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Educational Status, Anger, and Inflammation in the MIDUS National Sample: Does Race Matter?

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Abstract

Background Racial differences in anger frequency and expression styles have been found. Further, African Americans receive fewer health benefits from higher education than Whites.

Purpose This study seeks to investigate racial differences in how anger moderates the association between education and inflammation.

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Methods Midlife in the US participants ($n=1,200$; 43.0 % male; 18.5 % African American) provided education and anger data via survey assessments. Interleukin-6 (IL-6) and fibrinogen were determined from a fasting blood sample following an overnight clinic visit.

Results African Americans reported higher anger-out, IL-6, and fibrinogen and lower anger-control than Whites. Anger-out predicted higher IL-6 and fibrinogen among African Americans with higher education; whereas, trait anger and anger-out predicted lower fibrinogen among Whites with higher education. Anger-out marginally predicted higher IL-6 in less educated Whites.

Conclusions Findings underscore racial differences in the benefits and consequences of educational attainment, and how social inequities and anger are manifested in inflammatory physiology.

Keywords Anger · Race differences · Educational attainment · Inflammation

The pernicious health consequences of anger are widely recognized. Prospective studies have shown that high levels of anger and poorly controlled anger predict risk for hypertension, coronary heart disease, and stroke [1–3]. However, less research has addressed the physiological mechanisms underlying this association between anger and disease. Inflammation is a primary candidate pathway through which anger can affect disease processes. Inflammatory and coagulation markers, such as the pro-inflammatory cytokine, interleukin-6 (IL-6), and the clotting factor, fibrinogen, respond to the psychosocial environment and when elevated, contribute to the etiology and pathogenesis of many chronic conditions, including cardiovascular disease and diabetes [4–6]. Anger and hostility are associated with higher circulating levels of

inflammatory markers and clotting factors [7–13] and cytokine release from cells when stimulated and cultured in vitro [14].

Missing in the prior literature on anger and inflammation is a consideration of the broader societal context in which these associations occur. Anger is a common emotional reaction to inequality; those of lower socioeconomic status (SES) report more frequent angry experiences, more frequent outward expressions of anger (anger-out), and more frequent suppression of anger (anger-in), while those in higher SES strata report they can better manage their anger (anger-control) when experienced [15–18]. SES is a critically important predictor of health [19], and circulating levels of inflammatory and clotting factors are systematically linked with educational status and income, with the lower strata evincing elevated inflammation relative to those in more favorable positions [20–24]. However, whether or not the associations between anger and inflammation are consistent across the SES gradient is not typically studied, despite some evidence indicating that the frequent experience and expression of anger may be more harmful to the health of individuals already at risk due to disadvantaged SES, especially in childhood [25, 26].

In a prior study of the population-based Midlife in the United States (MIDUS) cohort of middle-aged and older adults, trait anger and anger-out were more strongly associated with higher IL-6 and fibrinogen for those with low educational attainment (i.e., high school education or less) as compared to those with college and professional levels of education. Anger-control also predicted lower IL-6 for those with a high school degree or less, but only among women [27]. Anger-in did not moderate the educational gradients in inflammation. One shortcoming of the prior analyses, however, was the limited representation of racial minorities within the national cohort (only 7.2 % of respondents). The current report addresses this limitation by including a city-specific sample of African Americans from Milwaukee, Wisconsin, permitting us to now examine the influence of race on the associations among IL-6, fibrinogen, education, and anger. Although a city-specific sample does not represent African Americans nationally, Milwaukee is an informative context, given that it is among the most segregated cities in the United States [28]. Publications from this Milwaukee sample have documented higher stress in many life domains (e.g., work, family, and neighborhood) and more early life adversity than for the overall MIDUS national sample [29, 30]. As such, it is an informative urban setting for evaluating the links between anger and health.

The literature regarding race differences in anger is inconsistent, and the role of SES in explaining those effects and the implications for inflammatory biology are largely unexplored. Qualitative research with middle- and upper-class African Americans indicates that anger continues to be a common corollary of higher SES due to persistent experiences of racial discrimination and unfair treatment, even in contexts of educational or occupational success [31]. Recent quantitative research by Williams and colleagues [32] shows that anger and frustration were the most common emotional responses to experiences of racial and non-racial discrimination, regardless of race or SES. In the same paper, Whites and African Americans did not differ on the average number of unfair experiences or treatment, but African American respondents viewed most of their unfair experiences as due to racial causes, whereas White respondents typically viewed them as nonracial. It is also noteworthy that the rates of unfair treatment were higher among low-SES African Americans and Whites than their middle-class counterparts. Additional evidence suggests that African Americans experience less "return" on education—higher educated African Americans have lower levels of occupational and income attainment [33, 34] and poorer health outcomes than similarly educated Whites [35–38].

Together, these findings suggest that the health ramifications of high anger, or poorly controlled anger, may be stronger in African Americans with more, compared to less, education, in contrast to prior findings in the largely White MIDUS national cohort. Alternatively, in at least one study of African American adolescents, trait anger was found to predict blood pressure more strongly among those from low, as compared to higher SES neighborhoods [26], indicating that anger may be most detrimental among low SES individuals; similar to the association previously reported among White respondents. We tested the predictive value of these perspectives by examining the influence of anger experience and expression as moderators of the association between education and two inflammatory and coagulation markers in African Americans compared to prior findings on primarily White adults [27]. Both groups were included in statistical analyses to test for race differences, which would be evident when there were three-way interactions among race, educational status, and anger, in predicting IL-6 and fibrinogen as outcomes.

Method

Participants were from the second wave of MIDUS (MIDUS II), which began in 2004 with a 75 % retention rate from the first wave (adjusted for mortality). MIDUS I began in 1995 with more than 7,000 non-institutionalized adults, recruited via random digit dialing from the 48 contiguous states, siblings of the random digit dialing sample, and a large sample of twins [39, 40].

Biological data were collected from a subset of respondents who completed both MIDUS I and II survey assessments and agreed to travel to one of three clinics (Madison, WI; Washington, DC; or Los Angeles, CA) for an overnight visit

[41]. The biological data sample included individuals from the national cohort ($n=1,054$) as well as African Americans from Milwaukee, WI, who were recruited at MIDUS II ($n=201$) [30]. A small number of respondents who identified as a race other than White or Black and/or African American were excluded ($n=55$). The majority (87.8 %) of African American respondents came from Milwaukee. This study was approved by Institutional Review Boards at University of Wisconsin-Madison, as well as at Georgetown University and the University of California, Los Angeles. All participants provided written informed consent.

Measures

Education During telephone and home interviews (Milwaukee sample only), respondents reported the highest grade of school or year of college completed. The twelve response categories ranged from no schooling to completion of a professional degree. Education was treated as a continuous variable in analyses.

Anger Anger was assessed using the trait anger (15 items), anger-out (8 items), and anger-control (4 items) subscales from Spielberger's State-Trait Anger Expression Inventory [42], which was completed during the clinic visit. Trait anger was measured by respondents indicating how often they generally felt the given statements (e.g., "I have a fiery temper") on a four-point scale. For the anger-out and anger-control scales, participants respectively indicated how often they expressed their anger outwardly (e.g., "I strike out at whatever infuriates me") or how often they attempted to manage the expression of their anger (e.g., "I control my temper") when they felt angry or furious. Internal consistencies were consistent across racial groups, ranging from .69 to .83 in the White sample and from .67 to .89 in the African American sample.

Covariates Covariates included age, gender, body mass index (BMI), medication usage, current smoking status, regular exercise, alcohol consumption, and chronic health conditions. Age, gender, BMI, medication usage, and chronic health conditions were considered confounding influences known to affect levels of inflammatory markers [43], and we were interested in examining associations among anger, education, and race independent of these factors. Height and weight were measured by nursing staff at each General and Clinical Research Center (GCRC) and used to calculate BMI (weight in kilograms divided by height in meters squared). BMI is a key predictor of inflammatory markers [43, 44]. Antidepressants, anti-hypertensives, cholesterol-lowering medications, and steroid usage (accounted for as four binary-coded variables to indicate current use or non-use) have modulatory effects on IL-6 and fibrinogen [45, 46]. Chronic health conditions (summed score)

were self-reported physician diagnosed diseases; inflammation is an important component for many of these chronic illnesses [47]. Smoking status, regular exercise (at least 20 min per day, three or more times per week), and alcohol consumption (drinks per month) were self-reported at the GCRC visit. These factors represent important behavioral pathways that likely contribute to the links among education, anger, and race to chronic inflammation [43]. Fully adjusted models included both confounding influences and health behaviors.

Inflammatory Markers Serum IL-6 levels were measured with the Quantikine® high-sensitivity enzyme linked immunosorbent assay kit (R&D Systems, Minneapolis, MN). Fibrinogen was measured using the BNII nephelometer (Dade Behring Inc., Deerfield, IL). The intra- and inter-assay coefficients of variance were all in an acceptable range (<12 % variance).

Statistical Analyses

All analyses involved comparisons between African American and White respondents in keeping with the central focus on race differences. A small number of IL-6 and fibrinogen values were winsorized to three standard deviations from the mean to reduce the influence of outliers (2 % of total data). IL-6 and BMI were log-transformed to achieve normal distributions. Generalized estimating equations models with random intercepts for each family cluster and an exchangeable within cluster covariance structure were used to address the dependencies in the data from the inclusion of twins and siblings (38 %) in the MIDUS national sample. All continuous variables were mean-centered. Race, education, anger, all possible two-way interactions, and three-way interactions were considered as respective predictors of IL-6 and fibrinogen. Control variables included age, gender, BMI, current smoking status, alcohol consumption, regular exercise, chronic health conditions, and mediation usage. Models were run separately for each dimension of anger and for each inflammatory marker. Significant three-way interactions were probed by examining simple slopes between anger and IL-6 and fibrinogen were examined at high and low levels of education, respectively, for each racial group [48]. For high and low education levels, we centered at the mean level of education among those with a college degree or more and those with a high school education or less, respectively. An alpha level of 0.05 was used for all statistical tests.

Results

Descriptive information is summarized in Table 1. The African American sample had higher IL-6 and fibrinogen

[49], and were younger, more likely to be female, less educated, had more chronic conditions, higher BMIs, reported more alcohol use, and were more likely to be current smokers. They were also more likely to be taking anti-hypertensive medication, less likely to get regular exercise, and less likely to be taking cholesterol-lowering or anti-depressant medication than the White respondents. Data from the anger scales portrayed separately by race and educational attainment are provided in Table 2. The African American respondents reported greater anger-out and lower anger-control than did the White respondents. There were no significant main effects of education or education-by-race interactions that predicted any of the anger scales.

Results from the three-way interaction models are presented in Table 3. Several significant three-way interactions emerged, including between race, education, and trait anger predicting fibrinogen and between race, education, and anger-out predicting both fibrinogen and IL-6. To probe these interactions, simple slopes between trait anger and anger-out and IL-6 and fibrinogen were calculated for African American and White respondents at low and high education levels. They are presented in Table 4 and displayed in Fig. 1. Among African Americans, greater anger-out predicted higher IL-6 and fibrinogen among those with high, but not low, educational status. Among White respondents, anger-out predicted higher IL-6 among those with low, but not high, educational status, though at trend level significance ($p=.054$). Trait anger and anger-out

also predicted lower fibrinogen among White respondents with high, but not low, educational status.

In order to examine whether the effects of anger were independent from general negative affect, we re-ran three-way

Table 2 Descriptive information on anger dimensions by education and race

Education level	African Americans	Whites	p value
High school or less ($n=334$)	($n=101$)	($n=233$)	
Trait anger	23.81 ± 6.53	23.82 ± 5.33	.99
Anger-out	13.64 ± 4.03	12.49 ± 3.17	<.01
Anger-control	8.88 ± 2.46	9.72 ± 2.22	<.01
Some college ($n=355$)	($n=75$)	($n=280$)	
Trait anger	24.18 ± 7.03	23.72 ± 5.36	.55
Anger-out	13.22 ± 3.76	12.69 ± 3.10	.21
Anger-control	9.41 ± 2.55	10.05 ± 2.32	.04
College or higher ($n=505$)	($n=44$)	($n=461$)	
Trait anger	24.24 ± 6.16	23.76 ± 5.03	.56
Anger-out	13.39 ± 4.11	13.01 ± 3.19	.47
Anger-control	9.43 ± 2.06	10.38 ± 2.06	<.01

Data are presented as means±standard deviation. Trait anger scores ranged from 15 to 54; anger-out scores ranged from 8 to 29, and anger-control scores ranged from 4 to 14. p values come from t tests of racial differences in anger within educational subgroups. Education by race interactions predicting the anger scales were all non-significant (p's>.46)

Table 1 Descriptive information on white and African American study participants ($n=1,200$)

Variable	White respondents ($n=978$) M(SD) or %	African American respondents ($n=222$) M(SD) or %	p value
Trait anger	23.8 (5.2)	24.0 (6.6)	.52
Anger-out	12.8 (3.2)	13.4 (3.9)	.01
Anger-control	10.1 (2.2)	9.2 (2.4)	<.001
IL-6 (pg/mL)[a,b]	2.7 (2.2)	3.9 (2.6)	<.001
Fibrinogen (mg/dL)[a,b]	339.0 (81.2)	385.0 (89.1)	<.001
Age (years)	58.4 (11.7)	53.6 (10.4)	<.001
Gender (% women)	54.6	67.6	<.001
Education			<.001
≤ High school	23.9	45.9	
Some college	28.8	34.2	
≥ College degree	47.3	19.8	
Chronic conditions	2.6(2.1)	3.0(2.3)	.01
Body mass index	29.0(5.9)	32.8(8.6)	<.001
Current smoking (% yes)	11.4	31.1	<.001
Alcohol (drinks/month)	13.3(22.9)	17.9(44.8)	.029
Regular exercise (% yes)	79.7	63.5	<.001
Medication (% yes)			
Anti-hypertensive	34.8	45.9	.002
Cholesterol	29.7	19.8	.003
Corticosteroid	12.5	10.4	.38
Anti-depressant	15.6	6.8	.001

Racial differences were assessed with independent sample t tests and Pearson Chi-square tests

[a] Values have been winsorized to 3 SD from the mean

[b] Differences in these inflammatory markers have been previously reported [49]

Table 3 Unstandardized results from generalized estimating equation models of three-way interactions among race, education, and varieties of anger predicting interleukin-6 and fibrinogen (*n*=1,200)

	IL-6				Fibrinogen			
	B	SE	Wald	p	B	SE	Wald	p
Race[a]	−.111	.023	23.828	<.001	−32.809	7.325	20.063	<.001
Education[b]	−.004	.008	0.261	.610	−0.223	2.249	0.010	.921
Trait anger	.006	.004	2.037	.153	0.684	1.028	0.443	.506
Race × education	.002	.009	0.047	.829	−1.014	2.461	0.170	.680
Race × trait anger	−.007	.005	2.378	.123	−1.331	1.120	1.413	.235
Education × trait anger	.002	.002	1.675	.196	0.543	.306	3.138	.076
Race × education × trait anger	−.003	.002	3.356	.067	−0.942	.356	7.009	.008
Race[a]	−.108	.022	23.451	<.001	−31.843	7.245	19.316	<.001
Education[b]	−.003	.008	0.200	.655	−.428	2.238	0.037	.848
Anger-out	.014	.005	8.123	.004	2.194	1.278	2.948	.086
Race × education	.000	.009	0.002	.968	-0.808	2.452	0.109	.742
Race × anger-out	-.011	.006	3.370	.066	-2.930	1.483	3.901	.048
Education × anger-out	.002	.002	1.923	.166	0.745	0.407	3.354	.067
Race × education × anger-out	−.005	.002	4.947	.026	-1.286	0.518	6.161	.013
Race[a]	−.104	.024	18.230	<.001	-33.142	7.267	20.798	<.001
Education[b]	.001	.009	0.017	.897	−.315	2.174	0.021	.885
Anger-control	−.013	.008	2.405	.121	.116	2.664	0.002	.965
Race × education	−.003	.009	0.114	.735	−.736	2.389	0.095	.758
Race × anger-control	.007	.009	0.503	.478	−.798	2.886	0.076	.782
Education × anger-control	.003	.003	0.617	.432	−.433	0.857	0.256	.613
Race × education × anger-control	−.001	.004	0.073	.787	.431	0.958	0.202	.653

[a] Race coefficient reflects White, compared to African American, respondents. Race differences in IL-6 and fibrinogen in this sample have been previously reported [49]

[b] Educational differences in IL-6 and fibrinogen in this sample have previously been reported [21, 22]. All models are adjusted for age, gender, medication usage, chronic health conditions, smoking status, regular exercise, alcohol consumption, and BMI

interaction models adjusting for depressed mood, assessed with the Center for Epidemiologic Studies Depression scale [50], and trait anxiety, which had been assessed with Spielberger's scale [51]. Results were unchanged by inclusion of depressed mood and trait anxiety as additional covariates in fully adjusted models, suggesting that the previously identified associations were specific to anger and not due to the influence of general negative affect (race × education × trait anger predicting fibrinogen: B(SE)=−0.97(.36), Wald=7.11, *p*=.008; race × education × anger-out predicting IL-6: B(SE)=−0.005(.002), Wald=5.17, *p*=.023; race × education × anger-out predicting fibrinogen: B(SE)=−1.26(.52), Wald=5.86, *p*=.015). Further, when trait anger was added in the models assessing the interactions with anger-out, none of the conclusions were altered (race × education × anger-out predicting IL-6: B(SE)=−.005(.002), Wald=5.58, *p*=.018; race × education × anger-out predicting fibrinogen: B(SE)=−1.25(.52), Wald=5.67, *p*=.017). Similarly, when anger-out was included in the significant model assessing interactions with trait anger, the findings were also unchanged

(race × education × trait anger predicting fibrinogen: B(SE)= −0.98(.36), Wald=7.40, *p*=.007).

Discussion

The primary objective was to examine racial differences in how different types of anger (trait anger, anger-out, and anger-control) moderated the association between educational attainment and two markers of inflammation, IL-6 and fibrinogen. For half of the tested interactions, the results revealed that among African American respondents, those with higher education and greater anger had elevated inflammatory profiles. In contrast, those with low education and high anger in the White sample had the highest levels of IL-6, although this trend just approached significance (*p*=.054). In addition for the White respondents, anger was negatively related to fibrinogen among those with higher education. These effects were significant after taking many key demographic and health variables into consideration. The specific findings just for the

Table 4 Simple slopes for IL-6 and fibrinogen regressed on trait anger and anger-out

	Slope	SE	Wald	p value
Anger-out, IL-6				
White, low education	.010	0.005	3.71	.054
White, high education	−.002	0.004	0.34	.560
African American, low education	.008	0.005	2.51	.113
African American, high education	.020	0.008	6.35	.012
Trait anger, fibrinogen				
White, low education	.484	0.759	0.41	.523
White, high education	−1.661	0.570	8.48	.004
African American, low education	−.851	1.000	0.72	.395
African American, high education	2.063	1.543	1.79	.182
Anger-out, fibrinogen				
White, low education	.794	1.288	0.38	.538
White, high education	-2.108	1.013	4.34	.037
African American, low education	.085	1.525	0.00	.956
African American, high education	4.087	1.808	5.11	.024

Low education refers to the mean education among individuals with a high school education or less. High education refers to the mean education among individuals with a college degree or more. Models adjusted for age, gender, medication usage, chronic health conditions, smoking status, regular exercise, alcohol consumption, and BMI

SE estimated standard error

White respondents have already been discussed in greater detail in a prior publication [27]. However, overall, the combined analyses support the additional hypothesis that there are important racial differences in how anger experience and expression affect inflammatory physiology and its association with educational attainment.

With regard to trait anger and anger-out, opposite patterns emerged between college-educated Whites and African Americans. That is, anger was negatively related to fibrinogen among college educated White respondents, and positively related to fibrinogen among college educated African Americans. Highly educated White adults who express anger may do so as a symbol of their status and dominance [52], which may not be biologically costly. However, these psychosocial benefits and salubrious inflammatory correlates seen in educated Whites did not extend to African Americans, consistent with prior evidence that African Americans get less health "returns" from advanced educational attainment [35–38]. There may be possible social sanctions against African American men and women who express anger—such as being labeled with the "angry black woman/man" stereotype [53, 54]. The threat of this label may function to limit anger expression [53] and prompt careful tailoring of self-presentation, especially among middle class African Americans in primarily White contexts [55, 56]. The current data support the inflammatory costs of expressing anger for higher educated African Americans, perhaps related to these social sanctions.

Inflammatory dysregulation is a major contributor to the development and pathogenesis of age-related chronic diseases [4]. As an index of relative effect size, we compared differences in predicted IL-6 and fibrinogen at high trait anger and anger-out (one standard deviation above the mean) between high and low education groups among African American and White respondents separately as a function of age. We found that African Americans with higher education and high anger had inflammatory profiles indicative of being approximately 10 years *older* than less educated African Americans with high anger. This pattern was reversed among White respondents. That is, White respondents with more education and high anger had inflammatory profiles that were approximately 10 years *younger* than less educated White respondents with high anger.

Anger-in (i.e., anger suppression) was not examined given that there was no evidence of an association between anger-in and inflammation in the national sample [27], and there was no evidence of anger-control moderating racial or educational gradients in inflammation. But African Americans did report lower anger control than White respondents. Among White respondents, we previously found that anger control predicted lower IL-6 among women with a high school education or less only [27]. Small cell sizes preclude consideration of gender as an additional substantive variable within the African American sample. However, prior papers indicate that women use more anger diffusion strategies than men when angry [57], and evidence from the General Social Survey suggests that sense of control is more strongly related to lower anger intensity in African Americans than in Whites [58]. Thus, considering gender and racial differences concurrently is an important avenue for future work.

Why would higher, as compared to lower, educated African Americans show that high anger moderates their inflammatory profiles? One possibility is that higher educated African Americans may interact more frequently in racially integrated occupational situations that highlight and perhaps reinforce societal inequalities, while also underscoring the pressures and toll associated with social advancement. These experiences can contribute to feelings and expressions of anger, which may exact a physiological toll. African Americans with less education, on the other hand, may reside and work in less diverse settings and thus be protected from the same types of social threats. Although we did not find evidence of educational differences in the frequency of experiencing or expressing anger among African Americans, the results nonetheless point to possible physiological ramifications of anger, especially among the highly educated. Cose (1993) reported on interviews with African Americans who had achieved educational or occupational success but still felt that anger was evoked as a frequent reaction to perceived discrimination

Fig. 1 Racial differences in the moderation of educational gradients in IL-6 and fibrinogen by two varieties of anger. *Lines* represent the simple effects of anger on IL-6 and fibrinogen for White and African American respondents by education. Low education refers to the mean education among individuals with a high school education or less. High education refers to the mean education among individuals with a college degree or more. Effects were adjusted for age, gender, medication usage, chronic health conditions, current smoking status, alcohol consumption, regular exercise, and BMI. Data for White respondents have previously been published [27]
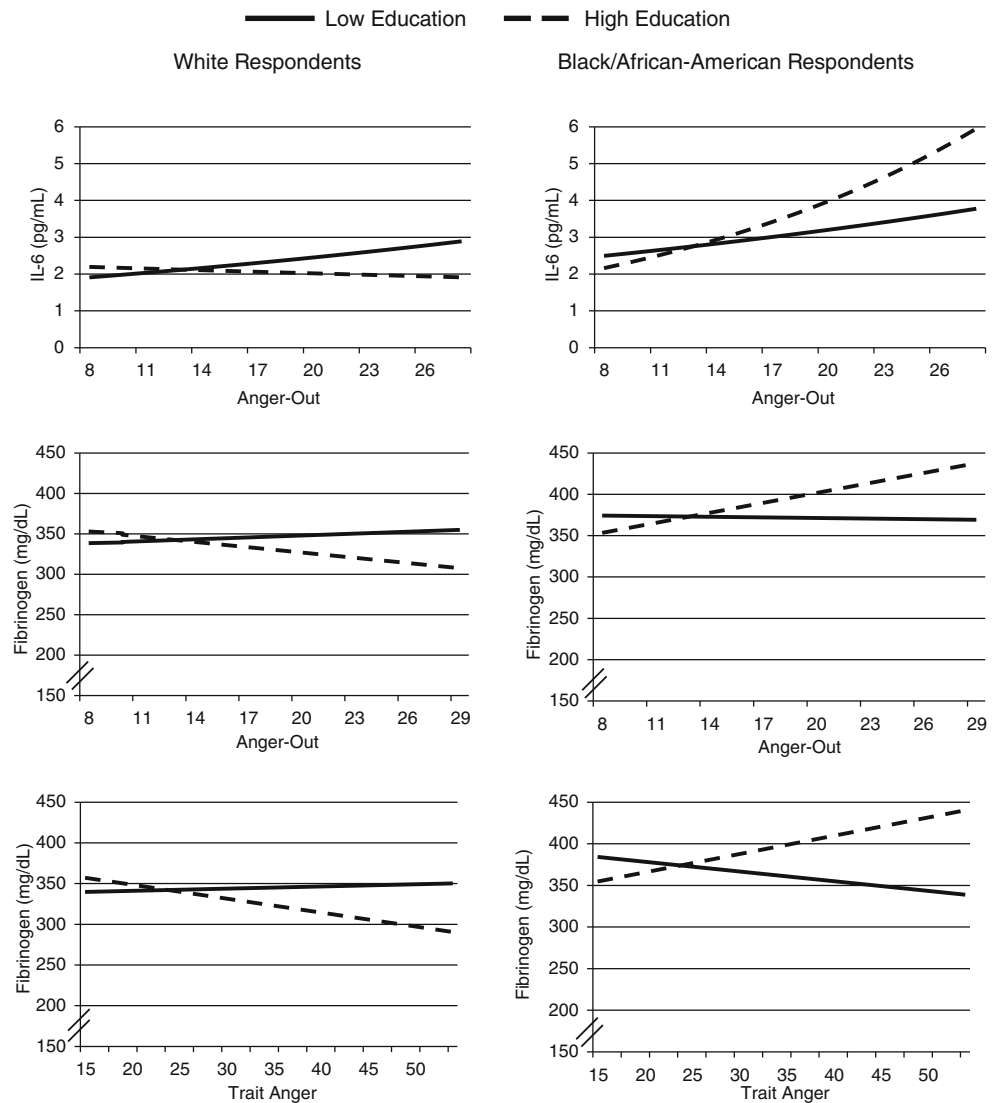
and inequitable treatment due to race [31]. Future work that considers other indicators of SES, such as employment status and occupational prestige, is warranted and may shed light on these ideas.

The role of discrimination, as a possible precursor to anger and inflammatory dysregulation, warrants further investigation, thus extending evidence of effects on stress-related hormones such as cortisol. In the same MIDUS sample, perceived discrimination predicted less healthy, flattened cortisol slopes across the day among Whites [59], and more healthy slopes among African Americans, supporting the view that awareness of racism may be protective among minority group members. However, in line with current findings, perceiving discrimination was less protective for higher (compared to lower) SES African Americans. Relatedly, it is known that African Americans are not as likely to experience other benefits of education advancement, including weight control and the opportunity for regular

exercise. These differences may explain lingering effects on inflammatory physiology mediated through adiposity [36]. Thus, African Americans and other minority group members may experience fewer health benefits of being higher SES than Whites, due in part to racial discrimination and the ensuing anger [31, 60, 61].

Several limitations should be acknowledged. First, the African American sample was recruited primarily from one city, thus limiting our ability to generalize to all regions. Nonetheless, Milwaukee is an informative urban environment for studying anger and health, given that it is among the most segregated cities in the United States [28]. Second, we are unable to rule out acute infection status of MIDUS respondents, which may affect levels of inflammatory markers. However, there were no significant associations between body temperature at the time of the biological assessments and any of the predictor variables (p's>.10), and fever is a standard sign of an acute inflammatory response. We note

that very few MIDUS respondents presented with fever ≥37.5 °C ($n=11$, <1 %). Third, causality cannot be definitively delineated, given the cross-sectional design, although education is generally completed early in adulthood, and thus, is temporally antecedent to the midlife assessment of inflammatory markers. Using education as the sole index of SES is another limitation, given that there may be racial differences in returns of higher education. An important extension of this work will consider other indicators of SES, especially related to employment status and occupational prestige. Finally, several interactions were tested, given the analytic focus on several anger varieties and two inflammatory markers. Our findings underscore that not all features of anger have an equivalent, detrimental effect on inflammatory profiles, a conclusion that needs to be replicated and extended.

In summary, these analyses show that anger expression was associated with elevated IL-6 and fibrinogen among African American respondents with a college degree, comparable to the associations among White respondents with a high school education or less. An additional unique strength of the study was the inclusion of several dimensions of anger. It is clear that race, educational attainment, and perceptions about one's status in society are critically important in understanding the biological concomitants of anger, a central aspect of emotional life.

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