						
Total (n = 43)	8.88 (27.51, 4.20)	.0996 (37.21, 5.67)	11.93 (7.53, 1.15)	2.92 (35.80, 5.52)	4.78 (38.54, 5.88)	10.55 (7.48, 1.15)
High Risk (n=24)	6.87 (24.68, 5.04)	-3.78 (33.33, 6.80)	12.21 (6.57, 1.34)	1.12 (39.17, 8.17)	11.62 (33.33, 6.80)	9.61 (7.35, 1.53)
Low Risk (n = 19)	11.41 (31.24, 7.17)	4.80 (41.88, 9.61)	11.58 (8.76, 2.01)	5.10 (32.17, 7.38)	-3.88 (41.35, 9.49)	11.68 (7.67, 1.76)

NB: T2 SCAS-P total (Low Risk) and T2 Maternal Threat-AB (High Risk) missing one dyad

* $p < .05$, ** $p < .01$

Table 3. Estimates of fixed effects for linear mixed model predicting child's anxiety symptoms.

<i>Predictors</i>	<i>B Estimate</i>	<i>SE</i>	<i>p</i>	<i>95% CI (Lower, Upper)</i>
Risk				
Low Risk ^a (n = 18 [*])	12.27	1.58	<.001	9.07, 15.46
High Risk-Low Risk (n = 24 ^{**})	-2.49	2.11	.245	-6.75, 1.77
Maternal Threat Bias x Risk				
Cross-sectional x Low Risk	-0.018	0.076	.811	-0.17, 0.14
Cross-sectional x High Risk [*]	0.125	0.058	.036	0.01, 0.24
Longitudinal x Low Risk	-0.014	0.039	.720	-0.07, 0.04
Longitudinal x High Risk	-0.014	0.029	.616	-0.09, 0.06
Variance-covariance parameters				
	<i>Variance (T1)</i>	<i>Variance (T2)</i>	<i>Covariance (T1, T2)</i>	
Estimate (SE)	56.43 (13.01)	51.17 (12.11)	28.82 (9.89)	

^aIntercept^{*}Excluding significant outlier^{**}Mixed model analysis based on estimated value for dyads missing T2 SCAS scores and T2 Maternal Threat Bias.^{*} $p < .05$.

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