Title: The Role of Sociospatial Adversity in Shaping Racial Disparities in Adolescents' Chronic Physiologic Stress

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Background

Black-white (B/W) disparities in adolescent health are pervasive in the U.S.[1] and they often persist across the life course.[2,3] Chronic physiologic stress as a consequence of black/African Americans (b/AA) increased exposure to environmental stressors (e.g. residential poverty and violence, discrimination) in comparison to whites is hypothesized to be one of the root causes underlying B/W disparities in health.[4-8] Prior research supports this line of inquiry as significant B/W disparities in the regulation of the hypothalamic pituitary adrenal (HPA) axis has been found, including blunting of the diurnal cortisol curve and elevated basal cortisol levels.[5, 9. 10] Cortisol is one of the primary hormones involved in the stress response.[11] Under "normal" conditions, cortisol release follows a circadian rhythm, such that levels are high upon waking with a marked increase 30 minutes after waking and then a gradual decline throughout the day with the lowest levels in the evening. Exposure to acute stress triggers a rise in cortisol - an adaptive mechanism needed to ensure glucose is available to the cells to respond to the stressor at hand.[11] However, chronic exposure to high levels or irregular release of cortisol is linked to immune suppression and increased infection risk (e.g. EBV reactivation);[12-16] central adiposity due to the high density of cortisol receptors on the fat cells at the midsection:[17] increased insulin resistance and type 2 diabetes:[18] and a myriad of other health issues - outcomes with known B/W disparities in their occurrence.

Although prior research has been particularly informative in identifying B/W disparities in HPA activity, research investigating explanations for these differences is limited. In addition, most studies examining HPA activity (including B/W differences) have focused primarily on measures of short-term or acute physiologic stress, specifically salivary cortisol measured via the diurnal curve captured over one to several days or in response to an acute stressor.[5,19-22] The emphasis on acute stress has led to an incomplete understanding of the extent to which adverse social environments contribute to *chronic stress*. In addition, prior research has tended to restrict the *scope* of the investigation to the residential neighborhood, which is typically measured as the census tract of residence. Although the reasoning for this restricted scope is partly rooted in the lack of appropriate existing large-scale data resources, incomplete measurement has limited our understanding of the extent to which sociospatial exposures have an effect on health outcomes.

The purpose of this study is to examine the extent to which B/W disparities exist in chronic physiologic stress (e.g. HPA activity measured using hair cortisol) among a probability sample of adolescents (N=500) and the role of adverse sociospatial exposures (e.g. poverty, violence, and disorder measured using an activity space approach) in explaining observed B/W differences in chronic stress. The study employs innovative measurement approaches – (1) hair for cortisol to better capture measures of chronic physiologic stress (e.g. 1 cm of hair approximates mean cortisol for corresponding month [23]) and (2) activity space measures using Smartphone technologies (e.g. global positioning systems and ecological momentary assessments) over a 1 week data collection period to better capture the breadth of sociospatial exposures.

Methods

Study Design

The study leverages data from two NIH-funded studies: (1) *The Adolescent Health and Development in Context (AHDC)* study (Browning, 1R01DA032371) – a prospective cohort study (and probability sample) that examines the impact of activity space exposures on the behavioral and health outcomes of 4,000 diverse youth aged 11-17 years in Franklin County,

Ohio and (2) *The Linking Biological and Social Pathways to Adolescent Health and Wellbeing* (Bio-Social Linkages) study (Ford, 1R21DA034960) – a probability subsample of 500 youth participating in wave 1 of the AHDC study in which chronic stress biomarkers of longitudinal HPA activity (hair for cortisol) are being collected for linkage to the AHDC study.

The AHDC study focuses on the multiple contexts of youth development over time in which data are collected at two time-points, approximately 1 year apart. Within each wave, the data are being collected over a weeklong period via face-to-face interviews/computer-assisted personal interviewing on days 1 and 7 of a focal youth and a primary caregiver and ecological momentary assessment (EMA) and global positioning system (GPS) methodologies using Smartphones for the youth over 7 days. The Bio-Social Linkages study employs a cross-sectional study in which biomeasures of chronic stress (e.g. hair for cortisol) and immune function (e.g. saliva for Epstein-Barr virus antibody and DNA) are being collected at wave 1 of the AHDC study. The



associated figure depicts the data collection design of the AHDC and Bio-Social Linkages study. Data collection is current underway and to date, 170 youth have completed wave 1 of the study.

Study Location and Sample

The study takes place in Franklin County, Ohio – a large, metropolitan area with an estimated 2012 population of 1.2 million.[24] Franklin

County contains the city of Columbus—Ohio's largest city and the 16th largest city in the U.S. (estimated 2012 population of 809,798). Franklin County is racially diverse (21.8% African American, 4% Asian, and 5% Latino). The 2008-2012 estimated median household income was \$50,700 with 17.1% living below the poverty line.[24] The Columbus metropolitan area is representative of the average U.S. metropolitan area in terms of social and economic characteristics[24].

The sampling design for the AHDC and Bio-Social Linkages studies was developed in collaboration with OSU's Center for Human Resources Research (CHRR) – an established survey research center with expertise in representative survey designs and data collection among children and adolescents. Replicate samples of 500 households stratified by racial, ethnic and socioeconomic composition of census tracts were drawn to ensure a representative sample of the city of Columbus. One youth from eligible households is then randomly selected to participate. To date, data have been collected on 170 youth of which 52% are male and the mean age is 14.6 years. With respect to racial/ethnic composition, 57% of the youth self-identify as white, 32% as black/AA, 1% as Asian, 2% as 'other', 2% as Hispanic and 6% as mixed race.

Data Collection

AHDC Study

Within each wave, the AHDC data are collected by trained interviewers over a weeklong period. A face-to-face interview and self-administered survey with both a focal youth and his or her caregiver is conducted on days 1 and 7. Caregiver interviews include questions on the youth's residential history, household composition, family structure and routine activity locations (e.g. school, organizations, activities, etc.), socioeconomic background, mental and physical health

and health behaviors of the caregiver and child, family conflict and legal troubles and child's exposure to adverse life events (e.g. violence), and activity space/neighborhood characteristics. The youth face-to-face interview includes questions related the youth's routine locations, social networks and risk behaviors at these locations (e.g. smoking at a "hang-out" location). In addition to the surveys, a seven day smartphone-based Global Positioning System (GPS) tracking and Ecological Momentary Assessment (EMA) data collection period follows the Entrance Survey for the youth collecting real-time data on locations; activities; network partner presence and interactions; mood; perceptions of safety and risk behavior.

The Bio-Social Linkages Study

Trained interviewers collect the hair sample from the youth during the exit interview at the end of the week. The interviewers cut approximately 25-75 mg of hair (0.4-1 cm in diameter) from the posterior vertex region of the scalp cutting as close to the scalp as possible with a new pair of thinning shears. The posterior vertex region of the scalp has the lowest variation in cortisol levels, thus it is the preferred area for sampling.[23] Consistent with prior research, adolescents with less than 1 cm of hair length on their scalp are generally excluded from this portion of the data collection due to insufficient quantity of hair to cut as are those who have taken oral or inhaled corticosteroids in the last month as these medications will significantly increase cortisol levels.[25] However, youth are still eligible for participation in the AHDC study, thus we will use the extensive data collected to examine the extent to which selection bias may occur and adjust for the bias in the statistical analyses using inverse probability of treatment weighting procedures when needed. To date, approximately, 90% (153) of the 170 youth provided a hair sample. Of the 10% who are missing, 3.5% refused to participate, 4% were ineligible due to insufficient hair and 2.5% were ineligible due to corticosteroid use.

Measures

Chronic physiologic stress is measured via cortisol (HPA activity) captured in adolescent hair. As previously stated, each 1 cm of hair approximates the mean cortisol level over the prior 1 month in growth.[23] Studies have found cortisol is leached out of hair at more distal lengths (> 3cm) due to repeated hair washing and environmental exposures,[26-28] thus consistent with prior research [26-32] we focus on the first 3 cm of hair growth. To date, most studies have only included participants who had at least 3 cm of hair. [29,30,32] However, in our diverse sample this would exclude individuals with shorter hair and increase the potential for selection bias. Thus, we include individuals with varying hair lengths (typically 1 cm - 3 cm) and adjust for hair length in our statistical analyses. Prior research found no significant differences in cortisol levels between individuals with varying hair lengths between 1 cm-to-3 cm,[31,33] thus, our approach reduces selection bias and provides a cost-effective and valid measure of chronic stress.

Hair is assayed for the measurement of a mean cortisol value at OSU's College of Nursing Lab under the direction of Ford using an adapted protocol by D'Anna-Hernandez et al.[34] and Meyer et al.[26] To prep for assay, the hair sample is washed with isopropanol and dried over 1 to 3 days. A total of 25-75 mg of hair is placed into a microcentrifuge tube, cut into 2-4 mm lengths, and then ground for at least 15 min in Retsch 400 Mill. A total of 1.1 ml of HPLC-grade methanol is added to the ground sample, and incubated for 18-24 hours at room temperature with constant agitation via a shaking table. The tubes are centrifuged at 1000g for 15 min at room temperature to pellet the powdered hair. The entire amount (~1 ml) of supernatant is transferred to a clean microcentrifuge tube and the methanol is removed by evaporation using a stream of air for 6-8 hours at room temperature. The cortisol extract is immediately reconstituted in 100ul of Salimetric immunoassay cortisol analysis diluent buffer. Samples are assayed in duplicate. Inter- and intra-assay coefficients of variation are calculated. Cortisol levels are continuous in nature and expressed in hair as pg/mg (typically log transformed for analysis).

Activity Space Construction and Sociospatial Adversity

The AHDC study measures youths' activity space using data from youth and parental report of routine activity locations over the month prior to the survey and the continuous GPS data collected over a 7-day tracking period. Activity space characteristics are estimated by specifying a buffer around the youth's space-time travel path and employing Kernel density methods to determine aggregate characteristics of the resulting activity space. Specifically, the Kernel density surface (KDS) derived from the location of a set of points using a kernel function and a predetermined search radius (or bandwidth). To generate a KDS from a point distribution of n activity locations, a nonparametric kernel estimation method will be employed. [[35,36] Following Bailey and Gatrell's formulation, [37] if R represents the study area, x represents a general location in \Re and x_1, x_2, \dots, x_n are the locations of the n activities, then the intensity or density, $\lambda(x)$, at **x** is estimated by:

$$\lambda_h(x) = \frac{1}{\delta_h(x)} \sum_{i=1}^n \frac{w_i}{h^2} k\left(\frac{(x-x_i)}{h}\right), \qquad x \in \Re$$

edge correction factor.¹⁴¹

In the AHDC study, the quartic kernel function: described in Silverman [36] is used for generating the KDS for each subject using an appropriate bandwidth.

where k(.) is the kernel function, the parameter h > 0 is the bandwidth determining the amount of smoothing, wi is a weighing factor, and $\delta_h(\mathbf{x})$ is an

$$k(x) = \begin{cases} 3\pi^{-1}(1 - x^T x)^2 & \text{if } x^T x \le 1\\ 0 & \text{otherwise} \end{cases}$$

The AHDC study will then create weighted scores for subject's activity spaces (as defined by the KDS) based on both objective (e.g. census-based)



characteristics of areas as well as reports of key social processes (e.g. collective efficacy) using caregiver and youth survey data (measures of activity space characteristics based on survey data will be aggregated from densely sampled respondents across the study area; the youth's own caregiver report may be excluded from the construction of a given activity space measure in order to avoid shared method variance). The example figure shows a density surface of commercial activity opportunities based on 10,727 commercial parcels in Columbus, Ohio. An analogous approach could be used to describe the density surface of actual activity locations for the entire sample of AHDC youth.

> The central measures of sociospatial adversity of the individual activity space for this study include:

concentrated poverty, physical and social disorder, and violence measures including, perceived safety, prevalence of violence and exposure to community violence. Respondent reports and administrative data will be aggregated across the study area and kernel density estimation will be employed to construct measures of sociospatial adversity of respondent-specific activity spaces. For self-reported measures, the effects of aggregated activity space characteristics will be estimated both with and without including youth and caregiver reports in order to ensure that observed associations are not due to shared method variance.

Concentrated poverty (2010 U.S. Census) will be measured as a composite consistent with prior research [38-40] via the following items: % of households below the poverty line, % of households on public assistance, % of female-headed households with children and unemployment rate.

Physical disorder (youth and caregiver surveys) will be measured as a composite of 3 items based on the Project for Human Development in Chicago Neighborhoods (PHDCN) [41] that

ask "how much of a problem is"..."litter, broken glass or trash on sidewalks and streets," "graffiti on buildings and walls," and "vacant or deserted houses or storefronts" in the neighborhood).

Social disorder (youth and caregiver surveys) will be measured as a composite of 3 items based on the PHDCN [41] that ask "how much of a problem is"..."drinking in public," "people selling or using drugs," and "groups of teenagers or adults hanging out and causing trouble" in the neighborhood).

Perceived safety (youth and caregiver surveys) will be measured as a composite of 5 items based on the PHDCN [41] that ask about subjective perceptions of neighborhood danger, including fear of being robbed, of the house being broken into, being afraid to walk in the neighborhood at night, to let the youth go outside).

Prevalence of violence (administrative data) will be measured via crime rates in the study area to determine rates of violence in specific locations, including publicly available information on the location and timing of a range of crimes as reported by the Columbus Division of Police and other administrative sources.

Exposure to community violence is measured by a checklist of items (youth survey) adapted from the PHDCN [41] on whether violence and other risk behaviors occurred in the last year within a 5 minute drive from their home; items include threats, chasing, hitting, attacking with a weapon, fights, beatings, and shooting or shooting at someone. For each behavior reported, follow-up questions assess frequency of victimization and witnessing. The battery of items is then repeated for *each routine location* reported by the caregiver and youth, including school locations. For each behavior reported, follow-up questions assess frequency of victimization assess frequency of victimization and witnessing each act. These unique data will allow for estimates of the extent to which the specific contexts of ETV (and the characteristics of those contexts) are linked with the chronic stress biomarkers.

Adolescent Race

Race is measured as mutually exclusive racial categories (e.g. non-Hispanic black/AA or non-Hispanic white) self-identified by the youth.

Control Measures

Control measures of adolescent and caregiver sociodemographics will be included in the analysis, including but not limited to sex, family structure, household socioeconomic status, adolescent health status, household conflict (e.g. exposure to domestic violence) and season of data collection. In addition, adolescent hair care practices (e.g. frequency of washing, chemical treatments, hair product use) and the weight and length of the hair will be included in the analyses as control measures as prior research has found associations between these measures and hair cortisol values.[42]

Analytic Strategy

As discussed prior, the data collection is currently underway and anticipated to be complete in winter 2015. Of the 153 hair samples currently collected, 73 have been assayed for cortisol and of these, 66 of the youth self-identified as non-Hispanic b/AA or non-Hispanic white. Preliminary analyses were conducted to examine B/W differences in hair cortisol levels in which multiple linear regression analyses were conducted adjusting for age, sex, hair length and hair weight (N=66).

The following describes the analytic strategy for the study aims to be conducted upon completion of the data collection. Specifically, we will examine the (1) extent to which B/W

disparities exist in chronic physiologic stress (e.g. HPA activity) and (2) the role of adverse sociospatial exposures (e.g. poverty, violence, and disorder) in explaining observed B/W differences in stress outcomes. We hypothesize that b/AA youth will have higher hair cortisol levels than white adolescents and that sociospatial adversity in routine activity spaces will account for the observed racial differences in chronic stress.

As noted, sociospatial adversity measures will be based on kernel density estimates of poverty levels, social and physical disorder, violence (both average levels of violence as captured by administrative data and direct experiences of violence as victim or witness), and perceptions of safety within a buffer bounding the locations of each subject's routine activities. This approach will substantially improve upon conventional strategies employing characteristics of the youth's residential census tract or block group alone. Characteristics of activity spaces will be included in OLS regression models predicting hair cortisol levels, incorporating a range of controls available in the AHDC data. The model will be specified as follows:

$$Y_{i} = \beta_{0} + \sum_{p=1}^{P} \beta_{p}(Adversity_{pi}) + \sum_{q=P+1}^{P+Q} \beta_{q}X_{qi} + e_{i}$$

Where the outcome, Y_i , represents logged hair cortisol levels measured in pg/mg for youth *i*, β_0 is the intercept, β_p are coefficients capturing the effects of *P* sociospatial adversity covariates on

hair cortisol levels and β_q are coefficients for the effects of Q control variables X on hair cortisol levels. Among control variables, we will include adolescent health measures (e.g. chronic condition, current illness symptoms, medications), substance use and exposure to smoking; hair care practices and hair length/weight); season of collection; family structure; and exposure to adversity in the home (as a key activity space context), including poverty, family/household violence, etc. Because sociospatial adversity is measured at the individual activity space level, case clustering within conventionally defined neighborhoods is less problematic, although residual dependencies will be assessed to ensure accurate estimation of standard errors.

Results

Characteristics of the preliminary sample of black/AA and white youth aged 11 to 17 years, means for the total sample and stratified by race

	Total sample	Black/African American youth	White youth
Cortisol level logged	4.48	4.79	4.35
Race			
Non-Hispanic black/AA	0.29		
Non-Hispanic white	0.71		
Male sex	0.50	0.37	0.55
Age	14.2	13.9	14.4
Hair length (cm)	2.8	2.6	2.85
Hair weight (mg)	28.1	31.1	26.9

Multiple linear regression of the racial differences in hair cortisol levels among the preliminary sample of black/AA and white youth aged 11 to 17 years (N=66)*

	b	SE
Race		
Non-Hispanic black/AA	0.50	0.21*
Non-Hispanic white (reference)		
Male sex	-0.06	0.20
Age	-0.05	0.04
Hair length (cm)	-0.002	0.18
Hair weight (mg)	-0.02	0.003***

*hair cortisol levels are in pg/mg and logged

Brief Discussion and Next Steps

The preliminary findings of this study suggest that black/African American youth are more likely to experience chronic physiologic stress as indicated by their higher hair cortisol levels in comparison to their white peers. The next steps for this paper prior to presentation are to complete the data collection (anticipated 2/15), complete the cortisol assays (anticipated 3/15), construct measures including youth's individual activity spaces (anticipated 3/15) and complete the analytic strategy as outlined above.

This study is among the first to examine racial differences in *chronic* physiologic stress and the extent to which sociospatial adversity accounts for these disparities. We employ novel measurement approaches - hair for cortisol to better capture chronic physiologic stress and youths' individual activity space to better capture the breadth of their routine sociospatial exposures. The findings will elucidate the extent to which racial disparities in chronic physiologic stress exist among youth and identify adverse sociospatial exposures that contribute to the disparate outcomes.

References

[1] Kitsantas P, Kornides ML, Cantiello J, Wu H. Chronic physical health conditions among children of different racial/ethnic backgrounds. Public Health. 2013 Jun;127(6):546-53.

[2] Harris KM, Gordon-Larsen P, Chantala K, Udry JR. Longitudinal trends in race/ethnic disparities in leading health indicators from adolescence to young adulthood. Arch Pediatr Adolesc Med. 2006 Jan;160(1):74-81.

[3] Williams DR. Miles to go before we sleep: racial inequities in health. J Health Soc Behav. 2012 Sep;53(3):279-95.

[4] Thoits PA. Stress and health: major findings and policy implications. J Health Soc Behav. 2010;51 Suppl:S41-53.

[5] 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. J Adolesc Health 2007;41:3e13.

[6] Ford JL, Stowe RP. Racial-ethnic differences in Epstein-Barr virus antibody titers among U.S. children and adolescents. Ann Epidemiol. 2013 May;23(5):275-80.

[7] Mujahid MS, Diez Roux AV, Cooper RC, Shea S, Williams DR. Neighborhood stressors and race/ethnic differences in hypertension prevalence (the Multi-Ethnic Study of Atherosclerosis). Am J Hypertens 2011;24:187e93.

[8] Brondolo E, Brady ver Halen N, Libby D, Pencille M. Racism as a psychosocial stressor. In: Contrada RJ, Baum A, editors. The handbook of stress science: biology, psychology, and health. New York, NY: Springer; 2011. p. 167e84.

[9] Hajat A, Diez-Roux A, Franklin TG, Seeman T, Shrager S, Ranjit N, Castro C, Watson K, Sanchez B, Kirschbaum C. Socioeconomic and race/ethnic differences in daily salivary cortisol profiles: the multi-ethnic study of atherosclerosis. Psychoneuroendocrinology. 2010 Jul;35(6):932-43

[10] Skinner ML, Shirtcliff EA, Haggerty KP, Coe CL, Catalano RF. Allostasis model facilitates understanding race differences in the diurnal cortisol rhythm. Developmental Psychopathology. 2011;23:1167-86.

[11] McCarthy, D.O. (in press). Biological measures for intervention research. In: *Designing, Conducting, and Analyzing Intervention Research: A Practical Guide for Success.* New York: Springer Publishing Company.

[12] Glaser R, Pearson GR, Jones JF, Hillhouse J, Kennedy S, Mao HY, Kiecolt-Glaser JK. Stress-related activation of Epstein-Barr virus. Brain, Behavior, and Immunity. 1991;5:219-32.

[13] Padgett DA, Glaser R. How stress influences the immune response. Trends in Immunology. 2003; 24: 444–448.

[14] Glaser R, Kiecolt-Glaser JK. Stress-induced immune dysfunction: implications for health. Nature Reviews Immunology. 2005; 5: 243-251.

[15] Dhabhar FS. Effects of stress on immune function: Implications for immunoprotection and immunopathology. In: The Handbook of Stress Science: Biology, Psychology, and Health (Eds. Contrada RJ, Baum A). 2011. New York: Springer. 47-63.

[16] Pedersen AF, Bovbjerg DH, Zachariae R. Stress and susceptibility to infectious disease. In: The Handbook of Stress Science: Biology, Psychology, and Health (Eds. Contrada RJ, Baum A). 2011. New York: Springer. 425-446.

[17] Brooks L, McCabe P, Schneiderman N. Stress and cardiometabolic syndrome. In: The Handbook of Stress Science: Biology, Psychology, and Health (Eds. Contrada RJ, Baum A). 2011. New York: Springer. 399-409.

[18] Andrews RC, Walker BR. Glucocorticoids and insulin resistance: old hormones, new targets.Clinical Science.1999; 96:513-23. Hackman D.A., Farah M.J., Betancourt L.M., Brodsky N.L.,Hurt H.,. Neighborhood disadvantage and adolescent stress reactivity. Frontiers in Human Neuroscience. 2012(SEPTEMBER).

[19] Brenner, Allison B., Zimmerman, Marc A., Bauermeister, Jose A., Caldwell, Cleopatra H.,. The physiological expression of living in disadvantaged neighborhoods for youth. Journal of Youth and Adolescence. 2013;42(6):792-806.

[20] Chen, E, Paterson, LQ. Neighborhood, family, and subjective socioeconomic status: How do they relate to adolescent health? Health Psychology. 2006;25(6):704-714.

[21] Suglia SF, Staudenmayer J, Cohen S,Wright RJ,. Posttraumatic stress symptoms related to community violence and children's diurnal cortisol response in an urban community-dwelling sample. Int J Behav Med. 2010;17(1):43-50.

[22] Rudolph, KE., Gary SW., Stuart, EA., Glass, TA, Marques, AH., Duncko, R. Merikangas,KR. The association between cortisol and neighborhood disadvantage in a U.S. population-based sample of adolescents. Health & Place. 2014;25:68-77.

[23] Sauvé B, Koren G, Walsh G, Tokmakejian S, Van Uum SH,. Measurement of cortisol in human hair as a biomarker of systemic exposure. *Clinical and investigative medicine*. 2007;30(5):183-91.

[24] U.S. Census Bureau. Franklin County, OH QuickFacts. http://quickfacts.census.gov/qfd/states/39/39049.html2014.

[25] Granger DA, Hibel LC, Fortunato CK, Kapelewski CH. Medication effects on salivary cortisol: tactics and strategy to minimize impact in behavioral and developmental science. *Psychoneuroendocrinology*. 2009;34:1437-48.

[26] Meyer JS, Novak MA. Minireview: Hair cortisol: A novel biomarker of hypothalamic-pituitaryadrenocortical activity. Endocrinology. 2012;153(9):4120-4127.

[27] Gow R, Thomson S, Rieder M, Van Uum S, Koren G. An assessment of cortisol analysis in hair and its clinical applications. Forensic Sci Int. 2010;196(1-3):32-37.

[28] Stalder T, Kirschbaum C. Analysis of cortisol in hair--state of the art and future directions. Brain Behav Immun. 2012;26(7):1019-1029.

[29] Luo H, Hu X, Liu X, et al. Hair cortisol level as a biomarker for altered hypothalamicpituitary-adrenal activity in female adolescents with posttraumatic stress disorder after the 2008 Wenchuan earthquake. Biol Psychiatry. 2012;72(1):65-69.

[30] Dettenborn, L., Tietze, A., Bruckner, F.,Kirschbaum, C.,. Higher cortisol content in hair among long-term unemployed individuals compared to controls. Psychoneuroendocrinology. 2010;35(9):1404-1409.

[31] Steudte S, Kolassa IT, Stalder T, Pfeiffer A, Kirschbaum C, Elbert T, Increased cortisol concentrations in hair of severely traumatized Ugandan individuals with PTSD. Psychoneuroendocrinology. 2011;36(8):1193-200.

[32] O'Brien KM, Tronick EZ, Moore CL. Relationship between hair cortisol and perceived chronic stress in a diverse sample. Stress Health. 2012.

[33] Boch S, Colbert A, Ford JL. Hair cortisol measurement of chronic stress in women postincarceration. [Midwest Nursing Research Society Annual Meeting, St. Louis, MO, March 2014].

[34] D'Anna-Hernandez KL, Ross RG, Natvig CL, Laudenslager ML. Hair cortisol levels as a retrospective marker of hypothalamic-pituitary axis activity throughout pregnancy: Comparison to salivary cortisol. *Physiology & Behavior*. 2011; 104: 348-353.

[35] Gatrell A. Density estimation and the visualization of point patterns. In: Hernshaw H, Unwin DJ, eds. Visualization in geographical information systems. New York: John Wiley & Sons; 1994:650-75.

[36] Silverman B. Density estimation for statistics and data analysis. London: Chapman & Hall; 1986.

[37] Bailey T, Gatrell A. Interactive spatial data analysis. New York: Longman; 1995.

[38] Ford JL, Browning CR. Neighborhood social disorganization and the acquisition of trichomoniasis among young adults in the United States. Am J Public Health. 2011;101:1696-1703.

[39] Browning CR, Leventhal T, Brooks-Gunn J. Neighborhood context and racial differences in early adolescent sexual activity. Demography. 2004;41(4):697-720.

[40] Browning CR, Burrington LA, Leventhal T, Brooks-Gunn J. Neighborhood structural inequality, collective efficacy, and sexual risk behavior among urban youth. J Health Soc Behav. 2008;49(3):269-285.

[41] Project for Human Development in Chicago Neighborhoods (PHDCN). Instruments and measures. <u>http://www.icpsr.umich.edu/icpsrweb/PHDCN/instruments.jsp2014</u>.

[42] Wosu AC, Valdimarsdottir U, Shields AE, Williams DR, Williams MA. Correlates of cortisol in human hair: Implications for epidemiologic studies on health effects of chronic stress. Ann Epidemiol. 2013;23(12):797-811.e2.