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Maternal postnatal depression predicts altered offspring biological stress reactivity in adulthood

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Summary The offspring of depressed parents have been found to show elevated basal levels of the stress hormone cortisol. Whether heightened cortisol stress reactivity is also present in this group has yet to be clearly demonstrated. We tested whether postnatal maternal depression predicts subsequent increases in offspring biological sensitivity to social stress, as indexed by elevated cortisol reactivity.

Participants (mean age 22.4-years) derived from a 22-year prospective longitudinal study of the offspring of mothers who had postnatal depression (PND group; $n = 38$) and a control group ($n = 38$). Salivary cortisol response to a social-evaluative threat (Trier Social Stress Test) was measured.

Hierarchical linear modelling indicated that PND group offspring showed greater cortisol reactivity to the stress test than control group participants. Group differences were not explained by offspring depressive or anxiety symptoms, experiences of negative life events, elevated

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basal cortisol at age 13-years, subsequent exposure to maternal depression, or other key covariates.

The findings indicate that the presence of early maternal depression can predict offspring biological sensitivity to social stress in adulthood, with potential implications for broader functioning.

1. Introduction

Research has established that the offspring of depressed parents are themselves at elevated risk of depressive disorder by early adolescence (Halligan et al., 2007b; Murray et al., 2011; Hammen et al., 2012). Efforts to understand the biological mechanisms by which such intergenerational risks are conferred have included a focus on the hypothalamic–pituitary–adrenal (HPA) axis. The HPA axis is fundamental to human stress responding, and HPA disturbances have been identified in association with numerous stress-related psychopathologies, most particularly depression (Gold et al., 1988). Basal cortisol concentrations, which are regulated by the HPA axis, may be elevated in children and adolescents during a depressive episode (Lopez-Duran et al., 2009), as well as in adults (Gold et al., 2002; Pariante and Lightman, 2008; Knorr et al., 2010).

Given the assumed role of HPA axis dysfunction in depressive disorder, a number of studies have examined basal cortisol secretion in at-risk groups, including the offspring of depressed parents. Elevated basal cortisol levels have been demonstrated in association with the presence of parental depression across a number of studies (Essex et al., 2002; Halligan et al., 2004; Ellenbogen et al., 2006; Mannie et al., 2007). Higher basal cortisol levels have, in turn, been found to predict subsequent depressive symptom levels (Halligan et al., 2007a; Ellenbogen et al., 2011), although there is contradictory evidence on this point (Carnegie et al., 2014). Longitudinal studies have tentatively indicated that the presence of maternal depression early in development may be particularly important (Essex et al., 2002; Halligan et al., 2004), possibly due to early parenting effects (Murray et al., 2010). Although further evidence is needed, such observations are consistent with rodent models of HPA development which identify the quality of early environmental stimulation as a key determinant of offspring stress reactivity (Meaney et al., 2007).

There is also some evidence of alterations in cortisol stress *reactivity* in the offspring of depressed parents, at least as tested in young infants and children. Elevated cortisol reactivity is suggested by cross-sectional studies of young infants (Azar et al., 2007; Brennan et al., 2008; Waters et al., 2013) and preschool-aged children (Dougherty et al., 2011, 2013) whose parents reported having previous or current depression. These studies have each found relatively higher cortisol levels following a mild stressor in at-risk versus control group infants, but interpretation is complicated by the fact that the provocations used typically did not result in a clear cortisol stress response. Studies where a cortisol stress response was clearly present have not always demonstrated consistent effects. Feldman and colleagues reported that, relative to controls, infants of depressed mothers showed higher cortisol levels overall during a stressor paradigm, but not enhanced reactivity to the stressor (Feldman et al., 2009). In addition, a longitudinal study using immunisation stress in 2 month old infants found antenatal maternal depressive symptoms to be predictive of cortisol reactivity, but the relationship was complex, with both low and high levels of depressive symptoms predicting greater reactivity (Fernandes et al., 2014). In contrast to studies of infants and young children, investigations of older samples have the advantage that standard paradigms exist which reliably provoke a cortisol stress response (Kirschbaum et al., 1993), although few studies have examined cortisol reactivity in youth or adults at risk for depression. One study examining the at-risk offspring of parents with bipolar disorder did not find cortisol responses to stress to distinguish them from controls (Ellenbogen et al., 2006). Moreover, in contrast to the pattern of findings deriving from infants and young children, a second study examining parental retrospective self-reports of ''depressive problems'' found that these were associated with *reduced* cortisol stress reactivity in adolescent girls, and were not associated with cortisol responses in boys (Bouma et al., 2011).

The limited availability of robust evidence for enhanced cortisol reactivity to stress in the offspring of depressed parents is significant, as elevations in basal cortisol levels that have been observed in this group have been proposed to reflect HPA axis dysregulation linked to increased vulnerability to stressors (Ostiguy et al., 2011). Further investigation is warranted. Here, we report on cortisol stress reactivity in a longitudinally studied sample of young adults. Participants in the study were recruited shortly following birth based on the presence or absence of maternal depression during the postnatal period; and maternal depression was repeatedly assessed throughout the 22-years of the study, providing a clear profile of offspring exposure to depressive disorder. In the same sample, we previously identified elevations in basal morning cortisol secretion in the 13-year old offspring of mothers who experienced postnatal depression (PND) versus control group offspring (Halligan et al., 2004). In the current study, offspring cortisol reactivity in response to a standard social stressor (Trier Social Stress Test: TSST) was measured at age 22-years.

We used hierarchical linear modelling (HLM) to model changes in cortisol concentrations over the course of the TSST in relation to PND group status, and to test the hypothesis that PND versus control group offspring would show enhanced cortisol stress reactivity as young adults. We controlled for gender, history of offspring depressive or anxiety disorder, current offspring depressive or anxiety symptoms, and incidences of negative life events in our analyses, given their potential relationship with cortisol reactivity (Burke et al., 2005; Petrowski et al., 2010; Allen et al., 2014). Since