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Do neighborhoods matter? Neighborhood disorder and long-term trends in serum cortisol levels

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Abstract

Background—Characteristics associated with low socioeconomic status neighborhoods may put children at risk for unique chronic stressors that affect cortisol levels. This research sought to explore whether neighborhood stressor exposure affected serum cortisol levels among children.

Methods—A total of 148 African and European American children with an average age of 8.28 years participated in a longitudinal study evaluating ethnic differences in body composition and disease risk. A total of five waves of data were included in analyses. Mixed modeling was used to explore neighborhood stressors, which was a composite index of five items for zip code level poverty and physical disorder, and serum cortisol outcomes for the full sample, by race/ethnicity and gender. Adjustments were made for individual level correlates age, pubertal status, gender, and total fat mass.

Results—Neighborhood disorder was predictive of lower serum cortisol levels among African American children ($p<.05$), such that higher neighborhood stressor exposure resulted in lower serum cortisol over time compared to individuals in socially ordered neighborhoods. Neighborhood disorder was marginally significant and predictive of higher serum cortisol among European American children ($p <.10$). Transition to a higher pubertal status, nested in age was also predictive of lower serum cortisol levels ($p<.01$) among European American children.

Conclusion—Children who are exposed to negative socioenviornmental climates over time are more likely to have altered serum cortisol levels. This may be an adaptive mechanism to cope with stress; however, disrupted cortisol levels may have negative effects on general physical and mental health.

Keywords

Neighborhood; cortisol; children

Neighborhood environments may be sources of negative stressful stimuli that pose significant health risks for children. The work of William Julius Wilson suggests that health outcomes within specific geographic locations cannot be understood without taking into account the social and economic circumstances from which they emerge.[1, 2] Researchers posit that neighborhood chronic stressors such as noise, violence, and poverty may disrupt

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endocrine pathways such as the Hypothalamic Pituitary Adrenal axis (HPA) and the sympathetic nervous systems.[3–6] In turn, these disruptions may contribute to the development of metabolic disorders.[7]

While many studies have associated neighborhood processes with psychosocial outcomes [8–12] few have assessed the direct effects of neighborhood characteristics on the physiological functioning of children.[3, 6, 13] The studies that have been conducted primarily focus on neighborhood socioeconomic status (SES) which leaves the effects of neighborhood socioenvironmental conditions relatively unexplored. Additionally, many of these studies have evaluated the relationships independently of body composition although adiposity is known to affect cortisol secretion.[14–16] The limited available data suggest that familial environment and individual and community level SES are highly correlated with cortisol.[3, 6, 13, 17, 18] Disentangling the relationship between low community-level SES and cortisol may show that neighborhood characteristics such as physical and social disorder may be the chronic stressors that affect the HPA axis. [3, 6, 13] Therefore, the aim of this research was to explore neighborhood stressor exposure and the relationship to cortisol.

Cortisol is a physiologically induced mechanism whereby the HPA axis mediates the effects of stressful life events on biological functioning via increased cortisol output.[18–22] Cortisol is activated when the HPA axis stimulates the production of corticotrophin releasing hormone in response to a stressor. In turn, adrenocorticotrophic hormone is released, with the end result of cortisol secretion into the blood stream.[18–21, 23] While cortisol is central in maintaining homeostasis during acute stress [15], exposure to chronic stressors can result in dysregulation of the HPA axis through either hyper- or hypo-cortisolism [9, 22, 24–26] and may increase the risks for hypertension, insulin resistance, neuronal damage, immune disorders, mental health disorders, and disrupt the ability to deliver cortisol to sites of inflammation within the body.[9, 22, 24–28] Exposure to acute stressors may result in higher cortisol, which may have negative metabolic health outcomes and conversely, exposure to chronic stressors, may result in lower cortisol secretion which may increase inflammation and negative health risks. In order to evaluate the relationship between neighborhood stressors and cortisol, it was hypothesized that neighborhood chronic stressor exposure would result in lower serum cortisol.

METHODS

Study Population and Design

The study consisted of 178 participants who took part in a longitudinal study evaluating racial and ethnic differences in the relationship between body composition and disease risk. After accounting for attrition a total of 148 children (African American = 67 and European American 81) with an average age of 8.30 years at baseline were included in the analytic cohort. Methods and research findings from this cohort have been published previously.[29–31] After receiving Institutional Review Board approval at the University of Alabama at Birmingham, participants were recruited by posting fliers at clinics and through participant referrals. A total of 39 children had data for 1 visit, 26 had data for 2 time points, 34 had data for 3 time points, 25 had data for 4 time points, and 24 had data for 5 time points.

Eligibility requirements included being of African- or European- American descent and being between 4 to 12 years of age at study entry. Participants were healthy, and were not taking any medications known to affect metabolism or body composition (e.g. Attention Deficit Hyperactivity, asthma medications, and corticosteroids). Individuals who agreed to participate were provided with consent information. During the first visit, the study protocol was reviewed and both parents and children provided consent and assent respectively.

The sample consisted of male and female African- and European- American children recruited from the Birmingham-Hoover MSA. Baseline data were acquired in 1994, and subjects returned annually for up to nine years after initial evaluation. Due to increased attrition, available data for up to five time points were used. There were no significant differences in baseline data for the larger cohort and the analytic cohort. Geographic data obtained from the US Census Bureau (2000) were used to assess neighborhood risk factors.

Measurements

This study included individual and group level data, obtained from clinical, survey, and objective measurements. At each yearly time point, participants came to the University for two separate visits. During the first visit, body composition by Dual-energy X-Ray absorptiometry (DXA) was recorded, anthropometric measurements were taken, surveys were conducted, and pubertal status was assessed by a physician according to the criteria of Marshall and Tanner [32, 33]. At the second visit, participants were admitted to the General Clinical Research Center (GCRC) for an overnight evaluation during which intravenous glucose tolerance tests were administered and blood pressure measurements were obtained. Children were served a standard meal and snacks which were consumed before 2000 hours. After the overnight fast, blood samples were taken for hormone analyses at approximately 700 hours.

Dependent variable

Total serum cortisol was obtained from participants after an overnight fast at the GCRC. Unlike salivary samples, total serum cortisol levels are not sensitive to the diurnal rhythm and as such, all samples were obtained at approximately the same time for all individuals. Biochemical analyses were conducted in the Metabolism Core Laboratory of the Clinical Nutrition Research Center and the GCRC at the University. Cortisol assays were performed using a Coat-a-Count radioimmunoassay method manufactured by Diagnostic Products Corporation, Los Angeles, California (now Siemens Medical Solutions Diagnostics Cary, North Carolina). This test has a sensitivity of 0.2μg/dL and inter-and intra assay coefficients of variation of 7.77% and 4.41% respectively.

Independent Variable

Objective indicators of neighborhood disorder were compiled from the US Census. The only available participant identifiers were zip codes. Zip Code Tabulation Areas (ZCTA) were developed by the US Census (2000) to compute summary statistics at the zip code level. The ZCTA represents the zip code used by the majority of addresses within a given area and may vary slightly from United States Postal codes. Due to differences in development, it is possible that addresses within a zip code may be assigned to a ZCTA that does not correspond with the actual zip code.[34] At the time of the study period, the Birmingham-Hoover MSA had a population of 921,106 and ZCTAs for the study participants covered 73% of the total population. The average population size was 17,749 (standard deviation ± 8613) and an average geographic size of 78799.27 km^2. For the study period, there was no neighborhood mobility which indicates that no children included in analyses had moved during the five waves of data collection. As such, only one neighborhood value was developed for each child.

The neighborhood index included percentages of unemployment, poverty, female headed households with dependent children, and vacant housing.[1, 2, 35–37] While the first three items gauged neighborhood SES, percent vacant housing indicated physical disorder and within impoverished areas may indicate dilapidated or abandoned housing which could be sources of illegal activity.[38] The neighborhood items were highly correlated with percent female headed households positively correlated with percent vacant housing, poverty, and

unemployment (r = 0.769, 0.901, and 0.793 respectively). Percentage vacant housing was strongly correlated with percent poverty and unemployment (r = 0.896 and 0.904 respectively). Lastly, percent poverty was highly correlated with unemployment (r = .865). For each individual item, Z scores were computed by subtracting the mean for the sample of ZCTAs and then dividing by the standard deviation. The Z scores for each measure were then summed to create a composite index. The Z score method has been established as a valid tool to assess neighborhood characteristics.[39, 40] Higher scores indicated greater neighborhood disorder relative to other children in the sample (expressed as standard deviation units above the mean) whereas lower scores indicated that respondents encountered less disadvantage (expressed as standard deviation units below the mean). The scale had a standardized Cronbach's Alpha = .96.

Total and regional body fat were assessed during each visit by DXA with the Lunar DPX-L scanner (GE-Lunar Radiation Corp., Madison, WI). Participants were scanned in light clothing with arms at their sides. In children, DXAs have shown high reliability and function as good indicators of body fatness with a correlation above .96.[41]

Additional covariates included self-reported age, gender, and pubertal status. Pubertal status was assessed by the criteria of Marshall and Tanner [42, 43] by a physician during the annual physical examination. Pubertal staging, defines physical measurements of reproductive maturity based on secondary sexual characteristics. [44, 45] There are 5 Tanner stages that have been demonstrated as reliable indicators of pubertal development. The staging based on the criteria of Marshall and Tanner is according to both breast and pubic hair development in girls (http://www.fpnotebook.com/Endo/Exam/FmlTnrStg.htm) and genitalia and pubic hair development in boys (http://www.fpnotebook.com/Endo/Exam/MlTnrStg.htm). The development is a continuous process in which an individual is assigned to one of the 5 categories. One value is assigned and represents the higher of the two values observed for breast/genitalia and pubic hair. Ethnicity was based on self-reports by the parent.

Statistical Methods

The sample included 148 children (67 African American and 81 European American) and a total of 413 clinical observations. At baseline, descriptive statistics and t-tests were performed to examine significant differences in the independent and dependent variables, with a significance criteria of $p<.05$. Variables were evaluated for normality and serum cortisol and total fat were log transformed.

The mixed modeling (SAS Proc Mixed; SAS, 2002) was used to examine longitudinal trends in serum cortisol for the full sample, by ethnic group, and gender.[46] This approach accounts for the high degree of within subject correlations in cortisol values and includes both random and fixed effects. After accounting for intra-individual correlation, this model adjusts for between subject variations in cortisol. Mixed modeling is flexible and can be fitted to handle heterogeneity of variances across subjects. Because this method uses maximum likelihood estimation and the missing values were random, this method can handle missing values without discarding available data. Two separate models were evaluated and included individual level variables, gender, age, pubertal status, and total fat mass to assess the independent contributions to cortisol. The second model included the individual level variables and adjustment for neighborhood disorder. In all analyses, pubertal stage was nested in age. Post hoc analyses examined whether the regression coefficients of the pubertal stages were significantly different and whether the neighborhood disorder coefficients significantly differed by race/ethnicity. A series of regression models were examined to determine if specific pubertal stages exhibited significantly greater effects on serum cortisol relative to other stages. A series of dummy variables was created with Tanner

Stage 2 as the reference stage (chosen based on results from initial mixed models) to examine whether this stage exhibited greater effects on serum cortisol.

Due to heterogeneity within the majority of the ZCTAs (i.e. on average less than 2 children per ZCTA), no analyses accounted for neighborhood clustering. Using neighborhood level-data as an independent level variable instead of as a hierarchical two-level model is valid for this type of research.[35] All statistical analyses were performed using SAS (version 9.1, 2002, SAS Institute, Cary, NC).

RESULTS

The average age at baseline was 8.28 years (±1.68) (Table 1). Approximately 57 percent of the sample was female. African American children were younger ($p < .05$) and had slightly more total fat mass than European American children. Neighborhood composite index scores ranged between −1.94 and 5.68 standard deviation units below and above the mean. Percentages for the individual items of the composite neighborhood disadvantage score are also provided, African American children were significantly more likely to live in disordered neighborhoods ($p < .05$) characterized by higher unemployment, poverty, single female-headed households, and vacant housing. There were no significant racial/ethnic differences in baseline or longitudinal serum cortisol measurements.

Results from the mixed model for the full sample indicate that over the study period, pubertal stage emerged as a significant predictor of serum cortisol levels at all ages (Table 2). Post hoc analyses indicated no significant differences in the relationships between pubertal stages and serum cortisol. Fat mass was predictive of lower serum cortisol among children. The second model included the individual level covariates and neighborhood disorder. Over the study period, neighborhood disadvantage predicted lower serum cortisol levels such that children who lived in disordered neighborhoods had lower serum cortisol levels than children in more socially ordered areas. In this model, the most significant predictor of serum cortisol levels was pubertal stage 2 where, at this stage of puberty, reduced serum cortisol levels were evident. With the inclusion of neighborhood effects in the model, pubertal stages 2 and 3 became significant and both negatively affected serum cortisol levels. Post hoc analyses indicated that children in pubertal stage 2 had significantly lower serum cortisol levels than children in pubertal stage 4 ($p<.05$).

Table 3 reports longitudinal results for African American children. In the first model, none of the independent variables were significant predictors of total serum cortisol. When the neighborhood disadvantage measure was included, pubertal stage 3, nested in age, was significantly predictive of lower serum cortisol levels $p < .05$. The composite neighborhood disorder index was also predictive of lower serum cortisol levels ($p<.05$), such that over time, neighborhood stressors affected cortisol and resulted in lower serum cortisol levels.

Table 4 provides results for the European American children. As children progressed through the pubertal transition, significant effects on serum cortisol emerged. Pubertal stages 1, 2, and 3 nested in age, significantly predicted serum cortisol such that, at each pubertal stage, children had lower serum cortisol levels. Children entering pubertal stage 2 had significantly lower serum cortisol levels relative to pubertal stages three and four ($p < .05$). The neighborhood composite index was marginally significant ($p < .10$) and predicted increased serum cortisol levels. In this model, all pubertal stages retained significance, and children in pubertal stage 2 had significantly lower serum cortisol relative to children in pubertal stage 4 ($p < .05$). Post hoc analyses indicated that there were no significant differences in the neighborhood disorder regression coefficient across racial/ethnic groups ($p>.05$).

Separate mixed models by gender were also conducted. While both models showed that neighborhood index operated negatively on serum cortisol, neither indicated significant relationships between neighborhood disorder and serum cortisol (data not presented).

DISCUSSION

This is the first longitudinal study that evaluates the relationship between neighborhood social and physical characteristics and cortisol. The findings suggest that disordered neighborhood exposure affects serum cortisol levels. Initial research indicated that low SES and mother's depressive state are related to hypersecretion of salivary cortisol among children.[6, 13] Salivary cortisol represents the biologically active fraction of cortisol.[21, 22] For the current study, results from total serum cortisol indicate that neighborhood disorder is related to lower cortisol among African American children. This supports the research of Chen and Paterson [3]; where children in lower SES neighborhoods have lower basal salivary cortisol levels. These findings also support the hypothesis of Lupien and colleagues[6] that the mechanism between neighborhood SES and altered cortisol lies in the characteristics of low SES neighborhoods (i.e. increased vacant housing, higher rates of unemployment, and poverty.

Because African American children were more likely to live in disordered neighborhoods, it was important to determine whether neighborhood indicators were surrogates for ethnicity. Although marginally significant, it was found that neighborhoods were predictive of higher serum cortisol among European Americans. These findings are in line with results from Lupien and colleagues but are the inverse of the results for African American children in this sample. While it is suggested that there are ethnic specific neighborhood pathways that are pertinent to health,[35] Chen and colleagues posit that the accumulation of stressors will result in blunted cortisol among all children as they transition into adulthood.[3] Further research needs to be conducted to understand why neighborhood stressor exposures may operate differently for racial/ethnic groups and to examine whether African and European Americans exposed to these stressors exhibit blunted cortisol during adulthood.

While research demonstrates that cortisol reactivity, as assessed by social stress tests, is different between men and women [22], the current study found no significant gender differences. Research has shown that cortisol secretion is suppressed by estradiol [47, 48] however, the HPA axis does not fully develop until pubertal stage three [48], which may explain why there were no significant sex differences. Pubertal stage nested in age, was related to lower serum cortisol in children. This seems to indicate that cortisol is significantly affected by pubertal development. Although not significant, greater fat mass affected serum cortisol such that children with greater fat mass exhibited lower total serum cortisol which is consistent with Hanrahan and colleagues.[20] Cortisol is intimately tied with adipose tissue, particularly abdominal fat, as it is related to an increased clearance of cortisol.[14] The increased clearance results in comparable or lower levels of cortisol in obese individuals, when compared to non-obese individuals.[14, 49,22]

Limitations

To our knowledge, there are no established guidelines for healthy cortisol ranges for a biethnic sample [9, 22] therefore, the extent of altered serum cortisol is unknown. Second, only one serum cortisol measurement was obtained at each time point; it is known that cortisol follows a diurnal pattern, and salivary cortisol may be a better indicator.[22, 50] While the neighborhood measure was developed using theory and findings from the literature, it is possible that important indicators of neighborhood disorder were not taken into account. The inclusion of these indicators may have resulted in stronger observed relationships between neighborhood disorder and serum cortisol. Also, ZCTAs were used to assess neighborhoods;

smaller units of analysis may be more preferable. Because, the neighborhood index was obtained from the decennial US Census, there may have been neighborhood changes over the study period. However, research indicates that neighborhoods remain relatively stable over time and can be "identified by patterned behavior and reproducibility of patterns across space and time" from which certain cultural values and beliefs are developed and maintained.[1, 51] Based on these conclusions we propose that the neighborhoods did not experience significant changes in exposures over the study period.

Individual level SES is not accounted for in the present analyses and while. researchers have found significant relationships between individual level SES and cortisol in young children [6], there has been no evidence of this relationship during adolescence. [3, 6, 23] Also, there are no measures that account for parental stress levels, research has established that maternal stress is related to cortisol levels among young children. [8, 10] Lastly, important mediators such as social support are not included..[24, 26, 52–54] A strength of this study is the use of DXA to measure adiposity. While body mass index has been used as a surrogate of adiposity for cortisol research, this may inaccurate because BMI underestimates/ overestimates adiposity prevalence in a multi-ethnic sample.

This study underscores the need to measure aspects of the social and physical environments above and beyond traditional measures of SES. It is possible that the consistent early exposure to unhealthy social and physical environments may contribute to the health disparities that are evident by geographic residence. Future studies should assess longitudinal relationships to cortisol using frequent sampling techniques and include other potential sources of chronic stress.

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Reference List

1. Wilson, WJ. The truly disadvantaged: The inner city, the underclass, and public policy. Chicago, IL: University of Chicago Press; 1987.
2. Wilson, WJ. When Work Disappears: The world of the new urban poor. New York, NY: Alfred A Knopf: distrubted by Random House; 1996.
3. Chen E, Paterson LQ. Neighborhood, family, and subjective socioeconomic status: How do they relate to adolescent health? Health Psychol. 2006 Nov; 25(6):704–14. [PubMed: 17100499]
4. Evans GW, Lercher P, Meis M, Ising H, Kofler WW. Community noise exposure and stress in children. J Acoust Soc Am. 2001 Mar; 109(3):1023–7. [PubMed: 11303916]
5. Leventhal T, Brooks-Gunn J. The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. Psychological Bulletin. 2000 Mar; 126(2):309–37. [PubMed: 10748645]
6. Lupien SJ, King S, Meaney MJ, McEwen BS. Can poverty get under your skin? basal cortisol levels and cognitive function in children from low and high socioeconomic status. Dev Psychopathol. 2001; 13(3):653–76. [PubMed: 11523853]
7. ez Roux AV, Jacobs DR, Kiefe CI. Neighborhood characteristics and components of the insulin resistance syndrome in young adults: the coronary artery risk development in young adults (CARDIA) study. Diabetes Care. 2002 Nov; 25(11):1976–82. [PubMed: 12401742]
8. Ashman SB, Dawson G, Panagiotides H, Yamada E, Wilkinson CW. Stress hormone levels of children of depressed mothers. Dev Psychopathol. 2002; 14(2):333–49. [PubMed: 12030695]

9. Bevans K, Cerbone A, Overstreet S. Relations between recurrent trauma exposure and recent life stress and salivary cortisol among children. Dev Psychopathol. 2008; 20(1):257–72. [PubMed: 18211737]
10. Brennan PA, Pargas R, Walker EF, Green P, Newport DJ, Stowe Z. Maternal depression and infant cortisol: influences of timing, comorbidity and treatment. J Child Psychol Psychiatry. 2008 Oct; 49(10):1099–107. [PubMed: 18492036]
11. Cicchetti D, Rogosch FA. Personality, adrenal steroid hormones, and resilience in maltreated children: a multilevel perspective. Dev Psychopathol. 2007; 19(3):787–809. [PubMed: 17705903]
12. Fitzpatrick, K.; LaGory, M. Unhealthy places: Differential risk in the urban landscape. New York: Routledge; 2000.
13. Lupien SJ, King S, Meaney MJ, McEwen BS. Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. Biol Psychiatry. 2000 Nov 15; 48(10):976–80. [PubMed: 11082471]
14. Holt HB, Wild SH, Postle AD, et al. Cortisol clearance and associations with insulin sensitivity, body fat and fatty liver in middle-aged men. Diabetologia. 2007 May; 50(5):1024–32. [PubMed: 17370058]
15. Kyrou I, Chrousos GP, Tsigos C. Stress, visceral obesity, and metabolic complications. Ann N Y Acad Sci. 2006 Nov.1083:77–110. [PubMed: 17148735]
16. Stimson RH, Andersson J, Andrew R, et al. Cortisol release from adipose tissue by 11beta-hydroxysteroid dehydrogenase type 1 in humans. Diabetes. 2009 Jan; 58(1):46–53. [PubMed: 18852329]
17. Cohen S, Schwartz JE, Epel E, Kirschbaum C, Sidney S, Seeman T. Socioeconomic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Psychosom Med. 2006 Jan; 68(1):41–50. [PubMed: 16449410]
18. Wolf JM, Nicholls E, Chen E. Chronic stress, salivary cortisol, and alpha-amylase in children with asthma and healthy children. Biol Psychol. 2008 Apr; 78(1):20–8. [PubMed: 18243483]
19. Chong RY, Uhart M, McCaul ME, Johnson E, Wand GS. Whites have a more robust hypothalamic-pituitary-adrenal axis response to a psychological stressor than blacks. Psychoneuroendocrinology. 2008 Feb; 33(2):246–54. [PubMed: 18082975]
20. Hanrahan K, McCarthy AM, Kleiber C, Lutgendorf S, Tsalikian E. Strategies for salivary cortisol collection and analysis in research with children. Appl Nurs Res. 2006 May; 19(2):95–101. [PubMed: 16728293]
21. Kumsta R, Entringer S, Hellhammer DH, Wust S. Cortisol and ACTH responses to psychosocial stress are modulated by corticosteroid binding globulin levels. Psychoneuroendocrinology. 2007 Sep; 32(8–10):1153–7. [PubMed: 17904296]
22. Qi D, Rodrigues B. Glucocorticoids produce whole body insulin resistance with changes in cardiac metabolism. Am J Physiol Endocrinol Metab. 2007 Mar; 292(3):E654–E667. [PubMed: 17077342]
23. DeSantis AS, Adam EK, Doane LD, Mineka S, Zinbarg RE, Craske MG. Racial/ethnic differences in cortisol diurnal rhythms in a community sample of adolescents. Journal of Adolescent Health. 2007 Jul; 41(1):3–13. [PubMed: 17577528]
24. Tsuru J, Akiyoshi J, Tanaka Y, et al. Social support and enhanced suppression of adrenocorticotropic hormone and cortisol responses to hypothalamic-pituitary-adrenal function and thyrotropin-releasing hormone tests in patients with major depressive disorder. Biol Psychol. 2008 May; 78(2):159–63. [PubMed: 18359551]
25. Vaillancourt T, Duku E, deCatanzaro D, Macmillan H, Muir C, Schmidt LA. Variation in hypothalamic-pituitary-adrenal axis activity among bullied and non-bullied children. Aggress Behav. 2008 May; 34(3):294–305. [PubMed: 18161876]
26. van Stegeren AH, Wolf OT, Kindt M. Salivary alpha amylase and cortisol responses to different stress tasks: impact of sex. Int J Psychophysiol. 2008 Jul; 69(1):33–40. [PubMed: 18417235]
27. Chrousos GP. The hypothalamic-pituitary-adrenal axis and immune-mediated inflammation. N Engl J Med. 1995 May 18; 332(20):1351–62. [PubMed: 7715646]
28. Hucklebridge FH, Clow A, Abeyguneratne T, Huezo-Diaz P, Evans P. The awakening cortisol response and blood glucose levels. Life Sci. 1999; 64(11):931–7. [PubMed: 10201642]

29. Ball GD, Huang TT, Gower BA, et al. Longitudinal changes in insulin sensitivity, insulin secretion, and beta-cell function during puberty. J Pediatr. 2006 Jan; 148(1):16–22. [PubMed: 16423592]
30. Casazza K, Higgins PB, Fernandez JR, Goran MI, Gower BA. Longitudinal analysis of the insulin-like growth factor system in African-American and European American children and adolescents. J Clin Endocrinol Metab. 2008 Dec; 93(12):4917–23. [PubMed: 18782874]
31. Casazza K, Goran MI, Gower BA. Associations among insulin, estrogen, and fat mass gain over the pubertal transition in African-American and European-American girls. J Clin Endocrinol Metab. 2008 Jul; 93(7):2610–5. [PubMed: 18349063]
32. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child. 1969 Jun; 44(235):291–303. [PubMed: 5785179]
33. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child. 1970 Feb; 45(239):13–23. [PubMed: 5440182]
34. Grubesic TH, Matisziw TC. On the use of ZIP codes and ZIP code tabulation areas (ZCTAs) for the spatial analysis of epidemiological data. Int J Health Geogr. 2006; 5:58. [PubMed: 17166283]
35. Paul K, Boutain D, Manhart L, Hitti J. Racial disparity in bacterial vaginosis: the role of socioeconomic status, psychosocial stress, and neighborhood characteristics, and possible implications for preterm birth. Social Science & Medicine. 2008 Sep; 67(5):824–33. [PubMed: 18573578]
36. Ross CE, Mirowsky J. Disorder and decay - The concept and measurement of perceived neighborhood disorder. Urban Affairs Review. 1999 Jan; 34(3):412–32.
37. Stimpson JP, Nash AC, Ju H, Eschbach K. Neighborhood deprivation is associated with lower levels of serum carotenoids among adults participating in the third national health and nutrition examination survey. Journal of the American Dietetic Association. 2007 Nov; 107(11):1895–902. [PubMed: 17964308]
38. Messer LC, Vinikoor LC, Laraia BA, et al. Socioeconomic domains and associations with preterm birth. Social Science & Medicine. 2008 Oct; 67(8):1247–57. [PubMed: 18640759]
39. Caughy MO, Nettles SM, O'Campo PJ. The effect of residential neighborhood on child behavior problems in first grade. American Journal of Community Psychology. 2008 Sep; 42(1–2):39–50. [PubMed: 18607720]
40. Drewnowski A, Rehm CD, Solet D. Disparities in obesity rates: analysis by ZIP code area. Soc Sci Med. 2007 Dec; 65(12):2458–63. [PubMed: 17761378]
41. Field AE, Laird N, Steinberg E, Fallon E, Semega-Janneh M, Yanovski JA. Which metric of relative weight best captures body fatness in children? Obes Res. 2003 Nov; 11(11):1345–52. [PubMed: 14627755]
42. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child. 1969 Jun; 44(235):291–303. [PubMed: 5785179]
43. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child. 1970 Feb; 45(239):13–23. [PubMed: 5440182]
44. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child. 1969 Jun; 44(235):291–303. [PubMed: 5785179]
45. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child. 1970 Feb; 45(239):13–23. [PubMed: 5440182]
46. Littell, RC.; Milliken, GA.; Stroup, WW.; Wolfinger, RD. SAS System for Mixed Models. Cary, NC: SAS Institute; 1996.
47. Kudielka BM, Schmidt-Reinwald AK, Hellhammer DH, Kirschbaum C. Psychological and endocrine responses to psychosocial stress and dexamethasone/corticotropin-releasing hormone in healthy postmenopausal women and young controls: the impact of age and a two-week estradiol treatment. Neuroendocrinology. 1999 Dec; 70(6):422–30. [PubMed: 10657735]
48. Van den Bergh BR, Van CB, Pinna PS, Van HS. Self-reported symptoms of depressed mood, trait anxiety and aggressive behavior in post-pubertal adolescents: Associations with diurnal cortisol profiles. Horm Behav. 2008 Aug; 54(2):253–7. [PubMed: 18499109]
49. Andrew R, Phillips DI, Walker BR. Obesity and gender influence cortisol secretion and metabolism in man. J Clin Endocrinol Metab. 1998 May; 83(5):1806–9. [PubMed: 9589697]

50. Gozansky WS, Lynn JS, Laudenslager ML, Kohrt WM. Salivary cortisol determined by enzyme immunoassay is preferable to serum total cortisol for assessment of dynamic hypothalamic--pituitary--adrenal axis activity. Clin Endocrinol (Oxf). 2005 Sep; 63(3):336–41. [PubMed: 16117823]

51. Skogan, W. Disorder and Decline. Berkley, CA: University of California Press; 1992.

52. Ditzen B, Schmidt S, Strauss B, Nater UM, Ehlert U, Heinrichs M. Adult attachment and social support interact to reduce psychological but not cortisol responses to stress. J Psychosom Res. 2008 May; 64(5):479–86. [PubMed: 18440400]

53. Nierop A, Wirtz PH, Bratsikas A, Zimmermann R, Ehlert U. Stress-buffering effects of psychosocial resources on physiological and psychological stress response in pregnant women. Biol Psychol. 2008 Jul; 78(3):261–8. [PubMed: 18462859]

54. Zwolinski J. Biopsychosocial responses to social rejection in targets of relational aggression. Biological Psychology. 2008 Oct; 79(2):260–7. [PubMed: 18655823]

What is already known on this subject?

A source of chronic stress such as low neighborhood socioeconomic status has been associated with dysregulation of the HPA axis resulting in either blunted or elevated levels of cortisol among children.

What does this study add?

This study is the first to evaluate long term trends in serum cortisol levels among children and the extent to which neighborhood stressors affect serum cortisol among a biethnic sample of children. This study extends the neighborhood literature to include other stressors aside from socioeconomic status. In addition, this is the first study to adjust for important covariates that result from clinical observations such as pubertal status and total fat mass. These current findings demonstrate that characteristics associated with low neighborhood socioeconomic status (e.g. percent unemployment, poverty, single-female headed households with dependent children, and percentage vacant housing) are affect serum cortisol levels.

TABLE 1

Baseline Descriptive Statistics for Total Sample and Ethnicity. Mean (Standard Deviation)

	Total Sample (N = 148)	European American (n = 67)	African American (n = 81)
Cortisol (μg/dL)	11.42 (3.49)	11.47 (3.80)	11.35
Gender (% female)	50.54	43.59	55.56
Age	8.30 (1.70)	8.72 (1.61)[a]	7.92 (1.67)[b]
Pubertal Stage	1.11 (0.41)	1.07 (0.26)	1.15 (0.5)
Total fat (kg)	10.28 (7.04)	9.29 (5.59)	11.46 (8.27)
Neighborhood Composite Score[c]	0.046 (−1.94–5.68)	−0.81[a] (−1.94–1.53)	0.66[b] (−1.59–5.68)[d]
Percent unemployment	4.11 (2.89)	2.33 (0.87)	5.48 (3.21)
Percent poverty	14.25 (9.48)	7.18 (4.60)	18.47 (9.15)
Percent female-headed Household[d]	15.52 (7.83)	9.68 (4.40)	20.24 (6.74)
Percent vacant housing	8.65 (3.12)	6.99 (1.76)	10.00 (3.35)

[a, b] denote significant differences at p<.05

[c] Neighborhood Composite Score represents Z-score index, sample means and range. Higher scores indicate greater neighborhood disorder.

[d] Female headed households with dependent children under age 18.

Table 2

Mixed Model for Longitudinal Trends in Serum Cortisol Levels among a Biethnic Cohort of Children

	Model 1			Model 2		
	B	**SE**	**β**	**B**	**SE**	**β**
Age (Tanner 1)	−.014	.014	−1.00	−.025	.016	−1.56
Age (Tanner 2)	−.020*	.012	−1.67	−.028**	.014	−2.00
Age (Tanner 3)	−.014	.012	−1.16	−.023*	.013	−1.76
Age (Tanner 4)	−.011	.011	−1.00	−.016	.012	−1.33
Total fat Mass	−.048	.030	−1.60	−.034	.033	−1.03
Gender (Female)	−.014	.045	−.31	.004	.047	.085
Neighborhood Index				−.018*	.011	−1.64
Log Likelihood	396.3			350.5		
χ^2	34.36			31.33		
p-value	.001			.001		

Age (Tanner) refers to Tanner stage nested in age

B = unstandardized coefficient

SE = standard error

β= standardized coefficient

Neighborhood Index consisted of z-score tabulations of percent unemployment, poverty, single female headed households with dependent children, and percent vacant housing

Significance levels were set at

† p<.10,

* p<.05;

** p<.01;

*** p<.001

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Table 3

Mixed Model for Longitudinal Trends in Serum Cortisol Levels among African American Children

	Model 1***			Model 2***		
	B	**SE**	**β**	**B**	**SE**	**β**
Age (Tanner 1)	−.015	.022	−0.68	−.022	.022	−1.00
Age (Tanner 2)	−.019	.019	−1.00	−.024	.019	−2.10
Age (Tanner 3)	−.024	.017	−1.41	−.031*	.018	−1.72
Age (Tanner 4)	−.017	.016	−1.06	−.023	.016	−1.44
Total fat Mass	−.048	.045	−1.06	−.027	.046	−0.586
Gender (Female)	−.007	.068	−0.10	.001	.067	0.014
Neighborhood Index				−.022*	.012	−1.83
Log Likelihood	191.7			195.5		
χ^2	27.98			26.75		
p-value	.001			.001		

Age (Tanner) refers to Tanner stage nested in age

B = unstandardized coefficient

SE = standard error

β= standardized coefficient

Neighborhood Index consisted of z-score tabulations of percent unemployment, poverty, single female headed households with dependent children, and percent vacant housing

Significance levels were set at

†p<.10,

*p<.05;

**p<.01;

***p<.001

Full model was significant at p<.001

Table 4

Mixed Model for Longitudinal Trends in Serum Cortisol Levels among European American Children

	Model 1***			Model 2***		
	B	**SE**	**β**	**B**	**SE**	**β**
Age (Tanner 1)	−.046*	.022	−2.09	−.051*	.022	−1.56
Age (Tanner 2)	−.049**	.019	−2.57	−.053**	.019	−2.00
Age (Tanner 3)	−.037*	.018	−2.05	−.041*	.018	−1.76
Age (Tanner 4)	−.022	.017	−1.29	−.026	.017	−1.33
Total fat Mass	−.009	.047	−0.19	−.001	.047	−1.03
Gender (Female)	−.005	.073	−0.06	.015	.073	−.085
Neighborhood Index				.042†	.031	1.64
Log Likelihood	170.9			174.2		
χ^2	67.23			64.71		
p-value	.001			.001		

Age (Tanner) refers to Tanner stage nested in age

B = unstandardized coefficient

SE = standard error

β= standardized coefficient

Neighborhood Index consisted of z-score tabulations of percent unemployment, poverty, single female headed households with dependent children, and percent vacant housing

Significance levels were set at

† p<.10,

* p<.05;

** p<.01;

*** p<.001