Characterising attentional biases to infant related stimuli associated with anxiety during pregnancy

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May 2011

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ABSTRACT

Attention is a key mechanism in the aetiology and maintenance of anxiety, a state commonly found in primiparous women during pregnancy. Recent research indicates an independent casual connection between antenatal anxiety and poor infant outcome, an area where attentional bias towards negative infant information could be an important mediating factor. Using the laboratory based dot probe paradigm (MacLeod, Mathews & Tata, 1986), this study aimed to replicate findings indicating that healthy individuals will selectively attend to infant over adult faces of a neutral expression. Additionally, the study extends previous work by exploring the impact of emotional expressions in infant faces. The possible moderating effects of anxiety levels and pregnancy status were considered. No main effects or interactions were seen in the study. This suggests participants did not show attentional prioritisation to any of the stimuli, regardless of their anxiety or pregnancy status. Implications and possible explanations for the absence of findings are discussed in the context of the antenatal anxiety literature.

Key Words:	Anxiety	Pregnancy	Attention	Dot Probe	Infant Faces

Table of Contents

List of tables and figures

1. Introduction

1.1 Antenatal Anxiety in pregnancy, effects on infant outcome.

Pregnancy is a time of physiological, hormonal and psychological changes. It is important to understand these changes as they are associated with an increased vulnerability to emotional disorders such as anxiety and depression (Andersson, Sunderstrom-Poromaa, Bixo, Bondestam & Astrom, 2006; Ross & McLean, 2006). Large population studies indicate that, at any one time, the prevalence of anxiety disorders (such as generalized anxiety disorder GAD) is increased more than two-fold in pregnant compared with non-pregnant populations (Andersson, Sundstrom-Poromaa, Wulff, Astrom & Bixo, 2003). Longitudinal research provides further evidence of an increase in women's experience of anxiety symptoms during pregnancy compared to pre-conception or after birth (Haas, Jackson, Fuentes-Afflick, Stewart, Dean & Brawarsky, 2004; Lee, Lam, Lau, Cong, Chui & Fong, 2007).

This increased vulnerability is of particular concern because studies over the last two decades provide growing evidence that negative maternal emotions during pregnancy strongly correlate with a range of poorer child outcomes, including a range of cognitive, emotional and behavioural delays in development (O'Connor, Heron & Glover, 2002). This is unsurprising because antenatal anxiety is a strong risk factor for postnatal maternal anxiety and depression. This in turn impairs the mother's ability to bond with and be sensitive to her child, thus increasing risk of child developmental delay (Cooper & Murray, 1998; Bremner, Wachs, Murray, Halligan, Cooper, 2010).

Furthermore, antenatal anxiety appears to have further reaching effects on the child's development. For example, several studies have demonstrated that, even when statistically controlling for the high concordance between antenatal and post-natal psychopathology, antenatal anxiety continues to strongly predict neurodevelopmental delays in the child at the ages of two, four and seven years old (O'Connor et al., 2002; O'Connor, Caprariello, Blackmore, Gregory & Fleming, 2007; Van den Bergh and Alfons Marcoen, 2004).

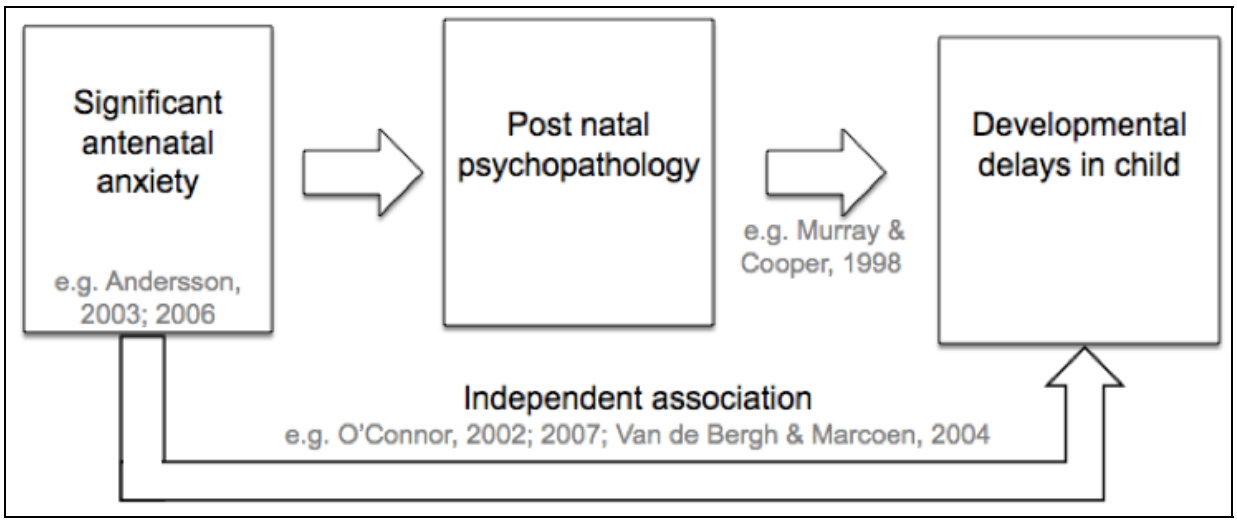

Figure 1: Schematic representation of the possible pathways between antenatal anxiety and poor child outcome

The treatment of anxiety in the antenatal period also holds unique challenges. The safety of pharmacological anxiolytic treatments (prescribed for high anxiety) is controversial, due to suggestions that they may pose a risk to foetal development (Eberhard-Gran, Eskild & Opjordsmoen, 2005; Iqbal, Sobhan & Ryals, 2002). Other methods of treatments such as Cognitive Behavioural Therapy (CBT) can have long waiting lists which, when coupled with treatment length, can be problematic for early intervention purposes preventing the transfer of risk to the child (Spain, 2009).

Thus evidence highlights that clinically significant levels of antenatal anxiety are both common and carry significant developmental risks to the unborn child independently of postnatal psychopathology. Furthermore, the efficacy and safety of the usual anxiety treatments during pregnancy are questionable. Therefore, understanding the mechanisms though which antenatal anxiety affects infant development could elucidate novel areas of intervention, that are both safe and effective to implement, and thus should be considered a key public health priority.

1.2 Mechanisms of risk transfer

Current mechanisms through which maternal antenatal anxiety is related to child developmental delays have been confined to tentative suggestions from a biological viewpoint based on the 'programming hypothesis' (O'Connor, Heron, Golding, Glover, 2003; Welberg & Sekl, 2001). This hypothesis suggests that maternal stress hormones cross the placenta to the womb, signalling a harsh external environment, thus altering foetal development. For example, maternal increases in the stress hormone cortisol leads the foetus to development a particularly sensitive stress response system, known as Hypothalamic Pituitary Adrenal Axis (HPA-Axis). This is an adaptive preparation for a harsh external environment, as it facilitates a faster reaction to danger (Clark, 1998). However, this is not without cost as a sensitive HPA-Axis is also associated with developmental difficulties including an increased likelihood of conduct disorder, ADHD, and other cognitive, behavioural and emotional difficulties (Davis & Sandman, 2010; O'Connor et al., 2007; Talge, Neil & Glover, 2007; Van den Bergh and Marcoen; 2004).

The programming hypothesis, although a theoretically sound explanation of risk transfer, has limited potential for targeted intervention given current technologies. For example,

despite recent epigenetic advances, we are far from gaining the scientific ability to modify HPA-Axis gene expression in humans, that is, effectively 'reprogramming' the 'disturbed' functioning of the HPA system (Meaney, Szyf, Seckl, 2007; Weaver, Champagne, Brown, Dymov, Sharma, et al,. 2005). Furthermore, Oates (2002) cautions that the focus on biologically based theories may lead to clinicians over-prescribing anxiolytic medications during pregnancy. This may cause more harm to the foetus than the mother's anxiety itself. Finally, the biological model, though useful, only partly captures the anxiety experience and only accounts for a portion of the variance between antenatal anxiety and infant development. Investigation of aspects relating to antenatal anxiety may lead to the development of a fuller explanation of the mechanisms involved.

Current theories suggest that biases in cognition, specifically in information processing, are also a proximal part of anxiety illnesses (Beck, 1976; Mathews and Mackintosh, 1998; Mathews and MacLeod, 2005). As such, anxiety related changes in the expectant mother's cognitive profile might also be a mechanism through which antenatal anxiety affects child development. In contrast to original theories (e.g. Beck, 1976) we now understand cognitive biases to be specific to the particular type of emotion experienced (Yeind, 2004). In relation to anxiety, early automatic biases, particularly those relating to attention to threatening stimuli have been most consistently evidenced, and as such will be the focus of this study (Cister & Koster, 2010); Mogg & Bradley, 1998).

1.3. Anxiety, attention and hypervigilance

The close relationship between anxiety and attention to threat has been evidenced in the last fifty years through the development of experimental tasks testing information-processing models. Traditional tasks (such as the emotional stroop task, see Williams, Mathews & MacLeod, 1996) indicate that, in the presence of threatening words or pictures, anxiety interferes with individuals ability to complete the task at hand. These tasks however could not ascertain whether the interference reflects attentional capture processes or alternatively later effortful avoidance of threat processing (De Ruiter & Brosschot, 1994). This methodological limitation was addressed through the design of more sensitive tasks such as the attentional 'dot' probe task (See Macleod, Mathews & Tata, 1986 for task description). This task is superior, due to its ability to take 'snapshots' at any one stage of attentional allocation.

The use of the dot probe task consistently demonstrates that individuals with significant anxiety are understood to be 'exquisitely sensitive' to threat stimuli, continuously seeking out and orientating their attention towards possible threatening stimuli in the environment – a phenomena termed hypervigilance (Browning, Holmes, Murphy, Goodwin and Harmer, 2010; Williams, Watts, MacLeod and Mathews, 1997). As with many aspects of high trait or clinical anxiety, this hypervigilance to threat is understood to stem from a traditionally adaptive mechanism, which, in excess can be severely debilitating for the individual (Ledoux 2000; Ohman and Mineka, 2001; Yeind, 2004). Several models demonstrate the influence of individual differences in trait anxiety, where high anxiety causes a continuous redirection of attentional resources to threat stimuli, hindering pursuit of the individuals current goals (see Appendix 14).

Furthermore, hypervigilance has been understood to be not only a symptom of anxiety, but proximal to its development, exacerbation and maintenance (Browning, Holmes & Harmer, 2010). Indeed, Williams (2008) highlights this bi-directional relationship,

suggesting a vicious cycle whereby worry increases threat orientation, which in turn further promotes worry. Similarly, the 'vigilance-avoidance' hypothesis suggests that anxiety related vigilance, causes further increased anxiety about the threat stimuli. In order to attenuate these anxiety emotions, the individual then strategically avoids further elaborative processing of threat (Derakshan, Eysenck, & Myers, 2007). Paradoxically, this vigilance-avoidance pattern only further promotes anxiety as strategic avoidance prevents habituation and objective evaluation of the anxiety provoking material (Mogg, Bradley, Miles, & Dixon, 2004).

1.4. Potential mediating and moderating factors: anxiety and hypervigilance

Although the relationship between anxiety and hypervigilance has been evidenced in a range of populations for a number of different stimuli, there are other possible factors that impact this relationship which are important to consider.

Key to the models above is the notion that attentional hypervigilance is dependant upon the subject matter of the worry being perceived as threatening and thus anxiety provoking for the individual (Ohman, Flykt & Esteves, 2001). Evidence from phobia literature illustrates this. For example, individuals with arachnophobia demonstrate hypervigilance specifically towards spider-related but not other threatening words and images, and those with social phobia show similar biases specifically to face stimuli (Mogg and Bradley, 2002, 2006). Thus, when considering extrapolation from a generalised anxiety model to individuals with antenatal anxiety, it is important to consider the possible specific nature of the worries within a sample.

Work by Broach Sander, & Scherer (2007) suggests that it is not only threat stimuli that result in attentional capture. In line with Lorenz's assertion that the perceptual features of infant faces promote attention, Broach et al., (2007) findings suggest that human infant faces cause attentional capture over other face types, such as human adult and animal (adult or infant) faces in healthy volunteers. Further neuroimaging studies promote the understanding that infant (compared with adult) faces are associated with increased activation in neural areas (e.g. amygdala) associated with attention (Kringelbach, Lehtonen, Squire, Harvey, Craske, & Holliday et al., 2008). From this it is suggested that the biological salience of a stimulus can also influence attentional engagement, which should be considered in relation to attentional capture.

Finally, large epidemiological studies suggest that anxiety and depression display high co-morbidity within a population possibly due to the degree of symptom overlap (Clark and Watson, 1991). With regard to attentional engagement, it is not yet clear how anxiety and concurrent depression interact. Experimental evidence supports a double dissociation between the affective disorder and attentional bias, with anxiety associated with early stage attentional biases, whereas depression relates to elaborative processin biases (Williams, Watts, MacLeod & Mathews, 1988). Therefore, when investigating attentional capture, the expected effects of co-morbidity in the sample would be negligible, as indicated by a recent meta-analyses (Bar-Haim, Lamy, Akermans-Kranenburg & van Ijzendoorn, 2007).

Conversely, some studies indicate that in co-morbid populations the early attentional biases usually expected with anxiety are no longer present (Mogg, Bradley, Williams & Mathews, 2003). These authors, along with Fox, Russo, Bowles, and Dutton (2001) refer

to the phenomenology of different affective disorders in explanation as depression is understood to be a-motivational, whereas anxiety is highly motivational in nature. Due to this polarisation, depression co-morbidity might therefore inhibit the motivation-based orientation to threat that characterises anxiety. Because the debate remains contentious, co-morbidity in samples is important for an accurate interpretation of attentional biases in anxiety.

1.5 Can we apply generalised anxiety models to antenatal anxiety?

Having established the importance of hypervigilance to perceived threat in anxiety, it is natural to suggest that attentional biases would also be associated with anxiety during pregnancy. Furthermore, the specificity of attentional biases according to the individuals concerns, together with biologically salient stimuli also promoting attentional allocation, it is conceivable that women, particularly during pregnancy, would preferentially attend to infant face stimuli.

Given the central placement of attentional biases in the development, maintenance and exacerbation of anxiety, hypervigilance becomes a primary area of exploration in relation to the antenatal anxiety-developmental delay pathway. Thus the question becomes whether pregnancy brings with it any innate complications for the application of this generally observable anxiety bias?

Recent theories suggest that pregnancy itself may lead to neurological and corresponding cognitive changes. High levels of Oestrogen and Oxytocin during pregnancy may activate structural and cognitive re-organisation of expectant mothers brain and cognitive processing in such a way that prepares them for motherhood (Glynn, 2010). These maternal adaptations have been termed ‘the construction of the maternal brain’ (Kim, Leckman, Mayers, Feldman, Wang, & Swain, 2010). Due to the novelty of this theory, there is little work to date which directly characterises the specific nature of these prenatal adaptations however, initial evidence indicates that attention biases to infant faces may play a role.

Certainly, neurological research documents attentional related differences between mothers and non-mothers when viewing infant stimuli (Kringelbach, Lehtonen, Squire, Harvey, Craske et al., 2008; Proverbio, Brignone, Matarazzo, Del Zotto, & Zani, 2006). However, currently it is unclear whether these neurological differences are activated by hormonal changes in pregnancy, or are a feature of rapid post-natal learning, thus they are not direct evidence to support the construction of the maternal brain prenatally.

More promising in this respect are experimental findings documenting a series of cognitive and physiological differences when viewing infant stimuli during pregnancy (Pearson, Cooper, Penton-Voak, Lightman & Evans, 2010; Pearson, 2010). This includes pregnancy related differences in attentional disengagement, that is, pregnant women spend a longer time looking at distressed infants than women who have never been pregnant. (Pearson, Cooper, Penton-Voak, Lightman, Evans, 2009; Pearson, 2010). Furthermore, postnatal follow-up suggests that this antenatal attentional disengagement to distressed infant pictures, directly predicts mothers ability to bond with their child three months postnatally (Pearson, Lightman & Evans, 2011).
From these findings, she suggests there is an evolutionary based, heightened activation of baby schema that occurs during pregnancy, which aids the mother in bonding with

their baby after birth. This baby schema is modulated by emotion on the baby's face, as a mother's ability to respond to her infant's distress is particularly important in promoting protective and nurturing behaviours (Pearson, 2010; Pearson et al., 2011).

To summarise, there is some evidence to support Kim et al. (2010) theory of the maternal brain, particularly in relation to attentional delayed disengagement when viewing infant stimuli. As a result, caution should be taken when attempting to directly apply a generalised understanding of anxiety related cognitive biases to pregnant populations, as currently we do not know how attentional biases in anxiety interact with the development of pregnancy related biases. Given this evidence it is important to investigate whether antenatal anxiety disrupts the development of cognitive processes promoting maternal behaviour, and therefore may also present as a mechanism through which antenatal anxiety increases risk of child developmental delays. As Kinsley and Meyer, speculate, "If the maternal brain does not develop the way it should…does that produce a bad or indifferent mother?" (2010, p710).

1.6.The effect of anxiety on the maternal brain

As well as investigation of the development of the maternal brain during pregnancy, Pearson (2010) investigates the possible moderating effects of depression and anxiety on this process. Depressive symptoms in early pregnancy (12 weeks gestation) were associated with faster disengagement from distressed infant faces, a pattern that is opposite to that developing in non-depressed pregnant women. Furthermore, postnatal follow up indicates that this disrupted cognitive profile predicts a corresponding decrease in the mother's ability to bond with their child three months after birth. This finding remains significant even when the mothers' postnatal psychopathology is controlled for (Pearson et al., 2009; 2010). From this, it is suggested that antenatal depression may impair the normal development of the maternal brain and mothers' ability to nurture their distressed child.

Pearson (2010) also investigated bias related to anxiety, but found no evidence that anxiety affects attentional disengagement processes. However, as already discussed, there is a double dissociation between the type of emotion experienced and the stage of attentional processing affected. Anxiety relates to engagement, not disengagement biases, therefore the absence of an effect of anxiety on disengagement is not surprising. No research to date has investigated attentional hypervigilance to emotionally valenced infant stimuli, the attentional bias most closely associated with models of anxiety, during pregnancy. An attempt to address this gap in the literature thus forms the basis of the current study.

1.7. Summary and Aims:

Work in the general population (with unspecified parity) suggests that individually both negative face emotions and infant faces are attentionally prioritised. As such this would be expected to be the case in this study. Thus my first hypotheses are as follows: There will be a general attentional capture effect to infant over adult faces and to emotionally negative compared with emotionally positive or neutral infant faces.

Additionally, based on cognitive models anxiety, which suggests that high anxiety relates to exaggeration in attentional prioritisation perceived threat, along with the

understanding that biologically salient stimuli (e.g. infant faces) are generally attentional prioritised, the second hypothesis is: Individuals with high trait anxiety will display hypervigilance towards distressed infant faces over distressed adult or neutral/happy infant faces.

Previous work indicates that pregnancy is associated with changes, such as attentional disengagement, that are adaptive in preparing the mother for protective and nurturing behaviours toward her infant after birth. In the same way, increased attentional engagement could aid the mother; increasing their sensitivity to their child. Thus the third hypothesis is that currently pregnant women will also display attentional hypervigilance to distressed infant faces.

Finally, given that these hypotheses predict that both anxiety and pregnancy may be associated with similar attentional prioritisation processes to distressed infant faces, it is likely that these factors will interact. Therefore the final (fourth) hypothesis is: There will be an interaction between anxiety and pregnancy whereby both factors together cause the most pronounced pattern of attentional vigilance.

This extreme vigilance, I propose may be a factor that links antenatal anxiety with poor developmental outcomes for the child because the adaptive mechanism of hypervigilance, in extreme, may become maladaptive, increasing the mothers anxiety, while at the same time preventing her tending to her child in accordance with theories such as the vigilance-avoidance hypothesis.

2. Method

2.1 Design

The study used a between groups design consisting of four groups. The between groups factors were pregnancy status (Primiparous women, n=26, never been pregnant, n=26) and anxiety levels (Low anxiety; Anxiety Symptoms). The within-group factors were the face-pair valence (Negative/Neutral vs. Positive/Neutral) and the face-pair age (Infant-Infant vs. Adult/Adult). The dependant variable is the response time latency.

2.2. Participants

26 primiparous women (currently pregnant for the first time) were recruited from the John Radcliffe Hospital Women's Department when arriving for their 12-15week scan. These women along with 26 nulliparous women (not currently and have never been pregnant) were also recruited via local newspaper advertisements; websites (http://www.psych.ox.ac.uk); posters and leaflets in public areas. All participants were female (mean age 30.0years; SD= 5.86).

Participants were split into 'low anxiety' and 'anxiety symptoms' groups based on a median split of participants according to their self-reported trait anxiety levels on the STAI-T questionnaire (Speilberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983, see measures). Those falling on the median (STAI-T=34) were excluded. This cut off is in line with previous literature (e.g. Mogg, Holmes, Bradley & Garner, 2008). Participants scoring below the samples median anxiety were grouped as 'low anxiety' (STAI-T= <34). Participants scoring above the sample median were classified as the 'anxiety symptoms' group (STAI-T=>36). These respective category descriptions were chosen due to their clarity aiding understanding; however, a more accurate description of groupings may be 'low anxiety symptom experience' and 'significant anxiety symptom experience' respectively, due to measurement along a continuum.

Exclusion criteria for all participants included: previous pregnancy; insufficient fluency in English to understand task instructions; current diagnosis of psychiatric illness and if women were less than 11 or more than 17 weeks pregnant. Two never been pregnant participants in the anxiety group were excluded from analysis as they were currently taking medications (Amitriptyline, and Propranolol) that have known effects on reaction time data (see Horder, Cowen, Simplicio, Browning and Harmer, 2009; Murphy, Yeind, Lester, Cowen and Harmer, 2009). Furthermore there were baseline differences in their reaction time data on filler trials (where two identical faces are displayed). For total groupings see table 1.

2.3. Materials

2.3.1.Self-report questionnaires

State-Trait Anxiety Inventory – trait version. (STAI-T; Spelberger, Gorsuch, Lushene, Vagg & Jacobs, 1983. Appendix 11). Trait anxiety is "the individual difference in anxiety proneness as a relatively stable personality trait" (Speilberger, Pollans and Worden, 1984, p.276). The STAI-T is a twenty item self-report questionnaire designed to yield a composite score of the individual's trait anxiety level. Items are measured on a four-point likert scale, which ranges from one (not at all) to four (very

much). The STAI-T also contains several anxiety absent items (e.g. I am content) that are reverse scored. The STAI-T has good internal validity with a mean alpha coefficient of .90 (Barnes, Harp, & Jung, 2002).

Edinburgh Post Natal Depression Scale (EPDS, Cox, Holden & Sagovsky, 1987, Appendix 12). The EPDS is a ten item, self-report questionnaire designed to screen for perinatal depression. Due to the possible effects of comorbid depression on anxiety related RT's the EPDS was included as a measure of depressive symptoms. This scale was chosen (e.g. Beck Depression Inventory, Beck et al., 1961) due to its focus on the psychological aspects in depression, as many of the physical symptoms of depression, such as changes in appetite and weight, are normal features during pregnancy. Each Item is scored on a likert scale from 0-3, yielding a total range of 0-30. Cut off scores to classify depression have varied in the literature from >9 to >12 (Matthey, Henshaw, Elliott, & Barnett, 2006).

Prenatal Pregnancy Anxiety (PPA, Rini, Schetter, Hobel, Glynn, & Sandman, 1999, Appendix 13). The PPA is a 10 item self report questionnaire assessing pregnancy related anxiety levels using a 4 point likert scale with possible responses for each item ranging from 1 (never or not at all) to 4 (almost all of the time or very much). Again, items were reversed and scores summated so that a higher score represents higher level of prenatal pregnancy anxiety (Rini, et al. 1999).

2.3.2. Attentional Probe Task

Face Stimuli*:*
Adult face stimuli displaying happy sad and neutral expressions were taken from the Ekman database of adult faces (Ekman & Friesen, 1975). Infant face stimuli displaying happy, sad and neutral expressions were taken from a database developed for a previous study. Parents gave permission for their children's faces to be added to the database for use in future studies using visual imagery (Kringelbach et al. 2008). The database contained pictures of 27 infants (aged 3-12 months) each displaying a happy, neutral and distressed facial expression (figure 2).

Figure 2: Example Stimuli in the attentional probe task taken from Ekman, (1975) and Kringelbach (2008) databases

All face pairs used were matched for size and luminosity, minimising possible confounding effects due to the visual appearance of the faces (a brighter or bigger picture could draw attention). Elements such as head direction and eye gaze were also controlled for (Ekman & Friesen, 1975; Kringelbach et al., 2008). The photographs were greyscale and 30x30mm in size when presented on the screen. One photograph in the pair was presented 30mm above the central fixation cross, the other 30mm below.

2.3.3. Task

The selective attention task used was a modified version of the visual 'dot-probe' task (MacLeod and Mathews, 1988). Participants were initially presented with a fixation cross in the centre of the computer screen for 500ms. Immediately following this, face pairs were presented, one above, the other below the fixation point for 100ms. The face pairs were then removed. In the location of one of the faces either one or two dots appeared (i.e ● or ● ●). Participants were required to report the number of dots seen via a key press of one of two keys labelled with one or two dots (labelled keys were in the position of the letters B and M on a British keyboard). The dots remained on the screen until participants made their response (see Figure 3). A blank screen remained for 500ms before the next trial began.

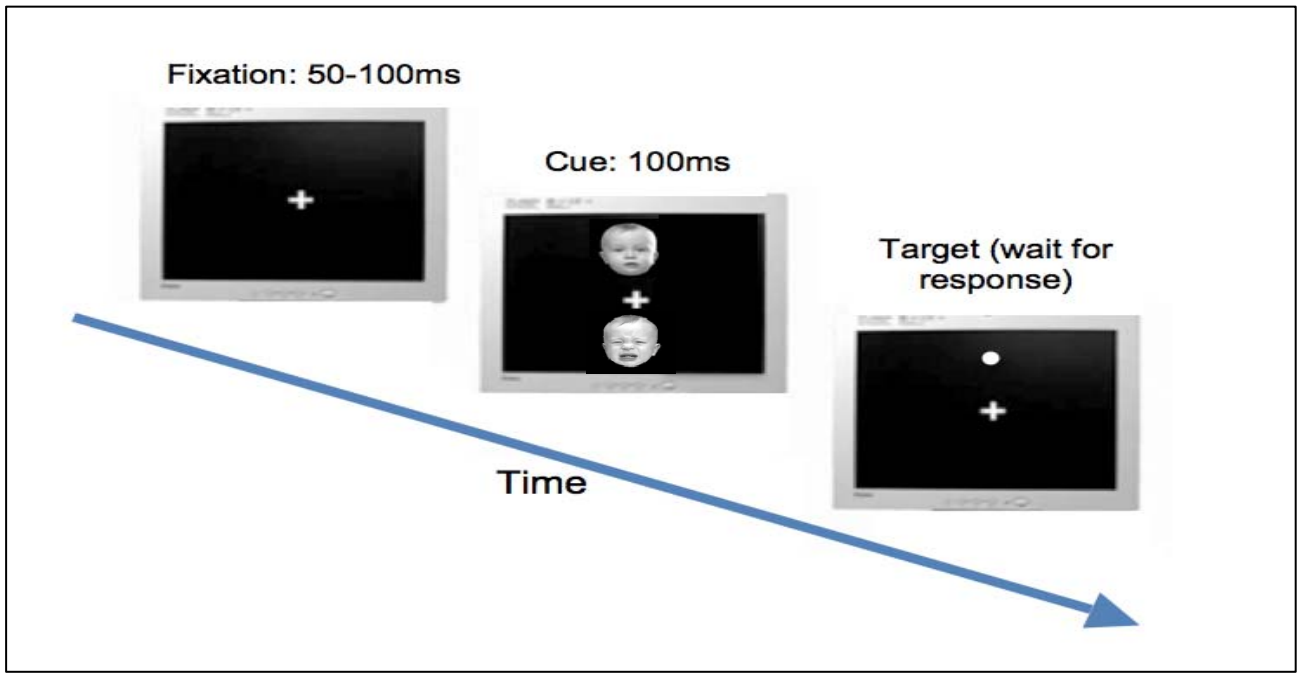

Figure 3: Schematic representation of a singular trial in the attentional probe task completed by study participants

Infant faces (happy vs. sad)

There were eight blocks of trials in which only infant face pairings were presented. In each block there were four happy-neutral trials and four sad-neutral trials. Additionally, there were two filler trials in each block where two neutral infant faces were displayed (neutral-neutral) in order to give a baseline measure of reaction time.

Adult faces (happy vs. sad)
In total there were 8 blocks of trials (ten trials per block) in which only adult face pairings were presented. In each block there were four happy-neutral trials and four sad-neutral trials. There were two filler trials in each block, where two neutral adult faces were displayed (neutral-neutral) in order to give a baseline measure of reaction time.

All face types/trials
On half of the trials, the probe appeared replacing the emotional faces (congruent) and on the other trials the probe replaced the neutral face (incongruent). From this an attentional bias index can be created (see results) which indicates whether the participants prioritised (were vigilant toward) the emotional stimuli.

The emotional face, as well as the following probe could be presented in either the upper or lower location, which was randomly controlled by the computer. This resulted in four possible presentation conditions, which occurred an equal number of times to avoid expectancy biases. Within every block, the trials were presented in a random order according to a computerised random selection, therefore preventing any order effect.

2.5 Procedure

After initial consent was taken, participants were given both verbal and on-screen instructions and asked to complete the attentional probe task. As can be seen in Appendix 6 participants were asked to complete the task as fasts as possible while not compromising on accuracy, and were given three evenly spaced breaks the length of which was under the participants control. The task took around 20 minutes to complete. After the dot probe task, participants were asked to complete the STAI-T and EPDS questionnaire measures, and other demographic information was recorded. Additionally pregnant participants took part in a battery of other tasks not reported here (See Appendix 7.). Participants were debriefed at the end of the study.

3. Results

3.1 Statistical Treatment

All analyses of data were conducted using Excel and SPSS V18. An alpha level of .05 was set for all statistical tests. All attentional bias indexes for groups according to pregnancy and anxiety were tested for normal distribution and no violations were found.

Several items on the STAI-T (1, 2, 5, 8, 10, 11, 15, 16) and the EPDS (1, 2, 4) were reversed before data was analysed. Where participants missed out items (<2), the mean value according to the particular scale and age group was inputted. No participant omitted >1 item on any one scale. Participants were grouped for analyses according to pregnancy status and anxiety (see methods).

3.2 Participant Characteristics

A summary of participant characteristics can be found in table 1. The groups appeared well matched according to age, and as expected, due to anxiety grouping procedures, differed in mean STAI-T scores. Pregnant groups were of a similar gestation at the time of testing, and the anxious group appeared to have a slightly higher prenatal pregnancy anxiety score (PPA). A between-groups analysis of variance was conducted with anxiety and pregnancy as the independent variables on participant characteristics, to investigate the significance of any differences between groups.

Table 1
Participant demographics by pregnancy (nulliparous vs. primiparous) and anxiety (anxiety symptoms vs. low anxiety group)

	Nulliparous Low Anxiety		**Nulliparous Anxiety Symptoms**		**Primiparous Low Anxiety**		**Primiparous Anxiety Symptoms**		**Total**	
		SD		*SD*		*SD*		*SD*		*SD*
Number *N*	13		11		13		13		50	
Age *Yrs*	30.8	7.31	29.8	6.91	31.1	4.51	32.3	4.70	31.0	5.8
EPDS *M*	2.92	2.75	7.18	5.36	3.84	2.60	8.38	4.86	5.52	4.5
STAI-T *Mdn*	28		37		28		44		34	
Axis I History *N*	1		0		0		3		4*	
PPA *M*	N/A		N/A		3.91	4.93	12.16	2.19	10.73	4.0
Gestation *Wks*	N/A		N/A		15.6	1.00	16.0	0.90	15.65	0.9

Nulliparous, never been, and not currently pregnant; Primiparous; currently pregnant for the first time; STAI-T, State-Trait Anxiety inventory-Trait version; EPDS, Edinburgh Postnatal Depression Scale; PPA, Prenatal Pregnancy Anxiety. *Significant differences between groups (p=<.05).

A main effect of anxiety grouping was found for STAI-T scores, as expected given group classification procedures (F (3,46) = 26.65, p<.001). Additionally anxiety groups differed in their experience of depressive symptoms according to EPDS scores (F(3, 46) = 14.93, p=<.001). Simple effects analysis revealed that there were significant differences between the anxiety symptoms and low anxiety groups (t(48) = -3.96, p=<.001), with participants in the anxiety symptoms group experiencing higher levels of depressive symtomatology, suggesting co-morbidity within the group. No differences were found according to general demographic information such as age (p=>.05).

Differences according to anxiety groupings (low vs. anxiety symptom) in the pregnant sample was somewhat associated with the prenatal pregnancy anxiety questionnaire (PPA) (F(2,23) = 3.60, p=.07). Simple effects analyses revealed that the anxiety symptom group is associated with higher prenatal pregnancy anxiety than the low anxiety group (t(2,23) = -1.90, p=.60). There was no between group differences in pregnant participants' level of gestation, their reported desire to have children or whether the pregnancy was planned (p=>.05).

Pearson correlation analyses were conducted between the questionnaire measures (Table 2). As suggested from the mixed groups ANOVA, the anxiety and depression measures (STAI-T and EPDS) were positively correlated r(48) = .73, p = <.001. However, it should be noted that PPA did not correlate with STAI-T (r(24) = .32, p=.11). This suggests that specific pregnancy-related anxiety shows discriminate validity from general trait anxiety.

Table 2
Correlations between questionnaire measures

Scales	STAI-T	EPDS	DC
EPDS	.728**		
PPA (Primiparous only)	.320	.270	
DC (Primiparous only)	-.053	-.157	-.130

**p=<.001; ; Primiparous only, questionnaire only completed by women currently pregnant with their first baby; STAI-T, State-Trait Anxiety inventory-Trait version; EPDS, Edinburgh Postnatal Depression Scale; PPA, Prenatal Pregnancy Anxiety; DC, Desire to have children.

3.3 Attentional probe task

Reaction times (RT's) from the attentional probe task for each participant were collected. Data with errors (one dot key was pressed when two dots were on screen and vice versa) accounted for 0.96% of total data, were discarded and not analysed further. In line with procedures commonly used when analysing attentional probe data, RT's below 100ms or above 2000ms were also excluded as were RT's over 3 standard deviations from the block mean RT for each participant. This resulted in a total of less than 4% of the data being removed.

Table 3

Mean attentional probe response times ms by age adult vs. infant, emotional valence sad vs happy, and congruency congruent vs. incongruent by pregnancy and anxiety groupings

	Primiparous Low Anxiety		**Primiparous Anxiety Symptoms**		**Nulliparous Low Anxiety**		**Nulliparous Anxiety Symptoms**	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Infant								
Sad Congruent	519.53	36.66	543.90	68.95	486.10	43.03	541.70	51.56
Sad Incongruent	510.77	35.01	527.11	52.31	488.42	35.39	532.48	46.63
Happy Congruent	520.95	47.62	538.85	68.35	500.03	31.30	540.05	53.49
Happy Incongruent	510.89	35.01	528.23	66.58	494.49	36.07	524.57	45.06
Filler	519.67	64.00	481.86	34.17	524.27	57.06	538.71	40.30
Adult								
Sad Congruent	509.23	52.38	521.87	71.11	491.93	28.56	538.26	64.16
Sad Incongruent	513.10	62.28	523.44	62.23	493.28	42.99	530.73	37.69
Happy Congruent	511.05	36.90	537.52	67.90	485.45	30.17	546.53	60.76
Happy Incongruent	509.79	39.17	531.27	79.07	494.49	36.07	529.23	39.43
Filler	532.18	63.00	498.62	32.12	533.71	61.99	536.35	48.82

Primiparous, currently Pregnant with first baby; Nulliparous, Never been pregnant; Sad, Sad/Neutral face pairs; Happy, Happy/Neutral face pairs; Infant, Infant/Infant face pairs; Adult, Adult/Adult face pairs; Congruent; Where the dot/s appear behind the emotional face; Incongruent; Where the dot/s appear behind a neutral face; Filler, trials with both faces of a neutral expression.

A pattern was expected whereby both adult and infant ‘sad’ face trials would show faster RT’s in congruent compared with incongruent trials (indicating vigilance toward the emotional face). Furthermore, this pattern was expected to be more pronounced if participants were either anxious or pregnant. However, as can be seen in table 3 no obvious pattern can be discerned visually.

There were no significant differences between the group’s mean RT on filler trials (where both faces in the trial were identical) indicating homogeneity of baseline RT scores across groups ($F(46,3) = 2.209$, p=.>.05). This allows a method commonly used in the attentional probe literature to aid interpretation of results: the calculation of an attentional bias index. This is calculated through the subtraction of the mean RT when the emotional face and probe were in the same position (congruent trials) from the mean RT of trials when the emotional face and probe were in different positions (incongruent trials) (MacLeod & Mathews, 1988). Positive scores indicate a shift in attention towards the infant face thus attentional vigilance, and negative values suggest attentional shift away from the emotional face, indicating attentional avoidance of emotional face / attention towards the neutral face.

A repeated measures ANOVA was conducted on the attentional bias indices. Pregnancy status and anxiety grouping served as the between-groups factors, and

the face-pair valence (negative/neutral vs. positive/neutral), and age of face-pair (adult vs. infant) were the within groups factors. No significant main effects of face-age or face emotion were found (*p*=>.05). Furthermore, no interactions were found for either anxiety or pregnancy, or between anxiety and pregnancy (p=>.05). See Appendix 15 for a complete list of findings.

3.4 Pregnancy Specific Anxiety

The repeated measures ANOVA was re-run with the currently pregnant sample with scores according to pregnancy specific anxiety entered as a continuous variable. However, no significant results were found (*p*=>.05).

3.5 Correlation Analyses

In order to investigate the impact of depression on the relationship between trait anxiety (STAI-T scores) and the attentional bias indexes, partial correlation analyses were conducted. No significant correlations between STAI-T scores and any of the attentional indices were found indicating that anxious participants concurrent symptoms of depression were not impacting on the relationship between anxiety and attentional bias indexes.

4. Discussion

It can be seen from the results that, contrary to the hypotheses, there were no significant main effects of attentional vigilance according to the face-age or emotion expression, or any interactions between these factors according to participants' anxiety grouping and pregnancy status. Thus, in every case it was not possible to reject the null hypotheses.

The lack of a main effect for face-age and face emotion was particularly relevant to the overall interpretation of the results. These findings are contradictory to two well-established effects within the literature, whereby infant faces and faces with negative emotions capture participants' attention (Mathews & MacLeod 2005; Broach; 2008). Post-hoc power analyses, using the program G*Power, was conducted to investigate whether the study had sufficient power to detect main effects of face-age and emotion (Faul, Erdfelder, Lang, & Buchner, 2007). These indicated the power to detect a main effect was .94, which is adequate according to Cohen's recommendations of a minimum of .80 (Cohen, 1998). Thus, a type II error is unlikely to have occurred, therefore the absence of the effect appears to be a 'real phenomena' within the constraints and characteristics for this study.

Possible reasons for the lack of main effect along with expected interactions according to anxiety and pregnancy will be considered. Issues discussed include stimuli used; task design; and participant characteristics.

4.1 Hypotheses 1: General attentional capture

Contrary to hypotheses, findings suggest that negatively valenced faces are not associated with attentional vigilance in comparison to faces with neutral or positive emotional valence. Additionally there was no main effect of face-age, indicating that infant faces were not prioritised over adult face stimuli. This contrasts with previous literature, suggesting that resource allocation toward potentially threatening stimuli is a generally observable evolutionary adaptive mechanism, aiding survival (Ledoux 2000; Ohman and Mineka, 2001). Furthermore, biologically salient stimuli, such as infant faces, have previously been argued to capture attention for reasons of maximising reproductive success, and the maintenance of genetic material (Brosch et al., 2007).

4.1.1. Stimulus factors – negative but not threatening

One explanation for these findings could be due to the negatively valenced face stimuli being negative but not intrinsically threatening. According to Mogg and Bradley's (1998) model, this is important, as the interpretation of the aversive stimuli as threatening is the contingent factor from which allocation of attention toward the threat is determined. This study used distressed infant and sad adult, rather than more obviously threatening face stimuli such as angry adult faces.

Reasons for the choice of distressed infant faces were practical, as there is no current database displaying angry infant faces, and theoretical, because previous research has investigated distressed rather than angry infant faces. Previous research suggests that a distressed infant face could signal an evolutionary threat to an infant's wellbeing, as such the development of hypervigilance to distressed infant stimuli during pregnancy would be adaptive in preparation for successful maternal sensitivity and care behaviours postnatally. However, this argument assumes that

the expectant mother interprets unfamiliar infant face stimuli in a similar manner to how they would react to their own baby after birth. This discrepancy is large, and therefore the study may have low ecological validity in relation to the actual attentional biases pattern women may display to their baby postnatally. Viewing pictorial face stimuli rather than a video of, or an actual baby, further reduces ecological validity. Thus pictures of unfamiliar distressed infant faces may not be interpreted as conveying an evolutionary threat to ones own child and therefore may not elicit the attentional biases expected.

In addition, sad, rather than angry adult faces were chosen due to their closer perceptual similarity to the distressed infant faces. A reasonable match between adult and infant faces allows between groups comparison to face-age. However, as with distressed infant faces, sad adult faces may not produce a high threat evaluation critical in Mogg and Bradley's (1998) model. Although some literature suggests that all negative emotions display a similar attentional capture effect, findings are mixed (e.g. Smith, Cacioppo & Chartrand, 2002). Thus the type of negative facial expression used may be critical to the absence of attentional prioritisation of negative stimuli.

Further research may wish to use a series of emotionally manipulated faces, such as chimeric or computer-interpolated (morphed) faces with varying degrees of sadness/distress compared to anger or neutrality. These tasks have the potential to discern the face types considered as threatening and demonstrate the degree to which the threat compared to a more general negative evaluation of the face is important for producing attentional vigilance effects. This is especially important with the infant face stimuli, because no research has investigated whether angry yet alone distressed infant faces are perceived as threatening.

4.1.2. Task design explanations

Competing evolutionary mechanisms

Previous findings suggest an attentional disengagement pattern specifically to distressed infant faces (not distressed adult or happy infant faces). However, the fact that two evolutionary mechanisms are at play within a single trial in this study may have resulted in an unexpected interaction. It is possible that the presence of an infant face competes with and over-rides the effects of the face's emotional salience, or vice versa, where the emotional salience masks the effect of face-age.

First, it is possible that the presence of two infant faces on the screen interfered with any usual attentional prioritisation effects expected due to the emotional expression of the face. However, this argument is somewhat attenuated, as findings in adult trials also found no effect of emotion, suggesting that the age of the face did not mask any effects of emotional expression.

Second, it is possible that the presence of emotionally valenced faces on screen masked any effects due to face-age. This report focuses on trials where the within-task effect of emotion is directly examined, whereas effects of age are inspected via between trials comparisons. However, the full attentional probe task administered to participants also included trials where one adult and one infant face, both with matched emotional expressions appearing simultaneously, which allows for direct within-trials analyses of the effect of face-age, and across trials analyses of

emotional expression. Although not central to the primary aims of this report, given these findings it is important for future work to analyse these trials. This is because, inspection of infant/adult trials would elucidate whether the presence of an emotional expression on screen masked any effects of face-age.

Stimulus duration

Based on previous studies it is reasonable (e.g. Mogg et al., 2004) to suggest that the attentional probe task used was sensitive enough to capture initial engagement prioritisation. However, it is still possible that the effects were confounded due to participants' ability to process both faces simultaneously or in quick succession within 100ms time frame. Prior investigations of stimulus duration involved words rather than pictorial stimuli (Cisler & Koster, 2010). Furthermore, infant and emotional faces are especially salient (Broach, 2007; Ohman & Mineka, 2001) thus it is possible that negatively valenced infant face stimuli activate automatic preconscious processing. This could allow a degree of processing of both the stimuli on screen, therefore confounding results. Indeed, Kringelbach (2008) investigated neurological activation in mothers and non-mothers using these exact stimuli. His findings suggest very early activation (within 14ms) of known automatic, bottom up pathways to the amygdala, supporting this argument.

Future research replicating this study with the addition of eye tracking equipment could identify whether 100ms is sensitive enough to discern initial attentional capture, yet prevent movement of eye gaze to other stimuli. Alternatively, the attentional probe paradigm could be used with the manipulation of stimulus duration to include several time-points of stimulus exposure, from preconscious subliminal priming (>17ms) to disengagement (<500ms). This would allow mapping of attentional biases to these stimuli at different processing stages and could identify areas whereby pregnancy and anxiety are likely to moderate this association.

4.2 Hypotheses 3: Anxiety

Results also found that anxiety groupings did not moderate attentional capture. Again this contrasts with previous literature as well as cognitive anxiety models suggesting attentional biases in anxiety (see Cisler & Koster, 2009; Mogg & Bradley 1998 for reviews; Browning, Holmes and Harmer, 2010).

As anxiety related hypervigilance is understood to be an exaggeration of typical responses in the general population, the same arguments considered in relation to hypotheses 1 also can be applied to the absence of main effects or interactions by anxiety groupings. These arguments, and other possible factors that may account for the absence of the expected effects, such as the sample characteristics, will now be discussed.

4.2.1. Stimulus factors

The difficulty in determining the perceived threat value of the face types is also important when considering the impact of anxiety on attentional capture. Unlike depression, where attentional biases relate to generally negative stimuli, anxiety is specifically related to biases to threatening stimuli (Williams et al., 1997). If distressed infant faces are perceived as generally negative but not threatening, an effect of anxiety may not be present.

4.4.2 Sample Characteristics

Trait anxiety groupings

Due to pragmatic difficulties in obtaining data from pregnant women, a median split (35) of women's trait anxiety scores was used. Though this cut off is in line with previous studies (e.g. Mogg, Holmes, Garner & Bradley, 2008) it is possible to argue that absence of expected findings could be due to the homogeneity of the groups. However, entering trait anxiety scores as a continuous variable (rather than dividing the groups according to anxiety) did not alter significance. This indicates that the median split, thus the homogeneity, of the two groups did not cause a type II error obscuring any anxiety effects. A measure of specific pregnancy related anxiety was also included given that women specifically anxious about the health and care for her baby may exhibit the expected biases. The findings suggested that this relatively new measure was not correlated with trait anxiety as expected and did not bare any relationship to attentional biases, further research developing and testing measures of anxiety about the baby antenatally would help in establish whether specific worries about the baby may hold a stronger relationship than trait anxiety.

State-Trait anxiety interaction

One limitation of this study was the inability to investigate interactions between state and trait anxiety levels. This could be a potentially confounding variable as some models of anxiety promote the 'interaction hypothesis' (Broadbent and Broadbent, 1988; Eysenck, 1992). This postulates that in the absence of stress, there may be little difference between high and low trait anxious individuals in their attentional biases towards threat stimuli, but when state anxiety is elevated, high trait anxious individuals will exhibit vigilance towards threat. Conversely other work suggests that both transient stress (irrespective of trait anxiety) and enduring anxious personality characteristics (irrespective of state anxiety) are both independently sufficient to produce attentional bias (Mogg, Mathews, Bird, & Macgregor-Morris, 1990).

Due to the exploratory nature of the study, together with the substantial length of the session, the impact of state anxiety as either a mediating or moderating factor was not measured or investigated. However, when the attentional bias task was administered, no stress induction test had been performed on either the primiparous or nulliparous participants, and efforts had been made to ensure their comfort (Appendix 7 - order of tasks). As such, if Broadbent and Broadbent's (1988) assertion is correct, it is possible that the absence of state anxiety may have resulted in the lack of findings. Future research using a repeated measures design, with women completing the attentional probe task under two conditions: one of low state anxiety, and the other in a high state anxiety, may help discern which theoretical model of the state-trait interaction on attentional biases reflects reality.

Furthermore, manipulation of state anxiety in relation to pregnancy, would also be an interesting and useful area for future research, as women at this time are exposed to multiple stressful situations, such as hospital appointments, scans, blood tests and results of diagnostic screening procedures. Additionally, the antenatal period is associated with increased other life events (e.g. maternity leave) also inducing high levels of state anxiety (Hass, et al., 2004). Given the effects anxiety on the developing foetus, ethical considerations mean that experimentally inducing a state of high stress in the antenatal period is implausible. However, future research could

take advantage of one of the many state anxiety provoking situations women endure during pregnancy, as described above.

Anxiety and Depression interaction

Finally, levels of depression may have 'hidden' the relationship between anxiety levels and patterns of attentional bias. Previous literature suggests that the incidence of anxiety and depression co-morbidity is high. This is perhaps due to overlap between symptoms such as difficulty sleeping, as well as predisposing vulnerabilities such as high negative effect and feelings of distress (Mineka, Watson & Clark, 1998). The current study supports this concordance at a sub-clinical level, as there were significant differences between anxiety groups according to reported experience of psychological symptoms of depression. Closer inspection confirmed a positive linier relationship between trait anxiety scores and depression measures.

Currently, how anxiety and depression interact on measures of attentional bias are unclear. Mogg et al.'s (1993) findings suggest that when clinical anxiety and depression coexist, the attentional bias usually found in anxiety conditions is no longer present. They propose that the difference in attentional biases evident in anxiety and depression reflect a difference in motivational states, that is, the attentional biases in anxiety are motivational in that they relate to individuals readiness to attend to negative stimuli in order to deal with potential threats. On the other hand, depression is considered a-motivational and as such could inhibit the motivation-based selectivity usually seen in anxiety conditions if experienced concurrently. Thus, in line with Fox et al., (2001) this suggests that co-morbidity of anxiety and depression in a sample could counteract the normal attentional biases associated with anxiety.

However, a recent meta-analysis examining boundary conditions of anxiety related attentional bias, conflicts with Mogg et al's (1993) theory of co-morbidity related inhibition. Here, the combined effect size of anxiety related biases did not differ between studies with anxiety only, compared with co-morbid populations (Bar-Heim et al., 2007). Additionally, Pearson (2010) suggests, anxiety would relate to cognitions that affect protective maternal processing, whereas depression is associated with disruptions in nurturing based cognitions. No correlations were seen between reported trait anxiety and attentional bias indices even when controlling for the potential confounders of depression. This is in line with the argument that the effects of depression are independent to that of anxiety, as they affect different attentional stages, or even different cognitive processes altogether.

4.3 Hypothesis 4 and 5: Pregnancy, and pregnancy anxiety interactions

One benefit of this study involves the pregnancy groupings. Pearson (2010) demonstrates that changes in the maternal brain that occur during pregnancy are enduring. Indeed, she found that mothers who are not currently pregnant displayed similar attentional disengagement patterns to women pregnant with their first child, with these groups significantly differing from women who have never been pregnant. However, most research to date (e.g. Broach, 2007) investigating the attentional prioritisation of infant faces, does not distinguish between these groups or report motherhood status as a potential confound. Thus, to better interpret results investigating the effects of pregnancy, this study recruited a never been pregnant

sample, along with a sample of women currently pregnant for the first time. Despite this stringency of group classification, the analyses found that pregnancy did not modulate attentional bias patterns. This contrasts with previous suggestions that adaptations, which foster protective and nurturing perceptual and attentional processing, emerge as early as twelve weeks gestation (Pearson, 2010).

However, no research has specifically investigated attentional engagement to infant faces of different emotions. Thus, it is possible that although prolonged attentional disengagement is a cognitive processing feature developed in pregnancy facilitating maternal bonding, the related but distinct construct of enhanced attentional engagement to emotional infant faces is not a factor in the construction of the maternal brain. This could explain the absence of any effects of pregnancy status.

4.3.1. Gestational time-point of pregnant group

However, given evidence of increased neurological activation in mothers in early stage processing (Kringelbach et al., 2008), it is likely that attentional engagement does play a role in aiding maternal responsiveness, yet differences are not evident until late pregnancy, or even as a function of learning process in the early post partum period. Future research is needed to investigate the time point at which this increased automatic neurological activation to emotional infant faces occurs. If attentional biases are a product of post partum learning, the theory that antenatal anxiety results in specific attentional disruptions in the normal development of protective maternal behaviour may be called into question.

Research indicates that the second trimester is associated with the lowest reported levels of trait anxiety, thus the effect of trait anxiety on attentional bias may be minimal compared with other times during pregnancy, for example, the first trimester involves significant psychological stress surrounding the risk of losing the baby (Lee et al., 2007). Furthermore, research by O'Connor (2005) suggests that anxiety in late pregnancy (mean gestation = 32 weeks) rather than in the second trimester (18 weeks gestation as above) most strongly predicts developmental delays in the child. This suggests that anxiety in late pregnancy could be critical for the increased risk of child developmental delays.

However, an earlier time point was chosen due to consideration of multiple factors. Previous literature suggests that although late pregnancy is more robustly associated with changes in cognition, (presumed to be associated with maternal brain development), this cognitive alteration is also commonly apparent as early as twelve weeks gestation. Thus, any differences due to pregnancy are expected to be evident by the second trimester. As anxiety during pregnancy may carry a significant risk to later child development (independent of postnatal psychopathology), identification of disruption in cognitive processing at earlier stages of pregnancy is necessary for early intervention. Thus the second trimester was deemed an appropriate time point of investigation, as evidence suggests that any effects of anxiety would be apparent by this point, while allowing time for early intervention in order to prevent risk transfer to the child.

Future research is needed which employs a longitudinal design, thus measuring attentional biases at several time points antenatally. This would aid understanding of

the developmental time-line for the maternal brain, and indicate the ideal time to implement specific intervention strategies which aiming to prevent the transfer of risk of child developmental delays.

As already discussed, the interaction between trait and state anxiety in relation to attentional processing is unconfirmed. If Broadbent and Broadbent's (1988) hypothesis of state anxiety modulation of hypervigilance is correct, low state anxiety in the second trimester and specifically during the session, could account for the absence of an interaction between anxiety and pregnancy. This further heightens the importance of research investigating the relationship between state and trait anxiety.

5. Conclusions

To conclude, although the results were unexpected, calculations of power suggest that acceptance of the null hypotheses would be recommended. Possible explanations why this study is not in line with previous literature have been discussed, including issues relating to the stimuli, task design and sample characteristics. Strengths and limitations of this work have been discussed throughout. As this is the first research to date that has specifically investigated attentional engagement processes during pregnancy, replication is needed before conclusions are drawn as to the role of attentional engagement in antenatal anxiety and subsequent later child developmental delays.

Furthermore, due to the state of research ambiguity exists regarding other possible factors that may mediate and/or moderate the association between high trait anxiety in the mother antenatally and later child developmental delays. Therefore work is needed to clarify theoretically contested areas such as the influence of state anxiety and co-morbid depression on anxiety related attentional engagement. Also more research is needed to document the specifics of the developing maternal brain before the impact of anxiety on this process can be fully understood. Until then, a comprehensive model cannot be built to explain the full impact of antenatal anxiety on child developmental.

There is great potential to bridge the current gap between anxiety and pregnancy research. However current evidence for a cognitive model is thin, yet alone an understanding of how cognitive elements of anxiety may interact with the biological model of risk transfer. Without such an integrative model, treatment for anxiety during pregnancy, and methods for prevention of the increased neurodevelopmental risk to the child will remain unknown. However, if cognitive mechanisms of risk transfer are identified, and effective prenatal interventions found, it may be possible to positively influence a child future wellbeing before we even begin to count their age.

References

Andersson, L., Sundstrom-Poromaa, I.S., Wulff, M., Astrom, M. A., & Bixo, M. (2006). Depression and anxiety during pregnancy and six months postpartum: a follow-up study. *Acta Obstetricia et Gynecologica, 85*(8), 937-944.

Anderson, L. Sundstrom-Poromaa, I., S., Bixo, M., Wulff, M., Bondestam, K., & Astrom, M. (2003). Point prevalence of psychiatric disorders during the second trimester of pregnancy: A population-based study. *American Journal of Obstetrics and Gynecology, 189*(1), 148-154.

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, & van IJzendoorn, I. (2007). Threat-Related Attentional Bias in Anxious and Nonanxious Individuals: A Meta-Analytic Study. *Psychological Bulletin, 133*(1), 1-24.

Barnes, L. L. B., Harp, D., & Jung, W. S. (2002). Reliability Generalization of scores on the Speilberger State-Trait Anxiety Inventory. *Educational and Psychological Measurement, 62,* 603-618.

Beck, A. T. (1972). Measuring depression: The depression inventory. In T. A. Williams, M. M. Katz & J. A. Shields (Eds.). *Recent advantages in the psychobiology of the depressive illnesses*, (pp299-302). Washington, DC: Governmental Printing Office.

Beck, A. T. (1976). *Cognitive therapy and emotional disorders.* New York: International Universities Press.

van den Bergh, B. R. H., Calster, B. V., Smits, T., Huffel, S. V., & Lagae, L. (2008). Antenatal Maternal Anxiety is Related to HPA-Axis Dysregulation and Self-Reported Depressive Symptoms in Adolescence: A Prospective Study on the Fetal Origins of Depressed Mood. *Neuropsychopharmacology, 33*, 536-545.

van den Bergh, B. R. H., & Marcoen A. (2004). High Antenatal maternal Anxiety Is Related to ADHD Symptoms, Externalising Problems and Anxiety in 8- and 9-Year-Olds*. Child Development, 77*(4), 1085-1097.

Bremner, J. G., Wachs, T. D., Murray, L., Halligan, S., & Cooper, P. (2010*). Handbook of Infant development*. Maldon, MA: Blackwell Publishing.

Brosch, T., Sander, D., & Scherer, K. R. (2007). The Baby Caught My Eye…Attention Capture by Infant Faces. *Emotion*, *7*(3), 685-689.

Browning, M., Holmes, E. A., Murphy, S. E., Goodwin, G. M., & Harmer, C. J. (2010). Lateral Prefrontal Cortex Mediates the Cognitive Modification of Attentional Bias. *Journal of Biological Psychology, 67,* 919-925.

Browning, M., Holmes, E. A., & Harmer, C. J. (2010). The modification of attentional bias to emotional information: A review of the techniques, mechanisms and relevance to emotional disorders, *Cognitive, Affective & Behavioural Neuroscience*,*10*(1), 8-20.

Cisler, J. M., & Koster, E. H. W. (2010). Mechanisms of attentional biases towards threat in anxiety disorders; An intergrative review. *Clinical Psychology Review, 30*, 203-216.

Clark, P. M. (1998). Programming of the hypothalamo-pituitary-adrenal axis and the fetal origins of adult disease hypothesis. *European Journal of Pediaterics, 157*(1), 7-10.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*, 316-336.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Earlbaum Associates.

Cox, J.L., Holden, J.M., & Sagovsky, R. (1987) Detection of postnatal depression: Development of the IO-item Edinburgh Postnatal Depression Scale. *British Journal of Psychiatry, 150*, 782-786.

Davis, E. P., & Sandman, C. A. (2010). The Timing of Prenatal Exposure to Maternal Cortisol and Psychosocial Stress Is Associated With Human Infant Cognitive Development. *Child Development, 81*(1), 131-148.

Derakshan, N., Eysenck, M. W., & Myers, L. B. (2007). Emotional information processing in repressors: The vigilance-avoidance theory. *Cognition & Emotion*, *21*(8), 1585-1614.

Eberhard-Gran, M., Eskild, A., & Opjordsmoen, S. (2005). Treating Mood Disorders During Pregnancy: Safety Considerations. *Drug Safety, 28(8),* 695-706.

Ekman, P., & friesen, W. V. (1975). *Unmasking the face: A guide to recognizing emotions from facial clues.* Englewood Cliffs, New Jersey: Prentice-Hall.

Faul, F., Erdfelder, E., Lang A. G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioural and biomedical sciences. *Behaviour Research Methods, 39*(2), 175-191.

Fox, E., Russo, R., Bowles, R. J., & Dutton, K. (2001). Do threatening stimuli draw or hold visual attention in sub-clinical anxiety? *Journal of Experimental Psychology: General, 130*, 681-700.

Glynn, L. M. (2010). Giving birth to a new brain: Hormone exposures of pregnancy influence human memory. *Psychoneuroendocrinology, 35*(8), 1148-1155.

Haas, J., S., Jackson, R., A., Fuentes-Afflick, E., Stewart, A. L., Dean, L. M., & Brawarsky, P. (2004). Changes in health status of women during and after pregnancy. *Journal of General Internal Medicine, 20*(1), 45-51.

Iqbal, M. M., Sobhan, T. & Ryals, T. (2002). Effects of Commonly Used Benzodiazepines on the Fetus, the Neonate, and the Nursing Infant. *Psychiatric Services, 53,* 39-49.

Kalikow T.J. 1975. History of Konrad Lorenz's ethological theory, 1927–1939. *Studies of History, Philosophy & Science, 6*(4): 331–341.

Kerr, J. S., Powell, J., & Hindmarch, I. (1996). The effects of reboxetine and amitriptyline, with and without alcohol on cognitive function and psychomotor performance. *British Journal of Pharmacology, 42*, 239-241.

Kim, P., Leckman, J. F., Feldman R., Wang, X., & Swain, J. E. (2010). The plasticity of human maternal brain: Longitudinal changes in the brain in the early postpartum period. *Behavioural Neuroscience, 124,* 695-700.

Kinsley, C. H., & Meyer, E. A. (2010). The Construction of the Maternal Brain: Theoretical Comment on Kim et al. (2010). *Behavioural Neuroscience, 124*(5), 710-714.

Kringelbach, M. L., Lehtonen, A., Squire, S., Harvey, A. G., Craske, M. G., Holliday, I. E., Green A. L., Aziz, T. Z., Hansen, P. C., Conelissen, P. L., Stein, A. (2008). A Specific and Rapid Neural Signature for Parental Instinct. *PLoS O*NE, 3(2), e1664.

LeDoux, J.E., (2000). Emotion circuits in the brain. *Annual Review of Neuroscience, 23*, 155–184

Lee, A. M., Lam, S. K., Lau, S.M.S.M., Cong, C. S. Y., Chui H. W., & Fong D. Y. T. (2007). Prevalence, Course, & Risk Factors for Antenatal Anxiety and Depression. *Osstetrics & gynecology, 110*(5), 1102-1112.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*(1), 15-20.

Mathews, A. & Mackintosh, B. (1998). A cognitive model of selective processing in anxiety. *Cognitive Therapy and Research, 22*, 539-569.

Mathews, A. & MacLeod, C. (2005). Cognitive Vulnerability of Emotional Disorders. *Annual Review of Clinical Psychology, 1*, 167-195.

Mathews, A., Ridgeway, v., Cook, E. & Yeind, J. (2007). Inducing a benign interpretational bias reduces trait anxiety. *Journal of Behavior Therapy and Experimental Psychiatry, 38*, 225-236.

Matthey, S., Henshaw, C., Elliott, S., & Barnett, B. (2006). Variability in use of cut-off scores and formats on the Edinburgh Postnatal Depression Scale – implications for clinical and research practice. *Archive of Womans Mental Health, 9*, 309-315.

Meaney, M. J., Szyf, M., Seckl, J. R. (2007). Epigenetic mechanisms of preinatal programming of hypothalamic-pituitary-adrenal function and health. *Trends in Molecular Medicine, 13*(7), 269-277.

Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behavioural Research and Therapy, 36*, 809-848.

Mogg, K., & Bradley, B. P. (2002). Selective orienting of attention to masked threat faces in social anxiety. *Behaviour Research and Therapy, 40*, 1403-1414.

Mogg, K., & Bradley, B. P. (2005). Attentional Bias in Generalized Anxiety Disorder Versus Depressive Disorder. *Cognitive Therapy and Research, 29*(1), 29-45.

Mogg, K., & Bradley, B.P. (2006). Time course of attentional bias for fear- relevant pictures in spider-fearful individuals. *Behaviour Research and Therapy, 44,* 1241-1250.

Mogg, K., Bradley, B. P., Miles, F., & Dixon, R. (2004). BRIEF REPORT: Time course of attentional bias for threat scenes: Testing the vigilance-avoidance hypothesis. *Cognition & Emotion, 18*(5), 689-700.

Mogg, K., Holmes, A., Garner, M., & Bradley, P. (2008). Effects of threat cues on attentional shifting, disengagement and response slowing in anxious individuals.*Behaviour Research and Therapy, 46*(5), 656-667.

Murphy, S. E., Yeind, J., Lester, K. J., Cowen, P. J. & Harmer, C. J. (2009). Short term serotonergic but not noradrenergic antidepressant administration reduces attentional vigilance to threat in healthy volunteers. *The International Journal of Neuropsychopharmacology, 12*, 169-179.

Murray, L. & Cooper, P. J. (1997). Effects of postnatal depression on infant development. *British Medical Journal, 77*, 99-101.

Oates, M. R. (2002). Adversee effects of maternal antenatal anxiety on children: causal effect of developmental continuum? *The British Journal of Psychiatry, 180,478-479.*

O'Connor, T. G., Caprariello, P., Blackmore, E. R., Gregory, A. M., & Fleming, P. (2007). Prenatal mood disturbance predicts sleep problems in infancy and toddlerhood. *Early Human Development, 83* (7), 451-458.

O'Connor, T. G., Heron, J., Glover, V., & THE ALSPAC STUDY TEAM (2002). Antenatal Anxiety Predicts Child Behaivoural/Emotional Problems Independently of Postnatal Depression. *Journal of the American academy of Child & Adolescent Psychiatry, 41*(12), 1470-1477.

O'Connor, T. G., Heron, J., Golding, J., & Glover, V. (2003). Maternal antenatal anxiety and behavioural/emotional problems in children: A test of the

programming hypothesis. *Journal of Child Psychology and Psychiatry, 44*(7), 1025-1036.

Ohman, A., Flykt, A., & Esteves, F. (2001). Emotion Drives Attention: Detecting the Snake in the Grass. *Journal of Experimental Psychology, 130*(3), 466-478.

Ohman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: toward and evolved module of fear and fear learning. *Psychological Review, 108*, 483–522.

Pearson, R. M. (2010). *Psycho-physiological preparations for motherhood and the relationship with affective disorder symptoms*. Unpublished Phd thesis, University of Bristol, UK.

Pearson, R. M. (2010). *Psycho-physiological preparations for motherhood and the relationship with affective disorder symptoms*. Unpublished Phd thesis, University of Bristol, UK.

Pearson, R. M., Cooper, R. M., Penton-Voak, I. S., Lightman, S. L., & Evans, J. (2009). Depressive symptoms in early pregnancy disrupt attentional processing of infant emotion. *Psychological Medicine*, *4*(4), 261-272.

Pearson, R. M., Lightman, S. L., & Evans, J. (2011). Attentional processing of infant emotion during late pregnancy and mother-infant interactions after birth. *Archives of Womens Mental Health, 14*, 23-31

Proverbio, A. M., Brignone, V., Matarazzo, S., Del, Z. M. & Zani, A. (2006). Gender and parental status affect the visual cortical response to infant facial expression. *Neuropsychologia, 44*, 2987-2999.

Rini, C., Schetter, C. D., Hobel, C. J., Glynn, L. M., & Sandman, C. A. (2006). Effective social support: Antecedents and consequences of partner support during pregnancy. *Personal Relationships, 13*(2), 207-229.

Ross, L. E. & McLean, L. M. Anxiety Disorders During Pregnancy and the Postpartum Period: A Systematic Review. *Journal of Clinical Psychiatry, 67*(8), 1285-1298.

de Rwiter, C. & Brosschot (1994). The emotional Stroop interference effect in anxiety: attentional bias or cognitive avoidance? *Behaviour Research and Therapy, 32*(3), 315-319.

Schmidt, N. B., Richey, A. J., Buckner, J. D. & Timpano, K. R. (2009). Attention Training for Social Anxiety Disorder. Journal of Abnormal Psychology, 118(1), 5 14.

Spain, L. A. (2009, November). IAPT wait lists too long. *Therapy Today, 20*(8), 18 19.

Smith, K. N., Cacioppo, J. T., Larsen, J. T., & Chartrand, T. L. (2003). May I have your attention, please: Electrocortical responses to positive and negative stimuli.

Neuropsychologica, 41(2), 171-183.

Spielberger, C.D., Gorsuch, R.L., Lushene, R., Vagg, P.R., & Jacobs, G.A. (1983). *Manual for the State-Trait Anxiety Inventory.* Consulting Psychologist Press, Palo Alto, CA.

Speilberger, C. D., Pollans, C. H., & Worden, T. J. (1984). Anxiety Disorders. In S. M. Turner, & M. Hersen (Eds.), *Adult psychopathology and diagnosis*, (pp.263 303). New York: Wiley.

Talge, N. M., Neil, C., Glover, V. & Early Stress, Translational Research and Prevention Science Network: Fetal and Neonatal Experience on CAMH. (2007). Antenatal maternal stress and long-term effects on child neurodevelopment: how and why? *Journal of Child Psychology and Psychiatry, 48*(3), 245-261.

Thun-Hohenstein, L., Wienerroither, C., Schreuer, M., Seim, G., & Wienerroither, H. (2008). Antenatal mental representations about the child and mother-infant interaction at three months post partum. *European Journal of Child and Adolescent Psychiatry, 17*, 9-19.

Weaver, I. C. G., Champagne, F. A., Brown, S. E., Dymov, S. Sharma, S. Meaney, M. J., & Szyf, M. (2005). Reversal of Maternal Programming of Stress Responses in Adult Offspring through Methyl Supplementation: Altering Epigenetic Marking Later in Life. *The Journal of Neurosicence, 25*(47), 11045-11054.

Welberg, L. A. M., & Seckl, J. R. (2001) Prenatal Stress, Glucocorticoids and the Programming of the Brain. *Journal of Neuroendocrinology, 13*, 113-128.

Williams, J. M. G., Mathews, A., & MacLeod, C. (1996). The emotional Stroop task and psychopathology. *Psychological Bulletin, 120*, 3-24.

Williams, J. M. G., Watts, F. N., MacLeod C., & Mathews, A. (1997*). Cognitive Psychology and Emotional Disorders*. Chichester: Wiley.

Yeind, J (2004). *Cognition, Emotion and Psychopathology: Theoretical, Empirical and Clinical Directions.* Cambridge, England: Cambridge University Press.