Res Cardiovasc Med. 2017 January; 6(1):e34029. doi: 10.5812/cardiovascmed.34029.

Published online 2016 June 29. **Research Article**

Hostility, Anger, and Cardiovascular Mortality Among Blacks and Whites

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Received 2015 October 24; **Revised** 2016 January 09; **Accepted** 2016 January 15.

Abstract

Background: Despite the well-known impact of baseline hostility and anger on subsequent cardiovascular mortality, few studies have tested whether predictive role of hostility and anger on mortality varies as a function of race and gender.

Objectives: Current study explored role of race and gender in modifying the effects of baseline hostility and anger on cardiovascular mortality in a nationally representative sample in U.S.

Materials and Methods: We used data from the Americans' changing lives study, a nationally representative longitudinal cohort of U.S. adults. The study followed 1,593 Blacks or Whites for 10 years from 2001 to 2011. Independent variables were baseline hostility and anger (anger-in, and anger-out), measured at 2001, using 4 item Cook-Medley cynical hostility scale and Spielberger Anger Expression scales, respectively. Dependent variable was time to death due to cardiovascular disease since 2001. Covariates were baseline socio-demographics (age and education), behaviors (smoking and drinking), and health (number of chronic medical conditions, self-rated health, and depressive symptoms) measured at 2001. We used Cox proportional hazard models in the pooled sample and specific to race, in the absence and presence of health variables.

Results: In the pooled sample, baseline hostility and anger-out predicted cardiovascular mortality in the next 10 years. We found significant interactions between race and baseline hostility and anger-in on cardiovascular mortality, suggesting that these associations are stronger for Whites than Blacks. Race did not interact with baseline anger-out on cardiovascular mortality. Gender also did not have any interactions with baseline hostility, anger-in, or anger-out on cardiovascular mortality.

Conclusions: Hostility and anger-in better predict cardiovascular mortality among Blacks than Whites in the United States. Black - White difference in the associations of hostility and anger with cardiovascular mortality suggest these factors may have some role in shaping health disparities across racial groups.

Keywords: Ethnic Groups, African Americans, hostility, anger, Mortality, Cardiovascular Mortality

1. Background

Hostility and anger are negative emotional and cognitive traits directed toward the self, others, and the environment (1-3). As disease-prone personality traits (4), hostility and anger have been associated with a wide range of undesired health outcomes (5) including but not limited to cardiovascular disease (6). Hostility, the feeling of anger (anger-in) and the expression of anger (anger-out) have similar and specific health effects (1-3).

There is some evidence suggesting that age (7), race (8, 9), socio-economic status (10-12), culture (13-17) and psychiatric disorders (18) may alter the health effects of hostility and anger. In line with the inconsistencies in the literature on the health effects of hostility and anger across studies, it has been hypothesized that populations may differ in susceptibility to the effect of hostility and anger (19). Hostility may also interact with other traits and type of stressor, which suggests its health effects may depend on context (20). One example is the systematic review in 2009 by Chida and Steptoe which suggested gender differences in the harmful effect of anger and hostility on coronary heart disease events in the healthy populations, with such effects being greater for men than women (1). However, a large body of research has enrolled one gender (21, 22), making any conclusion about moderating effect of gender on of the hostility - health link difficult. Whether the same link depends on race is also unknown, particularly because previous studies on this topic have mostly enrolled Whites, with minimal information available for Blacks (22, 23).

2. Objectives

As there are some evidence suggesting that race (24, 25) and gender (1) may modify the associations between hostility, anger and mortality, we conducted this study to explore racial and gender differences in the predictive role of hostility and anger on cardiovascular mortality, using a nationally representative sample of adults that provides gen-

eralizable results to the U.S. population.

3. Materials and Methods

3.1. Design and Setting

The Americans' changing lives (ACL), is a nationally-representative U.S. study of U.S. adults, 1986 - 2011. More information on the sampling and data collection has been published elsewhere (26-28).

3.2. Sampling and Participants

The ACL has used a stratified multistage probability sample of U.S. adults. The original study enrolled 3,617 adults who were 25 years or older and were living in the continental U.S. in 1986. All participants were non-institutionalized respondents (representing 70% of sampled households and 68% of sample individuals at baseline). The study oversampled older adults (age > 60) and Blacks. Wave 1 included 70% of sampled households and 68% of sample individuals. Current analysis is limited to Whites and Blacks who participated in Wave 4 (2001) (analytic N = 1,593, composed of 1185 White and 408 Blacks).

3.3. Process and Measures

Age, income, and all baseline health data, including number of chronic medical conditions, self-rated health, and depressive symptoms, were collected in Wave 4 (2001). All these variables were used to control for potential confounding. The outcome was time to the event of all-cause and cardiovascular mortality over 10 years from 2001 to the end of follow up in 2011. Death certificates or the national death index (NDI) were used to assess cause and date of death.

3.4. Socio-Demographics

Demographic characteristics included gender (a dichotomous variable with male as the referent category), and age (a continuous variable). Socio-economic characteristics included baseline education (years of schooling) and income (a continuous variable), both collected in 1986. Race was the moderator, defined as Black versus White (White respondents as the referent category).

3.5. Number of Chronic Medical Conditions

Baseline chronic medical conditions were measured using self-reported data. All participants were asked whether a health care provider had ever told them they had each of seven focal conditions including hypertension, diabetes, chronic lung disease, heart disease, stroke, cancer, and arthritis. Responses were dichotomous, and summed to result in a score ranging from 0 to 7. A more detailed description of the chronic medical condition measurement is available elsewhere (27, 28).

3.6. SRH

Respondents were asked to classify their self-rated health as excellent, very good, good, fair, or poor. SRH was operationalized in the following two ways: 1, as a dichotomous measure; and 2, as a continuous score. For the first approach we collapsed this five-category scale into two categories (fair/poor vs. excellent/very good/good), a cutoff point that is common in the literature. This measure has shown high test-retest reliability and validity, when considering its predictive power for mortality and other health outcomes (29, 30), (1 = excellent and 5 =poor).

3.7. Depressive Symptoms

Depressive symptoms were measured with 11 items from the center for epidemiological studies-depression scale (CES-D) (31). CESD items measure the extent to which respondents felt depressed, happy, lonely, or sad; that everything was an effort; that their sleep was restless; that people were unfriendly; that they did not feel like eating; that people dislike them; that they could not get going; and that they enjoyed life. Positively-worded items were reverse-coded. This abbreviated CES-D scale has shown acceptable reliability and a similar factor structure compared to the original version (32-34). Item responses were 1 to 3, resulting in a continuous measure of depressive symptoms, with a potential range from 11 to 33. Higher scores indicated greater severity of depressive symptoms.

3.8. Hostility

We used a 4 item Cook-Medley cynical hostility scale to measure hostility (35, 36). The items included most people inwardly dislike putting themselves out to help other people, most people will use somewhat unfair means to gain a profit or an advantage rather than lose it, I think most people would lie in order to get ahead, and I commonly wonder what hidden reasons another person may have for doing something nice for me. These items reflect the cynicism component of hostility, not the anger or aggressive behavior components. This is particularly important because Barefoot et al. showed that cynicism, hostile affect, and aggressive responding subsets were predictive of survival whereas other subsets were not (36). Response items included strongly agree (1), somewhat agree (2), somewhat disagree (3), and strongly disagree (4). We used the average of the items, ranging from 1 to 4, with higher scores indicating higher hostility. (Cronbach alpha = 0.709 for all, 0.716 for Whites, and 0.662 for Blacks)

3.9. Anger-in and Anger-out

We used a brief version of the Spielberger anger expression scales (37) to measure anger traits. The Spielberger

anger expression scale provides measures of self-reported anger expression style: inward expression (anger-in), outward expression (anger-out), and control (anger-control) of anger. Previous research indicates that these measures are valid and reliable (38) and predict cardiovascular outcomes (39). Items that reflect anger-in the subscale included when I am feeling angry or mad, I withdraw from people; when I am feeling angry or mad, I am irritated more than people are aware; and when I am feeling angry or mad, I am angrier than I am willing to admit (Cronbach alpha = 0.681 for all, 0.669 for Whites, and 0.708 for Blacks).

The following three items reflected anger-out domain: when I am feeling angry or mad, I argue with others; when I am feeling angry or mad, I strike out at whatever infuriates me; and when I am feeling angry or mad, I say nasty things. Item responses included never (1), sometimes (2), often (3), and almost always (4). We used the average of the items, ranging from 1 to 4, with higher score indicating higher hostility. (Cronbach alpha = 0.663 for all, 0.672 for Whites, and 0.649 for Blacks).

3.10. Mortality

Data on mortality were extracted from death certificates or NDI. Overall 383 deceased participants were detected, while 1210 individuals survived. The information derived from the death certificates or NDI included primary cause of death and underlying causes of death, as well as the date of death. In the U.S., a death certificate is filled out by a doctor as soon as possible after a person is deceased. In the first step, we grouped the primary causes of death into 18 different categories based on ICD codes. Then we categorized deaths due to all-causes compared to those due to cardiovascular or others.

3.11. Ethics

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. University of Michigan Institutional review board (IRB) approved the study protocol.

3.12. Statistical Analysis

As ACL has used a complex sample design, we used Stata-13 to calculate design based standard errors using sampling and non-response weights. Taylor series linearization was used to estimate standard errors. Survey linear and Cox regressions on sub-populations were used for data analysis. P values less than 0.05 were considered statistically significant. To test whether age, gender, education, smoking, drinking, number of medical conditions, self-rated health, and depressive symptoms are associated with hostility, anger-in, and anger-out, we used linear regression models in the pooled sample, and also specific to race.

To assess the effects of hostility, anger-in, and anger-out on mortality, we used a series of Cox proportional hazard models in the pooled sample, and also specific to race. Independent variables were baseline hostility, anger-in, and anger-out measured at 2001. Dependent variables were time to death for cardiovascular causes between 2001 and 2011. Covariates included age, education, smoking, drinking, number of medical conditions, self-rated health, and depressive symptoms measured at 2001. This strategy was taken to minimize risk of confounding, as demographic, socio-economic, and health status are correlated with race, hostility, anger, and cardiovascular conditions. Moderators included gender and race. First, we ran models without interactions to test the main effects on time to death over up to 10 years of follow up. In the next step, we entered the interaction terms to test the multiplicative effects of race and gender with baseline hostility, anger-in, and anger-out on mortality outcomes, while covariates and all main effects were controlled. At the last step, we ran race specific models. Hazard ratios and standardized regression coefficients with their Standard Errors (SE) are reported.

4. Results

4.1. Descriptive Statistics

Participants included 1593 individuals who were followed for 10 years, including 1,185 Whites and 408 Blacks. Table 1 presents descriptive statistics for all variables used in the analysis, by race and overall in the pooled sample. Blacks had significantly higher hostility and anger-in. Blacks had worse physical and mental health, measured by number of medical conditions, SRH, and depressive symptoms. Blacks also had lower age and education than Whites; however, age and gender were not significantly different across race groups. Cardiovascular death occurred in 156 cases.

4.2. Hostility and Mortality

Table 2 summarizes the results of Cox models in the pooled sample and also based on race on the effects of hostility on cardiovascular mortality. Models are reported in the absence and presence of interactions terms, and also physical and mental health. In this tabulation, hostility predicts cardiovascular mortality in Model 1, in the

Table 1. Descriptive Statistics for the Analytic Sample, Stratified by Race and Overall

Descriptive Statistics	Whites		Blacks		All	
	Mean (SE)	95% CI	Mean (SE)	95% CI	Mean (SE)	95% CI
Socioeconomic status						
Age	42.35 (0.46)	41.43 - 43.27	41.63 (.81)	39.99 - 43.28	42.29 (.43)	41.43 - 43.16
Education[a]	13.29 (0.10)	13.09 - 13.49	12.47 (0.23)	12.00 - 12.94	13.22 (0.09)	13.04 - 13.40
Health behaviors						
Smoking[a]	15.80 (0.01)	13.69 - 18.16	18.55 (0.02)	13.90 - 24.31	16.03 (0.01)	14.01 - 18.28
Drinking[a]	62.76 (0.02)	59.03 - 66.35	43.23 (0.03)	37.25 - 49.43	61.10 (0.02)	57.57 - 64.52
Health						
CMC[a]	1.19 (0.04)	1.11 - 1.27	1.34 (0.07)	1.20 - 1.47	1.20 (0.04)	1.12 - 1.28
SRH[a]	2.38 (0.03)	2.32 - 2.45	2.71 (0.05)	2.60 - 2.82	2.41 (0.031)	2.35 - 2.48
CESD[a]	-.32 (0.03)	-0.38 - 0.26	-.01 (0.06)	-.012 - 0.10	-.29 (0.03)	-.035 - 0.24
Traits						
Hostility[a]	2.36 (0.03)	2.30 - 2.42	2.71 (0.04)	2.62 - 2.80	2.39 (0.02)	2.34 - 2.44
Anger-in[a]	1.96 (0.02)	1.90 - 2.00	2.05 (0.04)	1.96 - 2.14	1.96 (0.02)	1.92 - 2.00
Anger-out	1.49 (0.02)	1.44 - 1.52	1.42 (0.02)	1.37 - 1.47	1.48 (0.02)	1.44 - 1.52
Gender						
Male	46.47 (0.02)	43.12 - 49.84	41.28 (0.03)	34.75 - 48.15	46.02 (0.01)	42.87 - 49.22
Female	53.53 (0.02)	50.16 - 56.87	58.71 (0.03)	51.85 - 65.25	53.97 (0.01)	50.78 - 57.13
Mortality						
Cardiovascular mortality[a]	5.88 (0.01)	4.80 - 7.16	7.56 (0.02)	4.80 - 11.70	6.02 (0.01)	4.96 - 7.28

Abbreviation: SRH, is entered as a continuous measure; CMC, chronic medical conditions; CES-D, center for epidemiologic studies depression.
[a] $P < 0.05$ for Blacks versus Whites.

pooled sample. Model 2 also shows significant interaction between race and baseline hostility on cardiovascular mortality. This finding suggests that the effect of baseline hostility cardiovascular mortality is stronger for Whites than Blacks, with socioeconomic factors controlled. The interaction term between baseline hostility and race on cardiovascular death stays significant, when health variables are in the model, suggesting that hostility is a stronger predictor of cardiovascular mortality for Whites than Blacks, net of socioeconomic and health status. In race specific models, hostility was a predictor of cardiovascular mortality for Whites but not Blacks.

4.3. Anger-in and Mortality

Table 3 summarizes the results of Cox models in the pooled sample and also based on race on the effects of anger-in on cardiovascular mortality. Models are reported in the absence and presence of interactions terms, and also physical and mental health. In this tabulation, anger-in does not predict cardiovascular mortality in Model 1, in the pooled sample. Model 2, however, shows significant interaction between race and baseline anger-in on cardiovascular mortality, suggesting that that the effect of baseline anger-in on cardiovascular mortality is stronger for Whites than Blacks, with socioeconomic factors controlled. The interaction term between baseline anger-in and race on cardiovascular death becomes marginally significant when health factors are controlled for as an outcome, suggesting that health status may partially explain why anger-in is a stronger predictor for Whites than Blacks.

4.4. Anger-out and Mortality

Table 4 summarizes the results of Cox models in the pooled sample and also based on race on the effects of anger-out on cardiovascular mortality. Models are reported in the absence and presence of interactions terms, and also physical and mental health. In this tabulation, anger-out predicts cardiovascular mortality in Model 1, in the pooled sample. Model 2 does not show any significant interaction between race and baseline anger-out on cardiovascular mortality.

5. Discussion

According to our findings, hostility and anger-in differently predict cardiovascular mortality among Whites and Blacks, with stronger effects for Whites than Blacks. Race, however, did not interact with anger-out on subsequent cardiovascular mortality. Gender also did not interact with baseline hostility or anger on cardiovascular mortality.

Existing literature on the effects of hostility and anger on cardiovascular mortality is mixed. For instance, a 15 year follow up study of 3850 and 4083 male and female individuals shows that trait angers, anger-in, and anger-out are

Table 2. The Association Between Baseline Hostility and Subsequent Cardiovascular Mortality Based on Race Using Cox Regression

Characteristic	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)
		All			Whites			Blacks	
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Race (Black)	1.34 (0.34)	18.41 (17.24)[a]	12.20 (12.02)[b]						
Age	1.10 (0.00)[c]	1.11 (0.00)[c]	1.11 (0.00)[c]	1.10 (0.00)[c]	1.12 (0.00)[c]	1.12 (0.01)[c]	1.07 (0.01)[c]	1.05 (0.02)[a]	1.05 (0.02)[a]
Gender (Female)	0.86 (0.16)	1.02 (0.82)	1.00 (0.84)	0.88 (0.18)	0.84 (0.18)	0.77 (0.17)	0.74 (0.30)	0.61 (0.23)	0.54 (0.20)
Education		0.56 (0.03)	0.96 (0.34)		0.94 (0.04)	0.95 (0.04)		0.96 (0.04)	0.95 (0.47)
Smoking		3.52 (0.91)[c]	3.43 (0.86)[c]		4.40 (1.24)[c]	4.23 (1.15)[c]		0.55 (0.46)	0.55 (0.44)
Drinking		0.76 (0.17)	0.84 (0.20)		0.81 (0.19)	0.90 (0.22)		0.42 (0.24)	0.48 (0.25)
Hostility	1.83 (0.30)[c]	2.00 (0.55)[b]	1.20 (0.09)[b]	2.08 (0.38)[c]	1.90 (0.38)[a]	1.22 (0.10)[b]	0. (0.17)	0.69 (0.17)	1.06 (0.13)
CMC			1.24 (0.18)			1.20 (0.19)			1.42 (0.35)
SRH			1.03 (0.08)			1.07 (0.08)			0.71 (0.11)[b]
CES-D			1.81 (0.54)[d]			1.70 (0.35)[b]			0.76 (0.16)
Hostility × Black		0.37 (0.12)[a]	0.42 (0.15)[b]						
Hostility × Female		0.93 (0.26)	0.91 (0.28)						

Abbreviations: SRH, self-rated health; CMC, chronic medical conditions; CES-D, center for epidemiologic studies depression.
[a] P < 0.01.
[b] P < 0.05.
[c] P < 0.001.
[d] P < 0.1.

Table 3. The Association Between Baseline Anger-in and Subsequent Cardiovascular Mortality Based on Race Using Cox Regression

Characteristic	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)
		All			Whites			Blacks	
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Race (Black)	1.65 (0.38)[a]	3.91 (2.15)[a]	3.20 (1.83)[a]						
Age	1.10 (0.00)[b]	1.11 (0.01)[b]	1.11 (0.01)[b]	1.11 (0.01)[b]	1.12 (0.01)[b]	1.12 (0.01)[b]	1.06 (0.01)[b]	1.05 (0.02)[c]	1.05 (0.02)[c]
Gender (Female)	0.77 (0.15)	0.55 (0.29)	0.51 (0.27)	0.76 (0.16)	0.75 (0.15)	0.70 (0.15)	0.74 (0.31)	0.61 (0.23)	0.54 (0.20)
Education		0.93 (0.03)[d]	0.95 (0.34)		0.91 (0.03)[a]	0.92 (0.04)[d]		0.98 (0.04)	0.97 (0.04)
Smoking		3.44 (0.78)[b]	3.24 (0.74)[b]		4.24 (1.05)[b]	3.95 (0.99)[b]		0.62 (0.50)	0.59 (0.47)
Drinking		0.67 (0.14)[d]	0.76 (0.20)		0.73 (0.16)	0.83 (0.20)		0.41 (0.23)	0.48 (0.25)
Anger-in	1.07 (0.13)	1.04 (0.25)	1.23 (0.09)[a]	1.15 (0.15)	1.17 (0.14)	1.25 (0.10)[c]	0.61 (0.16)[d]	0.62 (0.17)[d]	1.08 (0.13)
CMC			1.28 (0.18)[d]			1.23 (0.19)			1.40 (0.32)
SRH			1.06 (0.08)			1.11 (0.08)			0.73 (0.13)[d]
CES-D			0.94 (0.25)			1.06 (0.14)			0.69 (0.21)
Anger-in × Black		0.54 (0.15)[a]	0.59 (0.17)[d]						
Anger-in × Female		1.18 (0.32)	1.19 (0.35)						

Abbreviations: SRH, self-rated health; CMC, chronic medical conditions; CES-D, center for epidemiologic studies depression.
[a] P < 0.05.
[b] P < 0.00.
[c] P < 0.01.
[d] P < 0.1.

not related to cardiovascular or coronary outcomes, even thought anger control predicted risk of cardiovascular incidence (40). In another study, hostility was not related to atherosclerotic outcomes (22). A meta-analysis also confirmed inconsistencies in the previous literature on the effects of anger and hostility on blood pressure or heart rate reactivity in response to stress (20). Systematic review by Chida and Steptoe, however, documented that anger and hostility increase cardiovascular events in the healthy population (combined hazard ratio [HR]: 1.19) and worsened the prognosis of existing heart disease (HR: 1.24) (1).

Culture may explain moderating effect of race on the health effects of hostility and anger (13-17). For instance, using a large probability samples of Japanese and Americans, a study found a link between greater expression of anger and worse pro-inflammatory markers (interleukin-6 and C-reactive protein) and indices of cardiovascular malfunction (systolic blood pressure and ratio of total to HDL cholesterol) for Americans. However, these association were mostly reversed for Japanese, among whom

Table 4. The Association Between Baseline Anger-out and Subsequent Cardiovascular Mortality Based on Race Using Cox Regression

Characteristic	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)	HR (SE)
		All			Whites			Blacks	
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Race (Black)	1.66 (0.38)[a]	1.20 (0.76)	0.98 (0.70)						
Age	1.11 (0.00)[b]	1.11 (0.00)[b]	1.11 (0.01)[b]	1.11 (0.01)[b]	1.12 (0.01)[b]	1.12 (0.01)[b]	1.07 (0.01)[b]	1.06 (0.18)[c]	1.05 (0.02)[c]
Gender (Female)	0.78 (0.15)	0.80 (0.42)	0.69 (0.37)	0.77 (0.15)	0.76 (0.15)	0.71 (0.15)	0.80 (0.34)	0.66 (0.25)	0.60 (0.21)
Education		0.93 (0.03)[a]	0.94 (0.34)		0.90 (0.04)[a]	0.92 (0.04)[d]		0.98 (0.5)	0.95 (0.04)
Smoking		3.32 (0.78)[b]	3.17 (0.75)[b]		4.20 (1.07)[b]	3.96 (1.02)[b]		0.56 (0.45)	0.53 (0.42)
Drinking		0.67 (0.14)[d]	0.76 (0.17)		0.71 (0.16)	0.82 (0.20)		0.41 (0.23)	0.47 (0.24)
Anger-out	1.31 (0.22)[b]	1.36 (0.33)	1.23 (0.09)[c]	1.33 (0.24)	1.37 (0.25)[d]	1.25 (0.10)[c]	1.30 (0.47)	1.31 (0.50)	1.08 (0.14)
CMC			1.28 (0.18)[d]			1.23 (0.19)			1.42 (0.29)[d]
SRH			1.05 (0.08)			1.11 (0.09)			0.66 (0.10)[a]
CES-D			1.12 (0.30)			1.14 (0.24)			1.47 (0.51)
Anger-out × Black		1.02 (0.41)	1.16 (0.53)						
Anger-out × Female		0.96 (0.35)	1.02 (0.38)						

Abbreviations: SRH;, Self rated health; CMC, chronic medical conditions; CES-D; center for epidemiologic studies depression.
[a] $P < 0.05$.
[b] $P < 0.001$.
[c] $P < 0.01$.
[d] $P < 0.1$.

greater expression of anger predicted reduced risk (16). This may be because the direction and magnitude of the associations between social status and anger may vary across populations and cultures (17). Role of culture, context, and socio-economic status should be more deeply investigated in the future by changing the link between hostility, anger, and health. Thomas and Gonzalez-Prendes developed a conceptual model that discusses powerlessness as a unique source of anger and hostility among Blacks, particularly Black women. They argued that Black and individuals of a lower social class are at a higher risk of experiencing feelings of powerlessness associated with disparities and discrimination, which may have implications for the health effect of hostility and anger (41).

Results of biological studies may also provide an explanation for the variation of the effects of hostility and anger on health across populations (42). Hostility is closely linked to low central nervous system serotonin levels (43, 44) which predicts peripheral sensitivity to insulin and metabolic risk (45). Low central serotonergic responsivity is associated with greater body mass index, higher concentrations of triglycerides, glucose, and insulin, higher systolic and diastolic blood pressure, greater insulin resistance, and less physical activity (46-48). The link between hostility and central nervous system serotonin levels (43, 44) and also the link between central nervous system serotonin levels and cardiovascular risk depend on race, gender, and age (21, 44, 49). Some recent studies have documented a stronger association between hostility and fasting glucose, glucose dysregulation during an intravenous glucose tolerance test among Black women compared to Black men and White men and women (50-53). The mechanisms behind the race and gender specific effects of hostility or its biological proxies have not yet been identified and warrant additional research (50).

Hostility is also associated with greater systemic inflammation (19). The TNF-α, Il-6, and CRP are vulnerable to psychological and behavioral factors and may be involved in the effects of hostility (CMHo) on cardiovascular risk (19). Race and sex may, however, alter the associations between genes [5-hydroxyindoleacetic acid (5HIAA) and genotype of a functional polymorphism of the monoamine oxidase A gene promoter (MAOA-uVNTR)], hostility, and cardiovascular risk (21). Hostility may interact with sex on Il-6 and TNF-α, hostility with age on hsCRP, Il-6, and TNF-α. Hostility was positively related to TNF-α in women but not men (19). Hostility and anger also increase platelet activation and reactivity (54-56), which may be due to changes in the serotonergic and adrenergic function (55, 57). The effect of hostility on platelet activation may also depend on certain conditions related to type of stressor and presence of heart disease (54, 55).

The links between race, gender, hostility, anger, physical health, mental health, and cardiovascular mortality in the United States are complex and require more research. Future research is needed on mechanisms behind the differential effect of hostility and anger on the cardiovascular mortality of Blacks and Whites. It is not known whether culture, socioeconomic status, attitudes, beliefs, use or access to health care, and health profile explain such Black -

White differences in the effect of hostility and anger on cardiovascular mortality.

Our study had a number of limitations. First and foremost, hostility and anger are subject to change; however, we did not model their change over time. Second, the study did not control for baseline cardiovascular and psychiatric disorders. Third, the reliability of our measures was not identical among Blacks and Whites. However, using a nationally representative sample was a unique strength of this study. Unequal sample size of Blacks and Whites should also be considered before interpretation of the results. Another potential source of bias may be differential health status of Blacks and Whites at baseline. Despite that the reliability of our measures were similar among Whites and Blacks, racial differences may exist in validity of our scale that was used to measure hostility and anger. To minimize risk of confounding, we controlled for demographic, socio-economic, and health status.

To conclude, hostility and anger differently predict cardiovascular mortality among Blacks and Whites. Our findings may have implications for understanding the Black – White health disparities and also the Black-White health paradox (28, 58, 59).

Acknowledgments

Author would like to thank Dr. Redford Williams comments on the draft of the paper. Shervin Assari is supported by the Heinz C. Prechter Bipolar research fund and the Richard Tam foundation at the University of Michigan depression center.

Footnotes

Conflict of Interest: All authors declare that they have no conflicts of interest.

Funding/Support: The Americans' changing lives (ACL) study was supported by Grant # AG018418 from the national institute on aging (DHHS/NIH), and per the NIH Public access policy requires that peer-reviewed research publications generated with NIH support are made available to the public through PubMed central. NIH is not responsible for the data collection or analyses represented in this article. The ACL study was conducted by the institute of social research, University of Michigan.

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