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Dietary patterns, food groups, and telomere length in the Multi-Ethnic Study of Atherosclerosis (MESA)[3]

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Abstract

Background—Telomere length reflects biological aging and may be influenced by environmental factors, including those that affect inflammatory processes.

Objective—With data from 840 white, black, and Hispanic adults from the Multi-Ethnic Study of Atherosclerosis, we studied cross-sectional associations between telomere length and dietary patterns and foods and beverages that were associated with markers of inflammation.

Design—Leukocyte telomere length was measured by quantitative polymerase chain reaction. Length was calculated as the amount of telomeric DNA (T) divided by the amount of a single-copy control DNA (S) (T/S ratio). Intake of whole grains, fruit and vegetables, low-fat dairy, nuts or seeds, nonfried fish, coffee, refined grains, fried foods, red meat, processed meat, and sugar-sweetened soda were computed with responses to a 120-item food-frequency questionnaire completed at baseline. Scores on 2 previously defined empirical dietary patterns were also computed for each participant.

Results—After adjustment for age, other demographics, lifestyle factors, and intakes of other foods or beverages, only processed meat intake was associated with telomere length. For every 1 serving/d greater intake of processed meat, the T/S ratio was 0.07 smaller ($\beta \pm$ SE: -0.07 ± 0.03, $P = 0.006$). Categorical analysis showed that participants consuming ≥1 serving of processed meat each week had 0.017 smaller T/S ratios than did nonconsumers. Other foods or beverages and the 2 dietary patterns were not associated with telomere length.

Conclusions—Processed meat intake showed an expected inverse association with telomere length, but other diet features did not show their expected associations.

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The author's responsibilities were as follows—JAN: was responsible for analytic design, data analysis, and manuscript preparation; AD-R: was involved in ancillary study data acquisition and manuscript review; NSJ and ALF: critically reviewed the manuscript; DRJ: was involved in main study data acquisition and manuscript review.

A full list of participating MESA investigators and institutions can be found at http://www.mesa-nhlbi.org.

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INTRODUCTION

Telomeres are highly conserved regions of DNA made up of long arrays of repeating base pairs $(TTAGGG)_n$ that serve to protect the end of all linear chromosomes. Because DNA polymerase does not completely replicate telomeres with each successive cell division, telomere length reflects cell turnover and, therefore, the biological aging process (1). Data have shown that telomere attrition is associated with greater risk of various chronic diseases, including hypertension, diabetes, and cardiovascular disease (CVD) (2–8). Evidence from in vitro (9) and human (3,10,11) studies suggests that the balance between oxidative stressors and antioxidants in the cellular environment is a key determinant of the rate of telomere shortening. Although genetics play an important part in determining telomere length (4,12), environmental factors that influence oxidative and inflammatory responses probably influence the rate of cell turnover and, thus, telomere shortening (13).

Dietary components may favorably influence the biological aging process (reflected by telomere length) by antioxidative and antiinflammatory effects. Analogously, it is equally possible that dietary constituents associated with inflammation and oxidative stress may accelerate the biological aging process. For example, whole grains (14–16), nuts (17), and fruit and vegetables (16,18,19), as well as dietary patterns characterized by high intake of these food components, were inversely associated with total mortality (20 −29). Consistent with the hypothesis that dietary factors affect the biological aging process through their effects on inflammation, greater intake of whole grains (30,31), nuts (32), coffee (30,33), and antioxidants from fruit and vegetables (34) and dietary patterns rich in these food constituents were associated with lower concentrations of biomarkers of inflammation (32,35–39). In contrast, *trans* fatty acid intake (40–42) and dietary patterns high in foods known to provide significant amounts of *trans* and saturated fats were positively associated with biomarkers of inflammation (32,36–38).

To date few studies have examined associations between dietary factors and telomere length. A recent study reported positive associations between serum 25-hydroxyvitamin D concentrations and leukocyte telomere length (43). Thus, dairy foods (rich in vitamin D) may contribute to telomere length preservation. However, no studies of which we are aware have examined dietary factors more broadly.

With data from a subsample of the Multi-Ethnic Study of Atherosclerosis (MESA), we investigated associations of telomere length with dietary patterns and individual food or beverage groups (whole grains, refined grains, fruit, vegetables, nonfried fish, nuts or seeds, dairy, red meat, processed meat, fried foods, nondiet soda, and coffee). We hypothesized that dietary patterns rich in whole grains, fruit and vegetables, and nuts or seeds would be associated with greater telomere length, whereas dietary patterns rich in processed and red meats, fried foods, and refined grains would be associated with shorter telomeres. We further hypothesized that whole grains, nuts or seeds, coffee, fruit, vegetables, nonfried fish, and dairy foods would each be independently associated with longer telomeres, and conversely, that refined grains, red meat, processed meat, fried foods, and nondiet soda would each be independently associated with shorter telomeres.

SUBJECTS AND METHODS

Participants

MESA was initiated in 2000 to study the characteristics of subclinical CVD and the risk factors that predict progression to clinically overt CVD or progression of subclinical disease (44). White, African American, Hispanic, and Chinese men and women, aged 45–84 y (*n* = 6814), were recruited from 6 field centers across the United States (Baltimore City and

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County, MD; Chicago, IL; Forsyth County, NC; New York, NY; Los Angeles County, CA; and St. Paul, MN) to participate in the baseline examination. All participating persons were free of clinical CVD at baseline and gave informed consent. MESA study protocols were approved by each field center's institutional review board. As part of an ancillary study to MESA, telomere length was assessed in white, African American, and Hispanic participants from the Los Angeles and New York sites of MESA. Participants in whom telomere length was assessed were approximately similar in basic demographic and behavioral characteristics to other white, African American, and Hispanic participants at those sites. The current cross-sectional investigation includes data from 840 white, African American, and Hispanic MESA participants (434 women; 406 men; 157 whites; 228 African Americans; 455 Hispanics) for whom telomere length and adequate dietary information was available (32).

Measurement of telomere length

Leukocyte telomere length was measured by quantitative polymerase chain reaction (2). Each sample was amplified for telomeric DNA and for 36B4, a single-copy control gene that provided an internal control to normalize the starting amount of DNA. A 4-point standard curve (2-fold serial dilutions from 10 to 1.25 ng DNA) was used to transform cycle threshold into nanograms of DNA. Baseline background subtraction was performed by aligning amplification plots to a baseline height of 2% in the first 5 cycles. The cycle threshold was set at 20% of maximum product. All samples were run in triplicate, and the median was used for calculations. The amount of telomeric DNA (T) was divided by the amount of single-copy control gene DNA (S), producing a relative measurement of the telomere length (T/S ratio). Two control samples were run in each experiment to allow for normalization between experiments, and periodical reproducibility experiments were performed to guarantee correct measurements. The intraassay and interassay variability (CV) for quantitative polymerase chain reaction was 6% and 7%, respectively.

Dietary assessment

At the baseline examination, participants recorded their usual dietary intake in the previous year of 120 foods and beverages with a food-frequency questionnaire (FFQ) (32). The FFQ was developed in the validated Block format (45) and modified from the FFQ used in the Insulin Resistance Atherosclerosis Study, which was validated in non-Hispanic, white, African American, and Hispanic persons (32). Participants recorded serving size (small, medium, large) and frequency (9 options, ranging from rare or never to ≥2 times/d for foods and rare or never to ≥6 time/d for beverages) for each food and beverage item. We calculated servings per day for each item as the product of the reported frequency and serving size (small weighted by 0.5; medium by 1.0; large by 1.5).

Dietary patterns—Four dietary patterns were derived from 47 narrowly defined food groups by principal components analysis in the full MESA population, as previously described (32). Participants selected for this study retained their original empirical dietary pattern scores that we subsequently used in analysis. Consistent with our hypothesis that diet may influence telomere length through inflammatory pathways, in the current analysis we focused on 2 of the original 4 derived dietary patterns that showed most consistent associations with multiple markers of inflammation (each named according to the foods with highest factor loads): fats and processed meat (positively associated with C-reactive protein, interleukin-6, and homocysteine) and whole grains and fruit (inversely associated with C-reactive protein, interleukin-6, homocysteine, and soluble vascular adhesion molecule-1) (32). Each participant received a score on each dietary pattern, calculated as Σ[(food group$_i$ servings/d) × (food group$_i$ factor loading)], where i = food group 1–47. A higher score indicated greater conformity with the pattern being calculated. Scores were categorized into

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quintiles separately for each pattern. Food groups with highest factor loading values on the dietary pattern of fats and processed meats were added fats and oils, processed meats, fried potatoes, salty snacks, and desserts. Food groups with highest factor loading values on the dietary pattern of whole grains and fruit were whole grains, fruit, nuts or seeds, green leafy vegetables, and low-fat dairy foods. Loading values for all 47 food groups on each dietary pattern are given in Appendix A.

Food groups—We also evaluated 12 food groups in our analyses: whole grains, refined grains, fruit, vegetables, nonfried fish (including seafood and dark and light meat fish), nuts or seeds, dairy (split into high- and low-fat items), red meat, processed meat (including ham, hot dogs, lunch meats; sausage; organ meats; ham hocks), fried foods (including fried potatoes, fried chicken, fried fish), nondiet soda, and coffee.

Statistical analyses

We calculated age-adjusted participant demographic and lifestyle characteristics, as well as age- and energy-adjusted food group intakes, across quartiles of telomere length (T/S ratio) by including age (in y, continuous) and energy (in kcal/d, continuous) as covariates in a linear regression model. To evaluate the associations between telomere length and food groups and dietary patterns, we used linear regression to estimate adjusted mean differences in telomere length per 1 serving/d difference in food group intake or per 1-unit difference in dietary pattern score (1 unit = 1 SD by design of principal components analysis). Significant associations were further characterized by calculating adjusted mean telomere length across categories of food group intake. We calculated *P* trends across categories by including the independent variable as a continuous predictor in regression models. We used 3 multivariable models to evaluate these associations. In model 1, we adjusted for age, energy (in kcal/d), study center, sex, and race-ethnic group. To model 2, we added educational status (less than high school, high school, more than high school), inactive leisure activities (metabolic equivalent–min/wk), smoking status (current, former, or never smoker), and pack-years of smoking. Finally, in model 3 we included all food groups simultaneously to estimate mutually adjusted associations. With the exception of model 3, we followed similar procedures for dietary patterns. Because greater body mass index (BMI; in kg/m^2) may be associated with diet and was linked to telomere length in prior work (13), we also evaluated the robustness of results to additional adjustment for BMI. We tested interactions between dietary exposures and age categories (45–64 y compared with 65–84 y), sex, race-ethnicity, and prevalent diabetes with cross-product terms. All analyses were performed with SAS version 9.1 (SAS Institute Inc, Cary, NC).

RESULTS

As expected, age was strongly associated with telomere length. For every 1-y increment in age, the T/S ratio was 0.005 lower ($\beta \pm SE$: −0.005±0.001, $P<0.001$). This difference was greater in persons aged 65–84 y (−0.007 ± 0.001) than for persons aged 45–64 y (−0.004±0.001, $P<0.001$ for difference between age categories). After adjustment for age, longer telomere length was associated with female sex, shorter duration of smoking, and less time spent in inactive leisure activities ($P<0.001$–0.04; Table 1). With the exception of nonfried fish ($P = 0.04$), processed meat consumption ($P = 0.001$), and the dietary pattern scores for fats and processed meat, which were each inversely associated with telomere length, other food groups and dietary patterns were not significantly associated with telomere length after adjustment for age and energy (Table 1).

Of all the food groups studied, only processed meat intake was significantly associated with telomere length after multivariable adjustment, including adjustment for other food groups

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(Table 2). For each 1 serving/d greater intake of processed meat, the T/S ratio was 0.07 smaller ($P = 0.006$; model 3). Neither the dietary pattern for fats and processed meat nor the dietary pattern for whole grains and fruit was significantly associated with telomere length after adjustment for demographic and lifestyle factors. Additional adjustment for BMI did not change these results. Adjustment for gross family income or more refined categories of educational status [proxies for socioeconomic status SES)] also did not change these results.

Associations were slightly stronger if prevalent cases of diabetes were excluded ($n = 108$), which is consistent with the hypothesis that dietary change subsequent to diabetes diagnosis may confound the association. For example, mean difference (±SE) in telomere length per 1-serving increase in processed meat intake in the sample, excluding those with diabetes, was −0.08 ± 0.03, −0.07 ± 0.03, and −0.09 ± 0.03 ($P = 0.003$, 0.008, and 0.002, models 1, 2, and 3, respectively). However, a significant interaction was not observed between processed meat intake and diabetes (or between diabetes and other food groups). Analyses stratified by age categories (45–64 y compared with 65–84 y) suggested a slightly stronger association between telomere length and processed meat intake in the older group (adjusted mean difference ± SE per 1 serving/d: −0.02 ± 0.03 in participants 45–64 y, $P = 0.46$, compared with −0.10 ± 0.04 in participants 65–84 y, $P = 0.01$), but the interaction between age and processed meat intake was not significant ($P = 0.51$). In addition, no interactions were observed between processed meat intake and sex or race-ethnicity.

When we divided processed meat intake into quartiles, mean telomere length (T/S ratio) was lowest in those participants in the highest quartile of processed meat intake ([>0.15 servings/d or ≥1 serving/wk; Table 3). Associations of intake in servings per day with telomere length were similarly inverse for each of the components of the processed meat group, although the magnitude of estimates varied. Only the association with ham, hot dogs, bologna, salami, or other lunch meats was statistically significant ($P = 0.03$ adjusted for variables in model 2; Table 4). Because organ meats may not share all the nutritional attributes of other processed meat, we also reevaluated our analyses omitting organ meats from the processed meat food group and found similar results (data not shown).

DISCUSSION

Food groups such as whole grains, nuts, fish, fruit, and vegetables and prudent dietary patterns rich in these foods were inversely associated with several age-related diseases and total rate of mortality (14–19,21–29,46). Those associations could reflect effects of diet on the aging process. It was suggested that telomere length, or more precisely, the rate of telomere shortening, may be a better marker of the biological aging process than chronologic age itself (1). Therefore, we hypothesized that dietary intake of specific foods and patterns of dietary intake would be associated with telomere length in a manner consistent with reported associations between diet and inflammation, chronic diseases, and rate of mortality. Our data showed an inverse association between consumption of the food group processed meat and telomere length, but other food groups and dietary patterns were not associated with telomere length.

Our finding that processed meat consumption was associated with shorter telomeres is consistent with studies showing associations between greater intake of processed meat and risk of cancer (47) and diabetes (48–50) and with studies showing associations between red meat intake and CVD (51,52), all diseases with frequencies that increase with age. The difference in telomere length (T/S ratio) between the highest and lowest quartiles of processed meat intake (0.017) corresponds to a 3.4-y age difference (with the use of our sample-specific estimates). Within our sample, a 1-y difference in age corresponded to a 0.005 smaller T/S ratio, consistent with previous reports (6,53,54). The observed association

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between processed meat intake and telomere length was independent of intake of other food groups. Constituents of processed meat that may accelerate the aging process include saturated fat, sodium, nitrates and nitrites, cholesterol, and iron (55). Interestingly, red meat was not similarly associated with telomere length. Red meat has shown variable associations with rates of morbidity and mortality, which may be due to differences in preparation methods and cut of meat consumed (percentage of fat or lean) (56). Such variables are not measured well (or at all) by FFQ and, therefore, could not be investigated in our sample.

On the basis of the hypothesis that inflammation partly mediates telomere shortening (57), we hypothesized that the dietary patterns of fats and processed meat and of whole grains and fruit would be associated with shorter and longer telomeres, respectively. We previously reported strong associations between these empirical dietary patterns and markers of inflammation in the larger MESA cohort (32). The finding that the dietary pattern typified by high consumption of processed meat (fats and processed meat) was not associated with telomere length is somewhat surprising in light of the observed association between intake of processed meat and telomere length. It is possible that the absence of association is due to a dilution effect by other food groups not associated with telomere length but included in dietary pattern score computation. Although not among the food groups we identified for investigation a priori, intake of added fats and oils (the highest loading food group on this dietary pattern) was not associated with telomere length, nor was intake of salty snacks, fried potatoes, or desserts, other important contributors to scores on this dietary pattern.

Although telomere length or attrition was associated with several environmental factors (13,43,57,58), telomere length is also under genetic control (12,59) and shows interindividual variation beginning at birth (60). Thus, an ideally designed study would be able to take into account these inherent differences or, alternatively, measure within-person change in telomere length over a period of several years, preferably during the exposure periods in question. Our investigation was cross-sectional; therefore, we were not able to take into account intrinsic differences in telomere length, nor were we able to investigate changes in telomere length over time within participants. Such interindividual variations may have limited our ability to detect associations between telomere length and dietary factors.

Although we adjusted for demographic and lifestyle factors previously shown to be associated with telomere length, residual confounding remains a possibility. For example, persons of lower SES may be more likely to consume processed meat because of lower cost. Low SES may lead to greater life stress, which was associated with shorter telomeres (54,57). However, our data were adjusted for educational status and were not changed if also adjusted for family income (proxies for SES). Low energy intake was hypothesized to increase longevity (61); however, we observed no association between total energy intake and telomere length (data not shown), and the association between processed meat intake and telomere length was independent of total energy intake. Although adjustment for demographics, lifestyle factors, energy intake, and other food groups did not attenuate the association between shorter telomere length and greater intake of the food group processed meat, it remains possible that processed meat intake is serving as a marker of other lifestyle, dietary behaviors, or both that confound or mediate the association. We made every effort to measure these covariates accurately, but residual confounding in studies of this nature is, to some degree, unavoidable.

Two important limitations remain to be addressed. First, our sample size was relatively small, which may have limited our ability to detect small associations were they truly present. Second, it is possible that the statistically significant association we observed between processed meat intake and telomere length was a result of chance. We chose not to

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modify our criteria for statistical significance to account for multiple hypotheses testing, although we did study several food groups and 2 dietary patterns. We felt that an exploratory approach was justified, given the limited amount of research in this area and that we identified the food groups and dietary patterns of interest a priori. Furthermore, in the sample free of diabetes, the fully adjusted association between processed meat consumption and telomere length would remain significant ($P = 0.002$) even if we applied the conservative Bonferroni's correction (0.004 = 0.05/13 tests for 13 foods groups), and the association would be marginally significant in the larger group including participants with prevalent diabetes ($P = 0.006$).

In conclusion, in this cross-sectional study of 840 white, African American, and Hispanic adults we found little evidence of expected associations between telomere length and dietary intake of specific food groups and dietary patterns. However, we did observe a significant association between high intake of processed meat and shorter telomeres, deserving of further investigation in larger longitudinal studies with baseline and repeat measures of telomere length.

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APPENDIX A

% Food group factor loading values on the dietary patterns of fats and processed meat and of whole grains and fruit

	Fats and processed meats	Whole grains and fruit
Added fats and oils	0.65	0.18
Processed meat	0.63	−0.12
Fried potatoes	0.60	−0.09
Salty snacks	0.50	0.08
Desserts	0.48	0.10
High-fat cheese or cream sauce	0.42	0.16
Red meat	0.42	−0.13
Pizza	0.42	0.08
Sweet breads	0.41	0.11
Pasta or potato salad	0.41	0.29
Ice cream	0.40	0.15
White potatoes	0.37	0.04
Poultry	0.36	0.01
Sugar-sweetened soda	0.36	−0.16
Sweets	0.36	0.13
Eggs or omelets	0.34	−0.08
Chicken, tuna, or egg salad	0.30	0.30
Coffee	0.29	0.16
Cream-based soup	0.28	0.11

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	Fats and processed meats	Whole grains and fruit
Refined-grain bread, rice, cereal, pasta	0.28	−0.19
Cream in coffee or tea	0.23	0.08
Beer	0.19	−0.03
Fish	0.14	0.13
Whole milk	0.13	−0.07
Seeds or nuts	0.13	0.46
Cottage or ricotta cheese	0.12	0.30
Tomatoes	0.12	0.25
Other alcohol	0.11	0.16
Diet soda	0.10	0.10
Other soups	0.09	−0.09
Hot chocolate	0.09	0.08
High-fat Chinese dishes	0.05	−0.21
Fruit juice	0.05	0.24
Low-fat dairy desserts	0.04	0.28
Other vegetables	0.04	0.27
Meal replacement drinks	0.03	0.10
Green, leafy vegetables	0.01	0.38
Low-fat milk	0.01	0.33
Yogurt	0.01	0.21
Whole-grain bread, rice, cereal, pasta	0.01	0.59
Tea	−0.03	0.09
Beans	−0.04	0.09
Dark yellow vegetables	−0.15	0.21
Soy foods or soy milk	−0.15	0.05
Avocados or guacamole	−0.15	0.10
Fruit	−0.17	0.55
Cruciferous vegetables	−0.18	0.13

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TABLE 1

Participant demographics, lifestyle characteristics, and food intakes across quartile (Q) of telomere length in 840 men and women from the Multi-Ethnic Study of Atherosclerosis[1]

	Telomere length				*P* for trend across quartiles[3]
	Q1 (0.37–0.72 [0.65])[2]	Q2 (0.72–0.83 [0.78])	Q3 (0.83–0.96 [0.88])	Q4 (0.95–1.75 [1.06])	
Age	65.3 ± 0.7[4]	62.4 ± 0.7	60.6 ± 0.7	57.6 ± 0.7	<0.001
Age-adjusted characteristics					
Sex (% male)	58.4	52.6	46.0	36.3	<0.001
Race-ethnicity (%)					0.58
Whites	11.7	20.2	23.6	19.2	
Blacks	33.5	26.4	24.6	24.1	
Hispanics	54.7	53.4	51.8	56.7	
Yearly gross family income (% <$25 000)	37.6	42.9	41.3	39.6	0.89
Education (% ≥high school)	76.3	75.1	70.6	68.4	0.04
Smoking (%current)	11.0	13.3	11.9	9.9	0.63
Cigarette pack-years (*n*)	9.7 ± 1.1	9.5 ± 1.1	7.2 ± 1.1	5.8 ± 1.1	0.006
Active leisure time (MET-min/wk)	2495 ± 223	2435 ± 219	2422 ± 219	2450 ± 223	0.93
Inactive leisure time (MET-min/wk)	1749 ± 73	1694 ± 72	1653 ± 72	1470 ± 73	0.006
BMI (kg/m^2)	29.0 ± 0.4	28.3 ± 0.4	29.4 ± 0.4	28.9 ± 0.4	0.55
Waist circumference (cm)	100.5 ± 1.0	98.6 ± 0.9	100.3 ± 0.9	99.5 ± 1.0	0.97
Energy intake (kcal)	1717 ± 55	1734 ± 54	1678 ± 54	1592 ± 55	0.11
Age- and energy-adjusted food group intake (servings/d)					
Whole grains	0.57 ± 0.04	0.55 ± 0.04	0.63 ± 0.04	0.55 ± 0.04	0.71
Refined grains	1.34 ± 0.05	1.37 ± 0.05	1.35 ± 0.05	1.39 ± 0.05	0.62
Fruit	1.85 ± 0.11	1.96 ±0.11	1.97 ± 0.11	1.95 ± 0.11	0.96
Vegetables	2.41 ± 0.10	2.19 ± 0.09	2.26 ± 0.09	2.44 ± 0.10	0.47
Low-fat dairy	0.73 ± 0.07	0.75 ± 0.07	0.66 ± 0.07	0.68 ± 0.07	0.94
High-fat dairy	0.66 ± 0.05	0.64 ± 0.05	0.61 ± 0.05	0.66 ± 0.05	0.43
Seeds or nuts	0.21 ± 0.03	0.28 ± 0.03	0.25 ± 0.03	0.23 ± 0.03	0.80
Nonfried fish	0.28 ± 0.02	0.22 ± 0.02	0.21 ± 0.02	0.21 ± 0.02	0.04
Red meat	0.33 ± 0.02	0.35 ± 0.02	0.35 ± 0.02	0.37 ± 0.02	0.42

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	Telomere length				P for trend across quartiles[3]
	Q1 (0.37–0.72 [0.65])[2]	Q2 (0.72–0.83 [0.78])	Q3 (0.83–0.96 [0.88])	Q4 (0.95–1.75 [1.06])	
Processed meat	0.19 ± 0.02	0.13 ± 0.02	0.12 ± 0.02	0.11 ± 0.02	0.001
Fried foods	0.22 ± 0.02	0.21 ± 0.02	0.20 ± 0.02	0.22 ± 0.02	0.68
Coffee	0.97 ± 0.09	0.97 ± 0.09	1.06 ± 0.09	0.97 ± 0.09	0.95
Nondiet soda	0.39 ± 0.07	0.41 ± 0.07	0.41 ± 0.07	0.48 ± 0.07	0.46
Age- and energy-adjusted dietary patterns (composed of summed intake of 47 weighted food groups)[5]					
Fats and processed meat	−0.17 ± 0.05	−0.27 ± 0.05	−0.24 ± 0.05	−0.33 ± 0.05	0.05
Whole grains and fruit	0.03 ± 0.07	−0.06 ± 0.06	0.11 ±0.06	−0.03 ± 0.07	0.79

[1] MET, metabolic equivalent.

[2] Telomere length (T/S ratio) range; median in brackets (all such values).

[3] Calculated with telomere length modeled as a continuous variable.

[4] $\bar{x}$ ± SE (all such values).

[5] By design, dietary patterns derived by principal components analysis have a $\bar{x}$ = 0.0 and SD = 1.0.

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TABLE 2

Adjusted mean differences in telomere length associated with a 1 serving/d greater intake of different food groups and a 1-SD increase in empirically derived dietary patterns among 840 men and women from the Multi-Ethnic Study of Atherosclerosis

	Model 1[1]	Model 2[2]	Model 3[3]
Food groups[4]			
Whole grains	−0.004 ± 0.01	−0.004 ± 0.01	−0.007 ± 0.01
Refined grains	−0.007 ± 0.01	−0.01 ± 0.01	−0.01 ± 0.01
Fruit	−0.003 ± 0.004	−0.003 ± 0.004	−0.003 ± 0.004
Vegetables	−0.004 ± 0.004	−0.006 ± 0.004	−0.002 ± 0.005
Low-fat dairy	−0.004 ± 0.01	−0.003 ± 0.01	−0.006 ± 0.01
High-fat dairy	−0.003 ± 0.01	−0.002 ± 0.01	−0.006 ± 0.01
Seeds or nuts	−0.005 ± 0.02	−0.004 ± 0.02	−0.004 ± 0.02
Nonfried fish	−0.02 ± 0.02	−0.02 ± 0.02	−0.03 ± 0.02
Red meat	−0.01 ± 0.02	−0.007 ± 0.02	−0.002 ± 0.02
Processed meat[5]	−0.06 ± 0.02	−0.05 ± 0.02	−0.07 ± 0.03
Fried foods	0.02 ± 0.03	0.02 ± 0.03	0.02 ± 0.03
Coffee	−0.002 ± 0.004	−0.001 ± 0.004	−0.001 ± 0.004
Nondiet soda	0.008 ± 0.01	0.01 ± 0.01	0.006 ± 0.01
Dietary patterns[6]			
Fats and processed meat	−0.009 ± 0.01	−0.005 ± 0.01	—
Whole grains and fruit	−0.005 ± 0.01	−0.006 ± 0.01	—

[1] Adjusted for study center, age, sex, race-ethnicity, and energy intake.

[2] Additionally adjusted for education, physical activity, smoking (current, former, or never smoker), and smoking pack-years.

[3] Additionally adjusted for other food groups listed in table.

[4] $\bar{x}$ difference ± SE per 1 serving/d difference in food group intake (or per 1-SD difference in dietary pattern score).

[5] P = 0.02, 0.03, and 0.006 for models 1–3, respectively; all other estimates, $P > 0.05$.

[6] $\bar{x}$ difference in telomere length ± SE per 1-SD dietary pattern score.

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TABLE 3

Adjusted mean telomere lengths by categories of intake of processed meat and telomere length in 840 men and women from the Multi-Ethnic Study of Atherosclerosis

		Telomere length (T/S ratio)		
Processed meat intake	**Intake range**	**Model 1[1]**	**Model 2[2]**	**Model 3[3]**
	servings/d			
Quartile 1 (*n* = 261)	0.0–0.0	0.847 ± 0.01[4]	0.844 ± 0.01	0.845 ± 0.01
Quartile 2 (*n* = 159)	>0.0–0.04	0.850 ± 0.01	0.852 ± 0.01	0.854 ± 0.01
Quartile 3 (*n* = 211)	>0.04–0.15	0.853 ± 0.01	0.854 ± 0.01	0.852 ± 0.01
Quartile 4 (*n* = 209)	>0.15–2.8	0.831 ± 0.01	0.834 ± 0.01	0.828 ± 0.01
P for trend[5]		0.02	0.03	0.005

[1] Adjusted for study center, age, sex, race-ethnicity, and energy intake.

[2] Additionally adjusted for education, physical activity (metabolic equivalent–min/wk in sedentary activity during leisure time), smoking status (current, former, or never smoker), and smoking pack-years.

[3] Additionally adjusted for intake of whole grains, refined grains, fruit, vegetables, nonfried fish, nuts or seeds, high-fat dairy, low-fat dairy, red meat, processed meat, fried foods, nondiet soda, and coffee.

[4] $\bar{x}$ ± SE (all such values).

[5] Calculated with intake (in servings/d) modeled as a continuous variable.

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TABLE 4

Adjusted mean differences in telomere length per 1-daily serving difference in the intake of specific types of processed meat and telomere length in 840 men and women from the Multi-Ethnic Study of Atherosclerosis[1]

	Intake range	Telomere length difference per 1 daily serving	*P*
	servings/d		
Ham or hot dogs[2]	0.0–2.0	−0.10 ± 0.04[3]	0.03
Sausage[4]	0.0–1.5	−0.03 ± 0.04	0.46
Organ meats[5]	0.0–2.0	−0.08 ± 0.07	0.27
Ham hocks[6]	0.0–0.5	−0.27 ± 0.2	0.11

[1]Adjusted for variables in model 2 that included study center, age, sex, race-ethnicity, energy intake, education, physical activity, smoking (current, former, or never smoker), and smoking pack-years.

[2]Includes ham, hot dogs, bologna, salami, and other lunch meats.

[3]$\bar{x}$ ± SE (all such values).

[4]Includes sausage, chorizo, scrapple, and bacon.

[5]Includes liver, including chicken livers, and other organ meats.

[6]Includes ham hocks, pigs' feet, and chicarones.