RESEARCH ARTICLE

Diminished Returns of Educational Attainment on Heart Disease among Black Americans

Shervin Assari¹,², Sharon Cobb², Mohammed Saqib³ and Mohsen Bazargan¹,⁴

¹Department of Family Medicine, Charles R. Drew University of Medicine and Science, Los Angeles, CA, USA
²School of Nursing, Charles R. Drew University of Medicine and Science, Los Angeles, CA, USA
³Department of Health Behavior and Health Education, University of Michigan, Ann Arbor, MI, USA
⁴Department of Family Medicine, UCLA, Los Angeles, CA, USA

Abstract:

Background: Socioeconomic Status (SES) indicators, such as educational attainment, are social determinants of heart disease. Marginalization related Diminished Returns (MDRs) refer to smaller health benefits of high SES for racial and ethnic minorities compared to the majority group. It is still unknown, however, if MDRs also apply to the effects of education on heart disease.

Purpose: Using a nationally representative sample, we explored racial/ethnic variation in the link between educational attainment and heart disease among American adults.

Methods: We analyzed data (n=25,659) from a nationally representative survey of American adults in 2013. The first wave of the Population Assessment of Tobacco and Health - Adult (PATH-Adult) study was used. The independent variable was education (college graduate, high school graduate, less than a high school diploma). The dependent variable was any heart disease. Age and gender were the covariates. Race, as well as ethnicity, were the moderators. Logistic regressions were used to analyze the data.

Results: Individuals with higher educational attainment had lower odds of heart disease. Race and ethnicity showed statistically significant interactions with education, suggesting that the protective effect of higher education on reducing odds of heart disease was smaller for Hispanic and Black people than for non-Hispanic and White individuals.

Conclusion: Education reduces the risk of heart disease better among non-Hispanic Whites than for Hispanics and Blacks. Therefore, we may expect a disproportionately higher than expected risk of heart disease in Hispanics and Blacks with high educational attainment. Future research should test if the presence of high levels of environmental and behavioral risk factors contribute to the high risk of heart disease in highly educated Black and Hispanic Americans. Policymakers should not reduce health inequalities to just gaps in SES because disparities are present across SES levels, with high SES Blacks and Hispanics remaining at risk of health problems.

Keywords: Socioeconomic position, Socioeconomic status, Educational attainment, Ethnicity, Race, Ethnic groups, African Americans, Blacks, Heart disease.

1. INTRODUCTION

Although the health effects of socioeconomic status (SES) are well established [1 - 5], such effects are unequal across racial and ethnic groups [6 - 10]. While a higher education level predicts better health [3, 4], recent research has documented weaker protective effects of SES indicators for racial and ethnic minority groups [11 - 14]. Marginalization-related Diminished Returns (MDRs) refer to significantly weaker health effects of SES indicators, particularly education, for the
members of racial and ethnic minorities, particularly Blacks and Hispanics, relative to non-Hispanic White Americans [6, 7]. MDRs are being viewed as a paradigm shift as it investigates the mechanisms behind poor health of racial and ethnic minority people across SES levels [6, 7].

Considerable MDRs [6, 7] are shown in Blacks and Hispanics compared with Whites and non-Hispanics, documented as weaker effects of SES on the health of racial and ethnic groups [6, 7]. Such MDRs are shown for several SES indicators such as education [15 - 18], income [19 - 22], marital status [23], and employment [24] on various health outcomes. Supporting evidence has shown MDRs on drinking [15], smoking [18], diet [25] and exercise [16]. Similarly, MDRs exist for obesity as well [20, 26]. In addition, we can see MDRs for mental health indicators such as depression [22], anxiety [23], and risk of attempting suicide [27]. As a result, middle-class Blacks and Hispanics remain at a higher risk of chronic diseases (CDS) [19, 28, 29], disability [30], hospitalization [31], and mortality [32] even when they have a high SES.

In one study, highly educated Black and Hispanic people were found to be exposed to high levels of second-hand smoke exposure [33]. In addition, highly educated Blacks and Hispanics are more likely to drink alcohol [15, 34], smoke cigarettes [18, 35], and vape e-cigarettes [36]. This is probably why highly educated Black and Hispanic individuals may be at a higher risk of a wide range of non-communicable diseases such as hypertension [37], chronic obstructive pulmonary disease (COPD) [29] and asthma [28], compared with highly educated non-Hispanic Whites. Increased risk of heart disease may be particularly important and explain why highly educated Hispanics and Blacks stay at high risk of disability [30], hospitalization [31], and mortality [32], particularly for Black individuals.

Some of the mechanisms behind these MDRs include societal and structural processes and factors such as residential segregation, extra costs of moving upward in the society [38, 39], and a higher level of exposure [8, 40, 41] and sensitivity to discrimination [42] in ethnic minorities, particularly Blacks. As a result of such societal processes, high SES will be followed with less tangible health effects for Black people than non-Hispanic White people. However, less is known about MDRs of education level for heart disease in general.

As a result of MDRs [6, 7], the health impact of SES indicators including but not limited to education level, is diminished for racial and ethnic minority groups. Thus, diverse racial and ethnic groups not only vary in their SES [43 - 45] but also how their SES relates to health outcomes [22, 46].

Although high education reduces exposure to risk factors overall [47, 48], highly educated Blacks and Hispanics report high levels of environmental risk factors such as stress [39], discrimination [38], and active and passive exposure to smoke [18, 33, 36]. While the very same SES indicators, such as educational attainment, show a strong impact on lowering non-Hispanic Whites’ environmental risk exposures, highly educated Blacks and Hispanics are still at risk of factors that increase their risk to non-communicable illnesses such as hypertension [37], attention deficit hyperactivity disorder (ADHD) [19], asthma [28], and COPD [29].

2. AIMS

To better understand whether MDRs also applies to racial and ethnic disparities in heart disease, we compared Blacks, Hispanics, and non-Hispanic Whites for the negative effect of education level against heart disease. Although research has well-documented the effects of race/ethnicity [28, 49] and SES (education level) [28, 50, 51] on heart disease, few studies have ever tested MDRs of SES resources on heart disease [28, 29]. This study goes beyond the additive effect of race/ethnicity and SES and investigates the multiplicative effects of SES and race/ethnicity on heart disease [28, 29]. This work is in line with the research question if it is race/ethnicity and SES or race/ethnicity or SES that cause health disparities. To generate results that are generalizable to the U.S. population of adults, we borrowed data from the Population Assessment of Tobacco and Health - Adult (PATH-Adult) study, a survey with a nationally representative sample of adults (18+ years old). In line with the MDRs [6, 7] and previous relevant studies [28, 29], we hypothesized smaller effects of SES (educational attainment) on heart disease for Blacks and Hispanics than non-Hispanic Whites.

3. METHODS

3.1. Study Design and Setting

This cross-sectional survey borrowed existing data from the PATH 2013 [52 - 55] study, a national survey of American adults sponsored by the National Institutes of Health (NIH) and the Food and Drug Administration (FDA). PATH is a landmark survey of tobacco use and associated diseases. The data collection was carried out in 2013. The PATH design, sampling, sample design, and measures are described elsewhere [52 - 55].

The sample is limited to the civilian, non-institutionalized, adult (18+ years old) residents of the United States. The PATH data is a longitudinal study; however, we only used baseline data (wave 1). The sample design follows a multistage probability sample that recruited a representative sample of households and non-institutionalized people. The PATH study has used a multi-stage sampling strategy that involves survey weights. Due to the national sample and application of survey weights, the results of PATH are generalizable to the US population. In this study, 25,659 adults were analyzed.

3.2. Ethics

The PATH study protocol was approved by the Westat Institutional Review Board (IRB). All participating individuals signed informed written consent. All the PATH data were collected, stored, and analyzed anonymously.
4. MEASURES

4.1. Independent Variable

4.1.1. Education level

Education level was operationalized as a categorical variable with 3 levels: less than high school graduate, completed high school, and college graduates.

4.2. Moderators

4.2.1. Race

All participants self-identified their race. Race was treated as a dichotomous variable in the current study [Blacks = 1, non-Hispanic Whites = 0].

4.2.2. Ethnicity

All participants self-identified their ethnicity. Ethnicity was treated as a categorical variable in the current study [Hispanics = 1, Non-Hispanics = 0].

4.3. Covariates

Covariates in the current study included age (years) and gender (binary variable). Age was a categorical measure with the following levels: 1) 18 - 24 years old, 2) 25 - 34 years old, 3) 35 - 44 years old, 4) 45 - 54 years old, 5) 55 - 64 years old, 6) 65 - 74 years old, and, 7) 75+ years old. Gender was dichotomous (female = 0 vs. male = 1).

4.4. Outcome

Heart Disease. Heart diseases in this study included heart attack, congestive heart disease, and any other heart disease. Three items were used to measure lifetime history of heart disease. For example, participants were asked, “Have you ever been told by a doctor or other health professional that you had heart attack?” “Have you ever been told by a doctor or other health professional that you had congestive heart disease?” “Have you ever been told by a doctor or other health professional that you had any other heart disease?”

4.5. Data Analysis

We used the survey mode of SPSS 23.0 (IBM Inc, NY, USA) to analyze the data. Taylor series linearization was used to re-calculate the standard errors (SEs) that address the complex design of the PATH sample. Our estimates and inferences are generalizable to the US sample. We ran logistic regression models without and with interaction terms, all in the pooled sample. Model 1 had the main effects of race, ethnicity, education level, and covariates. Model 2 also included the following interaction terms: race $\times$ education level and ethnicity $\times$ education level. In all models, educational attainment and poverty level were the independent variables (IVs), heart disease was the dependent variable (DV), and age and gender were covariates. Race and ethnicity were the moderators. Publicly available data are available at the University of Michigan ICPSR Website available at: https://www.icpsr.umich.edu/icpsrweb/NAHDAP/studies/3623. As Black and Hispanic were both coded as 1, for the main effect of high educational attainment we expected a significant OR <1 (protective factor), and a significant OR >1 for the statistical interaction term between race/ethnicity and high educational attainment.

5. RESULTS

5.1. Descriptive Characteristics

This study included 25,659 American adults who were either White (82.9%), Black (17.1%), Non-Hispanic (83.9%), or Hispanic (16.1%). Table 1 provides a summary of the descriptive statistics of the study sample. Almost half of the participants were women.

Table 1. Descriptive statistics in the overall sample.

| -          | n   | %  |
|------------|-----|----|
| Race       |     |    |
| White      | 21265 | 82.9 |
| Black      | 4394   | 17.1 |
| Ethnicity  |     |    |
| Non-Hispanic | 21520 | 83.9 |
| Hispanic   | 4139   | 16.1 |
| Gender     |     |    |
| Women      | 12705  | 49.5 |
| Men        | 12954  | 50.5 |
| Age (years)|     |    |
| 18 – 24    | 6877   | 26.8 |
| 25 – 34    | 5197   | 20.3 |
| 35 – 44    | 4066   | 15.8 |
| 45 – 54    | 3979   | 15.5 |
| 55 – 64    | 3190   | 12.4 |
| 65 – 74    | 1636   | 6.4 |
| 75+        | 714    | 2.8 |
| Education  |     |    |
| Less than high school graduate | 4973 | 19.4 |
| High school graduate | 15137 | 59.0 |
| College Graduate | 5549 | 21.6 |

5.2. Multivariable Models

Table 2 shows a summary of the output of 2 logistic regression models. In both models, educational attainment was the independent variable and any heart disease was the dependent variable. Both models were estimated in the total sample, which included Whites, Blacks, Hispanics, and non-Hispanics. Model 1 only entered the main effects of educational attainment as well as race, ethnicity, and covariates. Model 2, however, also added 2 interaction terms between race and ethnicity with educational attainment.

As shown in Model 1, high education predicted lower odds of heart disease. Model 2, however, showed significant interaction terms between the variables race and ethnicity with the independent variable educational attainment on the outcome variable heart disease, suggesting that high education may have a smaller protective effect against heart disease for Blacks and Hispanics than non-Hispanic Whites. This finding was reflected with a <1 and significant (protective factor) OR for the main effect of high educational attainment, and a >1 and
Table 2. Logistic regression in the pooled sample.

|                | Model 1 (Main Effects) | Model 2 (Interactions) |
|----------------|------------------------|------------------------|
|                | OR  B  SE  95% CI p    | OR  B  SE  95% CI p    |
| Race (Black)   | 0.83 -0.19 0.07 0.73 -0.94 0.005 0.66 -0.42 0.13 0.51 -0.84 0.001 |
| Ethnicity (Hispanic)  | 0.52 -0.64 0.09 0.44 -0.62 0.000 0.39 -0.95 0.14 0.29 -0.51 0.000 |
| Age (years)    | - - - - - - - - - - | - - - - - - - - - - |
| 18 – 24        | - - - - - - - - - - | - - - - - - - - - - |
| 25 – 34        | 1.24 0.22 0.11 1.00 -1.54 0.52 1.24 0.22 0.11 1.00 -1.55 0.049 |
| 35 – 44        | 2.09 0.74 0.10 1.70 -2.57 0.000 2.11 0.74 0.10 1.72 -2.59 0.000 |
| 45 – 54        | 3.57 1.27 0.09 2.97 -4.30 0.000 3.60 1.28 0.09 2.99 -4.33 0.000 |
| 55 – 64        | 6.48 1.87 0.09 5.43 -7.73 0.000 6.57 1.88 0.09 5.50 -7.84 0.000 |
| 65 – 74        | 10.25 2.33 0.10 8.50 -12.35 0.000 10.41 2.34 0.10 8.63 -12.54 0.000 |
| 75+            | 14.30 2.66 0.11 11.59 -17.64 0.000 14.57 2.68 0.11 11.80 -17.99 0.000 |
| Educational Attainment | - - - - - - - - - - | - - - - - - - - - - |
| Less than High School Graduate | - - - - - - - - - - | - - - - - - - - - - |
| High School Graduate | 0.71 -0.34 0.06 0.63 -0.81 0.000 0.71 -0.34 0.07 0.62 -0.81 0.000 |
| College Graduate | 0.80 -0.22 0.06 0.71 -0.90 0.000 0.71 -0.34 0.07 0.62 -0.82 0.000 |
| Race (Black) x High School Graduate | - - - - - - - - - - | - - - - - - - - - - |
| Race (Black) x College Graduate | - - - - - - - - - - | - - - - - - - - - - |
| Ethnicity (Hispanic) x High School Graduate | - - - - - - - - - - | - - - - - - - - - - |
| Ethnicity (Hispanic) x College Graduate | - - - - - - - - - - | - - - - - - - - - - |
| Constant        | 0.03 -3.37 0.09 - - - - - - - - - - | 0.04 -3.29 0.09 - - - - - - - - - - |

Notes: Source Population Assessment of Tobacco and Health (PATH; 2013-2014) CI: Confidence Interval; SE: Standard Error; OR: Odds R.

The protective influence of SES on health [1, 2], non-communicable diseases [28, 50, 56], and heart diseases [57, 58]. The protective health effect of SES is well known.

The first finding on the inverse association between education and heart disease is in line with what is known about the protective influence of SES on health [1, 2], non-communicable diseases [28, 50, 56], and heart diseases [57, 58]. The protective health effect of SES is well known.

Another explanation for the lower health effects of educational attainment for Blacks and Hispanics compared with non-Hispanic Whites is the differences in the “quality” of education for Whites and Blacks and Hispanics may account for the lower effects of education on heart disease (and other measures of well-being) in Blacks and Hispanics. In other words, high-school and college education may not mean the same thing for Whites, Blacks and Hispanics. Hispanics and...
Blacks are more likely to attend urban public schools with lower resources [65]. Manley and others have shown that schooling differences may explain some of the differential effects of education for Whites and people of color [66, 67].

Another explanation for this finding is that compared with non-Hispanic Whites, Blacks and Hispanics with high education are more likely to be new to the educational class. This is because children with highly educated parents are more likely to attain higher education as well. Future research should test recency to the middle class, parental education, and being a first-generation college graduate, as these factors may hint at mechanisms by which non-White families gain less health from their educational attainment.

There is also a need to evaluate how early in the life course SES disadvantage results in atherosclerotic changes among Blacks. Research has shown that one reason high SES during adulthood is still associated with poor health among Blacks is childhood poverty [68 - 72]. That means early adversity results in some effects that cannot be undone with later availability of resources and human capital [73 - 77]. According to the Barker hypothesis [78], pathological changes that mediate adversities may start in utero. Some behavioral mechanisms such as drinking [34], vaping [36], smoking [18, 59], poor diet [25], poor health care use [79, 80], and low exercise [16] that are all common in highly educated Blacks may mediate the additional cardiovascular risk of middle-class Blacks. Another mechanism is environmental exposures in highly educated Blacks [81 - 83]. Genetics may also accelerate the process of atherosclerosis for Black people, and some research suggests that genes may increase the vulnerability of Black populations to hypertension [84 - 87] and kidney disease [87 - 91], which both have implications for development and outcomes of heart disease [92, 93]. Diabetes may also be more common in highly educated Blacks through some genetic predisposition [94].

7. LIMITATIONS

The major limitation of this study is its cross-sectional design. As a result of such design, we do not infer causal effects from our observations. Most SES indicators and health outcomes have bidirectional links and reverse causal effect of health on SES cannot be ruled out. Poor health is one of the reasons behind downward social mobility. This study measured heart diseases such as COPD and asthma based on self-report rather than physical examination, laboratory testing, or administered data. Self-reports have been shown to generate valid and reliable measures of chronic diseases. However, future research may replicate these findings for other ethnic groups such as Asian Americans, Native Americans as well as immigrants. Future research may also use comprehensive measures. The study is at risk of omitted confounders such as wealth and area SES. We only described the MDRs of education and heart disease. Future research may test whether education quality, discrimination, segregation, labor market discrimination, or neighborhood poverty can explain why Blacks and Hispanics have less health gain from their available resources than non-Hispanic Whites. Finally, it is possible that the effect of education on heart disease may also be different for males and females among Blacks and Hispanics (and Whites). Future work may also include interactions between education, race/ethnicity, and gender. Still, this study had a large sample size and extends what we know regarding the non-linear effects of race, ethnicity, and SES on health outcomes, particularly heart disease.

CONCLUSION

In the United States, the inverse association between education and heart disease is weaker for Blacks and Hispanics than non-Hispanic Whites. More specifically, a significant interaction was found between race/ethnicity and college-level education (but not with high school education), suggesting that the protective effect of college-level education was smaller for Blacks and Hispanics than non-Hispanic Whites. As a result, a higher than expected prevalence of heart disease in highly educated Black and Hispanic people, a rate which is disproportionate to their SES, has been observed. Researchers should know that health disparities are not merely due to the additive but also the multiplicative effects of race/ethnicity and SES. Real solutions to racial and health disparities in heart disease would be based not only on increasing SES of Blacks and Hispanic but also empowering them to translate their available SES resources to health outcomes. Public policies need to go beyond equalizing SES and specifically address societal barriers, environmental risk factors, and structural factors that endanger the health and well-being of Blacks and Hispanics at all SES levels. Economic and public policies are needed to minimize diminished health returns of SES (i.e., MDRs). Unfortunately, MDRs are historically overlooked as contributors to racial and ethnic health disparities in the US.

AUTHORS' CONTRIBUTIONS

SA designed the conceptual model, secured funding, performed the analysis, wrote the first draft, and revised the manuscript. MB, MS and SC contributed to the interpretation of the findings, drafts, and revision. All authors approved the final version of the text.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The PATH study protocol was approved by the Westat Institutional Review Board (IRB), the Westat Federalwide Assurance (FWA) number is FWA00005551.

HUMAN AND ANIMAL RIGHTS

Not applicable.

CONSENT FOR PUBLICATION

Written informed consent has been obtained from all the participants.

AVAILABILITY OF DATA AND MATERIALS

The data supporting the findings of the article is available in the ICPSR at https://www.icpsr.umich.edu/icpsrweb/NAHDAP/studies/36231 with reference number ICPSR-36231.
ACKNOWLEDGEMENTS
U54CA229974, U54MD007598, U54TR001627, CA201415-02 and of Health (NIH) grants: U54MD008149, R25MD007610, 22246797, 10604074, 11693982, 35814070, 1099-104.
http://dx.doi.org/10.1016/S0140-6736(05)74234-3 [PMID: 15781105]
http://dx.doi.org/10.1016/j.socscimed.2004.04.026 [PMID: 15482878]
http://dx.doi.org/10.1007/s11524-011-0348-x [PMID: 21293845]
http://dx.doi.org/10.11648/j.ijhpm.20190502.12 [PMID: 31633081]
http://dx.doi.org/10.3390/children5060073 [PMID: 29857488]
http://dx.doi.org/10.3390/children6010060 [PMID: 29890078]
http://dx.doi.org/10.3390/children6001001 [PMID: 30646634]
http://dx.doi.org/10.3390/children5006007 [PMID: 29724004]
http://dx.doi.org/10.1007/s40615-017-0381-x [PMID: 28634376]
http://dx.doi.org/10.1177/0022146515594814 [PMID: 26272989]
http://dx.doi.org/10.1016/j.sjwhh.2014.09.014 [PMID: 25244537]
http://dx.doi.org/10.1097/jomh.2017.03.006 [PMID: 22707959]
http://dx.doi.org/10.1097/jomh.2012.03.006 [PMID: 22655817]
http://dx.doi.org/10.1007/s11524-011-0348-x [PMID: 21293845]
http://dx.doi.org/10.1007/s11524-011-0082-8 [PMID: 22246797]
http://dx.doi.org/10.3390/children5060073 [PMID: 29857488]
http://dx.doi.org/10.3390/children6010060 [PMID: 29890078]
http://dx.doi.org/10.3390/children6001001 [PMID: 30646634]
http://dx.doi.org/10.3390/children5006007 [PMID: 29724004]
http://dx.doi.org/10.1007/s40615-017-0381-x [PMID: 28634376]
http://dx.doi.org/10.11648/j.ijhpm.20190502.12 [PMID: 31633081]
http://dx.doi.org/10.3390/children5060073 [PMID: 29857488]
http://dx.doi.org/10.3390/children6010060 [PMID: 29890078]
http://dx.doi.org/10.3390/children6001001 [PMID: 30646634]
Assari S, Mistry R. Diminished Return of Employment on Ever Smoking Among Hispanic Whites in Los Angeles. Health Equity 2019; 3(1): 138-44.

Assari S. Combined Racial and Gender Differences in the Long-Term Predictive Role of Education on Depressive Symptoms and Chronic Medical Conditions. J Racial Ethn Health Disparities 2017; 4(3): 385-96.

Assari S, Gibbons FX, Simons R. Depression among Black Youth: Interaction of Class and Place Brain Sci 2018; 8(6).

Assari S, Jared SM, Amin AH. Depression among White Young Adults: the role of sex and education J Am Heart Assoc 2013; 2(2): e004978.

Assari S, Gibbons FX, Simons R. Depression among Black Youth: Interaction of Class and Place Brain Sci 2018; 8(6).

Assari S, Jared SM, Amin AH. Depression among White Young Adults: the role of sex and education J Am Heart Assoc 2013; 2(2): e004978.

Assari S. Depression among White Young Adults: the role of sex and education J Am Heart Assoc 2013; 2(2): e004978.

Assari S, Gibbons FX, Simons R. Depression among Black Youth: Interaction of Class and Place Brain Sci 2018; 8(6).

Assari S. Depression among White Young Adults: the role of sex and education J Am Heart Assoc 2013; 2(2): e004978.

Assari S, Gibbons FX, Simons R. Depression among Black Youth: Interaction of Class and Place Brain Sci 2018; 8(6).

Assari S, Jared SM, Amin AH. Depression among White Young Adults: the role of sex and education J Am Heart Assoc 2013; 2(2): e004978.
Assari et al.

12 The Open Cardiovascular Medicine Journal, 2020, Volume 14

[71] Ramsay SE, Whincup PH, Lennon LT, Morris RW, Wannamethee SG. Longitudinal associations of socioeconomic position in childhood and adulthood with decline in lung function over 20 years: results from a population-based cohort of British men. Thorax 2011; 66(12): 1058-64. [http://dx.doi.org/10.1136/thoraxjnl-2011-200621] [PMID: 21784770]

[72] Zeki Al Hazouzi A, Haan MN, Kalbfeisch JD, Galea S, Lipsbeth LD, Aiello AE. Life-course socioeconomic position and incidence of dementia and cognitive impairment without dementia in older Mexican Americans: Results from the sacramento area latino study on aging. Am J Epidemiol 2011; 173(10): 1148-58. [http://dx.doi.org/10.1093/aje/kwy483] [PMID: 21430188]

[73] Beauregard JL, Drews-Botsch C, Sales JM, Flanders WD, Kramer MR. Does socioeconomic status modify the association between preterm birth and children's early cognitive ability and kindergarten academic achievement in the United States? Am J Epidemiol 2018; 187(8): 1704-3. [http://dx.doi.org/10.1093/aje/kwy068] [PMID: 29757345]

[74] Cox K. Reducing toxic stress experienced by children living in poverty. Nurs Outlook 2018; 66(2): 108-9. [http://dx.doi.org/10.1016/j.outlook.2018.02.010] [PMID: 29602448]

[75] Cox KS, Sullivan CG, Olshansky E, et al. Critical conversation: Toxic stress in children living in poverty. Nurs Outlook 2018; 66(2): 204-9. [http://dx.doi.org/10.1016/j.outlook.2018.02.009] [PMID: 29555091]

[76] Evans GW, Fuller-Rowell TE. Childhood poverty, chronic stress, and young adult working memory: The protective role of self-regulatory capacity. Dev Sci 2013; 16(5): 688-96. [http://dx.doi.org/10.1111/desc.12102] [PMID: 24035374]

[77] Evans GW, Kim P. Childhood poverty and health: Cumulative risk exposure and stress dysregulation. Psychiatr Sci 2007; 18(11): 953-7. [http://dx.doi.org/10.1111/j.1467-9280.2007.00208.x] [PMID: 17958708]

[78] Barker DJ, Winter PD, Osmond C, Margetts B, Simmonds SJ. Weight in infancy and death from ischaemic heart disease. Lancet 1989; 2(8663): 577-80. [http://dx.doi.org/10.1016/S0140-6736(89)90971-0] [PMID: 2570282]

[79] Assari S, Hani N. Household Income and Children's Unmet Dental Care Need; Blacks' Diminished Return. Dent J (Basel) 2018; 6(2) [PMID: 29602448]

[80] Assari S, Bazargan M. Educational attainment better increases the chance of breast physical exam for non-hispanic than hispanic American women: National health interview survey. Hosp Pract Res 2019; 4(4): 122-7. [http://dx.doi.org/10.15171/hpr.2019.25] [PMID: 32109011]

[81] Assari S, Bazargan M. Unequal Associations between Educational Attainment and Occupational Stress across Racial and Ethnic Groups. Int Journal Environment Res Pub Health 2019; 16(19):3539. [http://dx.doi.org/10.3390/ijerph16193539]

[82] Assari S, Bazargan M. unequal effects of educational attainment on workplace exposure to second-hand smoke by race and ethnicity; minorities’ diminished returns in the national health interview survey (NHIS). J Med Res Innov 2019; 3(2) [http://dx.doi.org/10.32892/jmri.179] [PMID: 31404444]

[83] Assari S BM. Second-hand exposure home second-hand smoke exposure at home in the united states; minorities’ diminished returns Int J Travel Med Glob Health 2019; 7(3) [PMID: 30340464]

[84] Brewer LM, Clark JF, van Montfrans GA. Is greater tissue activity of creatine kinase the genetic factor increasing hypertension risk in black people of sub-Saharan African descent? J Hypertens 2000; 18(11): 1537-44. [http://dx.doi.org/10.1097/00004872-200018110-00002] [PMID: 11081764]

[85] Cooper R, Rotimi C. Hypertension in populations of West African origin: is there a genetic predisposition? J Hypertens 1994; 12(3): 215-27. [PMID: 8021474]

[86] Daniel HI, Rotimi CN. Genetic epidemiology of hypertension: an update on the African diaspora. Ethn Dis 2003; 13(2)(Suppl. 2): S53-66. [PMID: 13677415]

[87] George C, Yako YY, Okpechi IG, Matsha TE, Kaze Folofack FJ, Kengne AP. An African perspective on the genetic risk of chronic kidney disease: A systematic review. BMC Med Genet 2018; 19(1): 187. [http://dx.doi.org/10.1186/s12881-018-0792-x] [PMID: 30340464]

[88] Price DA, Crook ED. Kidney disease in African Americans: Genetic considerations J Natl Med Assoc 2002; 94(8 Suppl): 16S-27S. [PMID: 12152908]

[89] Reidy KJ, Hjorten R, Parekh RS. Genetic risk of APOL1 and kidney disease in children and young adults of African ancestry. Curr Opin Nephrol 2018; 30(2): 252-9. [http://dx.doi.org/10.1097/MOP.0000000000001603] [PMID: 29406442]

[90] Swift PA, Macgregor GA. Genetic variation in the epithelial sodium channel: A risk factor for hypertension in people of African origin. Adv Ren Replace Ther 2004; 11(1): 76-86. [http://dx.doi.org/10.1053/j.arrt.2003.10.011] [PMID: 14730541]

[91] Udler MS, Nadkarni GN, Belbin G, Lotay V, Wyatt C, Gottesman O, et al. Effect of Genetic African ancestry on eGFR and kidney disease J Am Soc Nephrol 2015; 26(7): 1682-92. [http://dx.doi.org/10.1681/ASN.2014050474] [PMID: 25349204]

[92] Cooper R. The role of genetic and environmental factors in cardiovascular disease in African Americans. Am J Med Sci 1999; 317(3): 208-13. [http://dx.doi.org/10.1016/S0002-9629(15)40504-X] [PMID: 10100695]

[93] Wassef CL, Pankow JS, Peralta CA, Choudhry S, Seldin MF, Arnett DK. Genetic ancestry is associated with subclinical cardiovascular disease in African-Americans and Hispanics from the multi-ethnic study of atherosclerosis. Circ Cardiovasc Genet 2009; 2(6): 629-36. [http://dx.doi.org/10.1161/CIRCGENETICS.109.876243] [PMID: 20031644]

[94] Mige JB, Grant RW, Piccolo R, et al. Association of african genetic ancestry with fasting glucose and hba1c levels in non-diabetic individuals: the boston area community health (BACH) prediabetes study Diabetologia 2014; 57(9): 1850-8. [http://dx.doi.org/10.1007/s00125-014-3301-1] [PMID: 24942103]

© 2020 Assari et al.

This is an open access article distributed under the terms of the Creative Commons Attribution 4.0 International Public License (CC-BY 4.0), a copy of which is available at: https://creativecommons.org/licenses/by/4.0/legalcode. This license permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.