Trajectories of Maternal Pre- and Postnatal Anxiety and Depressive Symptoms and Infant Fear: Moderation by Infant Sex

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Various types of prenatal stress, including exposure to maternal psychological distress or anxiety and depressive symptoms during the prenatal period, are frequently linked with infant temperament traits, such as higher negative affect and poorer self-regulation (Bergman, Sarkar, O'Connor, Modi, & Glover, 2007; Diego, Field, & Hernandez-Reif, 2005; Korja, Nolvi, Grant, & McMahon, 2017; Nolvi et al., 2016). These traits are also precursors for later psychiatric disorders in the offspring (e.g. De Pauw & Mervielde, 2010; Putnam & Stifter, 2005). Likewise, maternal postnatal psychological distress also has been associated with higher infant negative affect (Bridgett et al., 2009; Feldman et al., 2009; Glasheen,

Richardson, & Fabio, 2010; Kingston, Tough, & Whitfield, 2012). Although existing work has focused on associations between pre- and, in particular, postnatal maternal psychological distress and infant temperament, it is still relatively poorly understood how the continuity of exposure to maternal distress during these periods is related to infant emotionality, and how individual factors, such as sex, may moderate link between maternal distress and emotionality. To address this gap in the existing literature, the purpose of the current study was to examine the associations between prenatally-limited exposure and more continuous exposure to maternal distress on infant temperament, specifically, infant fear, and consider infant sex as a moderator of these effects.

Mechanisms and Models of Early Life Programming

The link between maternal prenatal distress and infant negative affect is believed to result from alterations in maternal-fetal-placental stress biology. For instance, heightened fetal exposure to maternal stress hormones may affect fetal epigenetic regulation and brain development (Moisiadis & Matthews, 2014a, 2014b). In turn, maternal postnatal stress can result in disruptions to early mother-infant interactions or attachment patterns that may steer child development (Bergman, Sarkar, Glover, & O'Connor, 2008; Field, 2010). Exposure to stressors is particularly important early in life as the brain is especially plastic and thus susceptible to programming influences (Markant & Thomas, 2013; van den Bergh, 2011). Early life programming, in turn, is believed to help adapt the child to the expected or current environment (Gluckman & Hanson, 2004; Sandman, Davis, Buss, & Glynn, 2011), but may also raise the risk for future psychopathology (Barker, Edward, Copeland, Maughan, Jaffee, & Uher, 2012; Capron et al., 2015; Davis & Sandman, 2012).

Although many studies have considered early life emotional outcomes after exposure to maternal distress (Van den Bergh et al., 2017), the significance of continuity of maternal distress in relation to child development is still poorly understood. First, it it has been

suggested that cumulative stress exposures throughout life might be most crucial for mental health outcomes (Daskalakis, Bagot, Parker, Vinkers, & Kloet, 2013). Second, theoretical work suggests that the match between prenatal and later environments is central in determining the outcome ("the mismatch hypothesis", Gluckman & Hanson, 2004) such that individuals will fare better in circumstances where the postnatal environment "matches" that of the prenatal environment. Recently, it has been proposed that exposure to prenatal stress could increase susceptility to the postnatal environment (Grant, Sandman, Wing, Dmitrieva, & Davis, 2015; Hartman & Belsky, 2018; Hartman, Freeman, Bales, & Belsky, 2018; Pluess & Belsky, 2011) through multiple biological adaptations, e.g. heightened physiological reactivity and alterations in serotonin and oxytocin systems (Hartman & Belsky, 2018; Moore & Depue, 2016). Consequently, in contrast to children exposed to prenatal-only stress exposures, children exposed to more stressors continuing after birth would show different or amplified negative outcomes.

Given that children's outcomes contributed to by early exposures to stressful environments most likely arise from interactions among multiple factors, there have been some endeavours to integrate varying perspectives. Nederhof and Schmidt (2012) propose that the pathways of cumulative stress exposure and the mismatch theories may be explained by individual plasticity, which is also central to the idea of "prenatal programming of plasticity" presented earlier. According to this integrated approach, a mismatch would be detrimental for individuals who showed high sensitivity to programming effects and were "programmed" to fit a specific postnatal environment that is different than that which is experienced. On the other hand, the cumulative stress hypothesis would apply to individuals who were not specifically programmed to certain environment (Nederhof & Schmidt, 2012). Thus, based on these theories, individuals may show differential outcomes after continuous vs. exposure limited to only prenatal period, but their outcomes may also depend on their

individual susceptibility to programming effects. However, due to the paucity of studies that have tested these theories, there is relatively little empirical evidence to conclude which model best explains the effects of continuous stress.

Early Life Programming and Fear

One of the key aspects of negative affect considered among studies focusing on pre- and postnatal stress exposures is fear reactivity/fear, a temperament trait reflecting heightened behavioral reactivity to novelty and having partially distintictive developmental trajectory in comparison to the other aspects of negative reactivity (Rothbart, 2011). Importantly, fear is related particularly to later anxiety disorders (Clauss & Blackford, 2012; Kopala-Sibley et al., 2016). A number of studies have been conducted on pre- and postnatal maternal distress and infant fear (Bergman et al., 2007; Davis et al., 2004; Feldman et al., 2009; Gartstein et al., 2010; Möhler, Parzer, Brunner, Wiebel, & Resch, 2006; Nolvi et al., 2016; Pauli-Pott, Mertesacker, & Beckmann, 2004), but few have considered the continuity of maternal distress during these periods. Studies focusing explicitly on continuity have found support for the links between continuously high maternal distress and higher fear (Pesonen, Räikkönen, Strandberg, & Järvenpää, 2005) or prenatal-only but not postnatal or continuous maternal stress and higher fear (Henrichs et al., 2009). In turn, Stapleton et al. (2012) reported that maternal prenatal anxiety and depressive symptoms were not related to infant fear, but instead, maternal postnatal distress mediated the link between elevated prenatal distress and higher infant fear. More broadly, studies focusing on child emotional and behavioral problems indicate that continuously high or moderate maternal symptoms are stronger predictors of child emotional and behavioral problems later in development in contrast to maternal symptoms limited to a shorter period of time (Cents et al., 2013; Guyon-Harris, Huth-Bocks, Lauterbach, & Janisse, 2016; Park, Brain, Grunau, Diamond, & Oberlander, 2018; van der Waerden et al., 2015). These findings support the crucial role of continuous

exposure to maternal distress on child development, but provide little evidence on how continuity of maternal distress is related to fear specifically.

Beyond studies that have considered direct or mediated associations between early life stress and children's fear, recent theories suggest that infant sex may moderate the link between stress exposures and emotional outcomes (Glover & Hill, 2012; Sandman, Glynn, & Davis, 2013). Indeed, sex differences may exist in regards to the sensitivity of different brain areas to stress hormone programming (Bale, 2015; Ostlund et al., 2016; Weinstock, 2007) as well as placental sensitivity to glucocorticoids (Carpenter, Grecian, & Reynolds, 2017; Clifton, 2010; Mueller & Bale, 2008). In line with these theories, prenatal cortisol and maternal distress have been linked with heightened emotional reactivity, vigilance to socio-emotional stimuli and risk for later psychiatric symptoms in girls (Braithwaite et al., 2017; Kataja et al., 2019; Quarini et al., 2016; Sandman et al., 2013), but lower emotional reactivity in boys (e.g. Braithwaite et al., 2017). Recently, the idea of sex differences in susceptibility to prenatal programming influences have been extended to include postnatal stress influences, such as breast milk glucocorticoids (Grey, Davis, Sandman, & Glynn, 2013; Nolvi et al., 2018). Females also show higher fear from early childhood on (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006), and have higher risk for anxiety and depressive symptoms (Costello et al., 2003; Salk, Hyde, & Abramson, 2017).These findings suggest that there is a need to study infant sex as one factor increasing susceptibility when studying exposure to pre- and postnatal stress and their continuity.

The Current Study

The aim of the current study was to examine whether trajectories of maternal pre- and postnatal psychological distress, measured as anxiety and depressive symptoms, influence infant fear. First, the trajectories of consistently low, prenatal-only and consistently high maternal distress across five time points (from gestational week 14 to 6 months postpartum)

were identified using latent growth mixture modeling (LGMM) (see also Kataja et al., 2018). Second, the aims were to subsequently 1) assess how the identified trajectories of maternal distress are related to infant fear using both observed and mother-reported infant fear as outcomes, and 2) examine whether infant sex moderates the association between pre- and postnatal exposure to maternal distress and infant fear. It was hypothesized that both high levels of prenatal or more continuous stress exposure would predict higher infant fear, but that these effects would be amplified in the infants exposed to continuous stress, who were possibly more sensitized due to prenatal programming effects. It was also expected that infant sex would moderate the effects of pre- and postnatal stress exposure on infant fear and that girls would be more vulnerable to pre- and postnatal exposure to maternal distress showing higher fear after such exposure in contrast to infant boys.

Method

Study Design and Participants

A subsample of families participating in the larger FinnBrain Birth Cohort Study (N = 3,808; Karlsson et al., 2018) participated in the current study. A research nurse informed all families that attended the first trimester ultrasound visit at gestational week 12 about the study. Of all the families informed, 66% enrolled in the study and provided consent for their participation. The sample for this study is a part of the FinnBrain Focus Cohort (N = 1,227), which is comprised of mothers with either high or low prenatal distress based on their self-reported symptoms of depression and anxiety across pregnancy, and their babies (see description in Karlsson et al., 2018 and the more detailed description in the Supplement).

Of all the Focus Cohort mothers that were contacted (N = 908; the families where the infant met the age requirement of 8 months during the recruitment) to participate in the

observational assessment of fear at 8 months of age, 490 (54%) gave consent for participation, and 412 participated in the fear observation. The final sample for the present study included 391 mother-infant dyads that provided data for both the observed fear assessment and at least one distress symptom score to conduct trajectory analysis. No mother-infant dyads presented with conditions that would have required exclusion from the study (e.g., reported developmental disabilities or major CNS abnormalities of the infant, preterm birth). Demographic characteristics of the sample are displayed in Table 1.

Table 1

Procedures

Maternal psychological distress was assessed at gestational weeks 14, 24 and 34, and at 3 and 6 months postpartum. During the first assessment, mothers also responded to questions concerning their age, parity, education and monthly income. Information on due date, length of gestation, birth weight and infant sex was collected from hospital records when the children in the sample were born and complemented with data from the Finnish National Birth Register (www.thl.fi). Maternal reports of infant fearfulness were collected at 6 months and fear observations were collected at 8 months at the FinnBrain Cohort Research site at the University of Turku. The visits were conducted either by the psychologists or advanced psychology students who had received extensive training. After the 8-month-visit, families received a small gift (infant clothing, a toy) as a compensation for their participation. The study protocol was approved by the Joint Ethics Committee of Southwestern Hospital District and University of Turku.

Measures

Maternal Pre- and Postnatal Psychological Distress (Depressive and Anxiety Symptoms). Maternal depressive symptoms were assessed using the Edinburgh Postnatal Depression Scale (EPDS) and anxiety symptoms using Symptom Checklist -90 (SCL-90) anxiety subscale (at gwk 14, 24, 34, at 3 months and at 6 months). The EPDS is a widely used measure of both pre- and postnatal depression (Cox, Holden, & Sagovsky, 1987) consisting of 10 items rated from 0 to 3. 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. Both measures showed good internal consistency throughout the study (α = .83–.89 for EPDS and .86–.89 for SCL-90 anxiety subscale). All symptom measures correlated moderately to highly (r = .40–.75). Both maternal pre- and postnatal symptoms were used as continuous variables with higher scores indicating higher symptoms; individual items were used in CFA analysis and composites were used in GMM analysis. The descriptive statistics for EPDS and SCL-90 are reported in Table 1.

Trajectories of Maternal Pre- and Postnatal Distress. To identify trajectory classes separately for maternal depressive and anxiety composites across the five measurement points, LGMM was conducted using Mplus 6 software (Muthén & Muthén, 1998–2011). Growth mixture modeling is generally used to identify subpopulations based on their trajectories of a given attribute. Missing data were handled by using the FIML (Full Information Maximum Likelihood) estimator (Muthén & Muthén, 1998–2011). Consistent with recommendations and guidelines for identifying classes (Ram & Grimm, 2009), the number of latent classes was determined by increasing the number of classes in the analysis and observing the output, theoretical basis and interpretability of the results, and their usefulness in further analysis (e.g. that the number of participants in each class was sufficient to perform sex by trajectory class interaction analysis). The indices that were used to inform

the number of classes retained were the Bayesian Information Criterion (BIC) and Akaike Information Criterion (AIC) (lower values indicating better model), Entropy (with values closer to 1.0 indicating higher a confidence of classification), posterior probabilities of class membership, and Vuong-Lo-Mendell-Rubin likelihood ratio test (VLMR-LRT) and Bootstrapping Likelihood Ratio Test (BLRT) for *k* versus *k–1* groups (*p* values lower than .05 suggesting that *k + 1* is superior in comparison to *k* groups) (see references for these indices in e.g. Ram & Grimm, 2009).

For both anxiety and depressive symptoms, the AIC and BIC indices improved up to 5-class model. However, as entropy values, posterior probabilities of class membership and VLMR-LR test indices suggested no significant improvement after 3-class model, the 3-class model was selected as the most parsimonious model describing class trajectories of both depressive and anxiety symptoms[1]. The initial analyses for fear were made using anxiety and depressive symptom trajectories as predictor separately, but because the models were highly similar and the trajectories were correlated ($p < .001$), groups were collapsed to increase the sample size in distress trajectories. This resulted into three categories: "Consistently Low Distress" (N = 278 [71%, 146 boys]), "Prenatal-Only Distress" (N = 74 [19%, 38 boys], consisting of mothers with only prenatal anxiety/depressive symptoms) and "Consistently High Distress" (N = 43 [11%, 27 boys], consisting of mothers with consistenly high symptoms one at least one measure). The results obtained using collapsed trajectories resembled the original ones. For a more detailed description of the trajectory modelling and figures and original analyses with anxiety and depressive symptoms trajectories, see Supplement.

[1] 4- and 5-class solutions would have also included primarily increasing stress (Postnatal-only) stress group, but the number of cases in this group would have been too small to conduct further analyses. The 3-class solutions were additionally confirmed by listwise deletion of cases with no significant differences.

Mother-reported infant fear. The Fear subscale of the Infant Behavior Questionnaire Revised Short Form (Putnam, Helbig, Gartstein, Rothbart, & Leerkes, 2014) was used to measure mother-reported fear when infants were 6 months of age. This subscale consists of six items (e.g. “When in the presence of several unfamiliar adults, how often does the baby continued to be upset for 10 minutes or longer?” or “How often did your baby startle at a sudden change in body position (for example, when moved suddenly)?”) and falls under the broader dimension of Negative Affectivity. The parent rates infant behavior during the past week or past two weeks on a scale from 1 to 7, with higher scores on each item indicating higher levels of fear In the present study, the Fear subscale demonstrated good internal consistency (Cronbach’s $\alpha = .83$).

Observed infant fear. The Masks episode from the Laboratory Temperament Assessment Battery Prelocomotor version (Lab-TAB; Goldsmith & Rothbart, 1999) was used to assess experimentally-induced infant fear. During the episode, infants were exposed to four masks, ranging from relatively lower to higher in their intensity and potential to elicit a fear reaction. Four indicators of fear, escape behaviors (0–3), infant facial fear (0–3), bodily fear (0–3), and fearful vocalizations (0–5) were coded in response to presentations of each mask following the Lab-TAB instructions. These four behavioral indicators of fear were moderately to highly correlated (r =.45–.79) and were standardized before forming a composite variable. The indicators of fear showed high internal consistency (Cronbach’s α = .89) and inter-rater reliability (Mean of Cohen’s Kappa =.73– .83, with inter-rater r = .95–.98). The caregivers were told not to interrupt the episode unless instructed so by the experimenter. Caregiver behavior was assessed to index parental interference (0–2) during the task. The descriptive statistics infant fear are shown in Table 1.

Data Analysis Strategy

Observed and mother-reported fear were normally distributed after logarithm transformations. The missing data due to attrition was analyzed using T-tests and χ^2 test to get an overview of the differences between the responding and the non-responding mothers. The association between background variables, infant sex and fear were studied using Pearson correlation coefficients, analysis of variance, χ^2 test and T-tests. The relation between trajectories of maternal distress and mother-reported fear was analyzed using One-way ANOVA. As only 331 (85% of the original sample) provided 6-month mother-reported fear, multiple imputation using SPSS Missing Values was utilized to maintain the complete dataset with regard to mother-reported infant fear (Graham, 2009), with results using imputed and non-imputed data highly resembling each other.

Finally, analyses were conducted using factorial ANCOVA and within-group ANOVA or pair-comparisons to assess both main effects and stress trajectory by infant sex interactions. The analyses controlled for maternal education, which is frequently used as a covariate in studies considering either stress or temperament; for maternal age and parity, that were related to maternal trajectories of anxiety (see Supplement), and for monthly income and caregiver behavior during the observation, that were associated with observed fear.

Results

Attrition Analyses

The mothers who were invited to participate inthe 8-month-visit but decided not to do so were younger (t [1071] = 2.40, p = .016) and less educated (χ^2 [2] = 13.93, p = .001), and they had lower monthly income (χ^2 [2] = 8.30, p = .016), than families that participated in the visit. The mothers that did not respond to the postnatal symptom questionnaires were less educated (χ^2 [1) = 6.62 to 9.60, p's ranging from .010 to .002), but there were no differences between responders and non-responders with regard to age or income. The mothers who did

not respond to the 3-month questionnaires had more depressive (t [366] = 2.60, p = .013) and anxiety symptoms (t [367] = 2.38, p = .018) during pregnancy; mothers who responded to the 6-month questionnaires (incl. mother-reported fear) and those who did not, did not differ from each other in terms of symptoms.

The Association between Potential Confounding Factors, Infant Sex and Infant Fear

The associations between all study variables are displayed in Table 2. Girls showed higher observed fear (M = 3.82, SD = 2.35) in comparison to boys (M = 3.30, SD = 1.96), (T = –2.207, p = .028). Similarly, girls were rated higher in mother-reported fear (M = 2.70, SD = 1.21) than boys (M = 2.34, SD = 1.14) (T = 2.756, p = .006). There was a trend towards an association between maternal income and observed infant fear (F [2, 390] = 2.923, p = .06) with infants in the groups with the highest income being on average less reactive than infants in the group of typical monthly income. Moreover, caregiver behavior during laboratory assessment of infant fear was associated with infant fear: the infants of mothers who interfered during the observation showed higher in fear (F [2, 390] = 10.222, p < .001). Maternal education or age, parity, gestational weeks, infant age from expected due date and birth weight were not related to infant fear (p > .05). None of the selected confounding factors were associated with parent-reported infant fear.

Table 2

Table 3

The Associations between the Trajectory of Maternal Distress and Infant Fear

Observed fear. The means of infant reactivity for each maternal distress trajectory are displayed in Table 3. No significant group differences were found with regard to observed infant fear. However, the results of factorial ANCOVA (Table 4) revealed that there was a significant stress by infant sex interaction in predicting fear after adjusting for covariates. In post-hoc comparisons, girls and boys of mothers with prenatal-only distress differed from each other, with girls (*M* of standardized fear = 0.35, *SD* = 0.91) showing higher fear than boys (*M* = –0.22, *SD* = 0.79) within this trajectory (*T* = 2.90, *p* = .005). Furthermore, it was found that distress trajectories were related to fear only in the group of girls. Thus, infant girls exposed to prenatal-only maternal distress showed higher fear (*M* = 0.35, *SD* = 0.91) in comparison to infant girls exposed to consistently high distress (*M* = –0.44, *SD* = 1.02) (*F* = 4.22, *p* = .004–.017). There was also a trend (*p* = .04–.11) towards a significant difference between infant girls exposed to consistently low maternal distress and infant girls exposed to consistently high maternal distress, with infants of mothers with consistently high distress showing less fear than girls exposed to low distress (*M* = 0.08, *SD* = 0.85). No significant difference was observed between girls in the prenatal-only and consistently low distress trajectories. Group differences are displayed in Figure 1.

Mother-reported infant fear. The difference between the trajectories in mother-reported fear approached significance (Table 3). Mothers with either prenatal-only or consistently high distress rated their infants higher in fear in comparison to mothers with consistently low distress. After accounting for the selected confounding factors, the main effect only approached significance (Table 4). There were also no significant within-group differences besides the initial difference of girls and boys, that is, girls were rated as more fearful than boys in the groups of consistently low maternal distress (*T* = –2.60, *p* = .010) and prenatal-only distress (*T* = –1.97, *p* = .049), but not in the group of consistently high maternal distress.

Table 4

Figure 1

Discussion

This study investigated the effects of continuity vs. discontinuity of maternal pre- and postnatal distress, measured as anxiety and depressive symptoms, on infant fear. The following trajectories: "Consistently Low Distress" (71% of the mothers in the sample), "Prenatal-Only Distress" (19%) and "Consistently High Distress" (11%) were used as predictors of observed and parent-rated infant fear. Trajectories of maternal distress had no significant main effect on observed or reported fear reactivity after adjusting for covariates. However, moderation by infant sex was identified such that the trajectory of maternal "Prenatal-Only Distress" was associated with higher observed fear in infant girls in contrast to infant boys. Furthermore, girls who were exposed to consistently high maternal distress during the pre- and postnatal periods showed less fear than girls exposed to prenatal-only or consistently low distress. To the best of our knowledge, these findings are among the first to utilize trajectory modeling to study maternal pre- and postnatal distress and infant emotional development, with an aim of disentangling the effects of prenatal distress and more continuous exposure to maternal distress from each other while also considering the potential role of infant sex as a moderator.

The lack of significant main effects of either distress trajectories on observed fear was in contrast to several earlier studies in the field (Bergman et al., 2007; Gartstein et al., 2010; Henrichs et al., 2009; Pesonen et al., 2005). Mothers in the distress trajectories rated their infants higher in fear, but this finding only approached significance. Although studies have reported expected associations between at least some measure of stress exposure early in life and the aspects negative affect, there are also those that have not (Baibazarova et al., 2013;

Della Vedova, 2014; Kaplan, Evans, & Monk, 2009). One possible explanation for this lies in the differences between the study samples. In the current study, the sample consisted of relatively highly educated mothers, who may have experienced lower levels of distress (on average) in comparison to other studies in the field (see e.g. O'Donnell et al., 2017; Wen et al., 2017) which might have attenuated the effect sizes of maternal distress as a correlate of fear.

However, the role of moderating factors in detecting the associations between pre- and postnatal stress exposures and child development has been emphasized (Meaney, 2018; O'Donnell & Meaney, 2017; Zijlmans, Riksen-Walraven, & de Weerth, 2015) to take into account the differential sensitivity of the individuals to environmental stressors and explain inconsistent findings across studies. In the spirit of these calls, in the present study, distress exposure limited to the prenatal period was related to higher infant fear at 8 months, but only in girls (Bergman et al., 2007; Davis et al., 2004; Henrichs et al., 2009; Nolvi et al., 2016; Pesonen et al., 2005). This supports the previous literature stating that girls may be more susceptible to early life stressors in terms of emotional reactivity (Sandman et al., 2013). This has been explained by the evolutionarily grounded differences in susceptibility, making females more programmable in terms of fear and anxiety-proneness. Thus, the focus on fear in the context of maternal distress might primarily lead to findings within females. Interestingly, however, maternal prenatal distress and mother-reported fear showed no tendency towards sex-specificity. This suggests that in terms of temperament outcomes, sex may more strongly moderate the association between maternal prenatal distress and observed, experimentally-induced fear, given that these two measures, even though generally correlated, might partially capture different aspects of the same construct.

Another possible explanation for the differences between observed and reported fear is the influence of maternal characteristics on maternal report of their infants' fearfulness. There

is a long-standing evidence that maternal depression and anxiety are related to lower convergence between laboratory and maternal report measures (Leerkes & Crockenberg, 2003). In the current study, the normative difference in reported fear between the sexes in the trajectory of consistently low maternal distress was attenuated in the distress trajectories. This suggests that in addition to the previously reported tendency to overreport infant negative affect, mothers with continuous distress might also be less sensitive to existing sex differences in emotionality. On the other hand, it has been suggested that parental reactions to infant fear differ by infant sex, steering infant fear responses (Eggum et al., 2009) and that parenting may affect fearful girls and boys differently (Kiel, Premo, & Buss, 2016). Future work aiming to disentangle the effects of continuity of early life stress on young children's emotional reactivity should employ methods that can disentangle sources of potential rater influences on study outcomes.

Interestingly, in our study, consistently high maternal distress was associated with attenuated observed fear in girls. This was contrary to the hypothesis that stress exposure would have an amplified effect on fear in individuals exposed to continuous or more accumulated stress (Daskalakis et al., 2013; Pesonen et al., 2005) due to e.g. heightened plasticity resulting from prenatal programming (Pluess & Belsky, 2011). Instead, this finding is consistent with the mismatch hypothesis (Gluckman & Hanson, 2004), as girls exposed to continuous (matching) environments showed less fear, which might reflect better adaptation to the environment. When taken into to perspective of integrated theory of Nederhof and Schmidt (2012), the fact that the finding only appeared in girls would reflect their heightened susceptibility to programming specifically in terms of fear, and consequently heightened vulnerability to the environmental mismatch.

However, there are also alternative possibilities to our finding. One is that the heightened plasticity in the face of high maternal distress does not lead to heightened but to

dampened fear reactivity. For example, there is evidence that in the face of insensitive caregiving, a characteristic frequently related to maternal anxiety and depression (Field, 2010, 2018), infants may inhibit external reactions of fear that would otherwise be meant to elicit caregiving (Righetti-Veltema, Bousquet, & Manzano, 2003) or show less reactive behavior (Diego et al., 2005). More susceptible individuals experiencing frequent stress-evoking situations (such as disrupted maternal care) might also learn to habituate, resulting in an elevated threshold for fear. Thus, in such circumstances, a relatively mild, novel fear stimulus, such as that used in the current investigation, might not be salient enough to elicit a fear reaction in these individuals (Daskalakis et al., 2011).

Another possibility is that the infants with exposure to maternal postnatal distress might show delayed cognitive and neural development (Diego et al., 2005; Kingston et al., 2012). Fear reportedly emerges around 6 months of age (Gartstein et al., 2010; Putnam & Stifter, 2005), and may be related to more advanced cognitive development (Graham et al., 2015). As cognitive and emotional development are interrelated as early as infancy (Feldman & Eidelman, 2009), it is possible that continuous exposure to maternal distress adversely affects the normative occurrence of infant displays of fear. Finally, children living in conditions of higher stress might show precocious development of self-regulation abilities and neural networks as a response to an environment that places high demand on self-regulation, resulting in better regulation of emotional displays, including fear (e.g. Silvers et al., 2016). It must also be noted that even though most studies indicate that high fear is a risk factor for later anxiety, at least two studies have linked less fearful temperament early in life to the later development of psychopathology, particularly when children are exposed to non-optimal parenting (Beaver, Hartman, & Belsky, 2015; Colder, Mott, & Berman, 2002). The authors of both studies suggested that low fear could be indicative of lack of “normal” behavioral inhibition and thus, contribute to problems in socialization.

Strengths and Limitations

The current study has several strengths, including a relatively large sample using both standardized behavioral observations and parent-reported data as well as longitudinal follow-up of maternal depressive and anxiety symptoms, including five assessment points. However, one major limitation is the small sample size within the consistently high trajectory in contrast to other trajectories and the lack of controlling for the maternal/infant genotype. Furthermore, there is a possibility that the inclusion of primarily mothers belonging to the Focus Cohort, selected for high levels of prenatal stress, might have affected the trajectories of distress that were identified. However, the trajectories identified resembled the ones identified in the whole cohort (unpublished data), strengthening their validity. Moreover, the findings were not similar for reported and observed fear. Although only two months separated maternal report of infant fear and the laboratory observation of infant fear, this time period is one of fairly rapid emergence of this particular aspect of infant temperament (Gartstein et al., 2010), potentially contributing to differences in findings across observered and maternal reported fear in the current study.

Conclusion

In conclusion, the present study is one of the first to assess the significance of continuity of maternal pre- and postnatal psychological distress on infant fear, a notable risk factor for later anxiety. The findings suggest that prenatal or even continuously high maternal distress may not be major risk factors for infant fear for all infants that experience such exposures. The results are in line with the hypothesis that infant girls are more sensitive than boys to the effects of early stress exposures with regard to (observed) emotional responses. Moreover, the findings suggest that female infants exposed to prenatal-only maternal symptoms show different fear behavior than female infants exposed to more continuous maternal symptoms. Future studies should seek to more thoroughly investigate the effects of

continuity and timing of pre- and postnatal maternal distress on child brain and behavioral development, while addressing some of the limitions in and building upon the current investigation.

Declaration of interests: none.

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