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Low autonomic arousal as vulnerability to externalising behaviour in infants with hostile mothers

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ABSTRACT

Maternal psychopathology and the child's autonomic nervous system functioning are risk factors for aggressive behaviour later in life. While research has shown that maternal psychopathology already affects young children, less is known about the association between autonomic functioning and aggressive behaviour in young children. In addition, maternal psychopathology and autonomic nervous system functioning may interact to determine the risk of aggressive behaviour.

In a sample of 375 infants and their mothers, maternal psychiatric symptoms were assessed with the Brief Symptom Inventory and toddler aggressive behaviour with the Child Behaviour Checklist. Infant heart rate was recorded at 14 months.

Maternal psychiatric problems, including hostility and depression, were associated with toddler aggressive behaviour. Maternal psychiatric problems interacted with mean heart rate ($P=0.01$) and HF variability ($P=0.03$) in their effect on toddler aggressive behaviour.

Mothers with high psychiatric problems, in particular, high hostility, were more likely to have toddlers with high aggressive behaviour. Moreover, in the presence of maternal risk factors, low autonomic arousal renders children particularly susceptible to aggressive behaviour.

1. Introduction

Maternal psychopathology is a strong predictor of aggressive behaviour in young children (Tremblay et al., 2004; Kim-Cohen et al., 2005, 2006). Children of mothers suffering from psychopathology not only inherit an unfavourable genetic profile but are also exposed to multiple environmental risk factors (Kim-Cohen et al., 2005).

Nonetheless, most children of mothers with psychopathology do not display aggressive behaviour later in life. This difference in vulnerability may depend on biological susceptibility. An extensive body of research has investigated the role of genetic susceptibility to environmental factors. Studies in both adoption (Cadoret et al., 1983) and twin cohorts (Rowe et al., 1999) have shown the importance of gene–environment interaction in the development of aggressive behaviour. Some studies have even been able to identify specific genes responsible for these interactions. In a seminal study, Caspi et al. showed that maltreated children with a genotype conferring high levels of MAOA expression were less likely to develop antisocial problems than other maltreated children (Caspi et al., 2002). Despite such successes, gene–environment interactions are very complex and it has been difficult to identify or replicate candidate genes for gene–environment interactions (Zammit and Owen, 2006).

Studying the interaction of environmental risk factors with a physiological risk factor for aggressive behaviour may alleviate part of this complexity, as physiological risk factors are themselves the product of the interplay of multiple genes and environmental factors.

Autonomic nervous system (ANS) functioning, typically measured by mean heart rate (heart rate), is such a risk factor (Monk et al., 2001; Ortiz and Raine, 2004). ANS functioning in infants is primarily determined by the interplay of several genes and to a certain extent environmental factors (Dubreuil et al., 2003).

A large number of cross-sectional studies have shown that low heart rate during rest is associated with aggressive behaviour (Raine et al., 1997; van Goozen et al., 2000; Dietrich et al., 2007). Furthermore, longitudinal studies have shown low heart rate to predict later aggressive behaviour (Raine et al., 1997). The relationship between high frequency (HF) heart rate variability, a commonly used proxy for the vagal component of autonomic control, and aggressive behaviour is

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less clear (Lorber, 2004). Some studies though indicate that there may be an association between high HF variability in children and aggressive behaviour (Cole et al., 1996; Van Voorhees and Scarpa, 2002; Scarpa and Ollendick, 2003; Dietrich et al., 2007). According to the fearlessness theory, low levels of arousal, low heart rate and high HF variability, are indicative of low levels of fear. Children who lack fear are less likely to head potential negative consequences of their actions, which may in turn contribute to poor fear conditioning and lack of conscience development (Raine, 2002).

A few studies have previously investigated the interplay of these autonomic indices with other risk factors. Farrington (1997) reported that the association between low heart rate and aggressive behaviour was stronger when environmental risk factors were present as well. Boys with low resting heart rate were more likely to commit acts of violence as an adult if they also had a poor relationship with their parent, and if they came from a large family. Recently, a study investigated the interaction of heart rate and with environmental stressors in a large sample of adolescents. The study showed that adolescents with low heart rate displayed less psychopathology in relation to stressors in life than adolescents with normal or high heart rate (Oldehinkel et al., 2008).

As for HF variability, Shannon et al. showed that it moderated the association between parental and child externalising behaviour. In their study, high vagal tone played a protective role (Shannon et al., 2007). Furthermore, in several studies performed by El-Sheikh et al., vagal tone had a similar protective role in buffering the influence of marital conflict on child externalising behaviour (El-Sheikh et al., 2001; El-Sheikh and Whitson, 2006).

Maternal psychopathology is one of the strongest risk predictors of externalising behaviour in young children. There is a solid base of research showing that both maternal antisocial behaviour, and maternal depressive symptoms are associated with child aggressive behaviour (Tremblay et al., 2004; Kim-Cohen et al., 2005, 2006). While children of mothers with psychopathology are likely to be exposed to multiple biological and environmental risk factors, the impact of maternal antisocial behaviour seems to be primarily mediated through suboptimal parenting (Thornberry et al., 2009). Similarly, depression in mothers is related to suboptimal parenting as well (Paulson et al., 2006). Lack of adequate parenting and guidance may compound the absence of fear for negative consequences found in children with low autonomic arousal. Despite this, to our knowledge, the interaction of ANS indices with maternal psychopathology, has never been studied.

In this study we examined the relationship of heart rate and HF variability in infants with their behaviour at 18 months. We hypothesised that low heart rate and high HF variability render toddlers susceptible to aggressive behaviour. Furthermore, we expected the relation between low autonomic arousal and toddler aggressive behaviour to be even stronger in the presence of maternal psychopathology.

2. Methods

2.1. Participants

This study was conducted within the Focus Cohort of the Generation R Study, a population-based cohort study from foetal life until young adulthood (Jaddoe et al., 2006; Jaddoe et al., 2007). All children were born between February 2003 and August 2005. We obtained physiological measurements of 528 infants at age 14 months (Dierckx et al., 2009). Child Behaviour Checklist (CBCL) scores at 18 months were available for 474 children, who were included in our primary analyses. In 375 children measurements of maternal psychopathology were available as well.

2.2. Psychophysiologic measurements

Infant heart rate at 14 months was registered with a three-pole ECG lead. We monitored the breathing pattern using a piëzo-electric transducer. Signals were recorded for 8 min using a Vitaport 3 recorder (Temec Inc). During recording, the infant was at ease in its mother's lap. To help the infant relax, we played an episode of the Teletubbies© (BBC/Ragdoll Limited). Of the 8 min recorded, we discarded the first 3 min to allow the infant to reach a proper resting state. As heart rate variability is easily influenced by irregular breathing and movement, we manually selected an interval of 100–180 s where the breathing pattern was most regular and which was free of infant movement and other artefacts. Spectral analysis was conducted on this interval using discrete Fourier transformation (van Steenis et al., 1994).

We used HF variability (0.15 Hz–1.04 Hz) as a measure for cardiac vagal tone. As HF variability of heart rate is dependent on respiratory frequency, which is much higher in infants, we adjusted the upper bound of the HF band according to recommendations from literature (Bar-Haim et al., 2000).

2.3. Measurement of maternal psychopathology

Information on maternal psychopathology 2 months after birth was obtained by the Brief Symptom Inventory (BSI), a validated self-report questionnaire consisting of 53 items (Derogatis and Melisaratos, 1983). Each item was rated on a 5-point Likert scale. For the current study, we used the total scale and the hostility and depression subscales. Scale scores were calculated by summing the item scores involved and then dividing by the number of endorsed items. At 6 months post partum BSI subscales were administered to a subsample of our participants ($n = 265$). While this subsample was too small to conduct a meaningful analysis, it allowed us to investigate the stability of the subscales over. Pearson's correlation between BSI subscales at 2 months and at 6 months ranged from $r = 0.49$ for the depression subscale to $r = 0.64$ for the hostility subscale, indicating high stability.

2.4. Measurement of toddler behaviour

To assess behavioural problems, we used the Child Behaviour Checklist/11/2–5 (CBCL), a parent questionnaire for assessing psychopathology in children aged 11/2–5. The CBCL has good reliability and validity (Achenbach and Rescorla, 2000). Parents rate the child's emotional and behavioural problems over the preceding 2 months on a 3-point scale. For the current study, we used the aggressive behaviour scale (19 items).

2.5. Covariates

We considered sex, gestational age, weight at birth, maternal age, maternal educational level, and smoking and drinking behaviour during pregnancy as confounders because they can influence ANS functioning (Browne et al., 2000; Galland et al., 2006; Haley et al., 2006; Longin et al., 2006; John et al., 2007). Gestational age at birth and birth weight were obtained from community midwife and hospital registries at birth. Information on maternal smoking and drinking habits during pregnancy were obtained by maternal questionnaire.

2.6. Statistical analysis

For a non-response analysis, we compared the 474 children with the physiological measurements session and CBCL data with the 54 children who were not included in the analysis due to missing data. We used *T*-tests, Mann–Whitney *U* tests and chi-square tests where appropriate.

Both maternal BSI scores and toddler externalising behaviour scores were skewed to the right, which is inherent to these instruments when used in a normal population. Log, ln and $1/x$ transformation of the CBCL scores did not correct for this skewness. We decided to perform linear regression analyses using untransformed variables. Because of the non-normality of the variables, regression residuals did show some right skewing. Hence, we checked the consistency of our main findings using logistic regression models with CBCL scores dichotomised at 1 S.D. above the mean.

First, we tested whether maternal psychiatric symptoms were associated with toddler aggressive behaviour using linear regression models. Then, we tested whether infant mean heart rate and HF variability were associated with toddler aggressive behaviour again using linear regression models. Recent studies indicate that the relation between ANS functioning and aggressive behaviour may be different for boys and girls, at least at a later age (Dietrich et al., 2007; Beauchaine et al., 2008). To investigate such a differential relationship in our sample we entered infant mean heart rate and infant sex together with an interaction tem "infant mean heart rate*infant sex" into the same linear regression model. We repeated this procedure for "infant HF variability*infant sex."

Our main aim was to investigate the interaction between infant ANS indices with maternal psychopathology in determining toddler aggressive behaviour. We entered infant mean heart rate and maternal BSI scale score together with an interaction term "infant mean heart rate*maternal BSI score" into one linear regression model. The externalising CBCL scale score was the dependent variable. We followed an identical procedure for infant vagal tone.

The nature of the interactions was explored in strata. We defined two strata by dichotomisation at 1 S.D. above the mean. As CBCL scores were normally distributed in each stratum, we examined the relationship of mean infant heart rate and HF variability with the CBCL aggression scale by means of linear regression.

All analyses were adjusted for all covariates. All statistical analyses were carried out using the Statistical Package for the Social Sciences 13.0 for Windows (SPSS Inc).

3. Results

Table 1 depicts the characteristics of the study participants. In the non-response analysis we found no significant differences between the children included in the primary analyses and the children excluded due to missing data.

Table 2 demonstrates that toddlers of mothers who reported high psychiatric symptoms had more behavioural problems. High maternal