Soda and Cell Aging: Associations Between Sugar-Sweetened Beverage Consumption and Leukocyte Telomere Length in Healthy Adults From the National Health and Nutrition Examination Surveys

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Objectives. We tested whether leukocyte telomere length maintenance, which underlies healthy cellular aging, provides a link between sugar-sweetened beverage (SSB) consumption and the risk of cardiometabolic disease.

Methods. We examined cross-sectional associations between the consumption of SSBs, diet soda, and fruit juice and telomere length in a nationally representative sample of healthy adults. The study population included 5309 US adults, aged 20 to 65 years, with no history of diabetes or cardiovascular disease, from the 1999 to 2002 National Health and Nutrition Examination Surveys. Leukocyte telomere length was assayed from DNA specimens. Diet was assessed using 24-hour dietary recalls. Associations were examined using multivariate linear regression for the outcome of log-transformed telomere length.

Results. After adjustment for sociodemographic and health-related characteristics, sugar-sweetened soda consumption was associated with shorter telomeres (b = –0.010; 95% confidence interval [CI] = –0.020, –0.001; $P = .04$). Consumption of 100% fruit juice was marginally associated with longer telomeres (b = 0.016; 95% CI = –0.000, 0.033; $P = .05$). No significant associations were observed between consumption of diet sodas or noncarbonated SSBs and telomere length.

Conclusions. Regular consumption of sugar-sweetened sodas might influence metabolic disease development through accelerated cell aging. (*Am J Public Health.* 2014;104:2425–2431. doi:10.2105/AJPH.2014.302151)

Sugar-sweetened beverages (SSBs), including soft drinks or sodas, fruit-flavored drinks, sports drinks, and energy drinks, are the largest source of added sugar in the US diet.[1,2] Between 1999 and 2008, it was estimated that adults aged 20 to 34 years consumed an average of 333 to 421 calories per day, and adults aged 35 years or older consumed an average of 236 to 260 calories per day from SSBs.[3] Because of these strikingly high levels of consumption, SSBs have emerged as an important target of public health efforts and policies.[4,5]

In parallel to trends in SSB intake, the prevalences of obesity and type 2 diabetes have also increased in recent years.[6,7] Epidemiological studies have shown that regular consumption of SSBs is associated with increased risks of obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease.[8–11] However, the mechanisms for these associations are complex and not yet fully understood. There is evidence to suggest that excess calories (via lowered satiety) and high levels of insulin resistance, oxidative stress, and inflammation, may mediate these associations.[9] Because oxidative stress, inflammation, and insulin resistance are also associated with telomere shortening, impaired telomere length maintenance is a potential mechanism that may help to explain the association between SSB consumption and accelerated metabolic disease.[12–14]

Telomeres are the DNA-protein caps at the end of chromosomes that promote chromosomal stability and protect the genomic DNA from damage. Telomere length naturally shortens with each cell cycle, and if it falls to a critical short length, the cell is no longer able to divide and often malfunctions.[15] In addition to biological age, telomere shortness has been linked to lifestyle behaviors and psychological stress.[16–22] In turn, shorter telomeres have been associated with increased risks of chronic diseases, including cardiovascular disease, diabetes, and some cancers.[17,23–27] In population studies, evidence exists for a causal role of impaired telomere maintenance in raising risks of pulmonary and cardiovascular disease.[28] To date, the associations between dietary intake and telomere length have been examined in only a few studies; results for most food groups and nutrients have been mixed.[13,29,30]

Because of the known effects of SSBs on oxidative stress and insulin resistance, our objective in this study was to examine the associations between SSBs, diet soda, and 100% fruit juice consumption and telomere length in a large, nationally representative sample of healthy adults in the United States. We hypothesized that beverages with high sugar content would be the most detrimental to cellular aging, such that sugar-sweetened sodas and noncarbonated SSBs would show the strongest associations with telomere shortness.

METHODS

The National Health and Nutrition Examination Survey (NHANES) is an ongoing, multistage cross-sectional survey administered by the National Center for Health Statistics. The study population was restricted to 5309 adults, aged 20 to 65 years, who had complete dietary data and leukocyte telomere length (LTL) measured in the 1999 to 2002 NHANES. Adults

with a history of diabetes, coronary heart disease, angina, myocardial infarction, stroke or congestive heart failure were excluded.

Leukocyte Telomere Length

DNA samples purified from whole blood were collected from NHANES participants aged 20 years and older in the 1999 to 2002 waves to establish a national probability sample of genetic material for future research.[31] DNA aliquots were processed by the Division of Laboratory Sciences at the National Center for Environmental Health and provided by the Division of Health and Nutrition Examination Surveys, National Center for Health Statistics, Centers for Disease Control and Prevention. The LTL assay was performed in the laboratory of Elizabeth Blackburn at the University of California, San Francisco, using the quantitative polymerase chain reaction method to measure telomere length relative to standard reference DNA (T/S ratio), as described in detail elsewhere.[32,33] The polymerase chain reaction method was preferred over the Southern blot method because of the smaller amount of DNA required for the assay.[34,35] Each LTL sample was assayed 3 times on 3 different days. The samples were assayed on duplicate wells, resulting in 6 data points. Sample plates were assayed in groups of 3 plates, and no 2 plates were grouped together more than once. Each assay plate contained 96 control wells with 8 control DNA samples. Assay runs with 8 or more invalid control wells were excluded from further analysis (< 1% of runs). Control DNA values were used to normalize between-run variability. Runs with more than 4 control DNA values falling outside 2.5 SDs from the mean for all assay runs were excluded from further analysis (< 6% of runs). For each sample, any potential outliers were identified and excluded from the calculations (< 2% of samples). The mean and SD of the T/S ratio were then calculated normally. The interassay coefficient of variation was 6.5%. Throughout this article, we refer to the T/S ratio and relative telomere length as telomere length for brevity.

The conversion from the T/S ratio to base pairs was calculated based on comparison of telomeric restriction fragment length from Southern blot analysis and T/S ratios using DNA samples from the human diploid fibroblast cell line IMR90 at different population doublings. The formula used to convert the T/S ratio to base pairs was 3274 + 2413 * (T/S).

Sugar-Sweetened Beverage Intake

One 24-hour dietary recall was administered to NHANES study participants in the Mobile Examination Center. Beverage variables were derived from the NHANES individual food files. Consumption of sugar-sweetened sodas, noncarbonated SSBs (i.e., fruit drinks, sports drinks, energy drinks, sweetened waters), diet sodas, 100% fruit juice, and all SSBs (including sugar-sweetened sodas and noncarbonated SSBs) were identified using data from the US Department of Agriculture (USDA) Food and Nutrient Database for Dietary Studies. Serving sizes of 8 ounces (226.8 g) were applied to all beverages.

We used a statistical method developed by the National Cancer Institute to estimate usual dietary intake, because 24-hour dietary recalls might not accurately reflect long-term dietary intake.[36] The National Cancer Institute method requires 2 or more days of 24-hour dietary recalls on a subset of participants. Because study participants in the 1999 to 2002 NHANES only contributed one 24-hour dietary recall, we included data from 2003 to 2004 NHANES participants to calibrate the distributions of dietary variables. This method, which uses a 2-part nonlinear mixed model for foods consumed episodically (i.e., SSBs), was applied to participants from 1999 to 2004 NHANES with sociodemographic characteristics and dietary data. We modeled intake distributions for each beverage, correcting for age, gender, race/ethnicity and weekday-to-weekend effects. We then estimated individual beverage intake for all participants in 1999–2004 NHANES, although only the participants in 1999 to 2002 NHANES were retained in the analytical population. In using this method, we assumed that the distributions of SSB intake did not significantly differ between 1999 to 2002 and 2003 to 2004. The National Cancer Institute method's validity in evaluating associations between usual intake of foods and health outcomes was previously established.[37]

Study Covariates

Potential confounders included sociodemographic characteristic variables, such as participant's age (20–24, 25–29, 30–34, 35–39,40–44, 45–49 ,50–54, 55–59, 60–65 years), gender, self-reported race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, other race/multiracial), highest educational attainment (< 12 years, high school diploma or equivalent, some college, college graduate), ratio of family income to poverty, using the Department of Health and Human Services annual poverty guidelines (FPL; 0%–100% FPL, 100%–200% FPL, 200%–300% FPL, 300%–400% FPL, and > 400% FPL), and marital status (married or living with partner, never married, separated/widowed/divorced).

Health-related variables included smoking status (never, former, current), pack-years of smoking (0, < 30, 30–60, and > 60), physical activity assessed from questionnaire (some activity, no activity), total energy intake, alcohol intake, and Healthy Eating Index 2005 (HEI 2005) score, which is a dietary pattern developed by the USDA to measure compliance with national dietary guidelines.[38] The HEI 2005 is scored out of 100 points and comprises 12 components: total fruit, whole fruit, total vegetables, dark green and orange vegetables and legumes, total grains, whole grains, milk, meat and beans, oils, saturated fat, sodium, and calories from solid fats, alcoholic beverages, and added sugars. We collapsed HEI 2005 scores into gender-specific quartiles: for men, the cutpoints were 42.1, 45.9, and 50.5; for women, the cutpoints were 44.4, 48.6, and 53.5. We defined alcohol intake as low (0–0.5 drinks/day for men and women), moderate (0.5–2.0 drinks/day for men; 0.5–1.5 drinks/day for women), and heavy (> 2 drinks/day for men, > 1.5 drinks/day for women).

Adiposity measures included body mass index (BMI) and waist circumference. We calculated BMI from self-reported height (in meters) and weight (in kilograms squared), measured by trained personnel using a stadiometer and Toledo weight scale (Toledo Scale, Honolulu, HI).[39] We defined BMI categories as underweight (BMI < 18.5 kg/m^2), normal weight (BMI = 18.5–24.9 kg/m^2), overweight (BMI = 25.0–29.9 kg/m^2), and obese (BMI ≥ 30 kg/m^2). Waist circumference (centimeters) was measured at the upper lateral border of the right ilium. We defined elevated waist circumference as 102 centimeters or greater for men and 88 centimeters or greater for women.

Missing indicators were used to account for missing education level (n = 6, 0.16% missing),

marital status (n = 258, 5.5% missing), smoking status (n = 8, 0.15% missing), pack-years of smoking (n = 523, 9.1% missing), household income (n = 416, 6.9% missing), BMI (n = 89, 1.6% missing), and waist circumference (n = 126, 2.0% missing).

Statistical Analysis

To make nationally representative estimates, analyses accounted for the complex NHANES sampling design by incorporating sampling weights for the genetic subsample and strata and primary sampling unit indicators. The sampling weights accounted for different sampling probabilities and the potential nonresponse bias of the participants in the NHANES subsample who consented to the use of DNA specimens for future genetic research. First, we examined bivariate associations between LTL and individual-level characteristics. Because of the skewness of LTL, LTL was log-transformed before fitting to the regression models. Linear regression models were then fit for log-transformed LTL to estimate the difference in LTL for a 1-serving increase in beverage intake. The first model adjusted for age categories, gender, and total energy intake. The second model adjusted for all sociodemographic characteristics and health-related variables. We also examined heterogeneity in the associations between beverage intake and LTL by gender and race/ethnicity by introducing product terms between beverages and the individual modifiers in the fully adjusted models. Statistical significance of the product terms was determined with the Wald test.

All statistical tests were 2-sided, and statistical significance was considered at $P < .05$. Statistical analyses were performed using SAS 9.3 (SAS Institute, Cary, NC) and conducted using ANDRE (Centers for Disease Control and Prevention, Atlanta, GA), the Centers for Disease Control and Prevention remote access system for restricted data analysis.

RESULTS

As expected, age was linearly associated with shorter telomeres ($P < .001$) (Table 1). Mean telomere length was longest in Blacks and Hispanics, in never smokers, and in normal weight adults, as observed in previous studies.[18,31,40–43]

Pearson's correlation coefficients for associations among self-reported intakes of different

TABLE 1—Mean Leukocyte Telomere Length by Sociodemographic Characteristics and Lifestyle Behaviors of 5309 Adults Aged 20–65 Years: National Health and Nutrition Examination Surveys, United States, 1999–2002

Characteristics	No. (Weighted %[a]) or Mean ±SE	LTL, Mean (SE)	P[b]
Age, y	39.7 ±0.3	1.10 (0.01)	< .001
Gender			.27
Men	2473 (48.2)	1.09 (0.01)	
Women	2836 (51.8)	1.10 (0.02)	
Race			.009
Non-Hispanic White	2510 (69.2)	1.09 (0.02)	
Non-Hispanic Black	934 (11.0)	1.15 (0.02)	
Hispanic/Latino	1687 (15.2)	1.11 (0.02)	
Other race/Multirace	178 (4.6)	1.09 (0.02)	
Education level			.25
< 12 y	1554 (18.7)	1.08 (0.02)	
High school diploma	1227 (25.4)	1.09 (0.02)	
Some college	1429 (30.0)	1.11 (0.02)	
College graduate	1093 (26.0)	1.11 (0.02)	
Marital status			< .001
Married or living with partner	3310 (65.6)	1.07 (0.02)	
Never married	1021 (20.5)	1.19 (0.02)	
Separated, widowed or divorced	720 (13.9)	1.04 (0.02)	
Federal poverty level,[c] %			.09
0–100	887 (14.3)	1.15 (0.03)	
100–200	1111 (18.3)	1.09 (0.02)	
200–300	757 (15.1)	1.09 (0.02)	
300–400	619 (13.9)	1.10 (0.02)	
> 400	1519 (38.4)	1.08 (0.02)	
Pack-years of smoking			< .001
0	2872 (57.4)	1.11 (0.02)	
< 30	1533 (34.0)	1.09 (0.02)	
30–60	278 (6.3)	1.00 (0.02)	
> 60	103 (2.3)	0.96 (0.02)	
Physical activity			.03
Some activity	1974 (30.7)	1.08 (0.02)	
No activity	3332 (69.3)	1.11 (0.01)	
BMI (kg/m^2)			.004
Underweight (< 18.5)	78 (1.8)	1.13 (0.03)	
Normal weight (18.5–24.9)	1673 (35.0)	1.13 (0.02)	
Overweight (25.0–29.9)	1849 (34.4)	1.08 (0.02)	
Obese (≥ 30 kg/m^2)	1620 (28.9)	1.07 (0.02)	
Waist circumference (cm)			.003
Normal (< 102 for men; < 88 for women)	2693 (55.1)	1.12 (0.02)	
Elevated (≥ 102 for men; ≥ 88 for women)	2490 (42.9)	1.07 (0.02)	

Note. BMI = body mass index; LTL = leukocyte telomere length.
[a]Weighted percentages are representative of the United States civilian, noninstitutionalized population.
[b]From χ^2 test and univariate linear regression.
[c]Ratio of family income to poverty, using the Department of Health and Human Services annual poverty guidelines.

TABLE 2—Pearson's Correlations Coefficients for Sugar-Sweetened Beverages: National Health and Nutrition Examination Surveys, United States, 1999–2002

Beverage	Sugar-Sweetened Soda	Noncarbonated SSB	Diet Soda	100% Fruit Juice
Sugar-sweetened soda	1.00			
Noncarbonated SSB	0.20	1.00		
Diet soda	-0.23	-0.10	1.00	
100% fruit juice	0.04	0.13	-0.07	1.00

Note. SSB = sugar-sweetened beverage.

beverages are shown in Table 2. Overall, correlations between beverages were modest. The intakes of sugar-sweetened sodas, noncarbonated SSBs, and 100% fruit juice were positively correlated with each other. Diet soda was negatively correlated with SSBs and 100% fruit juice intakes.

Average sugar-sweetened soda consumption was 1.5 servings (12 ounces) per day. Average consumption of diet sodas, noncarbonated SSBs, and 100% fruit juice was lower, ranging from 0.3 to 0.5 servings per day. Consumption of all SSBs (including sugar-sweetened soda and noncarbonated SSBs) was averaged at 2.1 servings (16.8 ounces) per day. Associations between SSB intake and telomere length are shown in Table 3. After adjustment for sociodemographic characteristics and health-related variables and adiposity, sugar-sweetened soda consumption was inversely associated with telomere length (b = –0.010; 95% CI = –0.020, –0.001). Holding other covariates constant, this difference corresponded to a deficit of 14 base pairs. Because of the model-based estimate in this sample of the age-associated rate of telomere shortening of 13.6 base pairs per year, this was equivalent to 1.9 additional years of aging for an 8-ounce serving of sugar-sweetened sodas. For a daily consumption of the current standard 20-ounce serving size for sugar-sweetened sodas, this corresponds to 4.6 additional years of aging. Approximately 21% of adults in the study population reported daily consumption of 20 ounces or more of sugar-sweetened soda (data not shown).

No associations were observed between diet soda or noncarbonated SSBs and telomere length. Although a positive association was observed between 100% fruit juice and telomere length in the first model after adjusting for age, gender, and total energy, this association was attenuated after the inclusion of other potential confounders ($P = .05$).

Stratified associations by gender and race/ethnicity are available as a supplement to the online version of this article at http://www.ajph.org. There was no evidence of heterogeneity in the associations by gender or racial/ethnic groups.

DISCUSSION

In this nationally representative sample of healthy adults, the average daily consumption of sugar-sweetened soda was 12 ounces (1.5 servings), a level in excess of the American Heart Association recommended limit for added sugar.[5] Consistent with our hypothesis, we found that each daily 8-ounce serving of sugar-sweetened sodas was linearly associated with shorter telomeres, roughly equivalent to 1.9 additional years of aging, independent of sociodemographic characteristics and health-related variables. For a daily 20-ounce serving, the current standard serving size, this translates into approximately 4.6 additional years of aging. More than 20% of adults in the study population reported at least 20 ounces of sugar-sweetened soda consumption per day. Although these were modest associations, the magnitude of the association for consuming 20 ounces of sugar-sweetened soda was comparable to observed associations between telomere length and moderate or vigorous levels of physical activity (4.4 years, in the opposite direction) and smoking (4.6 years).[18,20] To our knowledge, ours was the first study to link sugar-sweetened soda consumption with telomere length in a large, nationally representative sample of healthy adults.

TABLE 3—Associations Between Beverage Intake and Log-Transformed Leukocyte Telomere Length (T/S Ratio): National Health and Nutrition Examination Surveys, United States, 1999–2002

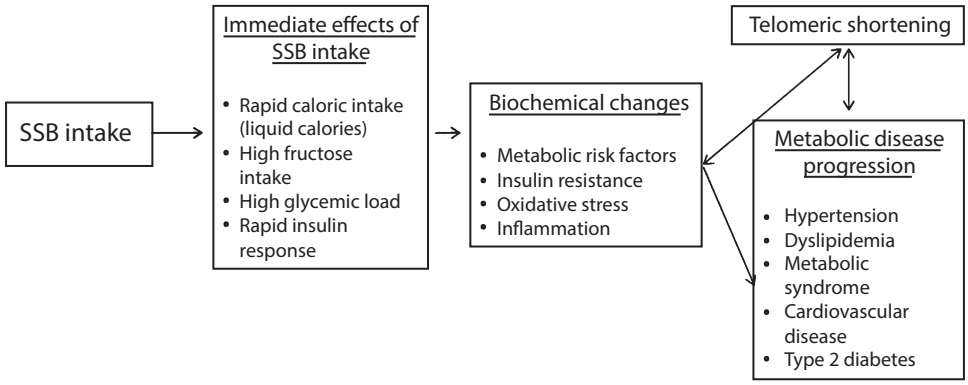
	Mean LTL by Quartile of Intake				Model 1	Model 2
Beverage Type[a]	Q1	Q2	Q3	Q4	b (95% CI)	b (95% CI)
All sugar-sweetened beverages[b]	1.05	1.10	1.11	1.13	-0.010 (-0.021, 0.001)	-0.008 (-0.020, 0.004)
Sugar-sweetened soda	1.04	1.13	1.09	1.12	-0.013* (-0.023, -0.003)	-0.010* (-0.020, -0.001)
Noncarbonated sugar-sweetened beverages	1.03	1.10	1.12	1.13	0.000 (-0.029, 0.029)	-0.001 (-0.030, 0.028)
Diet soda	1.10	1.08	1.09	1.10	-0.003 (-0.021, 0.016)	-0.000 (-0.019, 0.018)
100% fruit juice	1.10	1.08	1.08	1.11	0.022* (0.003, 0.041)	0.016 (-0.000, 0.033)

Note. CI = confidence interval; LTL = leukocyte telomere length. Model 1 included age, gender, and total energy. Model 2 included age, gender, race/ethnicity, education level, marital status, smoking status, pack-years of smoking, physical activity, poverty level, total energy, alcohol intake, Healthy Eating Index 2005 scores, body mass index categories, and waist circumference categories.

[a]Expressed in servings (8 oz).

[b]Includes sugar-sweetened soda and noncarbonated sugar-sweetened beverages.

*$P < .05$.



Note. High SSB intake leads to rapid caloric and fructose intake, and high glycemic load. This results in an increased risk of metabolic risk factors and a biochemical environment of high insulin resistance, oxidative stress, and inflammation. In turn, this can affect telomeric shortening and influence metabolic disease progression, including metabolic syndrome, type 2 diabetes, and cardiovascular disease.

FIGURE 1—Conceptual model of the effects of sugar-sweetened beverage (SSB) intake on telomeric shortening and metabolic disease progression: National Health and Nutrition Examination Surveys, United States, 1999–2002.

Results of telomere length associations with other various dietary aspects were inconsistent.[13,29,30] The Multi-Ethnic Study of Atherosclerosis (MESA) previously examined sugar-sweetened soda consumption in relation to telomere length among adults.[30] Their results showed no association after adjustment for sociodemographic characteristics, lifestyle factors, and BMI. The fact that MESA had a smaller sample size and an older population, on average than NHANES, might account for why an association was found in our study but not in MESA.

Our hypothesis that consumption of SSBs were related to shorter telomeres was derived from the known effects of SSB consumption on impaired fasting glucose and insulin resistance.[8–11] SSBs have been known to increase oxidative stress and systemic inflammation, which are both processes that can influence telomere attrition.[13,14] Telomere shortening in response to, and perhaps contributing to, these disease processes was reported, reflecting the overall burden of cardiometabolic disease.[27,44,45] Our results suggested that another link between sugar-sweetened soda consumption and metabolic disease might be through shortened telomere length, a biomarker and mechanism of cellular aging (Figure 1).

We observed no significant associations between consumption of noncarbonated SSBs and telomere length. The lack of association might be attributed to the large degree of heterogeneity in sugar content across beverages.[46] In the study population, the average consumption of noncarbonated SSBs (0.3 servings/day) was substantially lower than the average consumption of sugar-sweetened sodas (1.5 servings/day); it might be that sugar consumption in beverages affected telomere length only at higher intake levels. Consumption of noncarbonated SSBs has increased in recent years, whereas overall intakes of sugar-sweetened sodas have decreased, and an association between consumption of noncarbonated SSBs and telomere length might emerge in future studies.[3] Even lacking a significant current association with telomere length, decreasing consumption of SSBs to reduce risks of obesity-related chronic disease seems prudent.[8,10]

A marginally positive association was shown in our study between 100% fruit juice consumption and telomere length. Previous studies that examined fruit juice and health outcomes yielded mixed findings. Fruit juice was associated with an increased risk of type 2 diabetes in some,[47–49] but not all studies.[10,50–52] Consumption of 100% fruit juice was not shown to have the same effect on cardiometabolic risk factors[53] or markers of insulin resistance, oxidative stress, or inflammation as SSB consumption.[54–56] Fruit juice consumption might result in different metabolic effects compared with SSBs, with potentially beneficial effects of phytochemicals and micronutrients balancing out the harmful effect of liquid sugars. Consumption levels of 100% fruit juice were also generally lower than levels of sugar-sweetened soda consumption, as was shown in our study. Because fruit juice consumption was not associated with long-term health benefits in epidemiological studies, limiting its consumption in preference of whole fruit might be advisable.

Our study was strengthened by the use of a large, nationally representative sample of adults. In addition, we used a validated method to estimate usual beverage intake from the extensive NHANES dietary data. Furthermore, the NHANES response rates from 1999 to 2002 ranged from 76% to 80%; these were considerably higher than other national health surveys, and helped to improve the generalizability of our findings.[57] We also took steps to avoid spurious findings, including examining a small number of dietary components for which there were substantially strong a priori hypotheses for associations with oxidative stress and biomarkers of aging.

Study Limitations

Our study had limitations. First, the cross-sectional nature of the data made it difficult to infer causation. Longitudinal studies of dietary intake and telomere length are needed to understand how dietary intake can influence telomere length over time, and whether the associations are explained by the mechanisms proposed in Figure 1. Collection of biochemical

data, such as insulin resistance, oxidative stress, and inflammation, would also help to inform the understanding of the mechanisms of the association between SSB intake and telomeric shortening. LTL was measured from a single DNA specimen, which did not provide information on rates of telomere shortening. Similarly, beverage intake was estimated from a 24-hour dietary recall conducted at the time of the survey, which might not reflect diet or beverage patterns over the life course.

Telomere research in clinical studies is a relatively new field, and researchers are still identifying important individual and lifestyle determinants of telomere length. Thus, there is always the possibility of unmeasured confounding. For example, genetic differences may contribute to telomeric shortening; however, the degree of this confounding could be small because it is unlikely that any potential single nucleotide polymorphisms predictive of telomere length are strongly associated with beverage consumption. Psychosocial stress is another important determinant of telomeric shortening; unfortunately, this construct was not captured within the NHANES questionnaires. Our analyses included all potential sociodemographic characteristics and health variables known to be related to telomere length and dietary intake, some of which might act as proxies for psychological stress; the inclusion of these variables did not substantially change the model estimates. Because we examined healthy adults without a history of diabetes or cardiovascular disease, the associations should reflect sugar-sweetened soda consumption independent of cardiometabolic disease.

Conclusions

Understanding the role that nutrition plays in telomere length maintenance is critical in understanding how to improve dietary intake. Independent of adiposity and other individual characteristics, our study results suggested that regular consumption of sugar-sweetened sodas was associated with significantly shorter telomeres. Further epidemiological studies are needed to confirm this association in longitudinal settings, and experimental research can examine the pathway from soda to cell to better understand the mechanism of this relationship. Still, there is sufficient evidence to limit our consumption of all SSBs to improve cardiometabolic risk factors, reduce chronic disease risk, and improve overall health. Our study supported a new link, shortened immune cell telomere length, which is a biological risk factor for aging, between sugar-sweetened soda consumption and metabolic disease. ■

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Contributors

C. W. Leung, B. A. Laraia, B. L. Needham, D. H. Rehkopf, and E. S. Epel conceptualized the study. C. W. Leung, B. L. Needham, D. H. Rekhopf, and E. S. Epel analyzed the data. J. Lin and E. H. Blackburn performed the telomere assay. C. W. Leung wrote the first draft of the article. All authors contributed to the interpretation of the results, made substantial revisions to the content, and approved the final article.

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Human Participant Protection

Institutional review board approval was not needed because secondary data from National Health and Nutrition Examination Survey (NHANES) data were used to conduct this study. NHANES is approved by the National Center for Health Statistics (NCHS) Research Ethics Review Board. Our proposal to analyze the genetic data was approved by the NCHS Research Data Center.

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