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Poverty-alleviation program participation and salivary cortisol in very low-income children

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ABSTRACT

Correlational studies have shown associations between social class and salivary cortisol suggestive of a causal link between childhood poverty and activity of the stress-sensitive hypothalamic–pituitary–adrenocortical (HPA) system. Using a quasi-experimental design, we evaluated the associations between a family's participation in a large-scale, conditional cash transfer program in Mexico (*Oportunidades*, formerly Progresa) during the child's early years of life and children's salivary cortisol (baseline and responsivity). We also examined whether maternal depressive symptoms moderated the effect of program participation. Low-income households (income <20th percentile nationally) from rural Mexico were enrolled in a large-scale poverty-alleviation program between 1998 and 1999. A comparison group of households from demographically similar communities was recruited in 2003. Following 3.5 years of participation in the *Oportunidades* program, three saliva samples were obtained from children aged 2–6 years from intervention and comparison households ($n = 1197$). Maternal depressive symptoms were obtained using the Center for Epidemiologic Studies-Depression Scale (CES-D). Results were that children who had been in the *Oportunidades* program had lower salivary cortisol levels when compared with those who had not participated in the program, while controlling for a wide range of individual-, household- and community-level variables. Reactivity patterns of salivary cortisol did not differ between intervention and comparison children. Maternal depression moderated the association between *Oportunidades* program participation and baseline salivary cortisol in children. Specifically, there was a large and significant *Oportunidades* program effect of lowering cortisol in children of mothers with high depressive symptoms but not in children of mothers with low depressive symptomatology. These findings provide the strongest evidence to date that the economic circumstances of a family can influence a child's developing stress system and provide a mechanism through which poverty early in life could alter life-course risk for physical and mental health disorders.

Introduction

Poverty and child outcomes

The effects of growing up in poverty extend from infancy through adolescence and into adulthood (Bradley & Corwyn, 2002) and include poor health, cognitive and socioemotional delays and behavioral problems (Evans, 2004). For most child health outcomes, there is a gradient in which lower family socioeconomic status (SES) and material well-being is associated with concurrently poorer outcomes (Chen & Matthews, 2006). Experiencing poverty during childhood also appears to have an independent effect on later health outcomes including decreased life span and increased morbidity, even while controlling for adult SES or social status (Poulton et al., 2002). Children growing up in poverty, whether indexed by the family's income or, in low technology cultures, by other indices of the family's material well-being are exposed to a wide range of risk factors, and these conditions are exacerbated in the developing world (Walker et al., 2007). An estimated 219 million children (39% of all children in developing countries) have evidence of signs of poor development at multiple levels (Grantham-McGregor et al., 2007).

Salivary cortisol as a marker of stress system activity

The multiple mechanisms through which poverty produces poor outcomes are being actively explored. Lack of income

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leading to deficiencies in material well-being (e.g., inadequate housing, medical care, food, clothing) is associated with greater exposure to stressors (Elder, 1985). These stressors, ranging from increased exposure to pathogens to increased exposure to violence, activate the hypothalamic–pituitary–adrenocortical (HPA) system. The HPA system is one of the primary components of the mammalian stress response system (Gunnar & Davis, 2003), and is one mechanistic pathway that has received significant theoretical attention (McEwen & Seeman, 1999). When the brain first perceives a stressor, neural input to the hypothalamus induces secretion of corticotropin-releasing hormone (CRH), which stimulates release of adrenocorticotropic hormone (ACTH) from the pituitary gland followed by the release of cortisol from the cortices of the adrenal glands. Following exposure to a stressor, it typically takes 20–30 min for cortisol levels to reach their peaks (De Kloet, 1991); multiple negative feedback mechanisms inhibit further production of CRH and ACTH (Gunnar, Bruce, & Hickman, 2001). Cortisol rises in situations associated with physical and psychological stressors, including inflammatory immune responses, heat or cold stress, novelty or uncertainty, social conflict, negative emotions and feelings of threat or loss of control (Kirschbaum & Hellhammer, 1994).

SES and stress system activity

In the developed world, higher SES children have lower cortisol concentrations than lower SES children (Essex, Klein, Cho, & Kalin, 2002; Evans & English, 2002; Evans & Kim, 2007; Lupien, King, Meaney, & McEwen, 2000, 2001). These associations have been confirmed in some, but not all, studies in adults (Cohen, Doyle, & Baum, 2006; Dowd & Goldman, 2006). To our knowledge, only two studies have examined economic background and how it relates to stress response in children in a developing country. One study (Flinn & England, 1997, 2003) examined generally low SES children in Dominica. Within the range of economic circumstances they examined, there was no evidence that material benefits (improved housing or diet) were associated with salivary cortisol levels. They did find, though, that children from unstable family backgrounds had higher cortisol levels than did children from stable family homes. The other study set in Nepal measured morning cortisol and a composite index of physiologic functioning, allostatic load, created using cortisol and cardiovascular variables. The physiologic measures differed according to context in which a child lived (e.g., homeless, squatter, village or school) (Worthman & Panter-Brick, 2008); boys who were in school showed a healthier stress profile than those who were squatters or those who were homeless.

Thus, in spite of the small handful of studies that have shown that lower SES is linked with higher and/or dysregulated cortisol, there remains a large research gap relating to whether and how poverty and cortisol are related in young children. Most critically, none of the studies that have examined socioeconomic status and activity of the HPA axis in children or, for that matter, in adults have randomly assigned participants to their economic conditions. Normally, this scenario would not be possible and we would be left with correlational findings where the direction of effects and the potential mediators of the associations could not be wholly examined. Thus, the primary goal of this study was to examine the association between income and children's cortisol levels using participants drawn from a large-scale, randomized poverty-alleviation intervention. This sample allowed examination of the causal relation between income and children's cortisol levels. In addition, because few of the studies of SES and cortisol have examined children living in extreme poverty, the present study extends existing research on poverty and children's HPA axis activity to children experiencing the type of extreme poverty that is prevalent in many developing countries.

Maternal depression and low SES

The studies cited above that have found that lower SES is associated with higher cortisol levels in children have all also reported that the effects of lower SES may be *mediated*, at least in part, by maternal depressive symptoms (Bradley & Corwyn, 2002). Maternal depression is associated with having lower income and greater income insecurity in both the developed and developing world (Hadley & Patil, 2008; Pascoe, Stolfi, & Ormond, 2006). When parental care is poor or frequently disrupted during early development, increased levels of cortisol are released in response to threat as measured later in life (Meaney & Szyf, 2005). Depressed mothers tend to provide less sensitive and responsive care to their infants and young children, particularly in the first year of life (Dawson & Ashman, 2000). Further, children of depressed mothers are often less able to meet their children's age-appropriate and stage-salient needs (Arcenio, Sesin, & Siegel, 2004).

If maternal depression serves to influence the impact of SES on children's cortisol levels, then within the context of a poverty-alleviation intervention study, this would argue for examination of maternal depression as a moderator variable. That is, if under the no-treatment condition children of mothers with high depressive symptoms show the most dysregulated pattern of cortisol production, then under the treatment condition, children of depressed mothers may show the greatest impact on HPA activity. There are also other reasons that maternal depression might moderate child HPA axis activity (Goodman & Gotlib, 1999). Depressed mothers may pass genes to their children that increase the reactivity of the axis to the conditions (pathogens, food insecurity) associated with poverty (Goodman, 2003). Similarly, they may expose their infants to elevated stress hormones during prenatal development, and these exposures may increase the sensitivity of the HPA system to postnatal conditions. Thus, using a construct introduced by Boyce and Ellis (2005), the offspring of depressed mothers may be more sensitive to context, showing larger differences than the offspring of non-depressed mothers in response to better or worse family-level factors.

However, there is also a substantial animal and human literature indicating that when parental care is actively supportive, offspring can be relatively *buffered* from the impact of a variety of stressful events (see review, see Gunnar & Donzella, 2002). This process – maternal or parental buffering – reflects the capacity of a supportive adult to prevent or reduce elevations in cortisol, perhaps through stimulation of "anti-stress" hormones such as oxytocin (e.g., Carter, 1998). A responsive and high-functioning mother, however, may also regulate stress experiences in her offspring through active management of the environment (Leiferman, 2002; McLennan & Kotelchuck, 2000).

Previously, among 2.5- to 6-year old children growing up in urban poverty in Mexico, we reported that among offspring of depressed relative to non-depressed mothers, maternal depression did not affect baseline cortisol levels, but did affect salivary cortisol response to the same mild stressor used in this report (Fernald, Burke, & Gunnar, 2008). Unexpectedly, rather than maternal depression being associated with increased cortisol reactivity, it was associated with a blunted cortisol response. While the blunted response was unanticipated, it may have reflected the impact of chronic stress down-regulating activity of this system (see Miller, Chen, & Zhou, 2007). Thus,