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Adiposity and Leukocyte Telomere Length in US Adults by Sex-Specific Race/Ethnicity: National Health and Nutrition Examination Survey

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Objective: Little is known about the relationship between adiposity and telomere length in the United States population. The objective of our research was to examine this relationship in a representative, socioeconomically and sex-specific, diverse racial/ethnic population in the United States.

Design: Cross-sectional.

Methods: Body mass index (BMI), % total body fat (TBF) and waist circumference (WC) with leukocyte telomere length (LTL) were examined according to sex-specific race/ethnicity using separate adjusted multivariate linear regressions on a sample of 4,919 respondents aged 20-84 years from the National Health and Nutrition Examination Survey's 1999-2002 data.

Results: LTL was shortened .41%, .44%, and .16% in African American (AA) women and was associated with increasing BMI, %TBF, and WC, (β:-.0041, 95%CI: -.0070, -.0012; P=.007; β:-.0044, 95% CI: -.0081, -.0007; P=.02; β:-.0016, 95%CI: -.0031, -.0001; P=.04, respectively). LTL was shortened .29% in White women and was associated with increasing %TBF (β:-.0029, 95%CI: -.0048, -.0009; P=.006). There were no associations among AA men, White men or Mexican American men and women.

Conclusions: LTL is associated with an obesity phenotype in AA women. Tailored intervention is needed to ameliorate the burden of excess adiposity and subsequent cellular aging. *Ethn Dis.* 2020;30(3):441-450; doi:10.18865/ed.30.3.441

Keywords: Race/Ethnicity; Adiposity; Obesity; Leukocyte Telomere Length

Introduction

Trends in rates of risk factors associated with cardiovascular disease (CVD), such as smoking, hypertension, dyslipidemia, have been declining over time among US adults across all racial/ethnic groups.[1] Obesity rates, on the other hand, have increased.[2] Worldwide obesity has nearly tripled since 1975.[3] The United States has among the highest proportion of obesity.[2] More than one-third of US adults are obese.[4] Prevalence rates differ by sex according to race/ethnicity, with African American women experiencing higher rates of obesity.[4] Obesity is a major risk factor for many age-related CVD chronic conditions such as hypertension, type 2 diabetes and dyslipidemia, which increases the risk for heart failure, heart attack and stroke.[5] It is the leading cause of preventable deaths globally and occurs, in part, due to adverse modifiable behaviors such as sedentary lifestyle and unhealthy diet.[3] Obesity has been associated with the shortening of telomeres. [6,7]

Telomeres are the DNA-protein complex at the ends of chromosomes.[8] Telomeres are needed for the replication of DNA and provide protection to chromosomes from nuclease degradation and cellular senescence, which promotes the integrity and stability of chromosomes. During the cellular replication process, telomeres progressively shorten with each cell division. When telomeres shorten to a critical length, replicative senescence is triggered resulting in cell-cycle arrest.[9] In human peripheral leukocytes, telomere shortening has been demonstrated to be a biologic marker for cellular aging as well as a biomarker for age-related diseases such as CVD.[10] Evidence shows that the pathways through which obesity promotes morbidities include increasing systemic inflammation and oxidative stress; inflam-

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mation and oxidative stress have also been linked to telomere attrition.[11,12] Studies have investigated the relationship between adiposity and telomere length. Such studies have produced equivocal results.[2,13-17]

Research indicates that the association between obesity and telomere length differ by sex.[18] Adiposity and telomere length independently also differ by sex and race/ethnicity; in fact, women and African Americans having longer telomere length and a higher prevalence of obesity is observed among African American women.[19,20] However, little is known about the relationship between adiposity and telomere length in the US population. We hypothesized that the association between telomere length and adiposity differed by sex-specific race/ethnicity. The objective of our research was to examine this relationship in a representative, socioeconomically and sex-specific diverse racial/ethnic population in the United States. Findings will provide information about the relationship of cellular aging due to adiposity across major racial/ethnic groups in the United States.

We hypothesized that the association between telomere length and adiposity differed by sex-specific race/ethnicity.

Methods

Study Design and Sampling Procedures

Data were collected from the 1999-2000 and 2001-2002 cycles of the National Health and Nutrition Examination Survey (NHANES). This nationally representative cross-sectional survey and physical examination of the civilian, noninstitutionalized US population has been conducted by the US Centers for Disease Control and Prevention (CDC) since 1960. NHANES utilizes a 4- stage sampling design that includes: 1) primary sampling units (PSUs) consisting of single counties; 2) area segments within PSUs; 3) households within segment areas; and 4) persons within households. During the two cycles (1999-2002), NHANES oversampled low-income individuals, African Americans and Mexican Americans to obtain more accurate estimates in these populations. All respondents aged >20 years during this period were asked to provide DNA specimens to establish a national probability sample of genetic material for future research. DNA from the most recent NHANES is only available in the form of crude lysates of cell lines thereby precluding the assay of leukocyte telomere length (LTL). However, DNA collected during 1999-2002 is purified from whole blood thus facilitating the assay of LTL. Pooled data are available for public download from https://wwwn.cdc.gov/nchs/nhanes/default.aspx.[21]

Of the 10,291 respondents eligible to provide DNA, 7,825 provided DNA and consented to future genetic research. We excluded 653 respondents whose self-reported race/ethnicity was "other" or "other Hispanic," since our goal was to examine more discrete self-reported racial/ethnic groups (ie, White, African American, Mexican American). We also excluded 225 respondents aged >85 years because of survival bias among the extreme elderly.[22] An additional 2,037 were excluded due to missing data on one or more variables in the models, resulting in a final sample size of 4,919. There were no significant sociodemographic differences between the full sample and the final sample. Sampling weights were used to address oversampling and non-response bias and to ensure that estimates were representative of the general US population. Written informed consent was obtained from each participant in the study. Human subject approval was provided by the institutional review board (IRB) at the CDC and the study protocol was approved by the IRB of the National Institutes of Health. All procedures were in accordance with the ethical standards of the IRB and the Helsinki Declaration of 1975, as revised in 2000.

Measures

Outcome

Leukocyte telomere length was the outcome variable. Aliquots of purified DNA were provided by the laboratory of the CDC. DNA was isolated from whole blood using the Puregene (D-50K) kit protocol and stored at -80 C. The LTL assay was performed in the laboratory of Dr. Elizabeth Blackburn at the University of California, San Francisco, using

the quantitative polymerase chain reaction (PCR) method to measure telomere length relative to standard reference DNA.[23] This method measures LTL as a ratio (T/S) of telomere repeat length (T) to copy number of a single copy gene, 36B4(S), within each sample. Each sample was assayed twice. T/S ratios that fell within the 7% variability range were accepted; the average of the two was taken as the final value. A third assay was run for samples with >7% variability and the average of the two closest T/S values was used. The inter-assay coefficient of variation was 4.4% indicating a high degree of similarity between assays.

Primary Predictors

Body mass index (BMI), estimated % total body fat, and waist circumference were analyzed separately as primary predictor measures of adiposity. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2) using a calibrated electronic digital scale and a stadiometer. Estimated % total body fat was assessed using dual-energy X-ray absorptiometry of the whole body that lasted 3 minutes (Hologic scanner, QDR-4500, Bedford, MA, USA). Total % body fat was calculated as total body fat mass divided by total mass x 100. Waist circumference was measured in centimeters using a tape measure around the trunk, at the iliac crest, crossing at the midaxillary line. The details of these assays have been described elsewhere.[24]

Confounding Variables

All models were adjusted for confounding variables that may impact the relationship between adiposity measure and LTL, including adverse health behaviors; these include smoking, drinking, physical activity, and diet. Smoking was measured as cumulative exposure to tobacco smoke in pack years, calculated as the average number of cigarettes smoked per day times the number of years smoked divided by 20 (number of cigarettes in one pack).[25] Dummy variables were <30 pack years, 30-<60, ≥60 pack years and never smoked was coded as the reference. Drinking was based on daily alcohol consumption defined as heavy, moderate and abstainer.[26] Heavy drinkers were defined as women reporting having drunk ≥2 alcoholic beverages in the past 12 months per day and men reporting having drunk ≥3 alcoholic beverages in the past 12 months per day. Moderate drinkers were defined as women reporting <2 drinks per day in the past 12 months and men reporting <3 drinks per day in the past 12 months. Men and women reporting no alcoholic beverages in the past 12 months per day were the reference and defined as abstainers. Physical activity was based on guidelines provided by the Department of Health and Human Services.[27] Respondents met or exceeded recommended guidelines if they reported ≥150 to ≥300 minutes per week of physical activity, such as brisk walking, gardening, and muscle-strengthening based on total number of minutes reported for each activity. Those reporting <150 minutes of physical activity per week were below the recommended guidelines. Diet was based on The Healthy Eating Index (HEI) developed by the US Department of Agriculture in 2005.[28] The score is the sum of 10 components representing different aspects of a healthy diet. Each component of the index has a maximum score of 10 and a minimum score of zero. The maximum overall score for the 10 components combined is 100. An overall index score ≥80 implies a "good" diet, an index score between ≥51 and 80 implies a diet that "needs improvement," and an index score <51 implies a "poor" diet.

Other confounding variables included age in years at the time of the survey, age-squared, socioeconomic status based on Poverty Income Ratio (PIR), adiposity related health outcomes, markers of inflammation and oxidative stress, and characteristics of the blood from which DNA was extracted. The use of age along with an age-squared term is important when analyzing LTL given the strength of its association with age and the potential for nonlinearity in this association. PIR was calculated as the ratio of income to the poverty threshold for a household of a given size and composition. PIR values <1.00 are below the official poverty threshold as defined by the US Census Bureau.[29] Adiposity-related health status was based on respondents' answers to the questions: "Have you ever been told by a doctor or other health professional that you had hypertension, also called high blood pressure?" and "Have you ever been told by a doctor or health professional that you have diabetes or sugar diabetes?" Markers of inflammation and

oxidative stress included C-reactive protein (CRP) and gamma glutamyltransferase (GGT) measured from serum. Characteristics of the blood samples from which DNA was extracted included white blood cells (μL), lymphocytes (%), monocytes (%), neutrophils (%), eosinophils (%), and basophils (%).

Stratification Variable

We conducted separate multivariate linear regression models for BMI, % total body fat and waist circumference for each racial/ethnic group stratified by sex. Race/ethnicity was based on self-report as non-Hispanic White, non-Hispanic Black and Mexican American hereafter referred to as White, African American and Mexican American.

Operation of Variables

Pack years smoked, drinking level per day, hypertension status and type 2 diabetes status were entered as categorical variables. Physical activity level per week, HEI, age, age-squared, PIR, CRP, GGT and the characteristics of blood were entered as continuous variables.

Statistical Methods

We performed descriptive analysis on the total sample and stratified by race/ethnicity according to study variables. Continuous variables are presented as means ± standard deviation based on ANOVA and categorical variables as percent based on chi-square. Leukocyte telomere length was transformed by natural logarithm to improve the normality of the distribution prior to regression modeling. We report the percentage change in the average value of telomere length for a one-unit change in a predictor variable based on the beta estimate for telomere length as the outcome. Because the absolute value of all parameter estimates was <.10, the percentage change in the outcome was estimated by exponentiating the logged coefficient, subtracting one and multiplying the parameter estimate (β) by 100.[30] All regression models accommodated the complex sampling design of NHANES by incorporating strata and PSU indicators, as well as sample weights for the genetic subsample.[31]

An investigation of residual diagnostics inspection showed that the models met the assumption of linear regression. We also checked for potential outliers using regression diagnostics, as well as multicollinearity and found no evidence of variance inflation more than 5. All analyses were conducted using SAS version 9.3.[32] A two-tailed level of significance was established as P≤.05.

Results

Descriptive results in Table 1 reveal the average mean age for the total sample; younger age was observed among African Americans and Mexican Americans. Mean body mass index and mean waist circumference were slightly higher among African Americans. Compared with Whites, fewer African Americans and Mexican Americans were non-smokers. Fewer African Americans were heavy drinkers compared with Mexican Americans. More African Americans and Mexican Americans, compared with Whites, were below the recommended level of physical activity per week. The mean HEI score was somewhat comparable across groups, although slightly lower in African Americans. However, of the three groups, the prevalence of hypertension was highest among African Americans and lowest among Mexican Americans. Mexican Americans, as well as African Americans, had a higher prevalence of type 2 diabetes compared with Whites. African Americans had longest mean LTL compared with the total sample, Mexican Americans, and Whites.

Adiposity and LTL by Sex-Specific Race/Ethnicity

Findings comparing differences in the association of LTL and adiposity measures according to sex-specific race/ethnicity are presented in Table 2 (men) and Table 3 (women). There was no significant association between any of the adiposity measures and LTL in African American and Mexican American men (Table 2). Increasing BMI was marginally associated with .26% shorter LTL in White men (P=.06) as well as a marginal correlation with increasing waist circumference (P=.06). Table 3 shows that African American women experienced .41%, .44%, and .16% shorter LTL associated with increasing BMI, % total body fat and waist circumference (P=.007, .02, .04, respectively). Increasing % total body fat resulted in .29% shorter LTL in White women (P=.006). There was no association with any of the adiposity measures in Mexican American women.

Table 1: Descriptive statistics of the characteristics of the participants in the National Health and Nutrition Examination Survey (NHANES) 1999-2002 (N=4919)

	Total, N=4919		African American, N=919		Mexican American, N=1262		White, N=2738		
	%	Mean(SD)	%	Mean(SD)	%	Mean(SD)	%	Mean(SD)	P
Age, years (20 to <85)		46.0(.42)		42.4(.48)		38.3(.56)		47.1(.46)	<.0001
Sex									.003
Women	50.8		53.6		45.8		50.9		
Men	49.2		46.4		54.2		49.1		
Poverty income ratio									<.0001
Below poverty	17.6		32.6		31.3		14.6		
Meet and above poverty	82.4		67.4		68.7		85.4		
Body mass index, kg/m^2		27.9(.16)		29.5(.31)		28.2(.24)		27.6(.18)	<.0001
% Total body fat		33.7(.19)		33.1(.39)		33.6(.38)		33.8(.23)	<.0001
Waist circumference, cm		95.6(.38)		96.2(.62)		94.7(.54)		95.6(.45)	<.0001
Pack years smoked									<.0001
≥60	3.7		3.2		1.8		9.8		
30 to <60	5.0		4.2		3.0		6.0		
<30	36.8		31.4		33.2		31.2		
0	54.5		61.2		62.0		53.0		
Drinking level per day									<.0001
Heavy	20.9		17.6		33.3		20.2		
Moderate	50.1		38.6		32.7		53.2		
Abstainer	29.0		43.8		34.0		26.6		
Physical activity recommendation level per week									<.0001
Below	38.5		56.4		53.4		35.0		
Meet / exceed	61.4		43.5		46.6		64.0		
Healthy Eating Index Score (2005)		50.7 (.46)		48.2(.67)		51.5 (.45)		51.0 (.55)	<.0001
Hypertension	25.1		33.3		15.1		25.0		<.0001
Type 2 Diabetes	5.3		8.4		6.9		4.8		<.0001
CRP (mg/dL)		.39(.01)		.51(.04)		.43(.04)		.38(.01)	<.0001
GGT (U/L)		30.7 (.70)		41.8 (2.7)		34.8 (1.2)		29.0 (.74)	<.0001
White blood cell count (SI)		7.1 (.06)		6.4 (.06)		7.3 (.06)		7.1 (.06)	<.0001
Lymphocyte (%)		29.8 (.21)		35.3 (.24)		30.5 (.21)		29.1 (.23)	<.0001
Monocyte (%)		8.1 (.04)		8.4 (.10)		7.7 (.10)		8.1 (.04)	<.0001
Neutrophils (%)		58.6 (.22)		52.8 (.29)		58.4 (.32)		59.3 (.24)	<.0001
Eosinophils (%)		2.7 (.02)		2.7 (.06)		2.6 (.10)		2.7 (.03)	<.0001
Basophils (%)		.66(.02)		.68(.01)		.61(.01)		.66(.02)	<.0001
Leukocyte telomere length (T/S Ratio)		1.06(.01)		1.13(.02)		1.04(.02)		1.05(0.02)	<.0001

SD, standard deviation; CRP, C-reactive protein; GGT, gamma glutamyltransferase

Discussion

The objective of our study was to assess the association between adiposity measures and LTL among African American, Mexican American and White men and women in the United States. We found no association with any of the adiposity measures and telomere length in African American, Mexican American or White men. Men in these groups tend to be leaner compared with women in their counterparts.[19] We observed opposite findings for women. African American women experienced shorter telomere length related to increases in each of the adiposity measures. This finding is not surprising given African American women have the highest prevalence of obesity compared with men and women in other racial/ethnic groups.[19] One theory suggests Afri-

Table 2. Multivariable associations of adiposity measures with leukocyte telomere length in men (N=2462) according to race/ethnicity: National Health and Nutrition Examination Survey, (NHANES) 1999-2002

	African American[a], n=444		Mexican American[a], n=623		White[a], n=1395	
Adiposity	β (95% CI)	P	β (95% CI)	P	β (95% CI)	P
BMI	-.0016 (-.0060, .0028)	.46	-.0025 (-.0079, .0029)	.34	-.0026 (-.0053,.0001)	.06
% total body fat	-.0012 (-.0055,.0032)	.58	-.0041 (-.0092,.0011)	.11	-.0018 (-.0042,.0005)	.12
Waist circumference	-.0008 (-.0025,.0010)	.37	-.0009 (-.0029,.0011)	.36	-.0011 (-.0022, .0001)	.06

a. Adjusted for age, age-squared, PIR (poverty income ratio), hypertension, type 2 diabetes, CRP (c-reactive protein), GGT (gamma glutamyltransferase), white blood cells, lymphocytes, monocytes, neutrophils, eosinophils, basophils, pack years smoked, level of daily alcohol consumption, level of weekly physical activity, HEI (healthy eating index).

can American women are a unique group susceptible to obesity due to mechanisms associated with dietary preferences and early childbearing.[33] It may also be due to differences in metabolism and perceptions about an ideal body.[34] As with Mexican American men, we found no correlation in Mexican American women associated with any of the exposure measures and telomere length.

We found no association with any of the adiposity measures and telomere length in African American, Mexican American or White men.

We were surprised to find the lack of association in Mexican American women given the high prevalence of obesity in Hispanic women.[19] National prevalence rates are generally based on aggregate data of Hispanics in general and does not consider ethnic differences within Hispanics. The NHANES does collect information on "other Hispanic" but does not distinguish ethnicity within Hispanics other than Mexican American. Our findings on Mexican American women may reflect ethnic heterogeneity and may not be indicative of obesity status in Mexican American women. Only % total body fat was correlated with shorter telomere length in White women. This is not a unique finding given White women have an overall lower prevalence of obesity. [19]

There are conflicting findings regarding the relationship between adiposity and telomere length in the literature. Findings from two studies using NHANES data similarly revealed that an increase in BMI, waist circumference and % total body fat was associated with a decrease in LTL in the aggregate sample, which reflect our findings.[2,35] An investigation of the Cardiovascular Health Study, on the other hand, found no association between telomere length and BMI and waist circumference.[15] A study by Maceneay et al of 67 middle-aged and older adults also revealed no correlation between normal and overweight/obese BMI parameters with telomere length.[36] Few studies have compared the association between adiposity and telomere length according to race/ethnicity and concomitant sex. We could find only one study of 317 White and African American adults residing in South Carolina.[37] These investigators found no relationship between BMI and visceral fat based on race/ethnicity or sex in their study population of Whites and African Americans. These conflicting findings between adiposity and telomere length may be due to several factors, including study design, participant characteristics, a small study sample and other limitations.

Our findings, based on a representative, diverse racial/ethnic US sample, revealed differing association between adiposity indices and telomere length according to sex. However, we did not investigate possible mechanisms. Intense discussion has emerged on the appropriateness of comparing adiposity measures, such as BMI, according to race/ethnicity as an indicator of obesity due to underlying differences in body shape composition. A relatively recent

Table 3. Multivariable associations of adiposity measures with leukocyte telomere length in women (N=2457), according to race/ethnicity: National Health and Nutrition Examination Survey, NHANES 1999-2002

	African American[a], n=475		Mexican American[a], n=639		White[a], n=1343	
Adiposity	β (95% CI)	P	β (95% CI)	P	β (95% CI)	P
BMI	-.0041 (-.0070, -.0012)	.007	-.0003 (-.0037, .0031)	.85	-.0017 (-.0039, .0005)	.12
% total body fat	-.0044 (-.0081, -.0007)	.02	-.0028 (-.0069, .0012)	.16	-.0029 (-.0048, -.0009)	.006
Waist circumference	-.0016 (-.0031, -.0001)	.04	.0003 (-.0015, .0020)	.77	-.0007 (-.0017, .0004)	.19

a. Adjusted for age, age-squared, PIR (poverty income ratio), hypertension, type 2 diabetes, CRP (c-reactive protein), GGT (gamma glutamyltransferase), white blood cells, lymphocytes, monocytes, neutrophils, eosinophils, basophils, pack years smoked, level of daily alcohol consumption, level of weekly physical activity, HEI (healthy eating index).

study by Heymsfield and colleagues based on NHANES data reported men and women of the same age, weight and height but differing in race/ethnicity have significantly different levels of adiposity.[38] They argue differences arise due to variation between groups in relative body shape and aspects of body composition.

Thus, comparing race/ethnicity and sex-specific race/ethnicity with adiposity differences and telomere length may be challenging due to several biologic and social factors. For instance, racial admixture is well-established among African and Mexican Americans and, therefore, may affect body composition.[39,40] Genetic ancestry has also been demonstrated to be predictive of telomere length with African Americans having longer telomeres compared with Whites.[41] Additionally, our findings were based on cross-sectional data and the racial/ethnic groups examined were undoubtedly exposed to different socioeconomic and environmental factors until the time they were studied as part of NHANES. Lower socioeconomic status has been determined to be associated with shorter telomere length.[42] We controlled for socioeconomic status based on PIR in the models but it was not a significant factor. An alternative measure of socioeconomic status, such as level of education, may produce different results. We did not control for body composition and indices of ancestry and environment were not available in NHANES. Therefore, body composition, genetic ancestry, environmental and socioeconomic status may be other mechanisms associated with the relationship between adiposity and LTL. The differences in the association that we observed by sex may be due to these same factors, as well as hormonal and perception about ideal body weight.[34,43] Future research should be designed to elucidate genetic ancestry, body composition, social and environmental factors as possible mechanisms associated with adiposity and LTL based on race/ethnicity and sex.

Study Limitations

There are some caveats to our study that require consideration. First, we do not know the direction of the relationship between adiposity and LTL. Some researchers argue that selective adoption may be a causal factor related to telomere length due to the type and direction of exposure.[1] Selective adoption could occur either because telomere length directly affects behavior or because behavior affects telomere length, or both are affected by a third variable, such as exposure to early-life adversity. In addition, telomere senescence occurs over time and may present in some cases with a U-shaped pattern.[44] The NHANES is a cross-sectional survey and changes in LTL may come before exposure. Our observations may also be due to differing telomere length at birth or childhood.[45] Therefore, the correlations we observed should not be interpreted as causal. One way to address these important issues is to design longitudinal analysis to measure the bi-directional effect of differences in LTL and adiposity over time before obesity occurs.

Second, although we adjusted for potential confounders, other unmeasured confounding factors may exist resulting in "omitted variable bias" such as heritability, ancestry, menopausal status, adiposity biomarkers (ie, leptin and adiponectin) and sex hormones that may affect our findings.

Third, we measured telomere

length only in leukocytes. Whether our findings can be extrapolated to other tissues is unclear. However, studies have demonstrated robust correlations between LTL and telomere length in other tissues.[46] Finally, differences in assay methodologies may present a problem. Telomere length was assayed using qPCR. Southern blot is another assay method. These differing techniques may yield discrepancies in our findings. However, a study from two well-established labs comparing qPCR and Southern blot report both displayed highly reproducible results for the correlations between results obtained by either method on two occasions.[23] Finally, we excluded 2,037 participants due to missing data; this represents >25% of participants who provided DNA. Inclusion of these participants may have produced different findings. However, this is highly unlikely given there was no significant sociodemographic differences between the full and study samples.

Despite these limitations, our study has many strengths. Because the study is based on a racially and ethnically representative sample of US adults, findings are generalizable. To our knowledge, it is among the largest and first study to investigate the association of adiposity and telomere length according to sex-specific race/ethnicity.

Conclusion

Several studies have investigated the relationship between adiposity and telomere length.[7,13-15,35-37,43] But few have assessed such an association based on sex-specific race/ethnicity in the United States. Our findings reveal no relationship observed in African American and White men, nor Mexican American men and women. White women experienced shorter telomere length only due to increasing % total body fat. African American women, on the other hand, experienced shorter telomere length associated with increases in each of the adiposity measures and is consistent with this group having a higher prevalence of obesity, thereby suggesting an obesity-phenotype.[6] Tailored interventions are needed to ameliorate the burden of excess adiposity and subsequent cellular aging in African American women.

Overall, our findings suggest LTL may be a useful biomarker associated with adiposity and age-related health. However, more research is needed to examine the relationship in US men and women to better understand differences in body composition, socioeconomic status, environment and genetic ancestry as possible mechanisms.

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Conflict of Interest

No conflicts of interest to report.

Author Contributions

Research concept and design: Davis; Acquisition of data: Xu; Data analysis and interpretation: Davis, Xu, Khan, Gaye; Manuscript draft: Davis, Xu, Khan, Gaye; Statistical expertise: Xu, Khan, Gaye; Supervision: Davis

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