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The lifetime experience of traumatic events is associated with hair cortisol concentrations in community-based children

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ABSTRACT

Adversity early in life can disrupt the functioning of the hypothalamic–pituitary–adrenal axis (HPAA) and increase risk for negative health outcomes. Recent research suggests that cortisol in scalp hair represents a promising measure of HPAA function. However, little is known about the relationship between early exposure to traumatic events and hair cortisol concentrations (HCC) in childhood, a critical period of HPAA development. The current study measured HCC in scalp hair samples collected from 70 community-based children (14 males, mean age = 9.50) participating in the Imaging Brain Development in the Childhood to Adolescence Transition Study (iCATS). Data were also collected on lifetime exposure to traumatic events and current depressive symptoms. Lifetime exposure to trauma was associated with elevated HCC; however, HCC was not associated with current depressive symptoms. Consistent with some prior work, males were found to have higher HCC than females, although results should be treated with caution due to the small number of males who took part. Our findings suggest that hair cortisol may represent a biomarker of exposure to trauma in this age group; however, further study is necessary with a particular focus on the characterization of trauma and other forms of adversity.

1. Introduction

The hypothalamic–pituitary–adrenal axis (HPAA) controls the secretion of steroid hormones, notably cortisol, that interact with environmental contingencies in shaping the development of both stress reactivity and basal HPAA function across the lifespan, especially through mid-childhood and adolescence (Ellis and Essex, 2007; Romeo, 2010). Adverse experiences early in life, including trauma and long-term disadvantage, represent key risk factors for poor mental health and other problems, and particularly across their period of peak incidence in childhood and adolescence (Paus et al., 2008). However, research on the mediating role of HPAA function on the influence of adversity on health outcomes in humans has been limited by various methodological confounds surrounding measures of HPAA function.

Human cortisol levels are most commonly assessed via saliva, urine or serum but are difficult to accurately ascertain. Indeed, inconsistent findings in the literature are likely due to methodological differences in the timing, number and type of measures taken, as well as the large number of other potential confounds (see Kudielka et al., 2009; Vreeburg et al., 2009b). Hair, a relatively new medium for the assay of cortisol, has been proposed as a reliable index of long term function (Koren et al., 2002; Raul et al., 2004; Stalder and Kirschbaum, 2012). Specifically, hair appears to incorporate systemic cortisol over time from endogenous cortisol, as well as dermal sweat, sebum and external sources, however precise mechanisms remain a matter of conjecture (see Stalder and Kirschbaum, 2012). On average, hair grows at a rate of 1.06 cm per

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month (LeBeau et al., 2011), potentially providing a measure of total systemic cortisol over many months. It also has the advantage of offering a non-invasive, low-burden single-sample HPAA measure, versus the multiple temporally-specific and participant-dependent sampling typically required for saliva, urine and serum.

There is good evidence for the test–retest reliability of assaying cortisol in hair (Stalder et al., 2012) and the method has been validated via correlation with average daily salivary levels (D'Anna et al., 2011; van Holland et al., 2011), elevated levels during pregnancy (D'Anna et al., 2011) and disorders such as Cushing's Disease (Thomson et al., 2010). There is also mounting evidence that elevated hair cortisol concentration (HCC) reflects a biomarker of stress exposure across all age groups in non-human primates (e.g., Dettmer et al., 2014) and adult humans. Indeed, HCC is positively related to serious life events in university students (Karlen et al., 2011) and lifetime traumatic events in severely traumatized adults (those with PTSD had higher HCC than those without; Steudte et al., 2011), and has been found to increase in adolescent girls (~14 years old) over the 7-months following a significant traumatic event (i.e., the 2008 Wenchuan earthquake, China) relative to controls (Luo et al., 2012). Other studies with adults have also found relationships with HCC, but in the opposite direction; e.g., a negative association between childhood trauma and HCC (and average diurnal salivary cortisol) has been reported in healthy and depressed adults (Hinkelmann et al., 2013), and traumatized adults (with and without PTSD) have been shown to have lower HCC than non-traumatized adults (Steudte et al., 2013). These varied studies in adults indicate that trauma exposure (independent of PTSD diagnosis) is a relevant correlate of long-term cortisol secretion; however, the opposing directionality of findings demonstrates the need for further work in this area, and an examination of the temporal qualities of the trauma reported.

Dysregulation of the HPAA has been well demonstrated in depressive disorders and risk for their onset (e.g., Dienes et al., 2013; Vreeburg et al., 2009a). The few studies published to date examining associations between HCC and depression also suggest dysregulation of the HPAA, however they also demonstrate varied directionality. Hinkelmann et al. (2013) found no association with depressive symptoms in currently depressed adults and healthy controls; Dettenborn et al. (2012a) found higher HCC in depressed adults over six months (i.e., ~6 cm of hair) compared to healthy controls; whereas Wei et al. (2015) reported associations between HCC and depressive symptoms among patients experiencing a first episode of depression, but not among recurrent MDD patients or healthy controls.[1] In studies involving non-patient populations, a negative association between HCC and depressive symptoms has been reported in a sample of university students (Gerber et al., 2013b),[2] whilst HCC has been found to be positively associated with depressive symptoms among dementia patient caregivers (Stalder et al., 2014). Although results across these studies indicate that depression is a relevant correlate of long-term cortisol secretion, the inconsistent findings suggest a need for further investigation using different cohorts, with differing levels of depressive symptoms, to clarify the nature of this relationship.

Despite the range of studies with adults examining relationships with HCC, comparatively few studies have been conducted with children. Childhood represents a critical period of HPAA development (Romeo, 2010) that requires further study to increase our understanding of pathways from adversity to poor health and behavioral outcomes. This is supported by studies reporting associations between the experience of trauma and salivary cortisol levels (SCL) in children and adolescents. For example, Bevans et al. (2008) found that exposure to high levels of recent trauma in combination with frequent exposure earlier in life was related to both lower morning SCL and higher afternoon SCL. Feldman et al. (2013) examined basal and reactive SCL in war-exposed children (1.5–5 years) and found consistently high SCL in those without PTSD compared to controls, whereas children with PTSD had consistently low SCL.

To date, only three studies have examined relationships between the experience of stressors/trauma and HCC in children, and none have looked at traumatic events more than 12 months prior to HCC sampling. Groeneveld et al. (2013) collected a hair sample that included growth for three months prior to, and two months after, school entry in a cohort of children and found that HCC rose significantly at school entry, an effect associated with high temperamental fearfulness. Vanaelst et al. (2012) examined different cortisol methods and stressors over the prior 12 months in elementary school girls and found that average morning salivary cortisol reflected recent stress (three months), whereas HCC better reflected chronic stress (6–12 months), with positive associations present for both. Using the same cohort, Vanaelst et al. (2013) also examined the relationship between levels of hair cortisone (a metabolite of cortisol) and significant life events and found that hair cortisone was positively associated with both an overall life-event score and a negative life-event score for the prior six months. Three studies have been conducted with HCC in 1-year old infants, and report positive associations with parenting stress (Palmer et al., 2013), certain sociodemographic risk factors, both prenatal (Karlen et al., 2013) and post-natal (Karlen et al., 2015), and with subsequent risk for childhood disease onset to the age of 10 years (Karlen et al., 2015). This suggests that HCC (and cortisone) may be a useful biomarker of stressor/trauma exposure. However, it also raises some important questions, such as whether this association persists after comprehensive adjustment for other relevant variables, including general demographic factors (age, sex, BMI) and state psychological symptoms.

The purpose of this study was to examine relationships between lifetime exposure to traumatic events, depression and HCC in children. Based on previous findings, it was hypothesized that lifetime exposure to (i) more types of trauma, and to (ii) a greater number of traumatic events (across type), would both be significantly associated with elevated HCC. To examine the relationship between more proximate adverse states and HCC, we also investigated whether current depressive symptoms were associated with HCC. Although the inconsistent findings with adults and lack of studies with children in relation to HCC and depression do not point to a clear hypothesis, we conjecture that sufficiently high levels of depressive symptoms might be associated with elevated HCC (as per Stalder et al., 2014). Given previously identified relationships between HCC and sex (Dettenborn et al., 2012b), age (Dettenborn et al., 2012b) and BMI (Noppe et al., 2014; Veldhorst et al., 2014), we also investigated bivariate relationships between these variables and participants' HCC, and whether exposure to traumatic events was associated with HCC after controlling for these potential covariates.

2. Methods

2.1. Participants

Participants were 70 community-based children (14 males; M age 9.50 years, SD = 0.30) participating in a multi-wave population-

[1] There were differences in the proportion of medicated depressed participants between studies (~50% [Hinkelmann et al., 2013] and ~96% [Dettenborn et al., 2012a] medicated). Although analyses did not indicate a role of medication, differing proportions may reflect differences in underlying severity or impairment. Wei et al. (2015) did not collect data on medication use in patients.

[2] Controlling for vigorous physical activity ameliorated (but did not extinguish) relationships (see also Gerber et al., 2013a).