Psychoneuroendocrinology (2013) 38, 2666—2675

Available online at www.sciencedirect.com

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Cumulative effects of early poverty on cortisol in young children: Moderation by autonomic nervous system activity

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Received 4 January 2013; received in revised form 14 June 2013; accepted 20 June 2013

KEYWORDS
Child development;
Poverty;
Stress physiology

Abstract The relation of the cumulative experience of poverty in infancy and early childhood to child cortisol at age 48 months was examined in a prospective longitudinal sample of children and families (N = 1292) in predominantly low-income and rural communities in two distinct regions of the United States. Families were seen in the home for data collection and cumulative experience of poverty was indexed by parent reported income-to-need ratio and household chaos measures collected between child ages 2 months and 48 months. For the analysis presented here, three saliva samples were also collected over an approximate 90 min interval at child age 48 months and were assayed for cortisol. ECG data were also collected during a resting period and during the administration of a mildly challenging battery of cognitive tasks. Mixed model analysis indicated that child cortisol at 48 months decreased significantly over the sampling time period and that cumulative time in poverty (number of years income-to-need less than or equal to 1) and cumulative household chaos were significantly related to a flatter trajectory for cortisol change

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0306-4530/$ — see front matter
http://dx.doi.org/10.1016/j.psyneuen.2013.06.025

and to an overall higher level of cortisol, respectively. Findings also indicated that respiratory sinus arrhythmia derived from the ECG data moderated the association between household chaos and child cortisol and that increase in respiratory sinus arrhythmia during the cognitive task was associated with an overall lower level of cortisol at 48 months.

A large literature has shown that children growing up in poverty face substantially increased risk for early learning difficulties and behavior problems (Bradley and Corwyn, 2002; Brooks-Gunn and Duncan, 1997). One potential mechanism of this effect involves stress. A growing literature suggests that one way in which poverty adversely affects children's development is by altering levels of stress hormones (Blair et al., 2011a,b; Evans, 2003; Lupien et al., 2001). Alteration of stress hormone levels through factors present in the context of poverty is of substantial concern given that these hormones modulate activity in brain areas that underlie the control of attention and emotion (Arnsten, 2009; Floresco, 2013). At moderate levels of increase, catecholamines and glucocorticoids stimulate synaptic activity in areas of prefrontal cortex associated with working memory, executive function, and the regulation of attention and emotion (Alexander et al., 2007; Lupien et al., 1999; Ramos and Arnsten, 2007). Consequently, stress hormones can shape the circuitry of the brain, both developmentally and in adulthood. Human and animal models have shown that prenatal and postnatal stress as well as stress in adulthood alters cortical morphology and connectivity and functional activity in areas of PFC and associated limbic regions that underlie self-regulation and executive cognitive abilities (Bock et al., 2012; Liston et al., 2006; Hanson et al., 2010). As such, examinations of the effects of the environment, particularly the early environment, on stress hormone levels in children provide support for the idea that one pathway through which poverty increases risk for behavioral and learning difficulties in children is through effects on brain structure and function.

Examinations of child stress physiology in the context of poverty have frequently focused on the glucocorticoid hormone cortisol, usually as obtained from saliva samples. Cortisol is the end product of a cascade of activity in the hypothalamic–pituitary–adrenal (HPA) axis in which stimulation initiates the release of corticotropin releasing hormone (CRH) from the paraventricular nucleus of the hypothalamus leading to the secretion of adrenocorticotropic hormone from the pituitary and resulting release of cortisol from the adrenals. In part, the focus on cortisol in research on stress physiology and child development reflects the fact that the HPA axis is the slower acting branch of the stress response system. Notably, effects of glucocorticoids on development are primarily gene mediated (de Kloet et al., 2008; Joels and Baram, 2009), meaning that glucocorticoids stimulate activity at the cell nucleus that can shape downstream physiological activity with implications for psychological development. As such, cortisol may be a robust indicator of longer term effects of experience on development. Alterations to resting levels may lead to variation in gene activity and to alterations in brain structure and function in ways that shape the development of physical and mental health (McEwen and Gianoros, 2011).

Although few published studies have examined the relation of poverty to stress physiology in children, those that do form a relatively coherent picture. In one study, lower parent reported income was associated with higher single time point cortisol levels in a cross sectional sample of 6–10 year old children but not 11–16 year old children (Lupien et al., 2001). A second study with a sample of 9–11 year old children examined multiple aspects of the stress response including overnight levels of norepinephrine and epinephrine as well as cortisol. These measures, combined with others (blood pressure, body mass index) into an index of allostatic load, were positively and linearly related to a cumulative index of psychosocial and physical risk factors in the home (Evans, 2003). A third study (Miller et al., 2009) using genome wide transcriptional profiling found that low SES in childhood was associated with up regulation of genes associated with adrenergic neural receptor function and down regulation of genes associated with glucocorticoid receptor function in adulthood; a pattern consistent with an expected higher level of reactivity and less prototypically effective regulation of physiological reactivity in individuals experiencing poverty in early childhood. A fourth study, with a sample of children 13–18 years of age found that SES was inversely associated with cortisol increase over two years and that family disorganization as assessed by a measure of household chaos was a relevant aspect of this effect (Chen et al., 2010).

In addition to the aforementioned studies, previous findings from the longitudinal early childhood sample used in this analysis (N = 1292) have shown high levels of resting or basal cortisol at 7 through 48 months of age for children facing early socioeconomic disadvantage (Blair et al., 2011a) and shown that observed parenting behavior partially mediated effects of socioeconomic disadvantage on cortisol levels in children (Blair et al., 2011b). Findings from this sample have also shown that sensitive parenting behavior observed when children are infants is associated with a higher level of cortisol reactivity to an emotionally arousing situation when children are 7 months old and with an overall lower level of cortisol in response to emotional arousal when children are 15 months old (Blair et al., 2008). Findings from that previous analysis also indicated that child temperament, namely distress to novelty, was associated with a higher level of cortisol at age 15 months. It may be that child characteristics are relevant to examinations of the effect of poverty on child stress physiology, and we examine temperament as well as child intelligence as additional or alternative explanations for any effects of the environment on child cortisol.

Given the small but consistent literature on poverty and stress physiology in childhood, it is of interest to separately examine distinct but related aspects of early disadvantage, including income to need but also characteristics of the home such as chaos that covary with but are not identical with poverty. It is also of interest to examine indicators of child sympathetic and parasympathetic activity simultaneously