Low Social Support Is Associated With Shorter Leukocyte Telomere Length in Late Life: Multi-Ethnic Study of Atherosclerosis

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Objective: The primary goal was to test the hypothesis that limited social support (SS) is related to shorter leukocyte telomere length (LTL), particularly in an older adult population. **Methods:** Cross-sectional analyses were performed on 948 participants aged 45 to 84 years at Examination 1 of the Multi-Ethnic Study of Atherosclerosis (18.4% white, 53.1% Hispanics, and 28.5% African American). LTL was determined by using quantitative polymerase chain reaction, and SS was measured with the Enhancing Recovery in Coronary Heart Disease SS inventory. **Results:** Across the entire sample, SS was not associated with LTL ($p = .87$) after adjusting for demographic (age, sex, race/ethnicity, socioeconomic status), age × sex, age × race, health (body mass index, diabetes, pulse pressure), and life-style factors (smoking, physical activity, diet); however, the interaction term age (dichotomized) × SS was significant ($p = .001$). Stratification by age group revealed a positive association between SS (score range, 5–25) and LTL in the older (65–84 years; B[SE] = .005[.002]; $p = .007$) but not younger participants (45–64 years; $p = .12$) after adjusting for covariates. **Conclusions:** These results from a racially/ethnically diverse community sample of men and women provide initial evidence that low SS is associated with shorter LTL in adults aged 65 years and older and is consistent with the hypothesis that social environment may contribute to rates of cellular aging, particularly in late life. **Key words:** telomere length, social support, cellular aging, loneliness, isolation, older adults.

SS = social support; **LTL** = leukocyte telomere length; **B** = beta; **SE** = standard error; **MESA** = Multi-Ethnic Study of Atherosclerosis; **SES** = sosioeconomic status; **BMI** = body mass index; **ESSI** = ENRICHD Social Support Inventory.

BACKGROUND

Having limited social resources, few social ties, and/or feeling socially isolated increases risk for age-related disease morbidity and mortality, independent of traditional risk factors (1–3). In a recent meta-analysis of 148 studies examining associations between social relationships and mortality risk, relative risk for mortality associated with a lack of social integration was equal to or greater than the increased risk conferred by a number of other established risk factors (i.e., physical activity, obesity, and smoking) (3). The socioemotional selectivity theory (4) proposes that there is a shift in motivations to socially engage in late life as a consequence of changes in perceptions of how much time is left in the lifespan. This drives a redirection of attention to emotionally meaningful goals and relationships. However, accompanying this is often a reduction in social network size, which, in some instances, can increase feelings of isolation when there is a loss or lack of available meaningful relationships. These processes may cause older adults who are limited in social connections to experience feelings of social isolation, which may contribute to greater disease burden and premature death. The mechanisms through which social isolation affects disease vulnerability and death are likely multifaceted. Loneliness and low social support may influence health by altering cognitive appraisals of stress, enhancing the magnitude of autonomic and hypothalamic arousal to daily stress, interfering with restorative processes such as sleep, reducing humoral and viral immune defenses, and enhancing proinflammatory activity (5–9). Many of these factors may increase disease risk and premature death by accelerating cellular aging (10–13), which has been implicated in the pathophysiology of physical aging (14–18).

One indicator of cellular aging is telomere length, which acts as a mitotic clock by limiting cellular lifespan. Telomeres are protective protein-DNA complexes that cap the end of chromosomes and shorten with cell division and oxidative damage, thereby reflecting both cell replication history and oxidative stress burden (13,15,19,20). As telomeres progressively shorten over the lifespan, they can reach a critically short length and the cell is forced into a senescent state. This senescent state is characterized by an inability to divide and an altered behavior, including increased secretion of proinflammatory cytokines and extracellular matrix–degrading enzymes (21,22). In late life, there is an increasing proportion of late differentiated cells with short telomeres, putting older adults with shorter telomeres at greater risk for cellular senescence (23,24). Aging is also accompanied by a rise in inflammation, sometimes referred to as *inflammaging* (25–27), which is thought to arise from several sources including senescent cells. Inflammation may contribute to further shortening of telomeres with pathophysiological implications for age-related disease and system aging (14,28,29). Initial epidemiological evidence supports the links between aging and leukocyte telomere length (LTL) shortening (24,30,31). Old age has been linked to accelerations in LTL shortening compared with earlier adulthood (31). Importantly, this accelerated LTL shortening has been associated with elevated risk for mortality (32,33).

Later life (especially ages >65 years) is also a time of increased risk for chronic disease and death. Increasing age is accompanied by more rapid declines in physical functioning

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and increased frailty (34). This aging phenotype suggests that late life may be a time of particular vulnerability to factors that may affect the aging process.

Given that greater chronic psychological burdens in later life have been associated with shorter LTL (31) and greater inflammation (35), we hypothesized that biological aging, as indexed by LTL, in the later years of life would be magnified in the presence of social disconnection and limited social support. On the other hand, substantial social support may protect adults in late life from accelerations in biological aging.

Although several studies have linked socioemotional adversities such as pessimism, psychological stress, and depression with shorter LTL (11,12,36–39), no study to date has examined whether low social support, a particularly important psychosocial factor in older adults, is associated with an older biological age as evidenced by LTL shortening. Consistent with evidence that lack of social support increases risk for age-related disease morbidity and mortality in later years and declines in physical and biological aging are accelerated in old age, we predicted that observed differences in LTL by social support would be more pronounce in those aged 65 years and older.

The primary hypotheses are as follows: (1) individuals who report high levels of social support will have longer LTL than those who do not and (2) associations of LTL with social support will be greater in those who are in late life (65 years and older).

METHODS

Participants

Data were derived from the Multi-Ethnic Study of Atherosclerosis (MESA), a longitudinal study of subclinical and clinical cardiovascular disease risk in the United States of America (40), which recruited 6814 men and women free of clinical cardiovascular disease (ages 45–84 years) at baseline. Participants were recruited from six US communities (New York City, NY; Los Angeles County, CA; Baltimore, MD; Chicago, IL; Forsyth County, NC; St Paul, MN) between July 2000 and August 2002 through publicizing the study in local media, targeted recruitment through mailings of letters and brochures, and telephone calls (see Bild (40) for additional details on study objectives and design). The MESA Stress Study, an ancillary study designed to analyze the impact of stress on cardiovascular disease, added a measure of LTL on DNA (preserved from Examination 1) stored from 978 participants recruited from New York and Los Angeles sites (41). From this sample, 1 participant had no physical activity data, 11 participants were missing smoking history data, 15 participants were missing income data, and 3 participants had telomere values that were outliers (>3 standard deviation from the mean). Thus, analyses were performed on 948 participants, 176 (18.6%) white, 502 (53%) Hispanics, and 270 (28.5%) African American (the ancillary study did not enroll any Asian Americans). The demographics, health and life-style factors, the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Social Support Inventory (ESSI) and blood samples were obtained from the initial MESA visit (Examination 1). Informed consent was obtained from all participants included in the present analyses, and the study was approved by institutional review boards of each participating institution (University of California Los Angeles, Columbia University, and the University of Michigan).

Demographics, Health, and Life-Style Risk Factors

Age, sex, racial/ethnic identity, education, and income were recorded from Examination 1 interview. Race/ethnicity was classified into four categories and included white non-Hispanic, Hispanic, and African American. Because previous findings have reported associations of LTL with body mass index (BMI) (defined as kilograms divided by height in meters squared) (42,43), diabetic status (defined as ≥126 ng/dl fasting glucose or use of diabetic medications) (44), and pulse pressure (defined as systolic blood pressure minus diastolic blood pressure, expressed in mm Hg) (45), we selected these as covariates. Likewise, life-style risk factors associated with telomere length included in our model are physical activity (metabolic equivalent-min/wk of moderate and vigorous physical activity) (46), smoking history (pack-years cigarette smoked), and consumption of processed meats (previously reported by Nettleton et al. (47) to be associated with LTL in this MESA sample). A full description of these measures has been reported in previous studies (40,47,48). Based on prior analyses from MESA (47,49), educational level was estimated by categorizing into less than high school (HS), HS completion/some college, or bachelor degree or higher and creating two dummy variables (less than HS = 1, all other = 0; bachelor degree or more = 1, all others = 0). Current family income was estimated with the following four categories: less than $20,000, $20,000 to 39,999, $40,000 to 74,999, or $75,000 or more.

Social Support

The ESSI was designed to capture the extent of available social support with fewer items than other common scales to improve compliance and reduce participant burden in large epidemiological samples that typically have multiple questionnaires and physical examinations to complete at a visit (50). Responses to the five items are on a 5-point likert scale and assess whether there is someone available to the participant who will listen, give advice, show affection/love, provide emotional support, and can be confided in. Scores can range from 5 to 25. Higher values on this inventory are indicative of greater availability of social support. As reported previously (50,51), this scale has good reliability, with the five-item in the present analyses (Cronbach $\alpha = .90$; Mitchell et al. (50) reporting .87 for five-item), and has been cross validated with longer social support indexes (i.e., Perceived Social Support Scale, $r = .63$) (50).

Leukocyte Telomere Length

The telomere length measurement procedures have been reported previously (41). Briefly, real-time quantitative polymerase chain reaction methods were used for the determination of blood LTL (52). This method amplifies, through polymerase chain reaction, the DNA sequence of the telomere (T) and a single-copy (S) control gene (36B4) used to normalize values. Cycle threshold is converted to nanograms of DNA by using a standard curve of serial dilutions. With these values, a relative measure of LTL is computed (T/S ratio). Intra-assay and interassay coefficients of variation for this assay were 6% and 7%, respectively.

Analytic Strategy

Descriptive statistics for each predictor are reported by racial/ethnic category. Using χ^2 test, analysis of variance, linear regression, or independent samples t test, we tested for differences in social support and LTL by levels of covariates.

Linear regression analyses were used to estimate associations of the continuous variable social support with LTL, with Model 1 and all subsequent models, adjusting for age, sex, race/ethnicity, education, and income. Based on prior work in this sample (53), additive interactions between age and race/ethnicity and age and sex were also included in all models. Model 2 includes covariates in Model 1 and makes further adjustments by health factors that have previously been association with LTL including BMI (42), pulse pressure (45), and diabetes status (44). Model 3 includes Model 1 and Model 2 covariates and further adjusts for specific life-style factors previously reported to be associated with LTL, including physical activity (46), cigarette smoking, and consumption of processed meats (47). For one covariate, dietary data, there was a substantial number of missing values (number missing, 99). To prevent sample size changes in Model 3, we replaced missing dietary values with the sample mean. No other missing data were imputed.

Moderation of the association of social support with LTL by age was tested by using an age × social support interaction term. For these analyses, age was dichotomized into those younger than 65 years ($n = 547$) and those older than 65 years ($n = 401$; defined as "elderly" by the US Census). This "older" adults age group includes young-old and middle-old individuals with ages ranging from 65 to 84 years. Secondary analyses also tested for an interaction of age by

TABLE 1. Descriptive Statistics for Demographic, Health, Life-style, and Social Support Variables in the Full Sample and by Tertile of Social Support

	Full Sample (N = 948), M (SD) or %	Social Support 1st Tertile (n = 266), M (SD) or %	Social Support 2nd Tertile (n = 347), M (SD) or %	Social Support 3rd Tertile (n = 335), M (SD) or %	P
Age, y	61.4 (10)	60.5 (9.5)	62.5 (10.1)	61 (10)	.03
Sex (% male)	47.5	44.7	47.3	49.9	.46
Race					.14
White	18.6	19.2	21.3	15.2	
African American	28.5	26.3	30.5	28.1	
Hispanic	53	54.5	48.1	56.7	
Adult education					.32
Less than HS diploma	26.3	26.2	24.3	28.3	
HS diploma, some college	50.5	50	49.3	52.1	
Completed bachelor's degree	23.2	23.8	26.4	19.6	
Current family income/year					.002
$0–19,000	38.8	47.4	34	37	
$20,000–39,999	25.9	26.7	27.4	23.9	
$40,000–74,999	21.9	18.4	22.2	24.5	
≥$75,000	13.3	7.5	16.4	14.6	
Body mass index, kg/m^2	29 (5.6)	29 (5.4)	29 (5.7)	29 (5.6)	.99
Pack-years of smoking	8.2 (15.7)	9.4 (16.5)	8.7 (16.3)	6.6 (14.2)	.07
Pulse pressure, mm Hg	53.3 (16.6)	52.1 (15.6)	53.3 (16.9)	54.3 (17)	.27
Physical activity, MET	5691.8 (5525)	5588.7 (5397)	5588.2 (5590)	5881 (5570)	.74
% Diabetic	27.1	27.8	25.6	28.1	.73
Social support	21.1 (4.7)	14.7 (3.7)	22.1 (1.5)	25 (0)	NA
Leukocyte telomere length (T/S)	0.84 (0.17)	0.85 (0.18)	0.83 (0.16)	0.84 (0.17)	.10

For descriptive purposes, Table 1 reports p values for analysis of variance and Pearson χ^2 analyses by tertile of emotional social support. All regression models in Table 2 and reported in the text test the associations with social support as a continuous variable. M = mean; SD = standard deviation; MET = metabolic equivalent.

social support with age as a continuous variable. All analyses were performed on the full data set, except for analyses stratified by age.

RESULTS

Descriptive statistics for age, sex, education, income, BMI, smoking, pulse pressure, physical fitness, and percent diabetic by tertile of social support are displayed in Table 1. We found a significant positive association between social support and income ($F(3,944) = 6.34$, $p < .001$). Lower social support tended to be associated with more pack-years of smoking (B[SE] = − .02[.01], $p = .05$). No other demographic or health/life-style variables were associated with the ESSI.

Across the entire sample, chronological age was significantly inversely associated with LTL in an unadjusted model, with roughly a 0.005 shortening of LTL (T/S ratio) with each year increase in age ($F(1,947) = 96.74$, $R^2 = .09$, $p < .001$). Moreover, being male ($F(1,945) = 21.21$, $R^2 = .02$, B[SE] = −.05[.001], $p < .001$), smoking ($F(1,945) = 11.24$, $R^2 = .011$, B[SE] = − .001[.00], $p < .001$), consuming processed meats ($F(1,945) = 11.47$, $R^2 = .011$, B[SE] = −.075[.02], $p < .001$), and having lower pulse pressure ($F(1,945) = 13.42$, $R^2 = .013$, B[SE] = .001[.00], $p < .001$) were associated with age-adjusted shorter telomeres.

Linear regression analyses testing for associations of social support with LTL were performed, first adjusting for age, sex, race/ethnicity, SES, and the interaction of age with race and age with sex (Model 1). Subsequent models, in addition, were adjusted for health factors such as BMI, pulse pressure, and diabetes (Model 2) and for life-style factors such as smoking history, physical activity, and processed meat consumption (Model 3). As can be seen in Table 2, across the entire sample with ages ranging from 45 to 84 years, there were no significant associations of ESSI with LTL ($F(1, 936) = 0.29$, not significant). Further adjustments for health and life-style factors did not change this result.

In line with our second hypothesis, we proceeded to test for a significant interaction of age with social support, predicting that older adults would show stronger associations of the ESSI with LTL than younger participants. The results of these analyses can be seen in Table 2. The addition of the interaction term age (dichotomized) × ESSI to Model 1 revealed that the interaction was significant, $F(1,934) = 10.51$, B(SE) = .008(.002), $p = .001$. Additional adjustments in models 2 (B[SE] = .007[.002], p = .002) and 3 (B[SE] = .007[.002], $p = .002$) did not reduce the significant interaction effect. Stratified by age, group analyses revealed that there was a significant association between social support and LTL in the older (65-84 years) participants ($F(1,383) = 7.41$, R^2 change = .017, $p = .007$), independent of other health and life-style risk factors. No significant association of social support with LTL was present in the younger group (45–64 years; $p = .12$). A graphical representation of the interaction effect can be seen in Figure 1, where we report mean LTL values by tertile of social support within the two age categories. Among older participants, social support explained

TABLE 2. Estimated Mean Differences (Unstandardized β Coefficient) in Leukocyte Telomere Length in Older and Younger Participants Per Unit Increase in Social Support After Adjustments by Covariates

	Model 1			Model 2: Further Adjustment for Health Factors			Model 3: Further Adjustment for Life-style Factors		
	B (SE)	β	*p*	*B*(SE)	β	*p*	*B*(SE)	β	*p*
Entire sample (*N* = 948)									
ESSI (scale range: 5–25)	.001 (.001)	.017	.59	.00 (.001)	.012	.70	.00 (.001)	.005	.87
Younger (<65 y)									
ESSI (*n* = 547)	−.002 (.001)	−.06	.16	−.002 (.001)	−.06	.14	−.002 (.001)	−.07	.12
Older (≥65 y)									
ESSI (*n* = 401)	.006 (.002)	.15	.002	.005 (.002)	.15	.003	.005 (.002)	.13	.007

B = unstandardized β coefficient; SE = standard Error; β = standardized β coefficient; ESSI = ENRICHD Social Support Inventory.
Model 1: adjusted for age (centered), sex, age × sex, age × race, education, income. Model 2: adjusted for age (centered), sex, age × sex, age × race, education, income, body mass index, pulse pressure, diabetes. Model 3: adjusted for age (centered), sex, age × sex, age × race, education, income, body mass index, pulse pressure, diabetes, smoking, physical activity, dietary intake of processed meats.

1.7% of the variance in LTL when all other covariates were in the model. Moreover, with each unit decline in social support in the older group, we observed a 0.005 decline in LTL, which is comparable with a change of one chronological year in LTL in the current cohort sample. Secondary analyses were performed to test for an age by ESSI interaction with age as a continuous variable. Using Model 3 covariates, the interaction of age (continuous) × ESSI was not statistically significant, *B*(SE) = .000(.00), *p* = .17.

DISCUSSION

Our results based on a racially/ethnically diverse community sample of men and women provide initial evidence that low social support is associated with shorter LTL in older adults (65–84 years) but not in middle-aged participants (45–64 years), consistent with our hypothesis that the social environment may contribute to rates of cellular aging, particularly in late life when vulnerability to accelerated aging may be greater. Importantly, controlling for health risk factors and health behaviors that are thought to partially explain the associations between psychosocial factors and health outcomes failed to reduce the strength of the association between social support and LTL. These findings lend support to the hypothesis that social support is related to rates of cellular aging independent of common health risk markers and life-style.

Based on estimates of age effects on LTL derived from the same sample (i.e., T/S decline of −0.005 per year estimated from the entire cohort), the difference in LTL between the lowest and the highest tertiles of social support among the older age group (mean score of 14 versus 25) was equivalent to approximately 11 years of aging. These findings highlight a potential pathway through which limited social support contributes to disease initiation, progression, and premature death.

Mechanisms

In late life, there is a greater proportion of late-differentiated immune cells resulting from a lifetime of exposures to multiple sources of stimulation (e.g., bacterial, viral, and sympathetic nervous system) and subsequent proliferation. These older cells have unique biological characteristics including alterations of cellular metabolic activity, a rise in reactive oxygen species production, and a decrease in antioxidant capacity, making them more vulnerable to further telomere erosion (14). In older adults, a greater proportion of leukocytes have short telomeres (24,30), making later life a time of greater vulnerability to cellular dysfunction from aged cells. Here we report that low

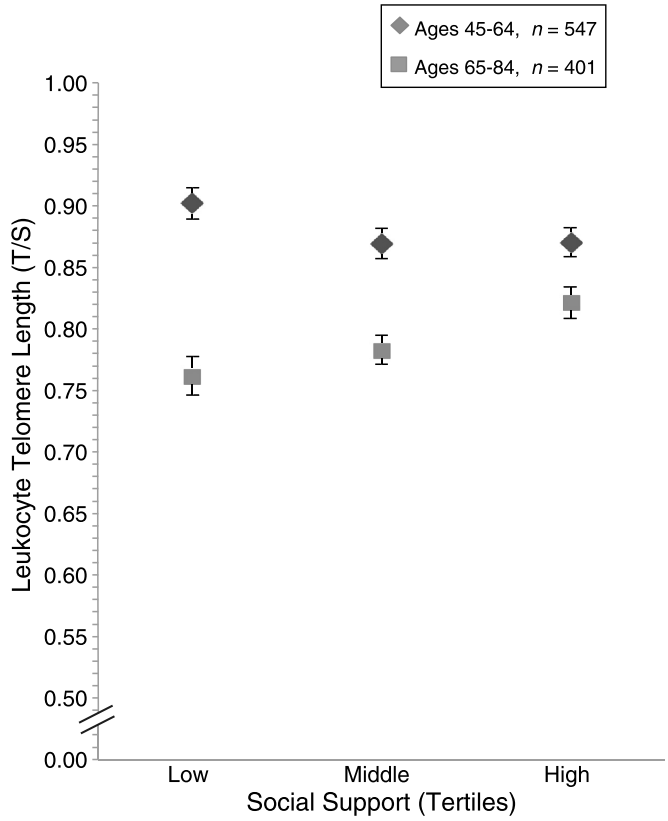

Figure 1. Mean leukocyte telomere length by tertile of social support and age category, corrected by all Model 3 covariates. The error bars denote standard error.

social support in later life is associated with shorter LTL. We posit that this association may reflect the impact of adverse social conditions on rates of cellular aging and thus help explain links of social adversity to a number of adverse health outcomes (1,4,8).

There are several potential mechanisms through which social support in late life may offer protection from telomere erosion or through which limited social support might contribute to loss. Several researchers have reported cross-sectional associations between chronic psychological stress and shorter LTL (12,36,38,54–56). Social support may act as a buffer to evaluations of threat and thereby reduce the magnitude of the stress response to adverse conditions (8,9). Only limited measures of psychological stress were available at MESA Examination 1 when the LTL measurement was performed, so we were unable to directly test this hypothesis. Low social support may also confer risk through contributing to elevated feelings of social isolation and loneliness, which has biological consequences that parallel chronic stressor exposure (5,57). It is well known that stress responses activate the hypothalamic-pituitary-adrenal axis and sympathoadrenal system, leading to the release of cortisol, epinephrine, and norepinephrine. These stress hormones signal a multitude of physiological changes that can increase inflammation (58–60) and oxidative stress (12,61–64). These factors are primary contributors to telomere loss in leukocytes.

Telomerase, the enzyme that rebuilds telomeres, is also modified by stress hormones. Recent evidence reports a decline in telomerase activity upon cortisol exposure in vitro (65), an inverse association of telomerase with nocturnal urinary epinephrine, and lower telomerase activity in those with exaggerated cardiovascular responses to a stressful task (66). Importantly, telomerase is not only important for rebuilding telomeres but can also protect older cells from entering senescence by capping the end of the chromosomes (21,22). In late life, this compromise in telomere capping may be more detrimental to health because there are greater proportions of late-differentiated cells that are vulnerable to senescence. These senescent cells cause further damage to nearby cells and tissue (67) and, in this way, may further promote telomere erosion.

Study Limitations and Strengths

There are several limitations to these analyses. First, the cross-sectional nature of these analyses prevents us from inferring causal relations. Ideally, studies should examine predictors of telomere change over time. Here, although social isolation in later life was associated with shorter LTL in this sample and this is compatible with greater attrition as a consequence of reduced social support, we cannot rule out the possibility that telomere erosion is associated with social support because it is a marker for chronic conditions causally linked to reductions in social support. For example, underlying morbidities and physical declines in functioning, which may be a consequence of or cause telomere loss, could drive individuals toward greater social withdrawal and thereby reductions in social support.

Given that cross-sectional associations of age with LTL estimate rates of shortening based on sample distribution rather than true longitudinal changes within individuals, our estimates of age effects on telomere erosion are bias and should therefore be interpreted with caution (68). Moreover, we cannot rule out the possibility that our age by social support interaction is caused by a unique cohort effect. For example, it is possible that social support is more strongly associated with LTL in older than in younger cohorts not because of age per se but because of interactions of social support with early exposures (e.g., trauma from war/economic hardship/malnutrition), which may be patterned by birth cohort. Likewise, this investigation was conducted on a single sample, and future work should seek to replicate this effect in an independent sample.

Another limitation of the present analyses is our measure of LTL. We cannot rule out the possibility that differences in a whole blood measure of telomere length are influenced by differences in the proportion of different types of white cell in circulation (e.g., percentage of neutrophils, T cells, etc), given that these cell types can have different telomere lengths (69). If white blood cell differential is linked to social support, it may confound estimates of associations of social support with LTL. Furthermore, the present assessment of LTL captures telomere length across the leukocyte pool, which likely reflects circulating progenitor and hematopoietic stem cell telomere length, but it is less clear whether this blood measure represents whole system biological aging (70,71).

Given that the cross validation of the ESSI with more substantial measures of social support was modest (r = .63) (50), our measure of social support undoubtedly includes some measurement error that could have affected our results. Future work should consider using more comprehensive measures of social support. It also seems plausible that the association of social support with LTL is caused by unmeasured individual factors (e.g., personality characteristics that persist across the lifetime that are also associated with low social support). Likewise, given that depression has been associated with shorter LTL (39,72), social support could be associated with LTL through history of depression, such that individuals with depression may alienate needed friends and family support. Future work should identify the causal path by using longitudinal analyses. Strengths of the present analyses include the large and diverse population sampled with detailed risk factor measurement. In addition, the analyses statistically controlled for several risk factors with known associations with LTL, including health and life-style factors. We were able to document that associations between social support and LTL in older adults remained significant after adjustments for these factors.

Future Directions

Further research examining the influence of socioemotional adversity on premature cellular aging is warranted. Longitudinal investigations may help to answer the question of whether socioemotional adversity might contribute to accelerated cellular aging that then increases the risk for physical declines, disease initiation and progression, and premature mortality.

Furthermore, as posited by our model of late-life biobehavioral vulnerability to cellular senescence, these socioemotional adversities may be particularly caustic in the later years of life. This increased vulnerability may be the result of altered cell behavior (e.g., increased inflammation, decreased telomerase activity, and immune compromise) and altered neuroendocrine patterns, which have been observed in older individuals experiencing chronic stress (73,74). Future research should focus on the consequences of neuroendocrine mediators on telomerase activity, gene transcription profiles, and inflammatory activity within these late-differentiated cells, with particular emphasis on characterizing which cells relevant to disease processes are particularly vulnerable. Lastly, it is conceivable that our measure of social support was associated with LTL in later life because it is related to social conditions of the individual that have existed for many years, and it is not until late life that the cumulative biological wear and tear becomes evident. Future work should consider social support over longer periods to better estimate cumulative effects.

Summary

In sum, in a multisite community sample of racially/ethnically diverse participants, we found that social support in late life but not in middle-aged participants is positively associated with LTL, an indicator of cellular aging. To our knowledge, this is the first report of an association between social support and LTL and suggests that telomere erosion may be a mechanism through which social isolation influences disease vulnerability and premature death in later life. These findings lend support to our model of late-life biobehavioral vulnerability to cellular senescence, which postulates that older adults may be more susceptible to the biologically significant downstream effects of behavioral and socioemotional factors because of the age of their biological system.

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