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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 micro-lancet and applying approximately 50  $\mu$ L drops of blood to filter paper (27). Blood samples were allowed to dry and were stored at  $-80^{\circ}\text{C}$ . 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 (QIAGEN; category # 56504). Genomic DNA was extracted from DBSs stored at  $-80^{\circ}\text{C}$  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  $175 \text{ ng} \pm 97 \text{ ng}$  in 50  $\mu$ L elution buffer.

The primers for the telomere polymerase chain reaction (PCR) were *tel1b* [5'-CGGTTT(GTTTGG)<sub>5</sub>GTT-3'], used at a final concentration of 100 nM, and *tel2b* [5'-GGCTTG(CCTTAC)<sub>5</sub>CCT-3'], used at a final concentration of 900 nM. The primers for the single-copy gene (human beta-globin) PCR are *hbg1* [5' GCTTCTGACACAAGTGTGTTCACTAGC-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  $\mu$ M each deoxynucleoside triphosphate, 1% Dimethylsulfoxide, 0.4 $\times$  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- $\mu$ L 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 outlier 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  $2.3\% \pm 1.6\%$ .

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 <sup>a</sup> (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 <sup>b</sup> , mean (SD)	2.98 (2.6)	2.70 (2.5)	2.73 (2.5)
Body mass index (kg/m <sup>2</sup> ), 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.

<sup>a</sup> Total sample was age restricted to participants <70 years old.

<sup>b</sup> 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 <sup>†</sup>	(-0.002 to -0.629)
Age × childhood diagnosis			-0.008*	(-0.016 to -0.001)	-0.009*	(-0.016 to -0.001)	-0.007 <sup>†</sup>	(-0.015 to 0.001)
≥10-y disease duration					0.063 <sup>†</sup>	(-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.

<sup>†</sup>  $P < 0.10$ .

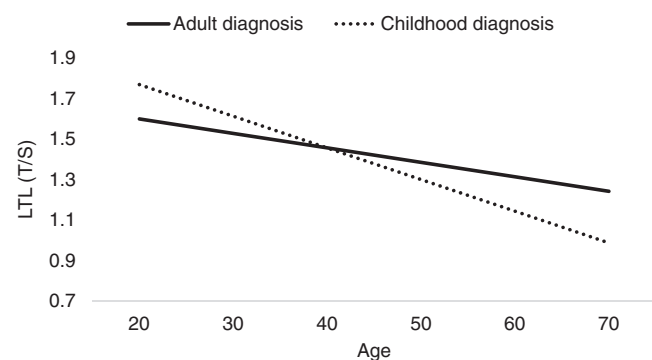
\*  $P < 0.05$ . \*\*\*  $P < 0.001$ .

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  $\chi^2$  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



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

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  $0.001$ ;  $P = 0.083$ ).

## 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.

**Study conception and design.** Bridges, Smitherman, Drenkard, Lin, Lim, Chae.

**Acquisition of data.** Drenkard, Wu, Lin, Lim, Chae.

**Analysis and interpretation of data.** Bridges, Chung, Martz, Smitherman, Wu, Lin, Chae.

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