

# Impact of Neighborhood Quality on Mental and Physical Health in African Americans: Evidence for the “Skin-Deep” Hypothesis

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## **Extended Abstract**

### *Background*

Institutions in the United States have systematically excluded minorities and the poor from enfranchisement for much of their history. Unfair housing policies, employment discrimination, and school segregation are not injustices of the past, but currently operate to oppress and keep large portions of the U.S. population isolated from the so-called “American dream.” The few who are able to advance socially and economically — through college completion or secure, gainful employment — do so under adverse circumstances utilizing outstanding coping skills and resilience. However, what toll does upward mobility take on one’s mental and physical health?

The theory of “skin-deep” resilience suggests that the upwardly mobile from severely disadvantaged backgrounds exhibit psychological resilience (indicated by the absence of depressive symptoms or externalizing behaviors), but carry biological markers of persistent physiological stress (Brody et al. 2013). While the upwardly mobile display resilience, below the surface is the biological accumulation of years of chronic stress. Researchers hypothesize the physiological stress is due to the psychological toll of navigating institutions in everyday life that produce and reproduce structural racism and classism, discouraging those who are non-white and/or poor (Link, Phelan and Hatzenbuehler 2018, Phelan and Link 2015). People of color and/or those from financially disadvantaged backgrounds may experience overt or covert

discrimination and insensitivity as they negotiate their way through higher education or the workplace (Hudson et al. 2013, Hudson et al. 2016).

While upward mobility offers relief from the anxieties associated with poverty, and may lead to an improved health trajectory, the process of social mobility may take a toll on one's health, and may not be the panacea for health disparities that policymakers expound. A recent article from Gaydos et al. (2018) finds that those from very disadvantaged contexts in childhood who received a college degree have similar levels of depression as their less disadvantaged counterparts; however, this group is at a much higher risk of metabolic disease compared to their similarly disadvantaged counterparts who did not receive a college degree. The findings were more extreme for the non-Hispanic black and Hispanic sample compared to the non-Hispanic white sample. Other scholars have found similar evidence for skin-deep resilience. Those from disadvantaged backgrounds who reported higher levels of conscientiousness (a proxy measure of having self-control and being hard working) were psychosocially very functional, as measured by educational attainment, depression, and close relationship quality; however, this group had poorer health outcomes regarding their immune response (Miller et al. 2016). In a sample of college students, those from very disadvantaged backgrounds had lower rates of drug use but higher levels of allostatic load compared to their less disadvantaged counterparts (Chen et al. 2015). Ultimately, these continuous stress responses are costly, increasing the risk of chronic, long-term illness.

Most research has focused on single accomplishments associated with socioeconomic status to indicate upward mobility, such as college attendance, as opposed to direct measurements of the social environment. Neighborhoods are contexts in which people can be exposed to the stresses associated with poverty and public underinvestment, such as violent

crime or the absence of functional public schools. Spatially concentrated poverty and segregation is associated with the negative attributes of poverty, such as delinquency, unemployment, substance abuse, and marital instability (Massey and Wagner 2018). Living in a stressful environment and experiencing persistent stress can lead to increased levels of c-reactive protein (CRP), a serum protein humans produce in response to stress (Steptoe, Hamer and Chida 2007). Researchers have documented the relationship between CRP as an early indicator of the development of cardiovascular disease (Buckley et al. 2009, Taylor et al. 2006), diabetes (Cox et al. 2012), and some cancers (Allin and Nordestgaard 2011). When using more direct measures of the social context to track social mobility — in this case, neighborhood characteristics — does the skin-deep phenomena still hold? Those who move from more to less disadvantaged neighborhoods may experience health benefits; however, it may be that the stressors of living in a context different from the context one grew up in may also cause undue stress. Additionally, those who are downwardly mobile could experience even greater stress due to mismatched expectations versus reality, in addition to being exposed to the stresses of living in a highly disadvantaged neighborhood.

### *Data*

In order to model the relationship between social mobility and physical health, I used Waves I and IV of the National Longitudinal Study of Adolescent to Adult Health, a prospective, school-based study of a nationally representative sample of U.S. adolescents in grades 7-12 in 1994 and 1995 (Harris et al. 2009). Follow-up surveys were conducted in 1996, 2001-2002, and 2008. At Wave I, 20,745 students participated in the in-home interviews (79% response rate); at Wave IV, 15,701 young adults participated in follow-up in-home interviews (80.3% response

rate) (Harris et al. 2009). For this analysis, I include those who participated in the in-home interview at Waves I and IV, when the participants were in grades 7-12 and aged 24-32, respectively. I additionally restricted the sample to those with valid sample weights, those who had complete data for all predictors and demographic covariates used in the analysis, and those who had CRP levels at 10 mg/L or less. The sample size used in these analyses is  $n = 12,283$ .

### *Measures*

#### Race/Ethnicity

Race and ethnicity were coded using responses from Wave I. Respondents answered separate questions regarding race and ethnicity. All respondents who indicated that they were of Hispanic origin were coded as being Hispanic. A separate question regarding race asked participants to mark all races that they identify with among the following options: white, African American, American Indian, Asian, and other. The focus of this paper is on those who identify as non-Hispanic white, non-Hispanic black, and Hispanic. Those who indicated they were not of Hispanic origin and only marked white or black as their race were coded as non-Hispanic white and non-Hispanic black, respectively.

#### Neighborhood disadvantage index and neighborhood social mobility

I created neighborhood disadvantage indices for Waves I and IV using tract- and county-level Census data. For both Waves I and IV, I created an indicator variable for the most disadvantaged quartile for the following neighborhood level variables: proportion of households receiving public assistance; proportion of unemployed adults; proportion of households that fell below the federal poverty level; proportion of adults over age 25 with less than a high school

degree or equivalent; proportion of vacant homes; proportion of female-headed households with children; and the county-level violent crime arrest rate. I then added together these seven indicators to create an index between 0-7, 0 indicating no neighborhood disadvantage, and 7 indicating high neighborhood disadvantage. I then dichotomized the neighborhood disadvantage index to indicate who had a disadvantage index in the bottom 50th percentile, meaning they lived in a comparatively less disadvantaged neighborhood, and those above the 50th percentile, who lived in a more disadvantaged neighborhood. Thus, respondents were categorized as having high or low neighborhood disadvantage at Waves I and IV.

In order to categorize who was socially mobile, I used change in neighborhood disadvantage as a measure of social mobility. Those who were in highly disadvantaged contexts at Wave I, but were in low disadvantage contexts in Wave IV were considered upwardly mobile. Those who were from low disadvantage contexts in Wave I, but were in highly disadvantaged contexts in Wave IV were considered downwardly mobile. Those who stayed either in high or low disadvantaged contexts in Wave I and IV were coded as not experiencing mobility.

### Depressive symptoms

At Wave IV, participants were asked how often in the last seven days they “were bothered by things that don’t usually bother you”, “felt that you could not shake off the blues”, “felt you were just as good as other people”, “had trouble keeping your mind on what you were doing”, “felt depressed”, “felt too tired to do things”, “were happy”, “enjoyed life”, “felt sad”, and “felt that people disliked you”. Answer options were “never or rarely” (coded 0), “sometimes” (1), a lot of the time (2), and “most of the time or all of the time” (3). The 10 items were added up to create a depression scale from 0 to 30, a scale modeled after the CES-D

(Center for Epidemiologic Studies — Depression), a standard instrument for measuring depressive symptoms (Radloff 1977).

### C-reactive protein

Blood samples were collected at Wave IV of Add Health, which were assayed for high-sensitivity CRP. For this analysis, participants whose CRP levels were above 10 mg/L, suggesting the participant had an acute infection or injury and not chronic inflammation (Pearson et al. 2003), were treated as missing. Generally, CRP levels less than 3 mg/L indicate low to average levels, while CRP levels between 3 and 10 suggest low-grade inflammation, and high risk for illness. I utilized a continuous measure of CRP, and transformed CRP levels using the natural logarithm for the analysis.

### Covariates

I included several covariates in the Poisson regression of depressive symptoms and linear regression of CRP. For depressive symptoms, I included sex and age at Wave IV. There are several health behaviors and physiological characteristics that may inflate or dampen CRP levels, and be unrelated to chronic stress (O'Connor et al. 2009). The covariates I included in the CRP regression, recommended by O'Connor et al. (2009), are the number of subclinical symptoms the respondent had recently experienced (e.g., fever, vomiting, cough), the number of infectious or inflammatory diseases the participant has, whether the participant is taking anti-inflammatory drugs, whether the participant was taking hormonal birth control, whether the participant was a daily smoker, waist circumference, BMI, age, and sex.

## *Results*

I utilized the *svy* command with the Wave IV probability weight in Stata 15, as recommended by Chen and Chantala (2014), in order to account for Add Health's complex sampling design. The first regression is a Poisson regression of the count of depressive symptoms on Wave I neighborhood disadvantage, Wave IV neighborhood disadvantage, an interaction term between Wave I and IV neighborhood disadvantage, and the previously listed covariates. The Poisson regression is stratified by race, the results of which are presented in Table 1. The interaction terms are of most interest in this paper, which, across the three races, are not statistically significant. It appears that in this sample neighborhood disadvantage at Waves I and IV does not predict the count of depressive symptoms, except for non-Hispanic blacks. For this group, those who were downwardly mobile (i.e., living in a less disadvantaged neighborhood at Wave I and a disadvantaged context at Wave IV) had a greater likelihood of having more depressive symptoms ( $IRR = 1.24$ ) than those who were living in non-disadvantaged neighborhoods at Wave IV.

In the second regression, I regressed natural log transformed CRP levels on neighborhood disadvantage at Waves I and IV, and their interaction, as well the previously listed covariates. Again, this linear regression is stratified by race. The results in Table 2 indicate that, for non-Hispanic blacks in this sample, the interaction between Wave I and Wave IV neighborhood disadvantage levels are statistically and substantively significant. Those who lived in low disadvantaged neighborhoods at Wave I and IV had an attenuated CRP level, a decrease of 0.32 in the natural log of CRP, compared to those who lived in highly disadvantaged neighborhoods in Waves I and IV. In order to more clearly interpret the interaction effects of Wave I and Wave IV neighborhood disadvantage on CRP levels, Figures 1-3 present the plotted marginal effects of

the interaction term. In Figure 2, non-Hispanic blacks who experienced no mobility and low disadvantage have the lowest levels of CRP. Those who did not experience mobility and lived in highly disadvantaged neighborhoods have comparable levels of CRP as those who were upwardly mobile, which are higher than the no mobility, low disadvantage group. Lastly, those who were downwardly mobile had the highest levels of CRP. For non-Hispanic whites and Hispanics in this sample, there were no significant effects of neighborhood disadvantage on CRP levels.

### *Discussion*

Using Add Health data and neighborhood disadvantage as a more direct measure of social mobility, I find evidence supporting the skin-deep hypothesis. The Poisson regression of depressive symptoms on the interaction term between childhood and adulthood neighborhood disadvantage indicates that depressive symptoms are unrelated to upward mobility. However, in the case of non-Hispanic blacks, there is an increased probability of having more depressive symptoms for those who are downwardly mobile. The directionality of this finding is not clear: it may be that the depressive symptoms contribute to moving down the social ladder; on the other hand, downward mobility and the mismatch between expected social status versus reality may contribute to depressive symptoms.

In the regression predicting CRP levels using the interaction between neighborhood disadvantage at Waves I and IV, the coefficient was only statistically significant for non-Hispanic blacks. The results suggest that those who continuously live in low disadvantaged neighborhoods have little stress, and thus low levels of CRP. Considering that policy pushes to improve the health and well-being of the disadvantaged are often centered around improving



educational outcomes and earnings potential, we might expect that those who are upwardly mobile, at the very least, experience less stress than those who have only lived in highly disadvantaged neighborhoods their entire lives. However, this is not the case in this analysis. In keeping with the skin-deep hypothesis, those who are upwardly mobile have approximately the same levels of CRP as those who continuously lived in highly disadvantaged neighborhoods. It may be that this relationship is due to a persistent feeling that one does not belong in a particular neighborhood, or that it is significantly foreign compared to one's childhood neighborhood. Lastly, the regression suggests that those who are downwardly mobile have the highest level of CRP. This finding, which adds to the social mobility and skin-deep hypothesis literature, suggests that the downwardly mobile experience more intense stress, though whether the relationship is causal is unclear. Whether someone is feeling greater stress as a result of downward mobility, or that overwhelming stress is contributing to downward mobility, is inconclusive. Future research could attempt to explain the mechanism for these stress pathways, and to elucidate why there are differential CRP levels by mobility for non-Hispanic blacks, but not for non-Hispanic whites and Hispanics.

Table 1. Poisson regression of count of depressive symptoms on neighborhood disadvantage at Wave I and Wave IV

	Non-Hispanic white			Non-Hispanic black			Hispanic		
	IRR	SE	p-value	IRR	SE	p-value	IRR	SE	p-value
High disadvantage at Wave I	1.03	0.03	0.369	1.01	0.09	0.896	0.95	0.09	0.556
High disadvantage at Wave IV	1.04	0.03	0.185	1.24	0.12	0.027*	0.98	0.11	0.867
High disadvantage at Wave I x high disadvantage at Wave IV	1.07	0.06	0.251	0.92	0.10	0.456	1.20	0.16	0.181
Sex (female)	1.20	0.03	0.000*	1.16	0.06	0.003*	1.24	0.05	0.000*
Age	1.00	0.01	0.597	1.01	0.01	0.676	1.00	0.02	0.776

\*p-value < 0.05

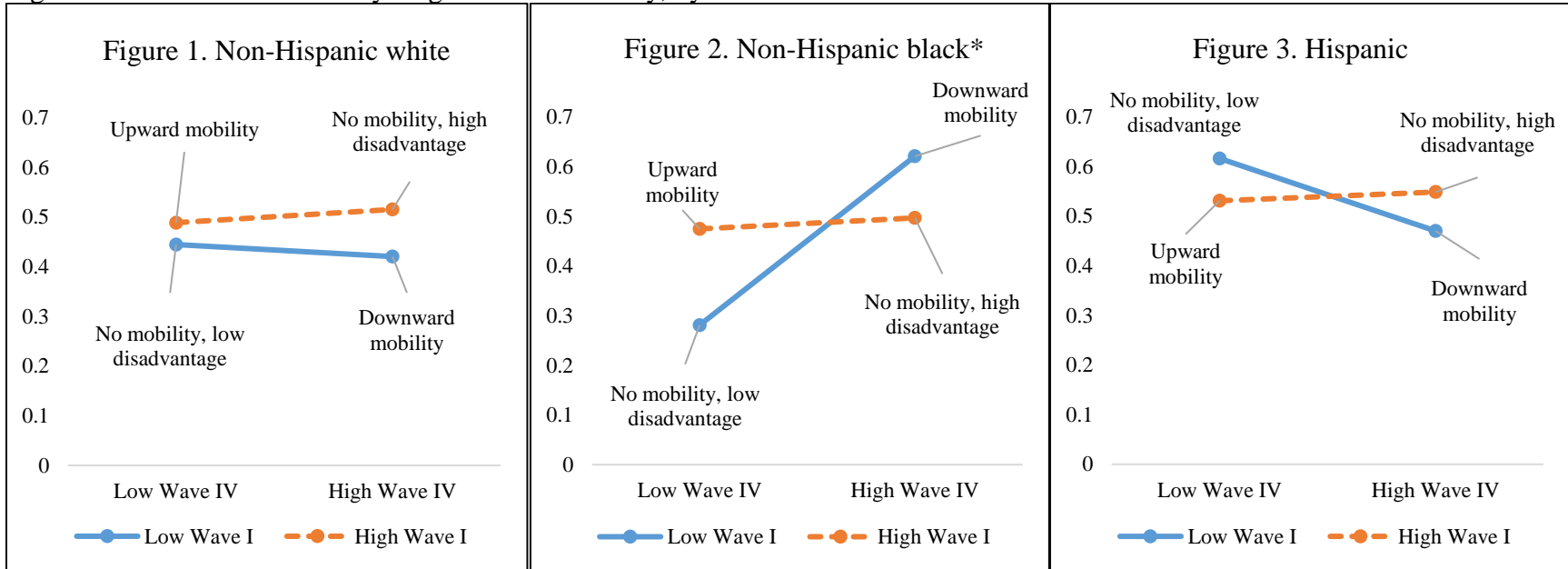
Table 2. Regression of lnCRP on neighborhood disadvantage at Wave I and Wave IV

	Non-Hispanic white			Non-Hispanic black			Hispanic		
	Beta	SE	p-value	Beta	SE	p-value	Beta	SE	p-value
High disadvantage at Wave I	0.04	0.06	0.461	0.19	0.12	0.120	-0.09	0.10	0.413
High disadvantage at Wave IV	-0.02	0.05	0.629	0.34	0.14	0.016*	-0.15	0.09	0.114
High disadvantage at Wave I x high disadvantage at Wave IV	0.05	0.08	0.546	-0.32	0.16	0.048*	0.16	0.14	0.259

Additional covariates included in the regressions but not displayed: count of sub-clinical symptoms, count of infectious or inflammatory diseases, inflammatory drug use, hormonal contraceptive use, daily smoking, waist circumference, BMI, age at Wave IV, and sex.

\*p-value < 0.05

Figures 1-3: Predicted lnCRP by neighborhood mobility, by race



\*p-value < 0.05

## REFERENCES

- Allin, Kristine H. and Børge G. Nordestgaard. 2011. "Elevated C-Reactive Protein in the Diagnosis, Prognosis, and Cause of Cancer." *Critical Reviews in Clinical Laboratory Sciences* 48(4):155-70. doi: 10.3109/10408363.2011.599831.
- Buckley, D. I., R. Fu, M. Freeman, K. Rogers and M. Helfand. 2009. "C-Reactive Protein as a Risk Factor for Coronary Heart Disease: A Systematic Review and Meta-Analyses for the U.S. Preventive Services Task Force." *Annals of Internal Medicine* 151(7):483-95. doi: 10.7326/0003-4819-151-7-200910060-00009.
- Chen, Edith, Gregory E. Miller, Gene H. Brody and ManKit Lei. 2015. "Neighborhood Poverty, College Attendance, and Diverging Profiles of Substance Use and Allostatic Load in Rural African American Youth." *Clin. Psychol. Sci.* 3(5):675-85.
- Chen, Ping and Kim Chantala. 2014. "Guidlines for Analyzing Add Health Data." Vol. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill.
- Cox, A. J., S. Agarwal, D. M Herrington, J. J. Carr, B. I. Freedman and D. W. Bowden. 2012. "C-Reactive Protein Concentration Predicts Mortality in Type 2 Diabetes: The Diabetes Heart Study." *Diabetic Medicine* 29(6):767-70. doi: doi:10.1111/j.1464-5491.2011.03560.x.
- Gaydosh, Lauren, Kristen M. Schorpp, Edith Chen, Gregory E. Miller and Kathleen Mullan Harris. 2018. "College Completion Predicts Lower Depression but Higher Metabolic Syndrome among Disadvantaged Minorities in Young Adulthood." *PNAS* 115(1):109-14.
- Harris, Kathleen Mullan, C.T. Halpern, E. Whitsel, J. Hussey, J. Tabor, P. Entzel and J.R. Udry. 2009. "The National Longitudinal Study of Adolescent to Adult Health: Research Design [Www Document]." (<http://www.cpc.unc.edu/projects/addhealth/design>).
- Hudson, Darrell L., Eli Puterman, Kirsten Bibbins-Domingo, Karen A. Matthews and Nancy E. Adler. 2013. "Race, Life Course Socioeconomic Position, Racial Discrimination, Depressive Symptoms and Self-Rated Health." *Social Science & Medicine* 97:7-14. doi: <https://doi.org/10.1016/j.socscimed.2013.07.031>.
- Hudson, Darrell L., Harold W. Neighbors, Arline T. Geronimus and James S. Jackson. 2016. "Racial Discrimination, John Henryism, and Depression among African Americans." *Journal of Black Psychology* 42(3):221-43. doi: 10.1177/0095798414567757.
- Link, Bruce G., Jo C. Phelan and Mark L. Hatzenbuehler. 2018. "Stigma as a Fundamental Cause of Health Inequality " Pp. 53-67 in *The Oxford Handbook of Stigma, Discrimination, and Health*, edited by B. Major, J. F. Dovidio and B. G. Link. New York, NY: Oxford University Press.
- Massey, Douglas S. and Brandon Wagner. 2018. "Segregation, Stigma, and Stratification: A Biosocial Model " Pp. 147-62 in *The Oxford Handbook of Stigma, Discrimination, and Health*, edited by B. Major, J. F. Dovidio and B. G. Link. New York, NY: Oxford University Press.
- Miller, Gregory E., Sheldon Cohen, Denise Janicki-Deverts, Gene H. Brody and Edith Chen. 2016. "Viral Challenge Reveals Further Evidence of Skin-Deep Resilience in African Americans from Disadvantaged Backgrounds." *Health Psychol.* 35(11):1225-34.
- O'Connor, Mary-Frances, Julie E. Bower, Hyong Jin Cho, J. David Creswell, Stoyan Dimitrov, Mary E. Hamby, Michael A. Hoyt, Jennifer L. Martin, Theodore F. Robles, Erica K. Sloan, KaMala S. Thomas and Michael R. Irwin. 2009. "To Assess, to Control, to Exclude: Effects of Biobehavioral Factors on Circulating Inflammatory Markers." *Brain Behav Immun* 23(7):887-97. doi: <https://doi.org/10.1016/j.bbi.2009.04.005>.

- Pearson, Thomas A., George A. Mensah, R. Wayne Alexander, Jeffrey L. Anderson, Richard O. Cannon, Michael Criqui, Yazid Y. Fadl, Stephen P. Fortmann, Yuling Hong, Gary L. Myers, Nader Rifai, Sidney C. Smith, Kathryn Taubert, Russell P. Tracy and Frank Vinicor. 2003. "Markers of Inflammation and Cardiovascular Disease: Application to Clinical and Public Health Practice: A Statement for Healthcare Professionals from the Centers for Disease Control and Prevention and the American Heart Association." *Circulation* 107(3):499-511.
- Phelan, Jo C. and Bruce G. Link. 2015. "Is Racism a Fundamental Cause of Inequalities in Health?". *Annual Review of Sociology* 41(1):311-30. doi: 10.1146/annurev-soc-073014-112305.
- Radloff, Lenore Sawyer. 1977. "The Ces-D Scale: A Self-Report Depression Scale for Research in the General Population." *Applied Psychological Measurement* 1(3):385-401. doi: 10.1177/014662167700100306.
- Steptoe, Andrew, Mark Hamer and Yoichi Chida. 2007. "The Effects of Acute Psychological Stress on Circulating Inflammatory Factors in Humans: A Review and Meta-Analysis." *Brain Behav Immun* 21(7):901-12. doi: <https://doi.org/10.1016/j.bbi.2007.03.011>.
- Taylor, Shelley E., Barbara J. Lehman, Catarina I. Kiefe and Teresa E. Seeman. 2006. "Relationship of Early Life Stress and Psychological Functioning to Adult C-Reactive Protein in the Coronary Artery Risk Development in Young Adults Study." *Biol Psychiatry* 60(8):819-24. doi: <https://doi.org/10.1016/j.biopsych.2006.03.016>.