History of Non-Fatal Physical Assault Is Associated with Premature Mortality for Whites but Not Blacks

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Abstract: Exposure to trauma increases the long-term risk of mortality, and experiencing non-fatal physical assault is not an exception. To better understand population heterogeneity in this link, the current study explored Black–White differences in the association between history of non-fatal physical assault and risk of all-cause mortality over a 25-year period in the United States. Data came from the Americans’ Changing Lives (ACL) study that followed 3617 non-institutionalized respondents for up to 25 years. History of non-fatal physical assault at baseline was the predictor. Outcome was time to death due to all-cause mortality during follow-up from baseline (1986) to follow-up (2011). Confounders included gender, age, and baseline socio-economic status (education and income), health behaviors (smoking and drinking), and health status (chronic medical conditions, self-rated health, and body mass index). Race was the moderator. Cox regressions were used for multi-variable analysis. History of non-fatal physical assault at baseline was associated with an increased risk of mortality, above and beyond baseline socioeconomic status, health behaviors, and health status. Race interacted with history of non-fatal physical assault on mortality, suggesting a stronger effect for Whites compared to Blacks. In race-specific models, history of non-fatal physical assault was associated with risk of mortality for Whites but not Blacks. The current study showed that experiencing non-fatal physical assault increases the risk of premature death above and beyond demographics, socioeconomic status, health behaviors, and health status. Experiencing non-fatal physical assault may have a larger effect on premature mortality among Whites than Blacks. Future research is needed on how Blacks and Whites differ in the health consequences of social adversities.

Keywords: trauma; assault; ethnic groups; all-cause mortality; race; blacks

1. Introduction

Due to higher risk of living under poverty and economic adversity, Blacks are, on average, more likely to be victims of physical assault throughout their life course [1]. Black children who grow up in poverty are at a high risk of exposure to various types of violence [2]. During adulthood, Black women are at a high risk of intimate partner violence [3]. Black males who live in high-crime urban areas have a high risk of victimization of robbery and theft [4]. Given their poor access to the healthcare system, a majority of Black victims do not receive proper treatment when they experience physical assault [4]. Exposure to non-fatal physical assault increases risk of depression and substance abuse [3]. Some victims of non-fatal physical assault may put on a masculine mask to hide their psychological pain [5–8]. Some may turn to substance use, violence, or other high-risk behaviors as a coping strategy to reduce
physical and psychological pain of their traumatic experience [2–4]. Others may respond by trying to “fit in” through joining gangs or other means they think would lower their risk of re-victimization [2].

Despite a higher exposure to a wide range of social stressors [3,9,10], Blacks may have higher expectations and preparedness for stress than Whites [11]. In the presence of chronic stress, a new stressor may be less disabling. This pattern may explain the weaker effects of social risk factors for Blacks compared to Whites [9,11]. Compared to their White counterparts who have not experienced the same level of adversities, one may hypothesize that Blacks may be more resilient to adversities such as non-fatal physical assault. This is in line with previous research showing weaker effects of social adversities for Blacks compared to Whites [9,11–19].

Objectives

Considering the literature on racial differences in exposure to physical assault [2–4] as well as racial differences in health effects of social adversities [20,21], this study compared Blacks and Whites for the association between history of non-fatal physical assault and long-term risk of mortality using a nationally representative sample of adults in the United States (US).

2. Methods

2.1. Study Design

We used data from the Americans’ Changing Lives (ACL), a nationally representative longitudinal cohort study conducted from 1986 until 2011 in the US. More information on the study design and methodology is published elsewhere [22,23].

2.2. Sampling and Participants

The ACL used a stratified multistage probability sampling. ACL recruited adults ages 25 or above who lived in the continental US in 1986. The recruited sample included 3617 non-institutionalized respondents. The study oversampled those aged 60 and older, and African Americans/Blacks. Wave 1 included 70% of sampled households and 68% of sampled individuals. Data were also collected in 1989, 1994, 2001, and 2011, but information from those interviews was not relevant for these analyses.

2.3. Measures

Information on demographics, socioeconomic status (SES), health behaviors (smoking and drinking), chronic medical conditions (CMC), body mass index (BMI), self-rated health (SRH), and depressive symptoms was measured at baseline in 1986 during face-to-face interviews.

Race and ethnicity. In this study, self-reported race and ethnicity was measured at baseline in 1986 with several survey items. Respondents were first asked the following open-ended question: “In addition to being American, what do you think of as your ethnic background or origins?” Respondents were then asked the following multiple-choice question: “Are you White, Black, American Indian, Asian, or another race?” Individuals who responded to more than one non-White group were asked to identify their “best described race.” The survey also assessed the state or foreign country in which the respondent, respondent’s mother, and respondent’s father were born, and the respondent’s father’s last name. Finally, participants’ ethnicity was measured using the following item: “Are you of Spanish or Hispanic descent, that is, Mexican, Mexican American, Chicano, Puerto Rican, Cuban, or Other Spanish?” Responses from the above questions were used to construct the following race and ethnicity categories: (1) “Non-Hispanic White”, (2) “Non-Hispanic Black”, (3) “Non-Hispanic Native American”, (4) “Non-Hispanic Asian”, and (5) “Hispanic”. The current analysis only included “Non-Hispanic Whites” and “Non-Hispanic Blacks”. All Hispanic individuals were excluded from this analysis.

History of Non-Fatal Physical Assault. Experience of non-fatal physical assault was measured using the following single item: “Have you ever been the victim of a serious physical attack or assault at
any time in your life?” Responses included (1) yes, (0) no, and do not know (missing). This item was previously used by others [24].

**All-Cause Mortality.** The main outcome in this study was time to mortality from all causes. Information on all deaths that occurred from mid-1986 through end of 2011 was obtained through the following three sources: (1) National Death Index (NDI), (2) death certificates, and (3) informants. In most cases, death data were verified with death certificates. Although in a handful of cases death could not be verified with death certificates, actual death was certain in all cases. In these cases, death information was reviewed carefully. Only in these cases, date of death was ascertained from the NDI report or the informants (instead of the death certificates) [13,14]. Time of death was operationalized as a continuous variable, defined as the number of months between the time of enrolment to time of death.

**Demographic Characteristics.** Demographic indicators in this study included age and gender. Age was treated as a continuous variable, operationalized as the number of years since birth. Gender was treated as a dichotomous variable with male as the referent category.

**Socioeconomic Characteristics.** Socioeconomic status was measured with education (less than 12 years of education, and 12 years or more) and household income (a continuous variable as total annual income in the household).

**Body Mass Index (BMI).** Body mass index (BMI) was calculated based on the self-reported weights and heights. BMI based on self-reported weights and heights is validated and closely correlates with measured BMI [25].

**Smoking Status.** To measure smoking behavior, a single item was used that asked respondents whether they currently smoke. A dummy variable was created (1) current smoker vs. (0) non-smoker. Similar single-item measures were previously used to measure smoking status [26].

**Drinking Behavior.** To measure drinking behavior, a single-item measure was used concerning current alcohol use: (1) current drinker vs. (0) non-drinker [27].

**Self-Rated Health (SRH).** Respondents were asked to report their overall health as excellent, very good, good, fair, or poor. Past literature defined SRH as dichotomous and continuous measures. We operationalized SRH as a dichotomous measure. Similar to the literature, we collapsed the five categories into two categories (fair/poor vs. excellent/very good/good). SRH has high test–retest reliability and validity. SRH is predictive of mortality and other health outcomes, regardless of confounders. Most studies of the SRH–mortality link operationalized SRH as a dichotomous variable [28–31].

**Number of Chronic Medical Conditions (CMC).** Number of chronic medical conditions at baseline was measured based on self-reported presence of seven chronic medical conditions. Participants were asked whether a healthcare provider had ever told them they had any of the following focal conditions: hypertension, diabetes, heart disease, chronic lung disease, stroke, cancer, and arthritis. A sum score was calculated, potentially ranging from 0 to 7. A detailed description of the measurement of chronic medical conditions is provided elsewhere [15,32]. We controlled for chronic medical conditions because some (i.e., heart disease, hypertension, diabetes, and cancer) increase risk of death. As we were interested in the effect of non-fatal physical assault on all-cause mortality, we wanted to isolate this association, net of chronic conditions.

2.4. **Statistical Analysis**

Univariate, bivariate, and multivariable analyses were performed using Stata 13.0 (Stata Corporation, College Station, TX, USA). For multivariable analysis, Cox proportional hazard models were used to determine factors associated with time to death due to all causes over the 25 years of follow-up. Sample weights were applied in all analyses. Stratification and clustering in the estimation of standard errors was accounted for using Taylor series linearization. Sample sizes reflect the un-weighted sample distributions.
Cox proportional hazard models require data on an outcome (death due to all causes) and the time that outcome occurred since baseline (time to death). Death was coded zero if the respondent did not die over the 25-year follow-up period. Time to the death event or to censoring was defined as the number of months from baseline to death, loss to follow-up, or the end of the year 2011.

We ran seven models in the pooled sample first. We gradually added more covariates to the model, so Model 1 had the least covariates and Model 7 had the most. Models 2 to 7 also included an interaction term between race and history of non-fatal physical assault. We also ran six models in each racial group. Model 1 was conducted separately among Whites and Blacks and only controlled for demographic factors. Model 6 included all covariates. Covariates included age, gender, education, income, health behaviors, CMC, SRH, and depressive symptoms. Hazard ratios (HR) with 95% confidence intervals (CIs) are reported. A value of \( p < 0.05 \) was considered statistically significant.

3. Results

3.1. Descriptive Statistics

The sample was either Black (\( n = 1156 \)) or White (\( n = 2205 \)). Table 1 reports the descriptive statistics overall and based on race. Average age of the population was 47.77 (standard error, SE = 0.534); 52.74% of the population was female. On average, participants had 0.79 chronic medical conditions (SE = 0.028), which was higher in Blacks than Whites (0.91 vs. 0.78, \( p < 0.05 \)). Blacks had lower education and income and higher depressive symptoms, higher BMI, and worse SRH than Whites. Blacks were more likely to be smokers, but less frequently reported drinking alcohol. Blacks and Whites did not differ in their frequency of non-fatal physical assault.

Table 1. Descriptive statistics at baseline for total sample and by race.

|                      | All (\( n = 3617 \)) | Whites (\( n = 2205 \)) | Blacks (\( n = 1156 \)) |
|----------------------|----------------------|-------------------------|-------------------------|
|                      | Mean (SE) 95% CIs     | Mean (SE) 95% CIs       | Mean (SE) 95% CIs       |
| Age                  | 47.77 (0.534) 46.69–48.84 | 47.96 (0.601) 46.75–49.17 | 46.33 (0.717) 44.89–47.78 |
| Education *          | 12.53 (0.096) 12.34–12.73 | 12.69 (0.105) 12.48–12.90 | 11.37 (0.233) 10.90–11.84 |
| Income *             | 5.41 (0.093) 5.22–5.60 | 5.57 (0.101) 5.36–5.77 | 4.25 (0.183) 3.88–4.62 |
| Depressive Symptoms *| -0.03 (0.025) -0.08–0.02 | -0.07 (0.025) -0.13–0.02 | 0.28 (0.051) 0.18–0.38 |
| Chronic Medical Conditions * | 0.79 (0.028) 0.74–0.85 | 0.78 (0.031) 0.71–0.84 | 0.91 (0.052) 0.81–1.02 |
| Self-Rate Health *   | 2.30 (0.024) 2.25–2.35 | 2.26 (0.026) 2.23–2.33 | 2.43 (0.054) 2.32–2.54 |
| Body Mass Index *    | 25.54 (0.11) 25.32–25.75 | 25.34 (0.12) 25.11–25.58 | 26.94 (0.20) 26.53–27.34 |
| Gender               |                       |                         |                         |
| Male                 | 47.26 (0.012) 44.86–49.68 | 47.82 (0.013) 45.12–50.52 | 43.18 (0.022) 38.79–47.69 |
| Female               | 52.74 (0.012) 50.32–55.14 | 52.18 (0.013) 49.46–54.88 | 56.82 (0.022) 52.31–61.21 |
| Smoking *            |                       |                         |                         |
| No                   | 69.59 (0.01) 68.83–72.23 | 70.35 (0.01) 67.34–73.19 | 64.05 (0.03) 58.54–69.21 |
| Yes                  | 30.41 (0.01) 27.77–33.17 | 29.65 (0.01) 26.81–32.66 | 35.95 (0.03) 30.79–41.46 |
| Drinking *           |                       |                         |                         |
| No                   | 39.98 (0.02) 36.73–43.33 | 38.49 (0.02) 35.15–41.95 | 50.93 (0.03) 45.37–56.47 |
| Yes                  | 60.02 (0.02) 56.67–63.27 | 61.51 (0.02) 58.05–64.85 | 49.07 (0.03) 43.53–54.63 |
| History of Non-Fatal Physical Assault | | | |
| No                   | 97.38 (0.00) 96.45–98.07 | 97.42 (0.00) 96.32–98.20 | 97.06 (0.01) 95.44–98.12 |
| Yes                  | 2.62 (0.00) 1.93–3.55 | 2.58 (0.00) 1.80–3.68 | 2.94 (0.01) 1.88–4.56 |

Notes: * \( p < 0.05 \) for all comparisons between Blacks and Whites. SE—standard error; CIs—confidence intervals.
3.2. Cox Regressions in the Pooled Sample

Table 2 shows the results of seven Cox regression models in the pooled sample. We added covariates gradually to these models, with Model 1 having the least covariates and Model 7 having all the covariates. Models 2 to 7 also included an interaction term between race and history of non-fatal physical assault. In Model 1, which only controlled for demographics, history of non-fatal physical assault at baseline (hazard ratio (HR) = 2.18, 95% CIs = 1.21–3.93) was associated with higher risk of all-cause mortality over 25 years of follow-up.

Models 2 to 7 that controlled for different sets of covariates showed a main effect of history of non-fatal physical assault and a significant interaction between race and history of non-fatal physical assault, suggesting the effect of physical assault on mortality is larger for Whites than Blacks.

In Model 7, history of non-fatal physical assault at baseline was associated with higher risk of all-cause mortality (HR = 1.88, 95% CIs = 1.02–3.45) beyond all covariates, including baseline socioeconomic status, health behaviors, and health status. In this model, race showed a significant interaction with history of non-fatal physical assault at baseline (HR = 0.33, 95% CIs = 0.11–1.00), suggesting a stronger effect for Whites than Blacks (Table 2).

3.3. Cox Regressions in Whites and Blacks

Table 3 shows the results of six Cox regression models in Whites. Based on Model 1, which only controlled for age and gender, history of non-fatal physical assault was associated with higher risk of all-cause mortality for Whites (HR = 2.67, 95% CIs = 1.40–5.09). Based on Model 6, history of non-fatal physical assault at baseline was positively associated with all-cause mortality, beyond all covariates, including baseline socioeconomic status, health behaviors, and health status in Whites (HR = 1.96, 95% CIs = 1.05–3.65). For Whites, regardless of covariates, the HRs were significant and larger than 1 (Table 3).

Table 4 shows the results of six Cox regression models in Blacks. Based on Model 1, which only controlled for age and gender, history of non-fatal physical assault, was not associated with all-cause mortality for Blacks (HR = 0.67, 95% CIs = 0.27–1.65). History of non-fatal physical assault at baseline was not associated with all-cause mortality in Model 6, which controlled for all covariates, including baseline socioeconomic status, health behaviors, and health status (HR = 0.53, 95% CIs = 0.21–1.33). For Blacks, HRs were less than 1 but non-significant (Table 4).
Table 2. Results of Cox regressions between race, assault, and risk of all-cause mortality over 25 years (n = 3617).

|                  | Model 1       | Model 2       | Model 3       | Model 4       | Model 5       | Model 6       | Model 7       |
|------------------|---------------|---------------|---------------|---------------|---------------|---------------|---------------|
|                  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  |
| Race (Black)     | 1.41 (1.23–1.61) *** | 1.45 (1.27–1.65) *** | 1.20 (1.03–1.40) * | 1.14 (0.98–1.33) # | 1.13 (0.97–1.32) | 1.12 (0.96–1.31) | 1.10 (0.95–1.28) |
| Age              | 1.09 (1.09–1.10) *** | 1.09 (1.09–1.10) *** | 1.09 (1.08–1.09) *** | 1.09 (1.08–1.09) *** | 1.08 (1.08–1.09) *** | 1.08 (1.08–1.09) *** | 1.09 (1.08–1.10) *** |
| Gender (Female)  | 0.60 (0.53–0.69) *** | 0.60 (0.53–0.69) *** | 0.55 (0.48–0.64) *** | 0.56 (0.48–0.64) *** | 0.54 (0.47–0.62) *** | 0.54 (0.47–0.62) *** | 0.56 (0.48–0.65) *** |
| Education (12 years or more) | 0.81 (0.71–0.93) ** | 0.87 (0.76–1.00) # | 0.89 (0.78–1.01) # | 0.89 (0.78–1.02) # | 0.90 (0.79–1.03) # | 0.90 (0.79–1.03) # | 0.91 (0.89–1.04) *** |
| Income           | 0.92 (0.89–0.94) *** | 0.93 (0.90–0.96) *** | 1.74 (1.50–2.01) *** | 1.54 (1.32–1.79) *** | 1.51 (1.30–1.77) *** | 1.47 (1.26–1.73) *** | 1.47 (1.26–1.73) *** |
| Self-Rated Health (SRH; Poor or Fair) | - | - | - | - | - | - | - |
| Chronic medical conditions (CMC) | - | - | - | - | - | - | - |
| Body Mass Index (BMI) | - | - | - | - | - | - | - |
| Depressive Symptoms | - | - | - | - | - | - | - |
| Smoking          | - | - | - | - | - | - | - |
| Drinking         | - | - | - | - | - | - | - |
| Physical Assault | 2.18 (1.21–3.93) * | 2.55 (1.35–4.80) ** | 2.28 (1.24–4.18) ** | 2.23 (1.20–4.11) * | 2.12 (1.15–3.89) * | 2.10 (1.14–3.88) * | 1.88 (1.02–3.45) * |
| Physical Assault × Race (Black) | - | - | - | - | - | - | - |

Notes: # p < 0.1, * p < 0.05, ** p < 0.01, *** p < 0.001. HR—hazard ratio.

Table 3. Results of Cox regressions between assault and risk of all-cause mortality over 25 years among Whites (n = 2205).

|                  | Model 1       | Model 2       | Model 3       | Model 4       | Model 5       | Model 6       |
|------------------|---------------|---------------|---------------|---------------|---------------|---------------|
|                  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  | HR (95% CIs)  |
| Age              | 1.10 (1.09–1.10) *** | 1.09 (1.08–1.10) *** | 1.09 (1.08–1.10) *** | 1.09 (1.08–1.09) *** | 1.09 (1.08–1.09) *** | 1.09 (1.09–1.10) *** |
| Gender (Female)  | 0.58 (0.50–0.67) *** | 0.54 (0.46–0.63) *** | 0.54 (0.46–0.63) *** | 0.52 (0.45–0.61) *** | 0.52 (0.44–0.61) *** | 0.54 (0.45–0.63) *** |
| Education (12 years or more) | 0.79 (0.69–0.92) ** | 0.86 (0.74–1.00) # | 0.87 (0.76–1.01) # | 0.88 (0.76–1.02) # | 0.89 (0.77–1.04) # | 0.89 (0.77–1.04) # |
| Income           | 0.92 (0.89–0.94) *** | 0.93 (0.91–0.96) *** | 1.88 (1.63–2.18) *** | 1.65 (1.41–1.94) *** | 1.60 (1.36–1.89) *** | 1.55 (1.30–1.85) *** |
| Self-Rated Health (SRH; Poor or Fair) | - | - | - | - | - | - |
| Chronic medical conditions (CMC) | - | - | - | - | - | - |
| Body Mass Index (BMI) | - | - | - | - | - | - |
| Depressive Symptoms | - | - | - | - | - | - |
| Smoking          | - | - | - | - | - | - |
| Drinking         | - | - | - | - | - | - |
| Physical Assault | 2.67 (1.40–5.09) ** | 2.40 (1.29–4.46) ** | 2.36 (1.26–4.41) ** | 2.23 (1.20–4.14) * | 2.20 (1.17–4.12) * | 1.96 (1.05–3.65) *** |

Notes: # p < 0.1, * p < 0.05, ** p < 0.01, *** p < 0.001.
Table 4. Results of Cox regressions between assault and risk of all-cause mortality over 25 years among Blacks (n = 1156).

|                     | Model 7          | Model 8          | Model 9          | Model 10         | Model 11         | Model 12         |
|---------------------|------------------|------------------|------------------|------------------|------------------|------------------|
| HR (95% CIs)        | HR (95% CIs)     | HR (95% CIs)     | HR (95% CIs)     | HR (95% CIs)     | HR (95% CIs)     | HR (95% CIs)     |
| Age                 | 1.08 (1.07–1.08)*** | 1.07 (1.06–1.08)*** | 1.07 (1.06–1.08)*** | 1.06 (1.05–1.07)*** | 1.06 (1.05–1.07)*** | 1.07 (1.06–1.08)*** |
| Gender (Female)     | 0.74 (0.60–0.91)** | 0.65 (0.53–0.81)*** | 0.65 (0.53–0.80)*** | 0.64 (0.51–0.81)*** | 0.65 (0.52–0.81)*** | 0.73 (0.57–0.92)** |
| Education (12 years or more) | 0.90 (0.70–1.17) | 0.92 (0.71–1.20) | 0.94 (0.72–1.24) | 0.93 (0.71–1.22) | 0.94 (0.73–1.22) | 0.94 (0.73–1.22) |
| Income              | 0.91 (0.86–0.96)*** | 0.92 (0.87–0.97) ** | 0.92 (0.87–0.97) ** | 0.91 (0.86–0.96)*** | 0.91 (0.86–0.96)*** | 0.91 (0.86–0.96)*** |
| Self-Rated Health (SRH; Poor or Fair) | 1.19 (0.98–1.44) $^|$ | 1.09 (0.89–1.34) | 1.12 (0.92–1.36) | 1.11 (0.92–1.34) | 1.12 (0.91–1.37) | 1.13 (0.92–1.37) |
| Chronic medical conditions (CMC) | 1.12 (1.01–1.23) * | 1.13 (1.02–1.25) * | 1.13 (1.01–1.25) * | 1.13 (1.01–1.25) * | 1.00 (0.98–1.02) | 1.00 (0.98–1.02) |
| Body Mass Index (BMI) | 1.00 (0.98–1.01) | 1.00 (0.98–1.01) | 0.77 (0.61–0.97) * | 0.75 (0.59–0.95) * | 1.56 (1.29–1.90)*** | 1.56 (1.29–1.90)*** |
| Depressive Symptoms | 0.67 (0.27–1.65) | 0.60 (0.25–1.41) | 0.59 (0.25–1.39) | 0.61 (0.26–1.41) | 0.62 (0.27–1.42) | 0.53 (0.21–1.33) |

Notes: $^|$ p < 0.1, * p < 0.05, ** p < 0.01, *** p < 0.001.
4. Discussion

This study aimed to investigate Black–White differences in the association between history of non-fatal physical assault and all-cause mortality over a 25-year period in the US. Although history of non-fatal physical assault was associated with an increased risk for mortality in the pooled sample, race modified the effect of non-fatal physical assault history on all-cause mortality. This association was present for Whites but not Blacks.

The result that history of non-fatal physical assault was a risk factor for premature mortality overall and for Whites is supported by the literature on the effects of stress and trauma on mortality [11,33–35]. Stress increases risk of health problems, such as excess body fat and obesity [36–44]. Although the exact mechanism of the effect of stress on obesity is still unknown [38], long-term stress induces long-lasting changes to the stress reaction through physiological pathways that have well-established roles in obesity and metabolic syndrome [38–44]. Several studies showed that chronic psychosocial stress is a risk factor for obesity and causes excess body-fat accumulation, especially in the abdomen [43–46]. Stress also increases high-risk behaviors, such as drinking, substance use, and suicide [47–51]. Finally, various types of stress also increase risk of mental health problems, such as depression and anxiety [16,52–54].

Our finding regarding larger effects of history of non-fatal physical assault on risk of mortality in Whites compared to Blacks agrees with other research showing risk and protective factors have stronger effects for Whites than Blacks [20,55], a pattern which is frequently shown for physical and mental health outcomes ([9,11–19,56–61]; see References [20,55] for a review). Stronger effects of low education [17], unemployment [12], poor neighborhood quality [11], depression [16,18,19,62,63], hostility and anger [64], low sense of self efficacy [14], and low perception of control over life [65] were shown for Whites than Blacks.

Future research should test whether weaker effects in Blacks are because they are more frequently exposed to multiple forms of assaults than Whites throughout their life course [23,54]. Due to poverty and racial segregation, Blacks have a higher tendency to live in dangerous neighborhoods, which increases their risk of exposure to a wide range of experiences such as violence and assault [26,27]. Chronic medical conditions also affect more Blacks than Whites [9]. As a result, Blacks may develop more efficient coping strategies compared to Whites, especially coping strategies that can be used to deal with stressful conditions in daily life [28]. It is still unknown whether racial differences in stress management and coping strategies explain the racial differences in physical and mental health consequences of social adversities that exist between Blacks and Whites [26–28].

Research should test whether these racial differences are due to Blacks’ habituation to adversities or not. Blacks may have developed a systemic resilience to adversities due to chronic exposure to various types of social and economic adversities. Since Blacks live in environments with adverse economic and social conditions [22,26,65], another adversity is everything but new to them. Blacks’ systemic resilience may be an adaptive response to their living conditions.

The above argument contrasts the traditional view that Blacks and other minority groups are more “vulnerable” than Whites to new risk factors since they already experience a wide range of adversities. Multiple Jeopardy hypothesis [16,66] suggests that cumulative exposures increase vulnerability of minority populations, including Blacks. The same argument is the basis of the Multiple Disadvantage theory [67,68]. Our results do not support these theories. In contrast, we argue that cumulative exposure to social stressors generates a systematic resilience, not susceptibility, among Blacks. This is in line with research showing stronger links between stressful life events and health problems for Whites compared to Blacks [20,54]. Habituation may allow Blacks to better cope with additional stressors when needed [54]. Frequent and long-term exposure to low-severity, stressful life events may result in positive outcomes such as resilience. Life events better predict post-traumatic growth among Blacks compared to Whites [54].

These findings should not reduce attention from reducing adversities in the lives of Blacks. There is still a need for violence prevention, enhancement of safety, and assault prevention in majority Black
communities. Similarly, we still advocate for provision of healthcare for Blacks who experience trauma and assault. At the same time, public health officials and policy makers should be aware that there might be racial differences in resilience and vulnerability to social stressors [20]; thus, racial groups’ health status may respond differently to social policies and programs that reduce exposures. Policies and programs that enhance safety and prevent assault would generate weaker health returns in the Black communities compared to high-socioeconomic, predominantly White neighborhoods.

We do not believe that innate racial differences in vulnerability and resilience exist. We argue that racial differences in vulnerability and resilience are the result of racial differences in life conditions and life history. In this view, high prevalence of adversities may explain Blacks’ higher resilience to social and economic adversities compared to Whites. Given the complexity of interactions between race, socioeconomic status, social and behavioral risk factors, and health, researchers may consider race as an effect modifier in development of conceptual models that address health disparities [69].

4.1. Implications

The current study may have some implication for policy and practice. A certain risk factor such as physical assault may have differential health effects across sub-populations, and researchers, clinicians, and policy makers should not assume that a sub-population’s health is similar if the exposure profile is similar. Independent of race, ethnicity, class, and location, programs and policies should reduce exposure to violence, and such investments may increase life expectancy of overall populations. So, the results do not suggest that we should not invest on reducing exposure to environmental stressors such as assault in Black communities.

4.2. Limitations

Our study is subject to a few limitations that should be considered before any interpretations. Firstly, we measured history of trauma, CMC, SRH, BMI, and behaviors at baseline. These constructs and conditions are subject to change over time. Future research should model them as time-varying covariates. Secondly, we did not control for type of assault, need for healthcare services, or medical consequences of trauma. Thirdly, we could not control for a wide range of confounders, such as function, daily activity, insurance, and access to medical care.

5. Conclusions

In conclusion, our findings support that race does modify the long-term risk of mortality associated with physical assault. Physical assault better predicts mortality risk among Whites rather than Blacks. Future research should explore potential mechanisms behind group differences in the effect of exposures to social adversities on physical and mental health outcomes.

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Informed Consent: All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all participants included in the study.

Compliance with Ethical Standards: The University of Michigan Institutional review board (IRB) approved the study protocol. Informed consent was obtained from all individual participants included in the study. All procedures performed in studies involving human participants were in accordance with the ethical standards.
of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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