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BRIEF REPORT

Leukocyte Telomere Length and Childhood Onset of Systemic Lupus Erythematosus in the Black Women's Experiences Living with Lupus Study

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Objective. The study objective was to compare leukocyte telomere length (LTL) among patients with systemic lupus erythematosus (SLE) diagnosed in childhood versus adulthood.

Methods. Data are from the Black Women's Experiences Living with Lupus (BeWELL) study. Multivariable linear regression analyses that examined childhood diagnosis of SLE (diagnosed before 18 years of age), age, and their interaction in relationship to LTL were conducted, adjusting for a range of demographic, socioeconomic, and health-related covariates.

Results. The total analytic sample size was 415. Forty participants (9.6%) were diagnosed in childhood. There was no main effect of childhood diagnosis on LTL (b = 0.007; 95% confidence interval [CI]: −0.089 to 0.103). However, the interaction between age and childhood diagnosis was significant (b = −0.008; 95% CI: −0.016 to −0.001), indicating a steeper inverse association between age and LTL among those diagnosed in childhood compared with those diagnosed in adulthood. This interaction remained statistically significant (*P* = 0.024) after controlling for disease duration measured dichotomously (less than 10 years vs. 10 years or more); it was marginally significant (*P* = 0.083) when controlling for disease duration measured continuously.

Conclusion. This cross-sectional analysis suggests that Black women with childhood-onset SLE may undergo accelerated LTL shortening compared with their adult-onset counterparts. This relationship persisted even after controlling for differences in SLE damage and disease duration. These findings inform research on immunosenescence mechanisms of SLE.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multisystem disease of immune dysregulation that affects between 161,000 and 322,000 adults in the United States (1). SLE disproportionately affects Black women, who have higher prevalence, an earlier age of onset, and greater disease severity, including morbidity (2–5). Although differences in socioeconomic characteristics account for some of these disparities, research has increasingly pointed to qualitatively unique sources of psychosocial stress experienced by Black women, such as those tied to racism, which increase vulnerability to severe disease (6–8). Black Americans are also more likely to be diagnosed in childhood compared with White Americans (9).

Childhood-onset SLE represents 10% to 20% of all cases of SLE in the United States (10). Childhood-onset SLE is more severe than adult-onset SLE in both disease activity and damage (11,12), degree of organ involvement (13–15), and rates of

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hospitalization (12,15–17). Childhood-onset SLE is an independent predictor for mortality, even when controlling for cumulative damage due to younger age at diagnosis (18). It is unclear, however, whether variations in severity of disease course between childhood-onset and adult-onset SLE are attributable primarily to differences in disease duration or whether their pathogenesis may be fundamentally different.

Leukocyte telomere length (LTL) has been posited to reflect immune system aging of B and T cells, and short LTL has been associated with both chronic health conditions mediated by inflammatory pathways as well as all-cause mortality (19). With increased T-cell activity as part of the disease process, patients with SLE have shorter T-cell telomere length compared with control T cells (20). Additionally, prior studies have found evidence for shorter LTL among patients with SLE compared with the general population (20–22). Given the role of LTL in immune system aging and its relationship to SLE, it is possible that LTL is involved in differences in disease severity between childhood-onset SLE and adult-onset SLE. To our knowledge, no studies to date have compared LTL among patients with SLE diagnosed in childhood versus adulthood. In this study, we sought to compare LTL between childhood-onset patients with SLE and adult-onset patients with SLE. Our aim was to investigate whether telomere length is associated with differences in disease severity between childhood-onset and adult-onset patients with SLE. We hypothesized that, because childhood-onset SLE is more severe, patients with SLE diagnosed in childhood would have shorter telomeres compared with patients with SLE diagnosed in adulthood.

PATIENTS AND METHODS

Study design and procedures. Data are from the Black Women's Experiences Living with Lupus (BeWELL) study (23). BeWELL represents one of the largest studies on the social epidemiology of SLE (24–26). A total of 438 Black women living in the Atlanta, Georgia, metropolitan area with a validated diagnosis of SLE (a score of 4 or more on the American College of Rheumatology SLE criteria or three criteria with a diagnosis of SLE by a board-certified rheumatologist) were recruited between April 2015 and May 2017. Trained field researchers collected dried blood spots (DBSs) from participants, a minimally invasive alternative to venipuncture that involves pricking the finger with a microlancet and applying approximately 50 μL drops of blood to filter paper (27). Blood samples were allowed to dry and were stored at –80°C. Field researchers also collected survey measures. Protocols and procedures were approved by the Institutional Review Board of Emory University. All study participants provided informed signed consent.

LTL assay. LTL assays were conducted by the Blackburn Laboratory at the University of California, San Francisco, using previously published protocol (28) and the the QIAmp DNA investigator kit (GIAGEN; category # 56504). Genomic DNA was extracted from DBSs stored at –80°C and quantified with the Quant-iT PicoGreen double stranded DNA Assay Kit (ThermoFisher Scientific Category #P7589). The average DNA yield from six 3-mm punches was 175 ng ± 97 ng in 50 μL elution buffer.

The primers for the telomere polymerase chain reaction (PCR) were *tel1b* [5′-CGGTTT(GTTTGG)$_5$GTT-3′], used at a final concentration of 100 nM, and *tel2b* [5′-GGCTTG(CCTTAC)$_5$ CCT-3′], used at a final concentration of 900 nM. The primers for the single-copy gene (human beta-globin) PCR are *hbg1* [5′ GCTTCTGACACAACTGTGTTCACTAGC-3′], used at a final concentration of 300 nM, and *hbg2* [5′-CACCAACTTCATCCACGTT-CACC-3′], used at a final concentration of 700 nM. The final reaction mix contained 20 mM Tris–HCl (pH 8.4), 50 mM KCl, 200 μM each deoxynucleoside triphosphate, 1% Dimethylsulfoxide, 0.4× Syber Green I, 22 ng *Escherichia coli* DNA, 0.4 Units of Platinum Taq DNA polymerase (Invitrogen, Inc.), and approximately 2.5 ng of genomic DNA per 11-μL reaction. Tubes containing 26, 8.75, 2.9, 0.97, 0.324, and 0.108 ng of a reference DNA (human genomic DNA from buffy coat; Sigma category #11691112001) were included in each PCR run so that the quantity of targeted templates in each research sample can be determined relative to the reference DNA sample by the standard curve method. The same reference DNA was used for all PCR runs. Assays were run in triplicate wells on 384-well assay plates in a Roche LightCycler 480. The average concentrations of telomere DNA (T) and single copy gene (S) from the triplicate wells were used to measure the T/S ratios after a Dixon's Q test to remove outliner wells from the triplicates. The T/S ratio for each sample was measured twice. When the duplicate T/S value and the initial value varied by more than 7%, the sample was run a third time, and the two closest values were reported. The average coefficient of variation for this study was 2.3% ± 1.6%.

Telomere length assay for the entire study was performed using the same lots of reagents. DNA from all time points of the same participant was extracted in the same batch (DNA extraction batch size is up to 24 samples) and assayed in the same batch (assay batch size is up to 96 samples) to address potential batch differences. Laboratory personnel who performed the assays were provided with de-identified samples and all demographic and clinical data were anonymized.

Sociodemographic characteristics. Age, relationship status, insurance status, and highest education level achieved were self-reported. The income-to-poverty ratio was calculated as the ratio of annual household income to the poverty threshold according to family size and number of children in the household. Higher ratios reflect lower levels of poverty.

Health-related variables. Disease duration was based on self-reported date of diagnosis (month and year), age of diagnosis and date of interview, or years since diagnosis, whichever was volunteered by the participant. Given that patients with SLE

Table 1. Descriptive characteristics of Black women with SLE in the BeWELL study

Characteristic	Childhood-onset SLE (n = 40)	Adult-onset SLE (n = 375)	Total sample[a] (N = 415)
Age in years, mean (SD)	38.28 (12.5)	47.29 (11.4)	46.42 (11.8)
Years since diagnosis, mean (SD)	23.88 (13.3)	14.80 (9.3)	15.68 (10.1)
Disease damage[b], mean (SD)	2.98 (2.6)	2.70 (2.5)	2.73 (2.5)
Body mass index (kg/m^2), mean (SD)	29.68 (8.0)	31.01 (8.1)	30.88 (8.1)
LTL (T/S), mean (SD)	1.46 (0.4)	1.39 (0.3)	1.40 (0.3)
Relationship status, N (%)			
Married or marriage-like	12 (30.0)	178 (47.5)	190 (45.8)
Romantic relationship	3 (7.5)	23 (6.1)	26 (6.3)
Divorced/separated or widowed	5 (12.5)	85 (22.7)	90 (21.7)
Single, never married	20 (50.0)	89 (23.7)	109 (26.3)
Education, N (%)			
Less than high school	2 (5.0)	32 (8.5)	34 (8.2)
High school	8 (20.0)	67 (17.9)	75 (18.1)
Some college	22 (55.0)	165 (44.0)	187 (45.1)
Bachelor's degree or higher	8 (20.0)	111 (29.6)	119 (28.7)
Income-poverty ratio, N (%)			
<1.00	20 (50.0)	111 (29.6)	131 (31.6)
1.00-1.99	11 (27.5)	127 (33.9)	138 (33.3)
2.00-4.00	6 (15.0)	92 (24.5)	98 (23.6)
>4.00	3 (7.5)	45 (12.0)	48 (11.6)
Insurance status, N (%)			
Public	14 (35.0)	138 (36.8)	152 (36.6)
Private	20 (50.0)	195 (52.0)	215 (51.8)
None	6 (15.0)	42 (11.2)	48 (11.6)
Current smoker, N (%)	3 (7.5)	57 (15.2)	60 (14.5)

Abbreviations: BeWELL, Black Women's Experiences Living with Lupus; LTL, leukocyte telomere length; SLE, systemic lupus erythematosus.
[a] Total sample was age restricted to participants <70 years old.
[b] Disease damage assessed with the Brief Index of Lupus Damage.

with onset prior to the age of 18 years are typically seen first by a pediatric rheumatologist (where available), disease was dichotomized as childhood onset if it occurred before the age of 18 and adult onset of it occurred at age 18 or older. Disease damage was reported using the self-administered version of the Brief Index of Lupus Damage (BILD), a validated measure of irreversible organ damage across 12 systems, scored from 0 to 30, with higher scores indicating greater damage (29,30). Body mass index (BMI) was calculated by dividing body weight (in kilograms) by height (in meters) squared (31). Current smoking status was self-reported (yes or no).

Data analysis. Multivariable linear regression analyses were conducted examining childhood diagnosis of SLE in relationship to LTL. All models were adjusted for relationship status, education, income-to-poverty ratio, insurance status, disease damage (BILD), BMI, and current smoking status. Seven participants with missing LTL were excluded from analyses. Analyses

Table 2. Regression results examining associations between childhood-onset SLE and LTL in the BeWELL study (N = 415)

Variable	Model 1		Model 2		Model 3		Model 4	
	b	95% CI	b	95% CI	b	95% CI	b	95% CI
Age	−0.008***	(−0.011 to −0.005)	−0.007***	(−0.010 to 0.004)	−0.006***	(−0.009 to −0.003)	−0.006***	(−0.009 to 0.003)
Childhood diagnosis	0.007	(−0.089 to 0.103)	0.339*	(0.027 to 0.652)	0.373*	(−0.060 to −0.687)	0.314†	(−0.002 to −0.629)
Age × childhood diagnosis			−0.008*	(−0.016 to −0.001)	−0.009*	(−0.016 to −0.001)	−0.007†	(−0.015 to 0.001)
≥10-y disease duration					0.063†	(−0.002 to 0.127)	—	—
Disease duration (continuous)					—	—	−0.002	(−0.006 to 0.002)

Abbreviations: BeWELL, Black Women's Experiences Living with Lupus; BILD, Brief Index of Lupus Damage; CI, confidence interval.
Note: All models adjusted for relationship status, education, income-to-poverty ratio, insurance status, disease damage (BILD), and current smoking status.
† $P < 0.10$.
* $P < 0.05$. *** $P < 0.001$.

were also restricted to women less than 70 years of age so that the age ranges of those diagnosed in childhood and those diagnosed in adulthood were equivalent, which resulted in excluding nine additional participants. Seven participants missing data on at least one covariate were also excluded, resulting in a final analytic sample size of n = 415.

RESULTS

A total of 40 participants (9.6%) were diagnosed in childhood; the oldest of these participants was 69 years of age. Descriptive characteristics can be found in Table 1. Independent sample *t* tests and χ^2 tests were used to assess differences in sociodemographic and health-related factors between the childhood- and adult-onset SLE cohorts. The average age of the childhood-onset SLE cohort was 38.3 years, whereas the average age of the adult-onset SLE cohort was 47.3 years ($t(413) = 4.73$; $P < 0.001$). The childhood-onset SLE cohort had longer disease duration ($t(413) = -5.62$; $P < 0.001$) than the adult-onset SLE cohort. Those diagnosed in childhood were also more likely to be single compared with their adult-onset counterparts ($\chi^2(3) = 13.73$; $P < 0.01$). These groups did not significantly differ in mean disease damage ($t(413) = -0.67$; $P = 0.50$) or mean LTL ($t(413) = -1.25$; $P < 0.21$). They also did not differ across any of the other sociodemographic or health-related covariates included in this study.

Results of multivariable linear regression analyses are shown in Table 2. As expected, age had a significant inverse association with LTL (b = −0.008; 95% confidence interval [CI]: −0.011 to −0.005). However, there was no main association between childhood diagnosis and LTL (Model 1: b = 0.007; 95% CI: −0.089 to 0.103). The interaction between age and childhood diagnosis was significant (Model 2: b = −0.008; 95% CI: −0.016 to −0.001). The association between age and LTL was more negative among those diagnosed in childhood compared with those diagnosed in adulthood (Figure 1). This interaction remained significant after controlling for disease duration measured dichotomously (less than 10 years vs. 10 years or more) (Model 3: b = −0.009; 95% CI: −0.016 to −0.001). When disease duration was included in the model as a continuous variable, the interaction between age and childhood-onset SLE was significant at the trend level (Model 4: b = −0.007; 95% CI: −0.015 to 0.001; $P = 0.083$).

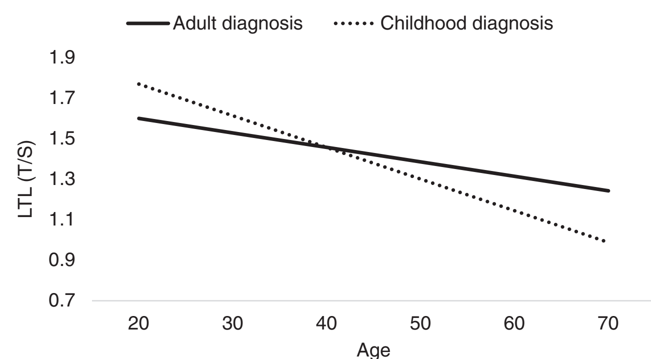

Figure 1. Interaction between leukocyte telomere length and age in childhood-onset versus adult-onset SLE. LTL, leukocyte telomere length; SLE, systemic lupus erythematosus.

DISCUSSION

This cross-sectional analysis suggests that Black women with childhood-onset SLE may undergo accelerated LTL shortening compared with their adult-onset counterparts. With increasing age across this study population, the telomere length of childhood-onset patients with SLE was progressively shorter than that of adult-onset patients with SLE (Figure 1). Our results suggest that there may be fundamental variation in cell aging dynamics between childhood- and adult-onset SLE, which is not solely attributable to differences in disease duration.

These findings should be considered in the context of a few study limitations. Owing to the cross-sectional nature of this study, we were unable to determine whether LTL shortening is an underlying cause of differences in disease severity between childhood- and adult-onset SLE and/or whether it is a consequence of differences in disease severity. If the former is true, further study of this causal relationship could shed light on disease mechanisms; if the latter, LTL could be evaluated as a biomarker for disease progression. LTL shortening could reflect the longitudinal effect of exposure to the stressors associated with having SLE. It is likely that LTL and SLE severity have a reciprocal relationship. However, as mentioned, these analyses used cross-sectional data, and we could not assess the causal direction of associations, which limits the clinical utility of measuring LTL in patients with SLE until a further understanding of the relationship is achieved. Additionally, self-reported measures of age at diagnosis, organ damage, and several covariates may be subject to recall and/or other forms of reporting bias. There is also potential that we did not adjust for other important potential confounders. The findings on the relationship between LTL and disease onset in SLE in this cohort of Black women cannot necessarily be extrapolated to male patients with SLE or to non-Black women with SLE. Without a non-SLE control group, assessing LTL shortening's direct relationship to SLE compared with other confounders is not possible in this study. Analysis of LTL prospectively over time would help to better establish causal relationships with onset of SLE disease, SLE disease activity, and cumulative disease damage.

Despite these limitations, our findings may advance research on immunosenescence mechanisms of SLE among Black women, a population disproportionately impacted by SLE. This research highlights one particular channel that may be involved in SLE disparities, which studies have shown is susceptible to psychosocial stress, including experiences of racism (32,33). To

our knowledge, this is the first study to compare LTL among patients with SLE based on age of diagnosis. Further study of this relationship in other racial and ethnic groups and using a longitudinal design should be considered.

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AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be published. Chae, Martz, and Chung had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.
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Analysis and interpretation of data. Bridges, Chung, Martz, Smitherman, Wu, Lin, Chae.

REFERENCES

1. Helmick CG, Felson DT, Lawrence RC, Gabriel S, Hirsch R, Kwoh CK, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part I. Arthritis Rheum 2008;58:15–25.
2. Lotstein DS, Ward MM, Bush TM, Lambert RE, van Vollenhoven R, Neuwelt CM. Socioeconomic status and health in women with systemic lupus erythematosus. J Rheumatol 1998;25:1720–9.
3. Feldman CH, Hiraki LT, Liu J, Fischer MA, Solomon DH, Alarcón GS, et al. Epidemiology and sociodemographics of systemic lupus erythematosus and lupus nephritis among US adults with Medicaid coverage, 2000-2004. Arthritis Rheum 2013;65:753–63.
4. Alarcón GS, Bastian HM, Beasley TM, Roseman JM, Tan FK, Fessler BJ, et al. Systemic lupus erythematosus in a multi-ethnic cohort (LUMINA) XXXII: [corrected] contributions of admixture and socioeconomic status to renal involvement. Lupus 2006;15:26–31.
5. Bastian HM, Roseman JM, McGwin G, Alarcón GS, Friedman AW, Fessler BJ, et al. Systemic lupus erythematosus in three ethnic groups. XII. Risk factors for lupus nephritis after diagnosis. Lupus. 2002;11:152–60.
6. Williams DR. Race, socioeconomic status, and health. The added effects of racism and discrimination. Ann N Y Acad Sci1999;896: 173–188.
7. Giurgescu C, Engeland CG, Templin TN, Zenk SN, Koenig, Garfield L. Racial discrimination predicts greater systemic inflammation in pregnant African American women. Appl Nurs Res 2016;32:98–103.
8. Duran S, Apte M, Alarcon GS. Poverty, not ethnicity, accounts for the differential mortality rates among lupus patients of various ethnic groups. J Natl Med Assoc 2007;99:3.
9. Lim SS, Bayakly AR, Helmick CG, Gordon C, Easley KA, Drenkard C. The incidence and prevalence of systemic lupus erythematosus, 2002-2004: The Georgia Lupus Registry. Arthritis Rheumatol 2014; 66:357–68.
10. Klein-Gitelman M, Reiff A, Silverman ED. Systemic lupus erythematosus in childhood. Rheum Dis Clin North Am 2002;28:561–77.
11. Lim LSH, Pullenayegum E, Feldman BM, Lim L, Gladman DD, Silverman ED. From childhood to adulthood: disease activity trajectories in childhood-onset systemic lupus erythematosus. Arthritis Care Res (Hoboken) 2018;70:750–7.
12. Groot N, Shaikhani D, Teng YKO, de Leeuw K, Bijl M, Dolhain RJEM, et al. Long-term clinical outcomes in a cohort of adults with childhood-onset systemic lupus erythematosus. Arthritis Rheumatol 2019;71:290–301.
13. Carreño L, López-Longo FJ, Monteagudo I, Rodríguez-Mahou M, Bascones M, González CM, et al. Immunological and clinical differences between juvenile and adult onset of systemic lupus erythematosus. Lupus 1999;8:287–92.
14. Chang JC, Xiao R, Mercer-Rosa L, Knight AM, Weiss PF. Child-onset systemic lupus erythematosus is associated with a higher incidence of myopericardial manifestations compared to adult-onset disease. Lupus 2018;27:2146–54.
15. Brunner HI, Gladman DD, Ibanez D, Urowitz MD, Silverman ED. Difference in disease features between childhood-onset and adult-onset systemic lupus erythematosus. Arthritis Rheum 2008;58:556–62.
16. Tucker LB, Uribe AG, Fernández M, Vilá LM, McGwin G, Apte M, et al. Adolescent onset of lupus results in more aggressive disease and worse outcomes: results of a nested matched case—control study within LUMINA, a multiethnic US cohort (LUMINA LVII). Lupus 2010; 17:314.
17. Bundhun PK, Kumari A, Huang F. Differences in clinical features observed between childhood-onset versus adult-onset systemic lupus erythematosus: a systematic review and meta-analysis. Medicine (Baltimore) 2017;96:e8086.
18. Hersh AO, Trupin L, Yazdany J, Panopalis P, Julian L, Katz P, et al. Childhood-onset disease as a predictor of mortality in an adult cohort of patients with systemic lupus erythematosus. Arthritis Care Res (Hoboken) 2010;62:1152–9.
19. Montoya-Ortiz G. Immunosenescence, aging, and systemic lupus erythematous. Autoimmune Dis 2013;2013:267078.
20. Kurosaka D, Yasuda J, Yoshida K, Yoneda A, Yasuda C, Kingetsu I, et al. Abnormal telomerase activity and telomere length in T and B cells from patients with systemic lupus erythematosus. J Rheumatol 2006;33:1102–7.
21. Georgin-Lavialle S, Aouba A, Mouthon L, Londono-Vallejo JA, Lepelletier Y, Gabet AS, et al. The telomere/telomerase system in autoimmune and systemic immune-mediated diseases. Autoimmun Rev 2010;9:646–51.
22. Lee YH, Jung JH, Seo YH, Kim JH, Choi SJ, Ji JD, et al. Association between shortened telomere length and systemic lupus erythematosus: a meta-analysis. Lupus 2017;26:282–8.
23. Chae DH, Martz CD, Fuller-Rowell TE, Spears EC, Smith TTG, Hunter EA, et al. Racial discrimination, disease activity, and organ damage: the Black Women's Experiences Living With Lupus (BeWELL) Study. Am J Epidemiol 2019;188:1434–43.
24. Drenkard C, Rask KJ, Easley KA, Bao G, Lim SS. Primary preventive services in patients with systemic lupus erythematosus: study from a population-based sample in Southeast U.S. Semin Arthritis Rheum 2013;43:209–16.
25. Drenkard C, Bao G, Dennis G, Kan HJ, Jhingran PM, Molta CT, et al. Burden of systemic lupus erythematosus on employment and work productivity: data from a large cohort in the southeastern United States. Arthritis Care Res (Hoboken) 2014;66:878–87.
26. Lim SS, Drenkard C. Understanding lupus disparities through a social determinants of health framework: the Georgians Organized Against Lupus Research Cohort. Rheum Dis Clin North Am 2020;46:613–21.
27. McDade TW, Williams S, Snodgrass JJ. What a drop can do: dried blood spots as a minimally invasive method for integrating biomarkers into population-based research. Demography 2007;44:899–925.
28. Cawthon RM. Telomere measurement by quantitative PCR. Nucleic Acids Res 2002;30:e47.
29. Yazdany J, Trupin L, Gansky SA, Dall'era M, Yelin EH, Criswell LA, et al. The Brief Index Of Lupus Damage: a patient-reported measure

of damage in systemic lupus erythematosus. Arthritis Care Res (Hoboken) 2011;63:1170–7.

30. Drenkard C, Yazdany J, Trupin L, Katz PP, Dunlop-Thomas C, Bao G, et al. Validity of a self-administered version of the brief index of lupus damage in a predominantly African American systemic lupus erythematosus cohort. Arthritis Care Res (Hoboken) 2014;66:888–96.
31. Nuttall FQ. Body mass index: obesity, bmi, and health: a critical review. Nutr Today 2015;50:117–28.
32. Vina ER, Hausmann LR, Utset TO, Masi CM, Liang KP, Kwoh CK. Perceptions of racism in healthcare among patients with systemic lupus erythematosus: a cross-sectional study. Lupus Sci Med 2015; 2:e000110.
33. Drenkard C, Bao G, Lewis TT, Pobiner B, Priest J, Lim SS. Physician-patient interactions in African American patients with systemic lupus erythematosus: demographic characteristics and relationship with disease activity and depression. Semin Arthritis Rheum 2019;48:669–77.