Infant Sex Moderates the Effects of Maternal Pre- and Postnatal Stress on Executive Functioning at 8 Months of Age

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Keywords: Prenatal stress, Postnatal stress, Executive function, Cognitive development, Infancy

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Abstract

Previous studies report that early life stress, including maternal pre- and postnatal stress, has adverse effects on cognitive development, and that these associations might be sex-specific. However, no studies exist on early life stress and infant executive functioning (EF). The aim of the current study was to examine the relationship between maternal pre- and postnatal stress and infant EF, and whether these associations are moderated by infant sex. Maternal prenatal depressive, general anxiety and pregnancy-specific anxiety symptoms were measured three times and postnatal depressive and general anxiety symptoms were measured 6 months postpartum. Infant EF was assessed with a modified A-not-B task 8 months postpartum (N = 214). Maternal postnatal general anxiety predicted poorer EF in girls in comparison to boys. Moreover, there was a trend towards an interaction between prenatal anxiety and infant sex such that prenatal anxiety predicted infant EF differently in girls and in boys. No association was found between depressive symptoms or pregnancy-specific anxiety symptoms and infant EF. These findings suggest that maternal anxiety may have sex-specific effects on early EF, and that pre- and postnatal stress may differently affect infant EF/cognitive development. The implications of these findings, and important future directions are discussed.

Keywords: Prenatal stress, Postnatal stress, Executive function, Cognitive development, Infancy

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1. Introduction

Executive functioning (EF) refers to a set of cognitive and self-regulatory processes such as working memory, inhibitory control and attentional flexibility, that are needed to facilitate goal-directed behaviors (Best & Miller, 2010). Efficient EF is important for a number of psychosocial, mental and physical health outcomes (Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Diamond, 2013). Corresponding closely with the rapid maturation of the brain that occurs during the first year of life (e.g. increase in synaptic density of the dorsolateral prefrontal cortex; Johnson, 2001; Knickmeyer et al., 2008), EF develops rapidly during the second half of the first year of life, resulting in striking improvement in EF task performance between 8 and 12 months of age. Such improvements are reflected in the ability to hold things in mind for longer periods of time (i.e., working memory) and to inhibit prepotent responses (Diamond, 1985; Diamond & Doar, 1989; Oakes, Hurley, Ross-Sheehy, & Luck, 2011). Moreover, the maturation of the prefrontal cortex and other brain structures underlying EF development takes place over an extended time period, continuing until early adulthood, making EF particularly vulnerable to being shaped by early contextual influences (Pechtel & Pizzagalli, 2011).

Early life stress, including pre- and postnatal stress is one influence that has demonstrated relations with children's early cognitive processes, such as EF (Pechtel & Pizzagalli, 2011). Early life stress is generally measured using several approaches, such as

maternal self-ratings of psychological distress/psychiatric symptoms, as stressful life events during pregnancy and the postnatal period, or as maternal blood/saliva, amniotic fluid or breast milk cortisol glucocorticoid levels (reflecting hypothalamus-pituitary-adrenal axis functioning). A number of studies have noted inverse associations between maternal psychological stress during the pre- and postnatal period and infant or child overall cognitive development (IQ or mental development index) (Davis & Sandman, 2010; Grace, Evindar, & Stewart, 2003; Henrichs et al., 2011; Ibanez et al., 2015; Keim et al., 2011; Kingston, McDonald, Austin, & Tough, 2015; Kingston, Tough, & Whitfield, 2012; Kingston & Tough, 2013; Tarabulsy et al., 2014). In most studies, including those noted here, maternal pre- and postnatal stress is typically measured using a variety of methods including general/state anxiety, trait anxiety, pregnancy-specific anxiety (i.e., maternal distress related to pregnancy and childbirth), family stress and depressive symptoms (see e.g. meta-analyses of Kingston et al., 2013 and 2015). However, only a few studies have examined several sources of stress simultaneously, and studies on postnatal stress have focused almost exclusively on maternal postnatal depression.

While most studies have noted the potentially deleterious effects of stress on early cognitive development, interestingly, some studies also suggest that prenatal stress exposure may accelerate the development of cognitive skills (DiPietro, Novak, Costigan, Atella, & Reusing, 2006; Ellman et al., 2008; Keim et al., 2011). This has been explained by the beneficial effects of mild to moderate levels of stress for fetal development, which may serve to biologically "program" the fetus to appropriately respond to diverse postnatal environments (Amiel-Tison et al., 2004) as well as by the hypothesis that fetuses of mothers with higher/moderate prenatal stress are exposed to more frequent external stimuli, which might accelerate their neural maturation (DiPietro et al., 2006). Thus, in conclusion, the current literature concerning how early life stress exposure affects overall cognition in

childhood are mixed, and different sources of stress with regard to child outcomes have not been taken into account in many studies, especially with regard to postnatal stress.

Whereas existing studies have focused on cognition, many have not focused on specific cognitive processes, and only one study has investigated the association between early pre- or postnatal stress and childhood EF. Buss, Davis, Hobel and Sandman (2011) reported a link between maternal pregnancy-specific anxiety over the course of gestation and poorer EF in children between 6 and 9 years of age. To our knowledge, no studies have reported associations between maternal postnatal stress and child EF, but maternal depression in toddlerhood is related to poorer EF in preschool age (Hughes, Roman, Hart, & Ensor, 2013; Jensen, Dumontheil, & Barker, 2014). Moreover, there is extensive evidence that general adversity during the first years of life is linked with poorer EF performance in childhood (Blair et al., 2011; Bos, Fox, Zeanah, & Nelson, 2009; Colvert et al., 2008; Pollak et al., 2010); however, the majority of studies considering these questions do not account for prenatal exposure to stress/adversity.

Further, despite some studies reporting associations between early life stress and EF in childhood, there is no literature on the effect of pre- or postnatal stress exposure on EF in infancy. Meanwhile, prenatal stress is associated with poorer infant emotion regulation (Babineau et al., 2015; Bolten et al., 2013) and altered attentional control (Gutteling et al., 2005; Huizink, Robles de Medina, Mulder, Visser, & Buitelaar, 2002; Lin, Crnic, Luecken, & Gonzales, 2014). Given the considerable overlap in the developmental timing of emotion regulation and EF (Bridgett et al., 2015; Henderson & Wachs, 2007; Hendry, Jones, & Charman, 2016; Zhou, Chen, & Main, 2012) and the neural structures underlying these two domains (Bridgett et al., 2015; Grossmann, 2013; Holzman & Bridgett, 2017) as well as earlier studies linking early childhood adversity and EF, existing evidence suggests that pre- and postnatal stress exposure might have effects on EF as early as infancy.

Beyond the potential direct effects of early life stress on early EF, child sex may be a moderator of these effects. According to the viability-vulnerability tradeoff theory (Sandman, Glynn, & Davis, 2013), males may be more prone to suffer from severe developmental problems when exposed to early life stress whereas females may exhibit greater plasticity, reflected in more fine-grained aspects of development, in the context of elevated stress exposure (Glynn & Sandman, 2012; Sandman et al., 2013). It has also been suggested that the different prenatal stress outcomes of females and males may reflect their different adaptive strategies to the environment (Glover & Hill, 2012). These theoretical perspectives are grounded in evolutionary theory, suggesting that in an environment lacking resources, males might invest in growth instead of adjusting to maternal/environmental conditions (Sandman et al., 2013).

However, the different strategies to adapt to early life stress exposure might predispose girls and boys to differential developmental trajectories and outcomes later in life. For instance, the greater plasticity to the environment of females might make them susceptible to long-term emotional distress. In line with the literature, in human studies, prenatal stress has been related to higher fearfulness, anxiety-proneness, depression, larger amygdala size and altered neural connectivity in girls only (Buss et al., 2012; Kim et al., 2016; Quarini et al., 2015; Sandman et al., 2013). In turn, in boys, prenatal stress exposure is related to gross developmental outcomes, such as higher morbidity or delays in neuromuscular and overall cognitive development (Ellman et al., 2008; Sandman et al., 2013). Although most of the research on sex differences has focused on prenatal stress, there is some evidence, from animal models, that similar differences in susceptibility might be applicable to postnatal influences (Zuloaga, Carbone, Hiroi, Chong, & Handa, 2011). Likewise, in human samples, and in line with the studies of prenatal stress, maternal postpartum depression has been negatively associated with cognitive performance in boys

(Grace et al., 2003; Murray et al., 2010), whereas glucocorticoids in breast milk are linked with elevated infant fear reactivity in girls (Grey, Davis, Sandman, & Glynn, 2013), potentially reflecting girls' greater emotional adaptation to the environment.

With regard to EF, however, Buss et al. (2011) reported that pregnancy-specific anxiety was associated with lower visuospatial working memory in both sexes but lower inhibitory control only in girls, which is inconsistent with the idea that boys would show poorer cognitive abilities after early life stress exposure. One possibility is that EF might reflect other abilities (e.g. emotion regulation) than overall cognitive development with regard to early programming influences. Further, it is suggested that prenatal stress exposure may accelerate the development of females (Ellman et al., 2008), or that the effects might be shown at younger ages in females than in males (Sandman et al., 2013). Overall, despite some evidence of its importance, moderation of early life stress exposure by sex with regard to cognitive task performance, and especially EF, has been scarcely studied.

This study aims to provide new insights into gaps in the current literature by examining associations between maternal pre- and postnatal psychological stress and infant EF task performance. Moreover, we wanted to test whether specific sources of psychological stress (pregnancy-specific anxiety, general anxiety and depressive symptoms) would be equally associated with infant EF. Specifically, we hypothesized that (1) both pre- and postnatal stress would be associated with poorer infant EF performance at 8 months, and that (2) infant sex would moderate the association between pre- and postnatal stress exposure and EF performance.

2. Methods

2.1. Study design and participants

The sample consists of families enrolled in the longitudinal FinnBrain Birth Cohort Study (n = 4,011). A research nurse informed all families that attended the first trimester ultrasound visit at gestational week 12 about the study. Of the families informed about the study, 67% enrolled and provided written informed consent for their participation. The current study is a part of the larger FinnBrain Focus Cohort (n = 1,227), which is comprised of mothers with high or low prenatal stress based on their self-reported psychological distress, and their babies. Moreover, this study included infants whose mothers were initially candidates for the Focus Cohort, but by the end of pregnancy, no longer met the criteria of experiencing high prenatal stress– instead, the level of prenatal stress for these mothers reflected moderate levels, rather than high or low prenatal stress. Despite the underlying Focus Cohort design, the distributions of the stress measures were continuous across the whole population, and therefore, all measures of maternal stress were handled as continuous throughout the study.

The sample for the present study consists of 304 (high prenatal stress n = 107, low prenatal stress n = 175, moderate stress n = 21) families that took part of the eight-month EF assessment. The overall recruitment rate for the eight-month visit was 52%. Nineteen individual measures were excluded from this portion of the study for one or more of the following reasons: language issues, infant was too restless, or emotionally difficult at baseline prior to administration of measures, the visit was terminated early, experimenter error or recording failure. At least some data was obtained from 285 infants, and a total of 214 families (n = 68 with high prenatal stress) had complete data including both the experimental data and mother-rated prenatal and postnatal stress symptom questionnaires.

The mean age of the mothers in the sample was 31 years (SD = 4). Most mothers (74%) had either a university or polytechnics degree, and 26% had an educational background of high school or vocational school or less. The typical income for the mothers in the sample varied

from 1000 to 2000 euros a month (48%). However, 19% of the mothers earned less than 1000 euros a month, which is considered living close to or below the poverty level in Finnish standards. The mean length of gestation in the sample was 40.0 weeks (ranging from 35 to 43). In regards to their infants, mothers reported no known developmental disabilities or major CNS abnormalities. The mean age of the infants at the time of the assessment, calculated from due date, was 8.1 (ranging from 7.6 to 8.6) months. The mothers that did not return 6-month-questionnaires were slightly younger ($t\,[283] = -1.866$, $p = 0.06$) and more likely to have lower education ($\chi^2 = 9.110$, $p = 0.003$) than those who returned questionnaires. No other significant differences between these groups were identified with regard to background factors.

2.2. Procedures

Maternal prenatal and postnatal stress was assessed at gestational weeks 14, 24 and 34, and 6 months postpartum, and pregnancy-related anxiety was specifically assessed at gestational weeks 24 and 34. Background information and details about maternal smoking were obtained in the first and the third trimester. Information on due date, length of gestation and infant sex based on hospital records was collected when the children in the sample were born. At 8 months, families participated in a developmental assessment visit at the FinnBrain Birth Cohort Research site. The assessments were conducted by a clinical psychologist or a trained psychology master's student. The study protocol was approved by the Ethics Committee of the Hospital District of Southwest Finland.

2.3. Measures

2.3.1. Infant executive function

The modified A*B* (i.e., the *delayed response task*) was used to assess infant EF. This task requires an infant to hold and update hiding location in mind and inhibit a prepotent response

(Diamond, 1985; Sun, Mohay, & O'Callaghan, 2009). Compared to the classic A*B* task, this version used a fixed order of hidings, an approach that is considered to be equivalent to the classic task in terms of its ability to assess infant EF (Diamond & Doar, 1989).

During the task, infants were seated in their mother's lap in front of a table. The testing apparatus consisted of a board with a midline located 20 cm from the edge and two identical hiding locations (one white or two blue cups) marked with X's separated by 11 cm on either side of the midline. The midline of the board was matched with the infants' position in the mother's lap and corrected whenever needed. During experimental trials, the toy was hidden in one of the two locations. After the toy was hidden, infants' gaze was brought to the midline by asking "(Infant's name), Where's the toy?". The direction of the infants' reaching after his/her gaze was brought to midline was scored either as correct or incorrect. All reaching behaviors that were initiated within 8 seconds were scored as reaching.

In contrast to the classic A-not-B procedure (e.g. Wolfe & Bell, 2007), stimuli were presented in a fixed pattern on three delay levels (0 seconds, 2 seconds and 4 seconds). For instance, in the delay of 0 seconds, the hidings started from the right (R-L-L-R-R-L). In each delay level, there were 6 trials. After the series of trials in each delay, infants were allowed to continue to the next delay if they scored correctly in 3 of the 6 trials. This threshold was based on the fact that 35% of the infants in the first delay level reached in fewer than 6 trials. Across the entire task, the number of trials presented to each infant varied from 6 to 18. The duration of presenting trials for the first delay level was approximately 5 minutes, with the duration of the entire task requiring between 5 and 15 minutes, depending on the delay level to which the infant was able to proceed.

The procedure was video recorded and infant performance was coded by the experimenter and afterwards by a trained coder. If there was a disagreement between the coder and the experimenter on infant scores, the average of the coder and experimenter scores

was calculated. If a disagreement involved the decision to move to the next level of delay, the coder evaluation based on the video recording was used in the final scoring. Inter-rater agreement between the raters was 81%, and in 82% of the disagreements, rater assessments were within one point of each other. The trials were not initiated or were discontinued if the infant was too upset/fussy, or did not reach at all. All trials where the infant showed some reaching behaviors, and at least the first 3 trials were presented, were included for analysis. The probability of correct recall with given difficulty level was used as the measure of infant EF performance in the task, with higher probability of correct choice reflecting more efficient infant EF. In practice, the probability of correct recall can be estimated using the relative frequency as the measurement (e.g. how many correct recalls out of six in each level of difficulty the infant was able to make). In this work, logistic regression was used to model the performance, as described later.

2.3.2. Maternal prenatal and postnatal stress

Maternal prenatal stress was assessed using the Edinburgh Postnatal Depression Scale (EPDS) and Symptom Checklist -90 (SCL-90) anxiety subscale (gwk 14, 24, 34) and the Pregnancy-Related Anxiety Questionnaire 2 (PRAQ-R2) (gwk 24, 34). Postnatal stress was assessed using EPDS and SCL-90 at 6 months postpartum. The EPDS is a widely used and sensitive measure of both postnatal and prenatal depression (Cox, Holden, & Sagovsky, 1987) and consists of 10 items rated from 0 to 3 (higher scores indicate more depressive symptoms). The anxiety subscale of SCL-90 is a reliable and valid measure of anxiety symptoms in both clinical and research settings (Derogatis, Lipman, & Covi, 1973) and consists of 10 items rated from 0 to 5. The PRAQ-R2 is a recently revised measure (Huizink et al., 2015) to assess pregnancy-specific anxiety and consists of 10 items rated from 1 to 5, comprising three factors. In this study, the sum of all PRAQ-R2 items was used in the

analyses. All prenatal stress measures showed good internal consistency throughout the study (0.84–0.89 for EPDS, 0.87–0.90 for SCL-90 anxiety subscale, 0.85–0.91 for PRAQ-R2 sum variable). Postnatal stress assessments also showed good internal consistency (0.88 for EPDS and 0.88 for SCL-90). Both maternal prenatal and postnatal symptoms were used as continuous variables (mean throughout the pregnancy/sum of symptoms at 6 months) in this study.

2.3.5. Data analyses

First, differences in performance with regard to infant sex, maternal smoking and maternal educational level were investigated with the χ^2-test and Mann Whitney U-test using the average of the correct choices across the task as a dependent variable. Second, the associations between maternal age, length of gestation, and maternal prenatal and postnatal anxiety, depressive and pregnancy-related anxiety symptoms, and infant EF performance (average of the correct choices) were investigated with Spearman correlations in the entire sample and in girls and boys separately. Third, infant performance was modeled using a logistic regression, with analyses being carried out using the function GLM in R (R Core Team, 2016). Logistic regression was used to investigate the influence of pre- and postnatal stress and the covariates on the probability of the correct recall *p* in the modified A*B* task. The outcomes are the dichotomous variables of each trial of the test. Furthermore, as the probability of the correct recall depends heavily on the difficulty level of the task as well as duration of the task reflected by difficulty level, and not all infants proceeded to the higher level delays, the item difficulty (delay level) was included as a factor in the model.

A stepwise approach was chosen to examine the effect of covariates starting with a simpler model and progressing to a more complex model. The sets were selected based on pre-existing hypotheses (maternal education) and preliminary exploratory analyses (pre- and

postnatal anxiety and infant age). The sets of covariates from each of the model steps are presented in Table 4. Finally, as some of the experiments were discontinued earlier than expected due to e.g. infant fussiness, the final model was re-run excluding these experiments. The results of this model are described in the text.

3. Results

3.1. Descriptive statistics

Table 1 shows infants' EF performance on each level of task difficulty (based on delay). Of all experiments conducted, 29 (14%) were discontinued at the 0 or 2 second delay levels. The most typical reason for a discontinued experiment was infant exhaustion, or increasing infant restlessness or irritability during the experiment (83%).

INSERT TABLE 1 HERE

Levels of prenatal and postnatal stress experienced by mothers in the sample are shown in the Table 2.

INSERT TABLE 2 HERE

3.2. Associations among background variables, maternal prenatal and postnatal stress and infant EF performance at 8 months

The correlations between continuous background factors and infant EF performance (mean probability of correct choice throughout the task) are presented in Table 3. Infant EF in the whole sample was not significantly correlated with background factors, prenatal or postnatal maternal depressive symptoms, or anxiety or pregnancy-specific anxiety symptoms, although there was a trend such that there was a negative association between postnatal anxiety and infant EF performance. When comparing girls and boys separately, there was a significant negative association between postnatal anxiety and infant performance for girls. Moreover, infant age from expected due date was positively associated with performance only in boys. In the whole sample, there were no differences between girls and boys in mean score (U [214] = 5415.5, $p = 0.53$), reaching cups (U [214] = 5067.5, $p = 0.16$), capacity to continue to each task difficulty level ($\chi^2 = 1.829$, $p = 0.18$ for 2 second delay and $\chi^2 = 0.31$, $p = 0.58$ for 4 second delay), maternal educational level (χ^2 [214] = 2.059, $p = 0.56$) or maternal smoking (U [214] = 2054.0, $p = 0.29$).

Moreover, in the preliminary models, pregnancy-specific anxiety and pre- or postnatal depressive symptoms were not related to infant performance. Similarly, there were no significant stress by sex interactions when using these measures of stress. Based on both zero-order correlations and this observation, only prenatal and postnatal general anxiety symptoms and their interactions with infant sex were used as predictors of EF in the final logistic regression analyses.

INSERT TABLE 3 HERE

3.3. Logistic regression model for infant EF performance

The logistic regression models for infant EF are displayed in Table 4 with the standardized coefficients, their significance values, the corresponding odds ratios (OR) for the covariates, and the 95% confidence intervals for the ORs. The estimated coefficients for gender specific effects are reported as differences with respect to the boys' estimates, e.g. the estimated coefficient for the postnatal anxiety's effect on the EF for girls is –0.35–2.21 = –2.56. In the second step of the model, there was a negative effect of prenatal anxiety on infant boys EF performance. In the final step of the model, after controlling for background variables and the main effects of prenatal and postnatal anxiety, there was a trend towards an interaction between maternal prenatal anxiety and infant sex ($\beta = 1.97$, $p = 0.053$). For girls, maternal prenatal anxiety was associated with better EF performance in contrast to boys (see fitted logistic regression curve Figure 1A). Moreover, there was a significant interaction between postnatal anxiety and infant sex in predicting infant EF. For girls, in contrast to boys, maternal postnatal anxiety was associated with lower EF (see Table 4 and Figure 1B). The difference of the predicted mean probabilities of boys vs. girls based on the fitted model evaluated at a range of pre- and postnatal stress points are displayed in Figure 2.

When experiments that were discontinued earlier than expected due to infant fussiness were excluded from the analyses, the results ($N = 185$) with regard to the interaction between postnatal anxiety and infant sex remained the same ($\beta = –2.05$, $p = 0.04$, OR = 0.93 [0.88, 0.99]), and the interaction between prenatal anxiety and infant sex was significant in predicting EF ($\beta = 2.08$, $p = 0.04$, OR = 1.05 [1.00, 1.11]).

INSERT TABLE 4 HERE

INSERT FIGURE 1 HERE

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4. Discussion

The aim of this study was to investigate the effects of pre- and postnatal stress on infant EF at the age of 8 months. We found no evidence of a main effect of prenatal stress, measured as either depressive symptoms, general anxiety or pregnancy-specific anxiety symptoms, on infant EF. However, infant sex by postnatal general anxiety interaction was identified – there was a small, but significant negative effect of postnatal general anxiety on infant performance at 8 months that was specific to girls. Further, there was a trend towards an interaction between prenatal anxiety and infant sex. Higher prenatal anxiety was related to a significant difference in performance between girls and boys, girls performed better than boys in the presence of prenatal stress exposure. After excluding discontinued experiments from analyses, this association was statistically significant.

Our finding that postnatal anxiety predicts poorer EF – a finding that accounted for prenatal stress exposure – corresponds to our hypothesis of the negative effect of early life stress on infant EF, and is in line with earlier studies suggesting that postnatal stress is a contributor to child cognitive development (Keim et al., 2011; Kingston et al., 2015). However, despite some consistency of our findings with some prior work, other previous studies have noted that maternal postnatal psychological distress is linked with poorer cognition in boys, but not in girls (Grace et al., 2003; Murray et al., 2010) – a finding that is contrary to the findings obtained in the present study. However, neither of these studies considered EF, which might explain differences between our findings and these earlier studies. It has been noted that EF might reflect a distinct ability from overall IQ or other neurodevelopment measures (Ardila, Pineda, & Rosselli, 2000; Friedman et al., 2006; Sun et al., 2009), and that EF is associated with emotion regulation (Bridgett et al., 2015; Zhou et

al., 2012). Interestingly, early life stress is more often associated with emotional problems in girls than in boys, who in turn show poorer cognitive outcomes (Buss et al., 2012; Ellman et al., 2008; Kim et al., 2016; Quarini et al., 2015). Consequently, it can be hypothesized that early life stress effects on EF may be independent of overall cognitive development and more similar to those on emotional development. Thus, future studies might want to investigate early life stress by child sex associations with EF and overall cognitive development separately.

Moreover, no evidence of an independent effect of prenatal anxiety on either girls' or boys' performance was found in the present study. However, two analyses, one at trend level, showed that girls who were exposed to prenatal stress exhibited heightened EF in infancy in contrast to boys who showed poorer EF. Hence, the direction of the effect of prenatal anxiety was dependent on infant sex. Together with the finding that suggests an adverse effect of postnatal anxiety on girls' EF, our findings provide some evidence for the viability-vulnerability tradeoff model (Sandman et al., 2013), which suggests that females and males would be differentially affected by early life stress. There are several possibilities that may explain these sex differences findings. First, some earlier studies have reported that cognitive development and maturation might be accelerated in girls exposed to prenatal stress (Ellman et al., 2008; Glynn & Sandman, 2012; Sandman et al., 2013). For girls in the present study, better EF, and possibly cognitive skills, may reflect an adaptation to early life stress exposure, marked by more efficient attentional strategies, vigilance and working memory skills in contrast to boys. These abilities might make them more resilient in the nearer term. However, the same set of skills that makes girls better than boys at this task (e.g. higher arousal), might also reflect their vulnerability to postnatal stressors, which was supported by our finding showing that the adverse effect of postnatal stress was seen only in girls.

Second, it is possible that high maternal postnatal stress and its possible consequences for the early environment (e.g. the lower quality of mother-infant interactions) show up as worse EF performance only in girls but not boys. Indeed, it has been suggested that those boys who survive prenatal stress are less susceptible to early environmental adversity (Sandman et al., 2013). Our data suggests this pattern, as after controlling for prenatal stress, postnatal stress did not contribute to boys' performance. Third, the effects of prenatal stress might become evident in girls at a younger age than in boys (Sandman et al., 2013), and consequently, that the performance difference between girls and boys might disappear later. This possibility must be delineated by future studies.

On the other hand, our finding that prenatal stress did not contribute significantly to infant performance is contrary to our hypothesis that prenatal stress would predict overall poorer EF (Buss et al., 2011). Instead, we found a link between postnatal stress and poorer EF in girls. The differences in the results of Buss et al. (2011) and our study might be due to the different age groups covered, especially given the nature of EF development throughout childhood. The difference might also be explained by the fact that when comparing the symptom scores, the mothers in the sample of Buss et al. (2011) seem to have experienced higher levels of psychological stress, on average, than the mothers in our sample (see Table 2 in the results section). This might have attenuated the effect size in the current study. The low number of mothers with high postnatal depressive symptoms might also explain why we found effects only for general anxiety, and not for depressive symptoms, even though previous studies have reported associations between maternal prenatal depressive symptoms and child cognition (e.g. DiPietro et al., 2006) as well as maternal postnatal depression and child EF/cognitive development (Grace et al., 2003; Hughes et al., 2013). Similarly, some previous studies have reported that only pregnancy-specific anxiety predicted child outcomes (Buss et al., 2011; Buss, Davis, Muftuler, Head, & Sandman, 2010). However, due to the

differences in measures of both maternal stress and infant EF in our study and in others, and the limited number of studies conducted on EF during this developmental period, further research is needed before making stronger conclusions about the effects of specific sources of early life stress on early EF.

Our findings have several clinical implications. First, infants of mothers with postnatal psychological distress might have poorer outcomes in developmental assessments. This should be taken into account in the clinical assessment of young children by assessing children's early life stress exposure and maternal history of psychiatric symptoms. Second, our study suggests that girls might be more susceptible to maternal postnatal psychological distress, and that girls and boys may be differentially susceptible to prenatal stress. However, though our study also suggests that prenatal stress exposure might positively predict girls' EF development, clinicians need to be aware of the possibility based on previous research that girls might still have heightened risk for later emerging internalizing problems (Costello et al., 2003; Salk, Hyde, & Abramson, 2017), especially when exposed to elevated levels of early life stress (Kim et al., 2016; Quarini et al., 2015). Despite some potential clinical implications, it should be noted that the effect sizes in our study were small. This is consistent with most prior work (Davis & Sandman, 2010; Henrichs et al., 2011; Huizink et al., 2002), but indicates that the clinical implications of our findings must be interpreted accordingly.

There were several strengths in the current study: despite the challenges of the measurement of EF in infancy, we were able to collect a large sample who showed relatively high variation in EF task performance. This study included both measures of prenatal and postnatal stress in a novel design, as to date, only one other study has been conducted on prenatal stress and EF, and no studies on pre- *and* postnatal stress in relation to infant EF exist. However, the present study is not without limitations. There was some attrition with regard to 6-month questionnaire data, which was related to maternal education and postnatal

stress. As noted earlier, despite the underlying aim to recruit mothers with high prenatal stress, mothers in our sample seem to have reported less symptoms compared to mothers in some other studies. In particular, in our study, the number of mothers with high postnatal depressive symptoms was low in comparison to postnatal anxiety symptoms and maternal symptoms in the prenatal period. This might have obscured effects of postnatal depressive symptoms on infant EF performance.

We also were not able to account for the quality of mother-infant interactions, which might be a potential mediator of family risk factors, including maternal postnatal depression or anxiety symptoms, alone or in interaction with infant sex, on early childhood EF (e.g. Murray, Kempton, Woolgar, & Hooper, 1993; Rhoades, Greenberg, Lanza, & Blair, 2011). Moreover, the finding of prenatal stress by sex interaction in predicting infant EF was only statistically significant when excluding the discontinued experiments (fussing/inattentive infants), which necessitates caution in interpretation and generalization of this finding to other populations.

5. Conclusion

The present study provided evidence of sex-specific prenatal and postnatal stress exposure associations with infant EF at 8 months. Maternal postnatal general anxiety predicted poorer EF, an effect that was specific to girls. Moreover, there was a trend-level interaction between prenatal general anxiety and infant sex - higher maternal general anxiety was associated with better EF performance in girls in contrast to boys. These findings are in line with the earlier literature in suggesting that girls might be affected by early life adversity differently than boys, showing more fine-tuned strategies in coping with the environmental stressors. However, further research on early life stress and infant EF, as well as infant sex as

moderator of the relations between these two, is needed to strengthen the findings of the current study.

References

Amiel-Tison, C., Cabrol, D., Denver, R., Jarreau, P.-H., Papiernik, E., & Piazza, P. V. (2004). Fetal adaptation to stress: Part I: acceleration of fetal maturation and earlier birth triggered by placental insufficiency in humans. *Early Human Development*, *78*, 15–27.

Ardila, A., Pineda, D., & Rosselli, M. (2000). Correlation between intelligence test scores and executive function measures. *Archives of Clinical Neuropsychology*, *15*, 31–36.

Babineau, V., Green, C. G., Jolicoeur-Martineau, A., Bouvette-Turcot, A.-A., Minde, K., Sassi, R., … Wazana, A. (2015). Prenatal depression and 5-HTTLPR interact to predict dysregulation from 3 to 36 months - A differential susceptibility model. *Journal of Child Psychology and Psychiatry*, *56*, 21–29.

Best, J. R., & Miller, P. H. (2010). A developmental perspective on executive function. *Child Development*, *81*, 1641–1660.

Blair, C., Granger, D. A., Willoughby, M., Mills-Koonce, R., Cox, M., Greenberg, M. T., … Fortunato, C. K. (2011). Salivary cortisol mediates effects of poverty and parenting on executive functions in early childhood. *Child Development*, *82*, 1970–1984.

Bolten, M., Nast, I., Skrundz, M., Stadler, C., Hellhammer, D. H., & Meinlschmidt, G. (2013). Prenatal programming of emotion regulation: Neonatal reactivity as a differential susceptibility factor moderating the outcome of prenatal cortisol levels. *Journal of Psychosomatic Research*, *75*, 351–357.

Bos, K. J., Fox, N., Zeanah, C. H., & Nelson, C. A. (2009). Effects of early psychosocial deprivation on the development of memory and executive function. *Frontiers in Behavioral Neuroscience*, *3*, 16.

Bridgett, D. J., Burt, N. M., Edwards, E. S., & Deater-Deckard, K. (2015). Intergenerational transmission of self-regulation : A multidisciplinary review and integrative conceptual framework. *Psychological Bulletin*, *141*, 602–654.

Buss, C., Davis, E. P., Hobel, C. J., & Sandman, C. A. (2011). Maternal pregnancy-specific anxiety is associated with child executive function at 6-9 years age. *Stress*, *14*, 665–76.

Buss, C., Davis, E. P., Muftuler, L. T., Head, K., & Sandman, C. A. (2010). High pregnancy anxiety during mid-gestation is associated with decreased gray matter density in 6-9-year-old children. *Psychoneuroendocrinology*, *35*, 141–153.

Buss, C., Davis, E. P., Shahbaba, B., Pruessner, J. C., Head, K., & Sandman, C. A. (2012). Maternal cortisol over the course of pregnancy and subsequent child amygdala and hippocampus volumes and affective problems. *Proceedings of the National Academy of Sciences of the United States of America*, *109*, E1312-9.

Colvert, E., Rutter, M., Kreppner, J., Beckett, C., Castle, J., Groothues, C., … Sonuga-Barke, E. J. S. (2008). Do theory of mind and executive function deficits underlie the adverse outcomes associated with profound early deprivation?: Findings from the English and Romanian Adoptees Study. *Journal of Abnormal Child Psychology*, *36*, 1057–1068.

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., Angold, A., (eds.), C. E., … M, R. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry*, *60*, 837.

Cox, J. L., Holden, J. M., & Sagovsky, R. (1987). Detection of postnatal depression. Development of the 10-item Edinburgh Postnatal Depression Scale. *The British Journal of Psychiatry : The Journal of Mental Science*, *150*, 782–6.

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*, 131–148.

Derogatis, L. R., Lipman, R. S., & Covi, L. (1973). SCL-90: an outpatient psychiatric rating scale--preliminary report. *Psychopharmacology Bulletin*, *9*, 13–28.

Diamond, A. (1985). Development of the ability to use recall to guide action, as indicated by infants' performance on AB. *Child Development*, *56*, 868–883.

Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, *64*, 135–68.

Diamond, A., & Doar, B. (1989). The performance of human infants on a measure of frontal cortex function, the delayed response task. *Developmental Psychobiology*, *22*, 271–94.

DiPietro, J., Novak, M., Costigan, K., Atella, L., & Reusing, S. (2006). Maternal psychological distress during pregnancy in relation to child development at age two. *Child Development*, *77*, 573–587.

Ellman, L. M., Schetter, C. D., Hobel, C. J., Chicz-DeMet, A., Glynn, L. M., & Sandman, C. a. (2008). Timing of fetal exposure to stress hormones: Effects on newborn physical and neuromuscular maturation. *Developmental Psychobiology*, *50*, 232–241.

Friedman, N. P., Miyake, A., Corley, R. P., Young, S. E., DeFries, J. C., & Hewitt, J. K. (2006). Not all executive functions are related to intelligence. *Psychological Science*, *17*, 172–179.

Glover, V., & Hill, J. (2012). Sex differences in the programming effects of prenatal stress on psychopathology and stress responses: An evolutionary perspective. *Physiology and Behavior*, *106*, 736–740.

Glynn, L. M., & Sandman, C. A. (2012). Sex moderates associations between prenatal glucocorticoid exposure and human fetal neurological development. *Developmental Science*, *15*, 601–610.

Grace, S. L., Evindar, A., & Stewart, D. E. (2003). The effect of postpartum depression on child cognitive development and behavior : A review and critical analysis of the literature. *Archives of Women's Mental Health*, *6*, 263–274.

Grey, K. R., Davis, E. P., Sandman, C. A., & Glynn, L. M. (2013). Human milk cortisol is associated with infant temperament. *Psychoneuroendocrinology*, *38*, 1178–1185.

Grossmann, T. (2013). Mapping prefrontal cortex functions in human infancy. *Infancy*, *18*, 303–324.

Gutteling, B. M., de Weerth, C., Willemsen-Swinkels, S. H. N., Huizink, A. C., Mulder, E. J. H., Visser, G. H. A., & Buitelaar, J. K. (2005). The effects of prenatal stress on temperament and problem behavior of 27-month-old toddlers. *European Child & Adolescent Psychiatry*, *14*, 41–51.

Henderson, H. A., & Wachs, T. D. (2007). Temperament theory and the study of cognition-emotion interactions across development. *Developmental Review*, *27*, 396–427.

Hendry, A., Jones, E. J. H., & Charman, T. (2016). Executive function in the first three years of life: Precursors, predictors and patterns. *Developmental Review*, *42*, 1–33.

Henrichs, J., Schenk, J. J., Kok, R., Ftitache, B., Schmidt, H. G., Hofman, A., … Tiemeier, H. (2011). Parental family stress during pregnancy and cognitive functioning in early childhood: The Generation R Study. *Early Childhood Research Quarterly*, *26*, 332–343.

Holzman, J. B., & Bridgett, D. J. (2017). Heart rate variability indices as bio-markers of top-down self-regulatory mechanisms: A meta-analytic review. *Neuroscience & Biobehavioral Reviews*, *74*, 233–255.

Hughes, C., Roman, G., Hart, M. J., & Ensor, R. (2013). Does maternal depression predict young children's executive function? - a 4-year longitudinal study. *Journal of Child Psychology and Psychiatry*, *54*, 169–177.

Huizink, A. C., Delforterie, M. J., Scheinin, N. M., Tolvanen, M., Karlsson, L., & Karlsson, H. (2015). Adaption of pregnancy anxiety questionnaire–revised for all pregnant women regardless of parity: PRAQ-R2. *Archives of Women's Mental Health*, 125–132.

Huizink, A. C., Robles de Medina, P. G., Mulder, E. J. H., Visser, G. H. A., & Buitelaar, J. K.

(2002). Psychological measures of prenatal stress as predictors of infant temperament. *Journal of the American Academy of Child & Adolescent Psychiatry*, *41*, 1078–1085.

Ibanez, G., Bernard, J. Y., Rondet, C., Peyre, H., Forhan, A., Kaminski, M., & Saurel-Cubizolles, M.-J. (2015). Effects of antenatal maternal depression and anxiety on children's early cognitive development: A prospective cohort study. *PLoS One*, *10*, e0135849.

Jensen, S. K. G., Dumontheil, I., & Barker, E. D. (2014). Developmental inter-relations between early maternal depression, contextual risks, and interpersonal stress, and their effects on later child cognitive functioning. *Depression and Anxiety*, *31*, 599–607.

Johnson, M. H. (2001). Functional brain development in humans. *Nature Reviews. Neuroscience*, *2*, 475–483.

Keim, S. A., Daniels, J. L., Dole, N., Herring, A. H., Siega-Riz, A. M., & Scheidt, P. C. (2011). A prospective study of maternal anxiety, perceived stress, and depressive symptoms in relation to infant cognitive development. *Early Human Development*, *87*, 373–380.

Kim, D.-J., Davis, E. P., Sandman, C. A., Sporns, O., O'Donnell, B. F., Buss, C., & Hetrick, W. P. (2016). Prenatal maternal cortisol has sex-specific associations with child brain network properties. *Cerebral Cortex*.

Kingston, D., McDonald, S., Austin, M.-P., & Tough, S. (2015). Association between prenatal and postnatal psychological distress and toddler cognitive development: A systematic review. *PloS One*, *10*, e0126929.

Kingston, D., & Tough, S. (2013). Prenatal and postnatal maternal mental health and school-age child development: A systematic review. *Maternal and Child Health Journal*, *18*, 1728–1741.

Kingston, D., Tough, S., & Whitfield, H. (2012). Prenatal and postpartum maternal

psychological distress and infant development: A systematic review. *Child Psychiatry and Human Development, 43,* 683–714.

Knickmeyer, R. C., Gouttard, S., Kang, C., Evans, D., Wilber, K., Smith, J. K., … Gilmore, J. H. (2008). A structural MRI study of human brain development from birth to 2 years. *The Journal of Neuroscience, 28,* 12176–12182.

Lin, B., Crnic, K. A., Luecken, L. J., & Gonzales, N. A. (2014). Maternal prenatal stress and infant regulatory capacity in Mexican Americans. *Infant Behavior and Development, 37,* 571–582.

Murray, L., Arteche, A., Fearon, P., Halligan, S., Croudace, T., & Cooper, P. (2010). The effects of maternal postnatal depression and child sex on academic performance at age 16 years: a developmental approach. *Journal of Child Psychology and Psychiatry, 51,* 1150–1159.

Murray, L., Kempton, C., Woolgar, M., & Hooper, R. (1993). Depressed mothers' speech to their infants and its relation to infant gender and cognitive development. *Journal of Child Psychology and Psychiatry, 34,* 1083–1101.

Oakes, L. M., Hurley, K. B., Ross-Sheehy, S., & Luck, S. J. (2011). Developmental changes in infants' visual short-term memory for location. *Cognition, 118,* 293–305.

Pechtel, P., & Pizzagalli, D. A. (2011). Effects of early life stress on cognitive and affective function: An integrated review of human literature. *Psychopharmacology, 214,* 55–70.

Pollak, S. D., Nelson, C. A., Schlaak, M. F., Roeber, B. J., Wewerka, S. S., Wiik, K. L., … Gunnar, M. R. (2010). Neurodevelopmental effects of early deprivation in postinstitutionalized children. *Child Development, 81,* 224–236.

Quarini, C., Pearson, R. M., Stein, A., Ramchandani, P. G., Lewis, G., & Evans, J. (2015). Are female children more vulnerable to the long-term effects of maternal depression during pregnancy? *Journal of Affective Disorders, 189,* 329–335.

R Core Team. (2016). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from https://www.r-project.org

Rhoades, B. L., Greenberg, M. T., Lanza, S. T., & Blair, C. (2011). Demographic and familial predictors of early executive function development: Contribution of a person-centered perspective. *Journal of Experimental Child Psychology, 108*, 638–662.

Salk, R. H., Hyde, J. S., & Abramson, L. Y. (2017). Gender differences in depression in representative national samples: Meta-analyses of diagnoses and symptoms. *Psychological Bulletin*.

Sandman, C. A., Glynn, L. M., & Davis, E. P. (2013). Is there a viability-vulnerability tradeoff? Sex differences in fetal programming. *Journal of Psychosomatic Research, 75*, 327–335.

Sun, J., Mohay, H., & O'Callaghan, M. (2009). A comparison of executive function in very preterm and term infants at 8 months corrected age. *Early Human Development, 85*, 225–30.

Tarabulsy, G. M., Pearson, J., Vaillancourt-Morel, M.-P., Bussières, E.-L., Madigan, S., Lemelin, J.-P., … Royer, F. (2014). Meta-analytic findings of the relation between maternal prenatal stress and anxiety and child cognitive outcome. *Journal of Developmental and Behavioral Pediatrics, 35*, 38–43.

Zhou, Q., Chen, S. H., & Main, A. (2012). Commonalities and differences in the research on children's effortful control and executive function: A call for an integrated model of self-regulation. *Child Development Perspectives, 6*, 112–121.

Zuloaga, D. G., Carbone, D. L., Hiroi, R., Chong, D. L., & Handa, R. J. (2011). Dexamethasone induces apoptosis in the developing rat amygdala in an age-, region-, and sex-specific manner. *Neuroscience, 199*, 535–47.

Table 1. Descriptive characteristics for the infant performance in the modified A*B* task including mean of correct recall and mean probability of correct recall out of six trials conducted (*N* = 214)

Task difficulty		25th percentile	75th percentile	Boys	Girls
0 second delay	N = 214 (100 %)			N = 114	N = 100
Reaching (SD)[a]	5.3 (1.2)	5	6	5.3 (1.2)	5.2 (1.3)
Mean score (SD)[b]	2.8 (1.4)	2	4	2.9 (1.5)	2.8 (1.4)
Mean prob. (SD)[c]	0.47 (0.24)	0.33	0.67	0.48 (0.24)	0.46 (0.24)
2 second delay	N = 116 (54 %)			N = 68	N = 48
Reaching (SD)	4.8 (1.5)	4	6	4.9 (1.5)	4.7 (1.4)
Mean score (SD)	2.7 (1.3)	2	3.75	2.6 (1.3)	2.8 (1.4)
Mean prob. (SD)	0.45 (0.22)	0.33	0.63	0.44 (0.22)	0.47 (0.23)
4 second delay	N = 57 (27 %)			N = 30	N = 27
Reaching (SD)	4.2 (1.8)	3	6	4.6 (1.6)	3.7 (1.9)
Mean score (SD)	2.5 (1.4)	1.25	3.5	2.7 (1.4)	2.2 (1.3)
Mean prob. (SD)	0.41 (0.23)	0.17	0.58	0.46 (0.23)	0.36 (0.22)
Overall	N = 214 (100 %)			N = 114	N = 100
Reaching (SD)	8.9 (1.8)	5	12	9.3 (4.9)	8.5 (4.7)
Mean score (SD)	4.9 (3.9)	2	7.6	5.1 (3.9)	4.7 (3.8)
Mean prob. (SD)[d]	0.27 (0.21)	0.11	0.42	0.28 (0.22)	0.26 (0.21)

[a]The mean of trials where infants reached the cup
[b]The mean sum of correct choice
[c]The mean probability (frequency) of correct choice *per* six possible trials in each delay
[d]The mean probability (frequency) of correct choice *per* eighteen possible trials during the experiment

Table 2. The descriptive statistics for maternal prenatal and postnatal stress measured as depressive, general anxiety and pregnancy-specific anxiety symptoms (*N* = 214)

Stress measure (theoretical range)	Mean (SD)	Range	% of mothers above the clinical cut-off[a]
Depressive symptoms (EPDS) (0-30)			
1st trimester	4.3 (4.2)	0–19	6.6%
2nd trimester	4.1 (4.3)	0–18	6.5%
3rd trimester	4.5 (4.4)	0–19	5.6%
Average across pregnancy	4.3 (3.8)	0–15.5	4.2%
Postnatal (6 months)	4.2 (4.3)	0–22	3.7%
General anxiety (SCL-90) (0-50)			
1st trimester	2.9 (4.6)	0–40	6.1%
2nd trimester	3.9 (5.9)	0–40	8.4%
3rd trimester	3.3 (5.7)	0–40	8.4%
Average across pregnancy	3.3 (4.5)	0–26.7	8.9%
Postnatal (6 months)	2.6 (4.0)	0–24	6.1%
Pregnancy-specific anxiety (PRAQ-R2) (10-50)			
2nd trimester	22.0 (6.8)	10–42	6.5%
3rd trimester	22.2 (6.8)	10–42	8.4%
Average across pregnancy	22.0 (6.5)	10–40.5	6.5%

[a]Clinical cut-offs used for the EPDS ≥ 12, for the SCL-90 ≥ 10, and for the PRAQ-R2 ≥ 34. The cut-offs are based on the analyses on the highest 25th percentile in overall stress measures of the first consecutive 500 mothers of the FinnBrain Cohort.

Table 3. The zero-order Spearman correlations between infant EF performance and background factors for whole sample and separately for girls (n = 100) and boys (n = 114)

Variable	1	2	3	4	5	6	7	8	9	10
1. EF										
2. EF in girls only										
3. EF in boys only										
4. Maternal age	-.07	-.03	-.10							
5. Infant age	.05	-.12	.20*	.12†						
6. Gestational weeks	-.05	-.04	-.05	-.07	.06					
7. Prenatal depressive	-.08	-.09	-.05	-.10	-.04	-.01				
8. Prenatal anxiety	-.07	-.04	-.13	-.05	.05	-.01	.73***			
9. Pregnancy-specific	-.08	-.01	-.13	-.08	-.01	.02	.59***	.54***		
10. Postnatal depressive	-.11	-.15	-.08	-.05	.01	-.00	.64***	.50***	.43***	
11. Postnatal anxiety	-.13†	-.22*	-.06	-.02	.05	.01	.53***	.61***	.42***	.64***

*** $p < 0.001$, * $p < 0.05$, † $p < 0.10$

Table 4. The logistic regression model for infant executive function at 8 months: the association between pre- and postnatal maternal anxiety and infant performance (*N* = 214)

	Step 1		Step 2		Step 3		
	Effect size[b]	β	Effect size[b]	β	Effect size[b]	β	OR (C.I. 95 %) for Step 3
	0.006		0.060		0.254		
Intercept		–1.75†		–2.51*		–1.88†	0.03 (0.00, 1.17)
Task difficulty		–4.00***		–4.00***		–4.50***	0.88 (0.83, 0.93)
Infant age[a]		1.69†		2.50*		1.87†	1.57 (0.98, 2.53)
Infant sex (Girl)		–0.61		–1.78†		–0.65	0.93 (0.76, 1.15)
Maternal education		–0.34		–0.28		–0.28	0.97 (0.77, 1.21)
Prenatal anxiety (SCL)				–2.60**		–1.26	0.97 (0.94, 1.01)
Prenatal anxiety x infant sex (Girl)				1.85†		1.97†	1.05 (1.00, 1.10)
Postnatal anxiety (SCL)						–0.35	0.99 (0.95, 1.03)
Postnatal anxiety x infant sex (Girl)						–2.21*	0.93 (0.88, 0.99)

***p < 0.001, **p < 0.01, *p < 0.05, †p < 0.10; [a]from expected due date, [b]for the whole model, McFadden (Pseudo-R)

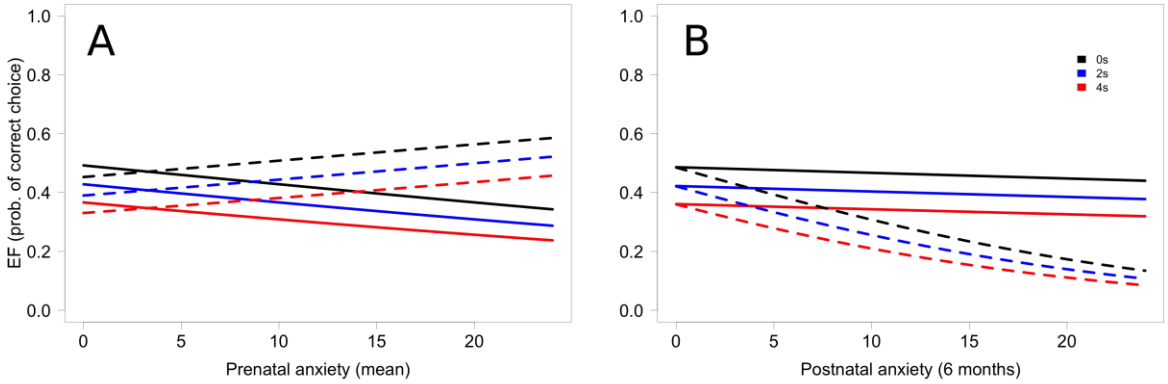

Figure 1. The fitted logistic regression curve showing the influence of the total prenatal and postnatal general anxiety to infant performance in EF task. (A) Prenatal anxiety has a positive influence on the performance of girls (dashed line) in contrast to boys (solid line). (B) Postnatal anxiety has a negative influence on the performance of girls (dashed line), but no influence on the performance of boys (solid line). The curves are fitted at the observed anxiety median (= 1).