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Does one size fit all? The role of body mass index and waist circumference in systemic inflammation in midlife by race and gender

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ABSTRACT

Objective: This study investigates the associations of body mass index (BMI) and waist circumference (WC) with markers of systemic inflammation in midlife by race and gender.

Design: Data were obtained from the Survey of Midlife in the United States, a cross-sectional, observational study of Americans 35 years old or older (White men: $N = 410$; White women: $N = 490$; Black men: $N = 58$; Black women: $N = 117$). Inflammation was measured by concentrations of fibrinogen and C-reactive protein (CRP) in fasting plasma and concentrations of E-selectin and interleukin-6 (IL-6) in fasting serum. Anthropometric data were used to obtain BMI and WC. Socio-demographic and health-related factors were assessed with a survey. Multivariate models by race and gender were estimated to test the roles of BMI and WC for each inflammation marker.

Results: Compared to White men, Black women have higher BMI and higher levels of all four inflammation markers; White women have lower BMI, lower WC, and lower E-selectin and fibrinogen but higher CRP; and Black men have higher fibrinogen. After adjusting for socio-demographic and health-related covariates as well as perceived discrimination, WC is associated with all four markers of inflammation among White men and women; with three markers (fibrinogen, CRP, and IL-6) of inflammation among Black women; and with CRP (and marginally with fibrinogen and E-selectin) among Black men. BMI is associated with higher CRP and fibrinogen among Black men (marginally so for White men) but not for women of either race.

Conclusions: WC shows more consistent associations with inflammation markers than BMI, although the relationships vary by inflammation marker and population group. Our findings suggest that WC is a risk factor for systemic inflammation among White and Black men and women, and BMI is an additional risk factor for Black men.

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Introduction

Systemic inflammation is involved in the etiology of prevalent aging-related chronic conditions such as hypertension (Savoia and Schiffrin 2006), cardiovascular disease (Everett et al. 2013), insulin resistance (Olefsky and Glass 2010), and cancer (Elinav et al. 2013). As most of these conditions manifest in midlife, it is important to investigate inflammation in this stage of the life course, especially for racial/ethnic minorities, who have high prevalence of such conditions (Frieden 2013).

It has been established that inflammation increases with measures of body fatness and weight status, including body mass index (BMI) (Laurson et al. 2011), waist circumference (WC) (Thorand et al. 2006; Rana et al. 2009), waist-to-hip ratio (Thorand et al. 2006), visceral fat (Cartier et al. 2009), and subcutaneous fat (Pou et al. 2007; Cartier et al. 2009). Adipose tissue secretes substances involved in inflammatory responses, including hormones, such as leptin, adiponectin, and resistin, and pro-inflammatory cytokines, such as interleukin-1(IL-1), interleukin-6 (IL-6), and tumor necrosis factor-α. In obese individuals who have hypertrophic adipocytes, these functions are dysregulated, leading to an inflammatory state that is systemic (as opposed to localized, typically with injury) and chronic (Pischon et al. 2007; Hajer et al. 2008; Olefsky and Glass 2010). Systemic, chronic inflammation is evident in higher concentrations of C-reactive protein (CRP), a protein produced in response to infection or injury (Berg and Scherer 2005); fibrinogen, a blood clotting factor involved in the coagulation response to vascular injury (Toker et al. 2005); and E-selectin, an endothelial adhesion molecule expressed as a result of endothelial damage (Ross 1999).

Notably, prior research indicates that the contributions of various measures of body fatness to inflammation may be gender-specific. Compared to men, women have greater body fat for an equivalent BMI (Gallagher et al. 1996) as well as higher levels of some inflammation markers, including adiponectin (Boyne et al. 2010), inflammation index consisting of CRP, fibrinogen, and urinary albumin (Yang and Kozloski 2011), high-sensitivity CRP among pre-diabetics (Saltevo et al. 2009), and CRP and IL-1 receptor antagonist among individuals with metabolic syndrome (Saltevo et al. 2008). In addition, women show a steeper relationship between fat distribution and CRP (Khera et al. 2005) and between BMI and CRP (Choi et al. 2013). Levels of CRP are related to subcutaneous adiposity among older women but visceral adiposity among older men (Cartier et al. 2009), and the largest part of variability in CRP is explained by fat mass for women but waist-to-hip ratio for men (Thorand et al. 2006).

Race is another factor potentially playing a role in adiposity, inflammation, and the linkages between the two. Compared to Whites, Blacks suffer a disproportionate burden of overweight and obesity (Schiller et al. 2012), which may place them at a higher risk for chronic inflammation and associated conditions. Increased levels of CRP (Khera et al. 2005; Kelley-Hedgepeth et al. 2008; Paalani et al. 2011) and IL-6 (Kiecolt-Glaser et al. 2003; Walston et al. 2007; Paalani et al. 2011) among Blacks as compared to Whites have been reported, although some research finds no racial differences in CRP levels after adjusting for socio-demographic characteristics (Ford et al. 2003; Meng et al. 2007).

Building on this evidence, this study aims to investigate the role of weight status in systemic inflammation by race and gender, using four biomarkers: fibrinogen, E-selectin, CRP, and IL-6. We use two measures of weight status – BMI and WC – because each

captures a slightly different aspect of weight. BMI is an established predictor of morbidity and mortality (Rexrode et al. 1998), while WC reflects abdominal adiposity, a well-known health risk (Zhu et al. 2002). Their joint consideration is an important contribution, as prior research suggests that each measure may predict health outcomes differently among men and women and among racial/ethnic groups (Mezick et al. 2014; Heymsfield et al. 2016), but it is not known how they relate to inflammation in each race-gender group.

Methods

Sample

We utilized data from the Survey of Midlife in the United States (MIDUS II), an ongoing national study of aging that represents non-institutionalized English-speaking residents of the 48 contiguous states who are age 35 years or older. We limited our sample to Non-Hispanic White and Black participants who also completed the biomarker sub-study (175 Blacks and 900 Whites) (Ryff et al. 2013). In this sub-study, blood samples and anthropometric data were obtained by trained staff during an overnight stay at a study clinic. In addition, participants completed mail surveys and telephone interviews collecting data on demographic, social, and psychological characteristics. Data were collected between 2004 and 2009.

Measures

Inflammation

Systemic inflammation markers included CRP, fibrinogen, E-selectin, and IL-6. Their concentrations were determined using fasting blood samples drawn on the second day of the clinical visit, before breakfast, at approximately 7 am. The collection and processing of the samples followed standardized procedures to ensure consistency. Fibrinogen concentrations (mg/dL) in citrated plasma were measured using immunoturbidometric assay at the University of Wisconsin, Coe Lab. CRP concentrations (ug/mL) in citrated plasma were measured using immunoturbidometric assay at the University of Vermont, Tracy Lab. BNII nephelometer from Dade Behring utilizing a particle enhanced immunonepholometric assay was used. Serum levels of soluble E-selectin, also known as endothelial leukocyte adhesion molecule-1 (ELAM-1) and CD62E, were measured in ng/mL at the University of Vermont, Tracy Lab. High sensitivity ELISA assay (Parameter Human sESelectin Immunoassay; R&D Systems, Minneapolis, MN) was utilized. IL-6 concentrations (pg/mL) in serum were determined using the Quantikine® High-sensitivity ELISA kit #HS600B (R&D Systems, Minneapolis, MN) at the University of Vermont, Tracy Lab.

Race/ethnicity and gender

Race/ethnicity was obtained by self-report and categorized as African American/Black (henceforth Black) and White. Gender was self-reported as man vs. woman.

Weight status

Measures of weight status included BMI and WC. Both were based on anthropometric data collected during the clinic visit. Consistent with guidelines by the Centers for Disease Control and Prevention (CDC 2016), BMI was calculated as weight in kilograms (kg) divided by the square of height in meters (m). WC was measured in centimeters (cm).

Control variables

Perceived discrimination was measured by two variables, representing the Daily Discrimination scale and the Lifetime Discrimination scale (Williams et al. 1997). These scales were originally developed for a study of racial discrimination in Detroit Area Study and measured discriminatory experiences of any type, regardless of the cause for discrimination (race/ethnicity, gender, age, etc.). Respondents were asked how often they experienced each of nine types of daily discrimination, for instance being treated with less courtesy, being treated with less respect, or receiving poorer service than other people at restaurants or stores (see Appendix 1 for the list of items). The Daily Discrimination scale was constructed as the sum of these nine items, with higher values indicating higher levels of discrimination. The Lifetime Discrimination scale measured discrimination in major life domains, such as employment, education, health care, and housing. Respondents reported discriminatory experiences in their lives because of race/ethnicity, gender, age, religion, physical appearance, sexual orientation, or other characteristics. Examples of such events included being denied a scholarship, not being hired for a job, or being prevented from renting or buying a home in a particular neighborhood. The scale was computed as the total of reported events.

Socio-demographic characteristics used as control variables included age, years of education, and annual household income. The models also adjusted for lifestyle and health-related factors that have been correlated with inflammation in prior research, including current smoking and the use of aspirin to prevent heart condition. We controlled for health status using a morbidity index that totals the number of chronic conditions during the past 12 months,[1] as well as vigorous exercise using an indicator constructed from questions about the frequency of vigorous leisure activity in winter and summer. Respondents who reported 'several times a week' for both winter and summer were coded as '1' for vigorous exercise; others were coded as '0'.

Analytic strategy

We obtained descriptive statistics by race and gender and performed bivariate tests comparing White men to White women, Black men, and Black women on each characteristic. In supplementary analyses, we examined whether unadjusted differences in inflammation markers among race-gender groups persisted after the adjustment for weight status measures and covariates. Next, we estimated multivariate linear regression models of inflammation markers for White men, White women, Black men, and Black women to assess relations between each weight status measure and each inflammation marker by race and gender. Model 1 included only the main explanatory variables (BMI and WC) to examine whether they related to inflammation independently of each other. Model 2 included covariates to investigate whether the effects of BMI and WC on each inflammation marker in each race-gender group are robust after the adjustment for social and health-related factors.[2] Covariates included perceived discrimination, age, socioeconomic status measured by education

and income, smoking, vigorous exercise, aspirin use, and chronic conditions. CRP and IL-6 had skewed distributions; therefore, these indicators were logged for the purposes of regression models to approximate normality; in addition, robust estimators were applied to minimize the influence of outliers in all models. $P < .05$ was taken as statistically significant; $p < .1$ was interpreted as marginally significant because of the small number of observations in some race-gender groups (i.e. $n = 58$ for Black men).

Results

Characteristics of White women, Black men, and Black women as compared to White men are presented in Table 1. Socio-demographically, Blacks in this sample are younger than Whites. White men have the most education, highest incomes, and best health as indicated by fewest reported chronic conditions during the past year. Black men and women report more perceived daily discrimination and lifetime discrimination compared to White men. Lifetime discrimination reports are also higher among White women compared to White men. Black women are less likely to report current smoking and, similarly to Black men, less likely to report vigorous exercise several times a week. White men have the highest rate of preventive aspirin use among all examined race-gender groups.

Concerning weight status, Table 1 indicates that White women have lower BMI and lower WC compared to White men, while Black women have higher BMI. In addition,

Table 1. Characteristics of the sample by race and gender: Means and standard deviations.

	White men ($N = 410$)		White women ($N = 490$)		Black men ($N = 58$)		Black women ($N = 117$)	
Variable (Range)	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Socio-demographic background								
Age, years (34–84)	55.98	12.05	54.79	11.55	50.86	9.60***	51.50	10.95***
Education, years (2–20)	15.17	2.51	14.81	2.41*	13.36	2.88***	13.66	2.67***
Annual household income, $10,000s (0–30)	8.30	6.19	7.35	6.05*	4.94	3.74***	3.85	3.47***
Systemic inflammation markers								
Fibrinogen,[a] mg/dL (45.00–857.00)	327.32	80.27	351.17	84.12***	352.67	85.60*	407.35	103.75***
E-selectin,[b] ng/mL (.09–166.97)	43.48	22.08	39.09	19.67**	49.64	23.37	49.13	27.40*
CRP,[a] ug/mL (.14–61.70)	2.30	4.51	3.15	4.38**	2.95	3.19	5.66	7.90***
IL-6,[b] pg/mL (.16–23.00)	2.72	2.57	2.85	3.01	3.37	2.63	4.14	3.19***
Weight status								
BMI, kg/m^2 (14.23–166.10)	30.78	13.89	28.97	11.99*	31.19	10.99	36.01	19.92**
WC, cm (60–187)	103.93	13.61	90.42	14.91***	101.51	17.68	101.95	18.54
Perceived discrimination								
Daily discrimination (9–32)	12.21	4.08	12.71	4.03	15.45	7.29**	14.30	6.04***
Lifetime discrimination (0–11)	.67	1.27	1.05	1.53***	3.40	2.95***	2.88	2.73***
Health-related factors								
Current smoker (0,1)	.11		.10		.03		.01‡	
Vigorous exercise (0,1)	.22		.21		.10‡		.12‡	
Aspirin (0,1)	.42		.25¥		.19†		.27†	
Chronic conditions, count (0–16)	1.83	1.83	2.58	2.35***	3.03	3.11**	3.39	2.90***

Source: Survey of Midlife in the United States (MIDUS II).

Notes: SD = standard deviation (shown for continuous variables only); CRP = C-reactive protein; IL-6 = Interleukin 6. BMI = body mass index; WC = waist circumference

[a]In citrated plasma.

[b]In serum.

Race-gender differences compared to White men based on simple linear regression: *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed tests).

Race-gender differences compared to White men based on simple logistic regression: ‡$p < .05$, †$p < .01$, ¥$p < .001$ (two-tailed tests).

White and Black women and Black men have higher levels of fibrinogen compared to White men. Black women also have higher concentrations of E-selectin, CRP, and IL-6. White women have higher levels of CRP but lower E-selectin. Supplementary analyses show that race-gender differences in fibrinogen, CRP, and IL-6 mostly persist after the adjustment for weight status and covariates (results upon request). The exception is E-selectin, which does not vary among race-gender groups in the multivariate adjusted model. For all other biomarkers, White men have the lowest levels of inflammation. Fibrinogen, CRP, and IL-6 concentrations are higher among Black and White women (p-values <.001) compared to White men, and a similar pattern of increased inflammation in seen for Black men (fibrinogen: $p < .1$, CRP: $p < .05$, and IL-6: $p < .05$).

Table 2 summarizes results of multivariate linear regression models of inflammation by race and gender. The general picture emerging from these results is that WC is more

Table 2. Coefficients from multivariate linear regression models of the relationships of BMI and WC to systemic inflammation markers by race and gender, before and after adjustment for covariates.

	White men ($N = 410$)		White women ($N = 490$)		Black men ($N = 58$)		Black women ($N = 117$)	
	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI
Fibrinogen[a]								
Model 1								
BMI	.61‡	−.10; 1.32	−.61	−1.48; .26	2.05***	1.18; 2.92	−.12	−.66; .41
WC	1.12**	.48; 1.75	1.73***	1.15; 2.30	1.80**	.62; 2.98	1.89**	.63; 3.15
Model 2								
BMI	.62‡	−.05; 1.28	−.48	−1.30; .35	2.47**	.94; 3.99	−.23	−.81; .35
WC	.93**	.30; 1.57	1.78***	1.21; 2.36	1.27‡	−.04; 2.57	2.06**	.71; 3.42
E-selectin[b]								
Model 1								
BMI	.07	−.08; .22	−.03	−.15; .10	−.40**	−.66; −.15	−.11	−.23; .01
WC	.30***	.14; .46	.36***	.22; .49	.33	.0005; .65	−.07‡	−.33; .18
Model 2								
BMI	.06	−.09; .21	−.04	−.16; .07	−.41‡	−.82; .01	−.12	−.29; .06
WC	.32***	.15; .49	.34***	.20; .47	.39‡	−.01; .79	−.15	−.46; .15
CRP[c]								
Model 1								
BMI	.01‡	−.001; .02	−.002	−.01; .01	.02**	.01; .03	−.002	−.01; .005
WC	.03***	.02; .04	.04***	.03; .04	.02**	.01; .04	.03***	.02; .04
Model 2								
BMI	.01‡	−.00004; .02	−.001	−.01; .01	.03*	.005; .05	−.002	−.008; .005
WC	.03***	.02; .04	.03***	.03; .04	.02**	.01; .04	.03***	.02; .05
IL-6[d]								
Model 1								
BMI	.004	−.003; .01	−.002	−.01; .003	.001	−.01; .01	.001	−.004; .003
WC	.02***	.01; .02	.02***	.02; .03	.01*	.002; .02	.02***	.01; .02
Model 2								
BMI	.004	−.002; .01	−.001	−.005; .003	−.001	−.02; .02	.001	−.003; .005
WC	.01***	.01; .02	.02***	.01; .02	.01	−.003; .02	.02***	.01; .02

Source: Survey of Midlife in the United States (MIDUS II). $N = 1046$.

Notes: Coeff. = coefficient; CI = confidence interval; CRP = C-reactive protein; IL-6 = Interleukin 6; BMI = body mass index (kg/m^2); WC = waist circumference (cm). Model 1 includes only BMI and WC. Model 2 further adjusts for years of education, annual household income ($ logged), daily discrimination, lifetime discrimination, smoking, vigorous exercise, preventive use of aspirin, and chronic diseases.

[a] mg/dL in citrated plasma.

[b] ng/mL in serum.

[c] ug/mL in citrated plasma, logged.

[d] pg/mL in serum, logged.

‡ $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$ (two-tailed tests).

consistently related to inflammation than is BMI but its role varies by inflammation marker and race-gender group. Before the adjustment for covariates (Model 1), all markers of inflammation increase with higher WC in one or more race-gender groups. Statistically significant effects at $p < .05$ are evident in a majority of models, and a model for Black men shows a marginally significant relationship between WC and E-selectin ($p < .1$). After the adjustment for covariates (Model 2), statistically significant relations between WC and inflammation markers at $p < .05$ persist in a majority of models, and two models (for Black men) reveal marginally significant relations of WC with fibrinogen and E-selectin (p-values <.1).

BMI, by contrast, shows few statistically significant relationships with inflammation markers. Before covariate adjustment (Model 1), the positive associations of BMI (independent of WC) with fibrinogen and CRP are significant for Black men (p-values <.001, and <.01, respectively) but BMI also shows a negative link to E-selectin for Black men (p <.01). The coefficients for BMI in relation to fibrinogen and CRP among White men are marginally significant ($p < .1$) and remain un-attenuated after the adjustment for covariates (Model 2). Among Black men, the coefficient for CRP increases in magnitude after adjustment (.02 in Model 1 vs .03 in Model 2, p-values <.01 and <.05, respectively), as does the coefficient for fibrinogen (2.05 in Model 1 vs 2.47 in Model 2, p-values <.001 and <.01, respectively).

Several covariates are linked to inflammation markers in Model 2 (Appendix 2). Among socio-demographic factors, higher age relates to increased fibrinogen and IL-6 among Whites of both genders (p-values <.05 for fibrinogen, p-values <.001 for IL-6), but for Black women, CRP and E-selectin decrease with age ($p < .1$ and $p < .05$, respectively). Higher education is related to lower fibrinogen ($p < .05$) among White women, and lower CRP ($p < .05$) and E-selectin ($p < .1$) among White men. Biomarkers that increase with income include E-selectin for White men ($p < .01$), fibrinogen and CRP for White women ($p < .1$ for both), and IL-6 for Black women ($p < .05$); in contrast, E-selectin concentrations decrease with higher income among Black women ($p < .05$). Perceived lifetime discrimination is associated with higher fibrinogen, CRP, and IL-6 concentrations among White women ($p < .05$, $p < .1$, and $p < .1$, respectively), higher fibrinogen among Black women ($p < .1$), and higher E-selectin among White men ($p < .05$).

Among health-related covariates, current smoking is linked to inflammation among White men and Black women as evidenced by higher levels of fibrinogen (White men: $p < .1$, Black women: $p < .01$) and CRP (White men: $p < .05$, Black women: $p < .001$). Black female smokers have increased IL-6 ($p < .001$) but also decreased E-selectin ($p < .001$) compared to their non-smoking counterparts. Black male smokers show lower levels of fibrinogen ($p < .05$) compared to Black male non-smokers. Vigorous exercise several times a week is linked to lower IL-6 concentrations for White women ($p < .01$) but also to higher CRP ($p < .01$) for White women and higher fibrinogen for Black women ($p < .05$). Compared to White men who do not use preventive aspirin, their counterparts who use aspirin have higher fibrinogen and CRP but lower E-selectin (p-values <.05). More chronic conditions are related to higher levels of IL-6 among Black men and women (p-values <.1), but also to lower fibrinogen among White women ($p < .01$).

Discussion and conclusions

This study examined how two measures of weight status – BMI and WC – related to inflammation among Black and White men and women. The key finding is that for most of these race-gender groups, WC showed more consistent associations with inflammation markers than did BMI, although the observed relationships varied by inflammation measure and population group. For White men and women, WC related to all four biomarkers of inflammation: fibrinogen, E-selectin, CRP, and IL-6. For Black women, WC was a significant contributor to three inflammation biomarkers: fibrinogen, CRP, and IL-6.

One surprising finding concerned Black women. Prior research has reported generally weaker associations between weight status and inflammation in Blacks compared to Whites (Choi et al. 2013). In this study, however, the relationships between WC and several inflammation biomarkers were stronger, not weaker, in Black women compared to White men. Among models of fibrinogen, the WC coefficient for Black women was the largest among all population groups. For IL-6, the WC coefficients were on par for Black and White women, both larger compared to the coefficient for White men. For CRP, Black women's WC coefficient had approximately the same size as coefficients for White men and women and was larger compared to the coefficient for Black men. Because of high multicollinearity, we were unable to evaluate whether these differences in coefficient size were statistically significant. Nevertheless, our results suggest that the weaker associations between weight status and inflammation reported in prior studies may have been predominantly driven by Black men.

In fact, Black men in our study showed patterns of inflammation that were unlike those found in other population groups. First, WC was relatively less helpful for understanding inflammation in this population group; it related to higher CRP but showed only marginally significant associations with fibrinogen and E-selectin. Second, BMI as a potential risk factor for inflammation performed better among Black men than in other race-gender groups. It related to three out of four examined biomarkers among Black men (fibrinogen, CRP, and E-selectin) and persisted in two (fibrinogen and CRP) even after the adjustment for covariates. These results further highlight the importance of evaluating inflammation separately for Black men and women, as different mechanisms may operate in these groups.

To summarize, our findings suggest that higher WC is a risk factor for systemic inflammation among White and Black men and women, and higher BMI is an additional risk factor for Black men. Individuals who reduce their WC may potentially benefit from reduced inflammation. This conclusion, however, must be considered tentative and contingent upon further study using experimental and longitudinal designs. The cross-sectional, observational design of this study prevents interpreting the results causally and thus constitutes an important limitation. Causal mechanisms linking weight status to inflammation are not well understood, but prior evidence suggests that reciprocal effects between weight status and inflammation may exist. Adipose cells in obese individuals appear to disrupt homeostatic regulation and lead to a pro-inflammatory state. Chronic inflammation, once present, may limit the ability to metabolize fat efficiently and thus contribute to further weight gain (Hajer et al. 2008).

Another limitation of this study concerns the number of observations for Black men. While this number ($n = 58$) was adequate for inferential statistics from the perspective

of central limit theorem, the statistical power in models for Black men was lower than in other groups and could have resulted in under-detection of some relationship. Low representation of Black men in research studies is a well-known problem (Woods et al. 2004) that poses a challenge for understanding the mechanisms undergirding the health outcomes of this population.

More research is needed to specify the mechanisms leading to inflammation, as well as the implications of inflammation for overall health and specific health conditions in different racial/ethnic groups, including non-Black minorities. Some Hispanic groups in the US, for instance, have a high prevalence of cardiovascular disease (Buys et al. 2015), and Native Americans have increased rates of diabetes (Wen et al. 2003). Since inflammation is implicated in these chronic conditions, Hispanics and Native Americans should be considered in future investigations of inflammation.

This study has several strengths. In contrast to earlier research that typically has focused on one or two biomarkers of inflammation, most commonly CRP, our work yielded a more comprehensive picture. Notably, we found important differences in the relationships between weight status measures and each biomarker of inflammation by race and gender, suggesting that each inflammation marker provides health-relevant information that may not be captured by other biomarkers. To our knowledge, this study is the first that considers WC and BMI jointly in relation to inflammation and clarifies their roles in different race-gender groups. Finally, unlike many earlier investigations that have relied on small convenience samples, this study uses data from a large national study, providing information about midlife population in the US.

In terms of policy and practice, this work can inform interventions to improve health outcomes in midlife among Whites and Blacks. It suggests that, overall, WC is a better marker of inflammation risk compared with BMI. WC is relatively easy to measure and may yield useful information for assessing overall health risk not captured by BMI. As an indicator of risk, WC may be especially useful for Whites of both genders, as well as for Black women, who had the highest levels of inflammation in our sample. In Black men, the inflammation risk is best captured by the combination of WC and BMI.

In conclusion, the evidence presented by this study expands the understanding of the role of WC and BMI in inflammation for different race-gender groups. It contributes to the literature on racial/ethnic health disparities in midlife with a special focus on inflammation as a risk factor for chronic disease in midlife. Consistently with several prior studies, it indicates increased inflammation among Blacks, especially Black women, and corroborates that weight status contributes to this risk, with WC emerging as the better marker of risk compared with BMI.

Notes

1. These include asthma/bronchitis/emphysema, tuberculosis, other lung problems, joint/bone diseases, sciatica/lumbago/backache, persistent skin trouble, thyroid disease, hay fever, stomach trouble, urinary/bladder problems, foot trouble, gall bladder trouble, varicose veins, AIDS/HIV, lupus/autoimmune disorder, gum/mouth trouble, teeth trouble, high blood press/hypertension, anxiety/depression, alcohol/drug problems, migraine headaches, chronic sleep problems, diabetes/high blood sugar, neurological disorder, stroke, ulcer, hernia, piles/hemorrhoids, and swallowing problems.

2. We attempted to estimate models for the whole sample that included interactions among weight status measures and race. However, high levels of multicollinearity with VIF > 30 raised concerns about potentially biased estimates. Therefore, these models are not reported.

Disclosure statement

No potential conflict of interest was reported by the authors.

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Key messages

(1) Weight status has been linked to inflammation in prior research but race-gender differences in these linkages are not understood. This study investigates the associations of body mass index (BMI) and waist circumference (WC) with systemic inflammation markers (fibrinogen, CRP, E-selectin, IL-6) among Black and White men and women in midlife.
(2) WC showed more consistent associations with inflammation markers compared to BMI. It was independently associated with ≥3 inflammation markers among White men and women and among Black women, as well as with one marker (CRP) among Black men. In contrast, BMI related to CRP and fibrinogen among Black men but showed no relationship with inflammation in other groups.
(3) WC is a risk factor for systemic inflammation among White and Black men and women during midlife. BMI as an additional risk factor for Black men.
(4) Mechanisms linking weight status to inflammation may vary by race and gender groups.

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Appendix 1

Lifetime Discrimination

How many times in your life have you been discriminated against in each of the following ways because of such things as your race, ethnicity, gender, age, religion, physical appearance, sexual orientation, or other characteristics?

a. 'You were discouraged by a teacher or advisor from seeking higher education.'
b. 'You were denied a scholarship.'
c. 'You were not hired for a job.'
d. 'You were not given a promotion.'
e. 'You were fired.'
f. 'You were prevented from renting or buying a home in the neighborhood you wanted.'
g. 'You were prevented from remaining in a neighborhood because neighbors made life so uncomfortable.'
h. 'You were hassled by the police.'
i. 'You were denied a bank loan.'
j. 'You were denied or provided inferior medical care.'
k. 'You were denied or provided inferior service by a plumber, care mechanic, or other service provider.'

Coding: Each item is answered by frequency (# of times) of its happening.

Daily Discrimination

How often on a day-to-day basis do you experience each of the following types of discrimination?

a. 'You are treated with less courtesy than other people.'
b. 'You are treated with less respect than other people.'
c. 'You receive poorer service than other people at restaurants or stores.'
d. 'People act as if they think you are not smart.'
e. 'People act as if they are afraid of you.'
f. 'People act as if they think you are dishonest.'
g. 'People act as if they think you are not as good as they are.'
h. 'You are called names or insulted.'
i. 'You are threatened or harassed.'

Coding: 1 Often; 2 Sometimes; 3 Rarely; 4 Never (reverse coded for the purposes of the scale).

Appendix 2

Table A1. Coefficients from multivariate linear regression models of the relationship of BMI and WC with fibrinogen concentrations[a] by race and gender ($N = 1046$).

	White men ($N = 410$)		White women ($N = 490$)		Black men ($N = 58$)		Black women ($N = 117$)	
	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI
Weight status								
BMI, kg/m^2	.62‡	−.05; 1.28	−.48	−1.3; .35	2.47**	.94; 3.99	−.23	−.81; .35
WC, cm	.93**	.30; 1.57	1.78***	1.21; 2.36	1.27‡	−.04; 2.57	2.06**	.71; 3.42

(*Continued*)

Table A1. Continued.

	White men (*N* = 410)		White women (*N* = 490)		Black men (*N* = 58)		Black women (*N* = 117)	
	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI
Perceived discrimination								
Daily discrimination	.31	−1.97; 2.58	−1.01	−2.87; .85	1.40	−2.69; 5.48	−.74	−4.92; 3.45
Lifetime discrimination	−.75	−6.93; 5.44	7.66*	1.46; 13.85	1.38	−8.12; 10.88	7.12‡	−.50; 14.75
Socio-demographic background								
Age, years	.87*	.14; 1.60	1.17*	.50; 1.85	−.05	−2.37; 2.27	.64	−1.61; 2.89
Education, years	−.86	3.94; 2.22	−4.20*	−7.60; −.80	−2.35	−10.43; 5.73	.85	−6.67; 8.37
Annual household income[b]	.94	1.08; 2.97	.86‡	−.10; 1.81	4.95	−9.84; 19.73	.84	−3.85; 5.53
Health-related factors								
Current smoker	20.99‡	−.39; 42.37	−7.39	−30.17; 15.39	−36.19*	−70.88; −1.50	120.48**	31.92; 209.04
Vigorous exercise[c]	−7.61	−25.54; 10.32	1.77	−18.23; 21.76	−43.95	−121.89; 33.98	59.31*	.58; 118.05
Chronic conditions	2.04	−2.34; 6.43	−4.70**	−7.83; −1.56	1.63	−4.81; 8.08	1.60	−5.46; 8.67
Aspirin use	16.61*	.20; 33.02	−12.32	−30.57; 5.94	3.69	−61.55; 68.93	−6.69	−50.19; 36.84

Source: Survey of Midlife in the United States (MIDUS II).
Notes: Coeff. = coefficient; CI = confidence interval; BMI = body mass index; WC = waist circumference.
[a]mg/dL in citrated plasma.
[b]$ logged.
[c]Several times a week, winter and summer.
‡$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed tests).

Table A2. Coefficients from multivariate linear regression models of the relationship of BMI and WC with E-selectin concentrations[a] by race and gender (*N* = 1046).

	White men (*N* = 410)		White women (*N* = 490)		Black men (*N* = 58)		Black women (*N* = 117)	
	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI
Weight status								
BMI, kg/m^2	.06	−.09; .21	−.04	−.16; .07	−.40‡	−.82; .01	−.12	−.29; .06
WC, cm	.32***	.15; .49	.34***	.20; .47	.39‡	−.01; .79	−.15	−.46; .15
Perceived discrimination								
Daily discrimination	.29	−.36; .94	.22	−.25; .69	−.10	−1.35; 1.15	.13	−.95; 1.21
Lifetime discrimination	2.58*	.33; 4.84	.20	−1.16; 1.57	−.29	−2.37; 1.78	1.42	−.59; 3.44
Socio-demographic background								
Age, years	−.11	−.28; .06	−.11	−.27; .05	−.40	−1.11; .30	−.55*	−1.03; −.08
Education, years	−.80‡	−1.66; .60	.11	−.62; .85	1.30	−1.25; 3.85	−.73	−2.65; 1.18
Annual household income[b]	.50**	.22; .79	.16	−.19; .51	1.61	−2.68; 5.91	−1.42*	−2.69; −.15
Health-related Factors								
Current smoker	.01	−7.37; 7.39	3.42	−3.00; 9.84	−5.65	−27.01; 15.70	−37.46***	−53.16; −21.75
Vigorous exercise[c]	−.38	−5.80; 5.03	−.26	−4.79; 4.27	−7.00	−28.10; 14.10	−5.52	−19.03; 7.98
Chronic conditions	.25	−1.38; .88	.13	−.73; .98	−.34	−2.57; 1.89	1.47	−.97; 3.92
Aspirin use	−5.08*	−9.63; −.54	2.28	−1.76; 6.32	3.08	−14.10; 20.26	−1.36	−13.41; 10.69

Source: Survey of Midlife in the United States (MIDUS II).
Notes: Coeff. = coefficient; CI = confidence interval; BMI = body mass index; WC = waist circumference.
[a]ng/mL in serum.
[b]$ logged.
[c]Several times a week, winter and summer.
‡$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed tests).

Table A3. Coefficients from multivariate linear regression models of the relationship of BMI and WC with CRP concentrations[a] by race and gender ($N = 1046$).

	White men ($N = 410$)		White women ($N = 490$)		Black men ($N = 58$)		Black women ($N = 117$)	
	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI
Weight status								
BMI, kg/m^2	.01‡	.00004; .02	−.001	−.01; .01	.03*	.005; .05	−.002	−.01; .005
WC, cm	.03***	.02; .04	.03***	.03; .04	.02**	.01; .04	.03***	.02; .05
Perceived discrimination								
Daily discrimination	.01	−.02; .03	−.01	−.04; .02	.01	−.05; .07	−.02	−.05; .02
Lifetime discrimination	−.02	−.10; .07	.06‡	−.003; .13	−.01	−.13; .11	.04	−.05; .13
Socio-demographic background								
Age, years	.002	−.01; .01	.001	−.01; .01	−.01	−.05; .02	−.02‡	−.04; .003
Education, years	−.05*	−.09; −.005	−.003	−.04; −.04	.006	−.09; .11	−.04	−.13; .05
Annual household income[b]	.02	−.01; .04	.02‡	−.002; .04	−.15	−.33; .03	.02	−.01; .05
Health-related factors								
Current smoker	.38*	.06; .71	−.10	−.42; .23	−.43	−1.68; .81	1.76***	1.18; 2.34
Vigorous exercise[c]	.17	−.08; .42	.33**	−.58; −.08	.59	−1.96; .79	.28	−.46; 1.04
Chronic conditions	.03	−.03; .09	−.03	−.07; .02	.05	−.04; .14	.05	−.04; .14
Aspirin use	.27*	.05; .49	−.05	−.28; .18	.12	−.81; 1.05	.13	−.42; .68

Source: Survey of Midlife in the United States (MIDUS II).
Notes: CRP = C-reactive protein; Coeff. = coefficient; CI = confidence interval; BMI = body mass index; WC = waist circumference.
[a]ug/mL in citrated plasma, logged.
[b]$ logged.
[c]Several times a week, winter and summer.
‡$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed tests).

Table A4. Coefficients from multivariate linear regression models of the relationship of BMI and WC with interleukin-6 concentrations[a] by race and gender ($N = 1046$).

	White men ($N = 410$)		White women ($N = 490$)		Black men ($N = 58$)		Black women ($N = 117$)	
	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI	Coeff.	95% CI
Weight status								
BMI, kg/m^2	.004	.002; .01	−.001	−.005; .003	−.001	−.02; .02	.001	−.003; .005
WC, cm	.01***	.01; .02	.02***	.01; .02	.01	−.003; .02	.02***	.01; .02
Perceived discrimination								
Daily discrimination	.002	−.02; .02	−.002	−.02; .01	−.001	−.04; .03	−.02*	−.04; −.0001
Lifetime discrimination	.04	−.01; .09	.04‡	−.003; .09	−.06	−.15; .03	.03	−.01; .08
Socio-demographic background								
Age, years	.02***	.01; .02	.01***	.01; .02	.01	−.01; .03	.01	−.01; .02
Education, years	.004	−.02; .03	−.01	−.04; .02	.03	−.04; .10	−.01	−.05; .03
Annual household income[b]	−.004	.03; .02	.005	−.01; .02	.03	−.14; .20	.02*	.004; .03
Health-related factors								
Current smoker	.13	−.08; .34	.05	−.23; .13	.22	−.41; .85	1.6***	1.3; 1.99
Vigorous exercise[c]	−.07	−.23; .08	−.20**	−.35; −.06	.02	−.95; .90	.13	−.12; .38
Chronic conditions	.03	−.01; .06	−.002	−.03; .03	.08‡	.01; .16	.03‡	.01; .07
Aspirin use	.11	−.04; .25	.09	−.25; .07	.12	.59; .82	−.11	.35; .13

Source: Survey of Midlife in the United States (MIDUS II).
Notes: Coeff. = coefficient; CI = confidence interval; BMI = body mass index; WC = waist circumference.
[a]pg/mL in serum, logged.
[b]$ logged.
[c]Several times a week, winter and summer.
‡$p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$ (two-tailed tests).