Published in final edited form as:

Prev Sci. 2015 February ; 16(2): 171–180. doi:10.1007/s11121-014-0474-2.

Prevention Effects Ameliorate the Prospective Association Between Nonsupportive Parenting and Diminished Telomere Length

Gene H. Brody,

Center for Family Research, University of Georgia, 1095 College Station Road, Athens, Georgia 30602-4527

Tianyi Yu,

Center for Family Research, University of Georgia, 1095 College Station Road, Athens, Georgia 30602-4527

Steven R. H. Beach, and

Center for Family Research, University of Georgia, 1095 College Station Road, Athens, Georgia 30602-4527

Robert A. Philibert

Department of Psychiatry, University of Iowa, Iowa City, IA, USA

Gene H. Brody: gbrody@uga.edu

Abstract

Telomere length (TL) is an indicator of general systemic aging, with diminished TL associated with several chronic diseases of aging and with heightened mortality risk. Research has begun to focus on the ways in which stress contributes to telomere attrition. The purposes of this study were (a) to establish whether exposure to nonsupportive parenting, defined as high levels of conflict and rancor with low levels of warmth and emotional support, at age 17 would forecast TL 5 years later; and (b) to determine whether participation in an efficacious family-centered prevention program could ameliorate any associations that emerged. Rural African American adolescents participated in the Adults in the Making (AIM) program or a control condition. Primary caregivers provided data on nonsupportive parenting during a pretest when adolescents were age 17. Adolescents provided data on anger at the pretest and at a posttest administered 7 months later. When the youths were age 22, TL was assayed from a blood draw. The results indicated that heightened nonsupportive parenting forecast diminished TL among young adults in the control condition but not among those who participated in AIM; socioeconomic status risk, life stress, and the use of alcohol and cigarettes at age 17, and blood pressure and body mass index at age 22, were controlled. Subsequent exploratory analyses suggested that AIM-induced reductions in adolescents' anger served as a mediator connecting group assignment to TL. The results suggest that the cellular-level sequelae of nonsupportive parenting and stress are not immutable.

© Society for Prevention Research 2014

Correspondence to: Gene H. Brody, gbrody@uga.edu.

The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute on Drug Abuse or the National Institutes of Health.

Keywords

Health promotion; Parent–child relations; Stress; Psychological; Telomere shortening

Introduction

A growing body of research has tested the hypothesis that family emotional climate and parent–child relationship quality across childhood and adolescence may contribute to chronic diseases later in life (Shonkoff et al. 2009). The risky family model offers a psychosocial account of the impact that stress during childhood and adolescence exerts on health (Repetti et al. 2002). It posits that some families confer risk for later health problems through chronically conflicted relationships characterized by a lack of warmth and emotional support. These familial dynamics trigger a cascade of psychological vulnerabilities, including deficits in the regulation of negative emotions and the propensity to compensate for them with health-compromising behaviors. They also lead to increased reactivity of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS). Frequent activation of these circuits triggers the release of hormones such as cortisol, epinephrine, and norepinephrine. The impact of these hormonal surges accumulates with time, leading to the wear and tear on bodily systems that is known as allostatic load (McEwen 1998) and to subsequent health problems (Shonkoff et al. 2009).

The present study was designed to advance understanding of the association between risky family processes and subsequent health status by testing hypotheses involving prospective pathways between nonsupportive parenting and telomere length (TL). Nonsupportive parenting places children and adolescents at risk through high levels of conflict and low levels of warmth and emotional support. TL is a biomarker of cellular wear and tear and of aging. Two hypotheses were tested with a sample of rural African American adolescents who took part in a randomized prevention trial. The first hypothesis proposed that adolescents who, at age 17, received highly nonsupportive parenting would evince shorter TL at age 22. The second hypothesis proposed that adolescents who, at age 17, participated in the Adults in the Making (AIM) prevention program (Brody et al. 2010b; Brody et al. 2012c) and who received highly nonsupportive parenting would evince longer TL, indicating less cellular stress, at age 22 than would youths in the control condition. In the following sections, we explore telomeres as a biomarker of stress, hypothesized protective-stabilizing effects of AIM, and anger as a mediator of protective-stabilizing effects.

Telomeres

To understand how social environments and psychological processes impact aging, researchers seek to identify bio-markers that provide a window into social environments' associations with longevity. TL appears to be such a marker. Telomeres, the protective caps at the tips of chromosomes, shorten with age; this shortening predicts both disease and longevity (Blackburn 2005; Epel 2009). TL may be viewed from a life-span approach because it reflects, in part, the cumulative number of cell divisions that have occurred and the long-term biochemical environment. Many studies find a negative association between age and TL (Monaghan and Haussmann 2006), but substantial variation in TL exists among

age-matched individuals. This suggests that factors other than chronological age affect telomere shortening. Reduction in TL has been associated contemporaneously with the presence of subclinical cardiovascular disease, cancer, stroke, diabetes, and autoimmune disease (Price et al. 2013). Although TL in these conditions could be merely an indicator of ongoing disease, other evidence implicates TL as a predictor of the likelihood of disease in the future despite current health status. Prospective studies indicate that shorter TL predates the occurrence of cancer (Ma et al. 2011; Wu et al. 2003), hypertension (Yang et al. 2009), and all-cause mortality (Cawthon et al. 2003).

A growing body of research has demonstrated that telomeres appear to shorten with exposure to chronic stress via the biological embodiments of stress exposure such as oxidative, endocrine, and inflammatory processes, along with other forms of biological stress (Aviv 2008). Studies have found financial stress (Epel et al. 2004), strain associated with care-giving (Kiecolt-Glaser et al. 2011), perceived overall stress (Bauer et al. 2009), and exposure to violence (Shalev et al. 2013) to be associated with diminished TL. This suggests that TL may be useful in determining how psychosocial stress is associated with biological aging at the cellular level. Consistent with suggestions that the chronic diseases of aging originate in changes in biological processes that occur at earlier stages of development (Shonkoff et al. 2009), recent studies have examined possible associations between childhood adversity and TL. Adverse childhood events that have been found to be associated retrospectively with TL include maltreatment, trauma, parental death, familial mental illness, and parental unemployment. Four of these studies reported that adults who retrospectively recalled childhood adversity had shorter TL than did controls (Kananen et al. 2010; Kiecolt-Glaser et al. 2011; O'Donovan et al. 2011; Tyrka et al. 2009). One retrospective study, however, did not replicate this association (Glass et al. 2010). The sole prospective study found that cumulative exposure to violence was associated with heightened telomere erosion in children across ages 5 to 10 years (Shalev et al. 2013). In our analyses, we sought to extend existing research by examining prospective associations between nonsupportive parenting and TL. The risky families model (Repetti et al. 2002), along with the research reviewed previously, support the first study hypothesis. We predict that exposure to nonsupportive parenting during adolescence, assessed at age 17, will forecast diminished TL during young adulthood, measured at age 22.

Protective-Stabilizing Effects of Prevention

Prevention researchers have demonstrated a specific form of moderation in which program effects are stronger for individuals who are at higher risk at program entry (Brody et al. 2012b). This type of moderated program effect can be viewed from the perspective of risk reduction. The program reduces the naturally occurring relation between risk and outcome that emerges in the control condition. Conceptually, this pattern is identical to a protective-stabilizing effect described in the resilience literature, in which a resilience resource reduces the negative impact of risk factors on development over time (Rutter 2005). From this perspective, a prevention program can be viewed as a “constructed resilience resource” because it is designed to develop and support processes that promote resilience. AIM was conceptualized as a resilience resource. It was designed to mitigate the negative impact of life stress on rural African American adolescents by increasing family-based buffering

processes such as emotional support, instrumental assistance, vocational coaching and advocacy, and racial socialization. The inclusion of these components of the curriculum was informed by studies of protective processes among rural African American families demonstrating that the provision of these resources increased the likelihood that youths would approach life stress with direct action rather than with anger, rumination, avoidance, drug use, or risky behavior (Luthar 2006). AIM has demonstrated stress-buffering capabilities for rural African American adolescents experiencing even the highest levels of life stress (Brody et al. 2010b; Brody et al. 2012c). For this reason, in this quasi-experimental analysis, we hypothesized that stress-buffering properties would be evident at the cellular level in TL.

Exploratory Hypothesis

Contingent upon empirical support for the protective-stabilizing hypothesis, we planned to conduct exploratory analyses to determine whether AIM-induced reductions in anger accounted for the program's effects on TL among adolescents who received high levels of nonsupportive parenting. We focused on anger for two reasons. First, one consistent consequence of nonsupportive parenting is the development and expression of chronic anger (Brody et al. 2010a; Simons et al. 2011). Second, such chronically angry states have been found to “get under the skin” by forecasting blood pressure reactions to stress (Fredrickson et al. 2000), elevated fasting glucose levels (Shen et al. 2008), heightened plasma lipid levels (Weidner et al. 1987), and levels of C-reactive protein, a measure of systemic inflammation (Brody et al. 2013a). Whether AIM effects on anger serve as a mediator of the hypothesized protective-stabilizing effect on TL is an open question, but it appeared to be a good candidate for this role.

Method

Participants

Participants in the AIM trial included 367 African American youths who, at the beginning of the study, were in high school. The families had an average of 2.4 children. Of the youths in the sample, 59.1 % were female and 63.6 % lived in single-mother-headed households. A majority of the youths' care-givers (78.7 %) had completed high school or earned a GED. The median family income of \$2,012 per month was representative of the sampled population (Boatright 2005); they can be described as working poor. Of the 367 youths who participated in the AIM trial, 216 agreed to the assessment of TL, measured via blood sample analysis, at age 22. These 216 participants constituted the sample in this quasi-experimental study. Of this subsample, 114 were in the intervention group and 102 were in the control group at age 17. Primary care-givers consented to their own participation and the participation of minor youth; youth assented or, if 18 or older, consented to their own participation. Each family was paid \$100 at each assessment.

To evaluate the equivalence of the baseline assessments of the study variables for participants who provided or did not provide telomere data at age 22 by prevention group assignment, a two-factor multivariate analysis of variance was executed. No significant main effects or interaction effects emerged for any study or confounder variables. Table 1

presents the means for univariate 2(Telomere data: provided or not provided)×2(Prevention group or control group) interaction results for each study variable. Additional analyses, using *t* tests, of the study variables' baseline equivalence among participants in the AIM and control groups who provided telomere data did not detect any differences. The means for these comparisons are presented in the first two columns of Table 1.

Intervention Implementation, Attendance, and Fidelity

The AIM prevention program, modeled after an existing family-based, skills-training group intervention for rural African American preadolescents (see Brody et al. 2004) consists of six consecutive weekly meetings held at community facilities, with separate parent and youth skill-building curricula and a family curriculum (see Brody et al. 2012c, for complete details). Each meeting includes separate, concurrent training sessions for parents and youths, followed by a joint parent–youth session, during which the families practice the skills they learned in their separate sessions. Concurrent and family sessions each last 1 h. Thus, both parents and youths receive 12 h of prevention training.

African American group leaders presented the prevention curriculum, organized role-playing activities, guided discussions among parents, and answered parents' and youths' questions. The parents' curriculum was designed to promote provision of emotional and instrumental support, problem-focused coping, and occupational and educational mentoring. Youths were taught how to develop a planful future orientation and to identify people in their communities who could help them attain their goals and cope with barriers and racial discrimination.

Data Collection Procedures

Data on parenting, anger, and demographic characteristics were collected at pretest in participants' homes using a standardized protocol at pretest, when youths were 17 ($M=17.7$, $SD=0.77$). The posttest assessment of anger was obtained 7 months after pretest, when most of the youths were 18. TL, BMI, and blood pressure were measured when youths were 22 ($M=22.0$, $SD=1.17$). Two African American field researchers worked separately with the primary caregiver and the target youth. Interviews were conducted privately, with no other family members present or able to overhear the conversation.

Measures

In addition to the primary study measures of nonsupportive parenting, anger, and TL, other variables were assessed as demographic and biobehavioral confounders that were statistically controlled in the data analyses. The control measures were socioeconomic status (SES) risk, life stress, smoking, and alcohol use assessed at age 17, and BMI and blood pressure assessed at age 22. SES risk, life stress, and drug use have been found to be associated with shorter TL (Price et al. 2013). BMI and blood pressure served as indicators of current health status.

Socioeconomic Status Risk Index—Six dichotomous variables formed a socioeconomic risk index that was used as a control in the data analyses. A score of 1 was assigned to each of the following characteristics: family poverty based on federal guidelines,

primary caregiver unemployment, receipt of Temporary Assistance for Needy Families, primary caregiver single parenthood, primary caregiver education level less than high school graduation, and caregiver-reported inadequacy of family income. The scores were summed to form an index that has been found to forecast biomarkers of stress in African American adolescents (Brody et al. 2013b).

Life Stress—Life stress was assessed at age 17 and was controlled in the analyses; youths endorsed a checklist of 12 events (e.g., acute economic stress, death of a friend, parental divorce, serious injury or illness; Brody et al. 2010a), indicating whether each had occurred during the previous 6 months. Because this index yields count data, internal consistency was not computed.

Smoking and Alcohol Use—Two items used to assess past-month smoking and alcohol use at age 17 were controlled in the analyses. Youths were asked how much they had engaged in each form of substance use. A 7-point response set ranging from *not at all* to *about two packs a day* was used for cigarette smoking; a 6-point scale ranging from *none* to *20 or more days* was used for alcohol use (Brody et al. 2012a). Because the distributions of both smoking and alcohol use were skewed, we applied a log transformation to normalize the ratings.

Youth Body Mass Index and Blood Pressure—Assessments of BMI and blood pressure were controlled in the analyses. At age 22, resting blood pressure was monitored with a Critikon Dinamap Pro 100 (Critikon; Tampa, FL, USA) while the youth sat reading quietly. Three readings were taken every 2 min, and the average of the last two readings was used as the resting index. Systolic and diastolic blood pressure were standardized and summed to form the blood pressure scores. The weight and height of each participant were recorded and used to calculate BMI (weight in kilograms divided by the square of height in meters).

Intervention Status and Gender—Intervention status and gender were dummy coded. AIM participants were coded 1 and control participants were coded 0; male participants were coded 1 and female participants were coded 0.

Nonsupportive Parenting—The nonsupportive parenting construct, expressed as an index and assessed at pretest, was derived from measures of parent–child conflict and parent support. Parent–child conflict was measured using two scales. On the first, parents completed the ineffective arguing inventory (IAI; Kurdek, 1994). They rated, on a scale ranging from 0 (*disagree strongly*) to 4 (*agree strongly*), statements about the conflicts they had with their children; $\alpha=0.79$. Example items include, “You and your child’s arguments are left hanging and unsettled,” and “You and your child go for days being mad at each other.” On the second, the 4-item arguing sub-scale from the discussion quality scale (DQS; Brody et al., 1998), parents reported, on a scale ranging from 1 (*never*) to 4 (*always*), how frequently they and their children argued about choice of friends, school or job, alcohol and other drugs, and sex; $\alpha=0.76$. The IAI and DQS items were highly correlated ($r=0.50$, $p<0.001$) and were summed to form an indicator of parent–child conflict. Parental support was also measured using parents’ reports on two scales. The first, a revised version of the 4-

item emotional support subscale from the Carver support scale (CSS; Carver et al. 1989) assessed levels of parental support. On a scale ranging from 1 (*not at all true*) to 5 (*very true*), parents responded to items such as, “My child discusses his/her feelings with me,” and “My child gets sympathy and understanding from me”; $\alpha=0.79$. On the second, the 5-item emotional support subscale from the family support inventory (FSI; Wills et al. 1992), parents rated statements on a scale ranging from 0 (*not at all true*) to 5 (*very true*) about the support they provided to their children; $\alpha=0.80$. Examples include, “My child can trust me as someone to talk to,” and “When my child feels bad about something, I will listen.” The CSS and FSI were highly correlated ($r=0.69$, $p<0.001$) and were summed to form an indicator of parental support. Parent–child conflict and parental support scores were standardized, and parent support was subtracted from parent–child conflict. High values indicated high parent–child conflict and low levels of emotional support.

Anger—Anger was measured using the 15-item anger subscale taken from the state-trait anger expression inventory that Spielberger et al. (1983) developed. Youths were asked about their feelings over the past 3 months and to rate discrete emotions (e.g., “I am furious”; “I feel angry”) on a scale ranging from 1 (*always*) to 5 (*never*). Cronbach’s alphas were 0.92 for pretest and 0.93 for posttest. The pretest value of anger was subtracted from the posttest value and served as the indicator of change from pretest to posttest.

Telomere Length—Certified phlebotomists went to each participant’s home to draw a blood sample. After the blood was drawn into serum separator tubes, it was frozen and delivered to the Psychiatric Genetics Lab at the University of Iowa for assaying. The measurement of TL involved several steps. Mononuclear (e.g., lymphocyte) cell pellets were generated using Ficoll separation (see Philibert et al. 2012). The resulting lymphocyte cell (LC) pellets were then prepared using a Qiagen QIAamp DNA Prep Kit according to the manufacturer’s instructions. Relative telomere/standard (T/S) ratios for each sample were calculated using a minor adaption of the improved quantitative polymerase chain reaction (PCR) method that Cawthon (2009) developed. In brief, 40 ng of LC DNA were placed robotically into 384-well optical PCR plates. The resulting DNA was then amplified using a set of primers specific for either telomeric sequence or a single-copy-number standard gene (albumin). The primers for the telomeric sequence are forward TGTTAGGTATCCCTATCCCTATCCCTATCCCTATCCCTAACA and reverse ACGTAAGGTTTGGGTTTGGGTTTGGGTTTGGGTTAGTGT. For the single-copy-number standard gene, albumin, the primers are forward GCCCGGCCCGCCGCGCCCGTCCCGCCGAAAAGCATGGTCGCTGTT and reverse CGGCGGCGGGCGGCGCGGGCTGGGCGGAAATGCTGCACAGAATCCTTG. The telomeric primers were used at a final concentration of 900 nM each, whereas the single-copy-number standard gene primers were used at a final concentration of 500 nM each. SybrGreen® Power Master Mix (Life Technologies, Carlsbad, CA, USA) was used to supply the buffer, DNA polymerase, and deoxynucleotides. The cycling parameters for each of the amplification stages were as follows: stage 1, 95° for 10 min; stage 2, 2 cycles of 15 s at 95°, 15 s at 49°, and 10 s at 62°; and stage 3, 38 cycles of 15 s at 95°, 15 s at 62°, and 10 s at 72°.

Results

Hypothesis Testing

Two regression models were executed to test the study hypotheses; the results are presented in Table 2. The first model tested the hypothesis that nonsupportive parenting when youths were 17 would forecast youth TL at age 22. The second regression model tested the hypothesis that participation in AIM would ameliorate the association between nonsupportive parenting and TL. This model tested the contributions to TL of nonsupportive parenting, assignment to AIM or the control group, and the interaction of nonsupportive parenting with group assignment. Gender; age 17 socioeconomic risk, life stress, smoking, and alcohol use; and age 22 BMI and blood pressure were controlled in each model.

The interaction analysis was executed based on the conventions that Aiken and West (1991) prescribed, whereby nonsupportive parenting is first mean centered, and interactions are calculated as the product of the centered nonsupportive parenting and prevention status. To interpret interactions, we plotted estimated levels of TL at low (1 standard deviation below the mean; -1 SD) and high (1 standard deviation above the mean; $+1$ SD) levels of nonsupportive parenting for each prevention status.

Nonsupportive Parenting at Age 17 and Telomere Length at Age 22—As hypothesized, nonsupportive parenting (Table 2, model 1; $\beta = -0.139$, $p < 0.05$) at age 17 forecast TL at age 22. Heightened levels of nonsupportive parenting forecast diminished TL across 5 years.

Participation in Prevention Programming and Risk of Diminished Telomere Length—Model 2 in Table 2 presents tests for hypothesized interaction effects between nonsupportive parenting and prevention program status on TL at age 22. Participants in the AIM condition were assigned a code of 1, and those in the control condition were assigned a code of 0. The analysis revealed a significant interaction of nonsupportive parenting with prevention status ($\beta = 0.216$, $p < 0.03$). This interaction is illustrated in Fig. 1. As hypothesized, participation in AIM moderated the association of nonsupportive parenting with TL. Nonsupportive parenting (simple-slope = -0.074 , $SE = 0.024$, $p < 0.01$) when youths were 17 was significantly associated with youth TL at age 22 among those in the *control* group. Nonsupportive parenting was not associated with TL among youths randomly assigned to the prevention group (simple-slope = 0.000 , $SE = 0.023$, $p = ns$). Additional analyses were performed to determine whether dose (number of prevention sessions attended) or participant gender conditioned any of these findings. No moderational effects were detected. The lack of a dose effect likely reflects its small variation among participants.

Test of Exploratory Hypotheses—To test the exploratory hypotheses, we performed a series of analyses comparing youths who received *high* levels of nonsupportive parenting (the top 30 %, $n = 65$) according to their participation in AIM ($n = 37$) or the control condition ($n = 28$). The exploratory hypotheses posited that changes in anger from pretest to posttest would mediate the group difference in TL at age 22 among youths who had received high levels of nonsupportive parenting during adolescence. The hypotheses were tested using regression-based mediational analysis procedures as depicted in Fig. 3. First, regression

coefficients were calculated for the association between intervention status and changes in anger (path A) and the association between changes in anger and TL (path B). Then, the indirect effect was quantified as the product of the two regression coefficients ($A \times B$). In addition, nonparametric bootstrapping, which has been found to be sensitive in mediational analyses (Preacher and Hayes 2004), was used to obtain the bias-corrected and accelerated confidence intervals (BCA) of the indirect effect for significance testing. The indirect, mediating effect was calculated 5,000 times using random sampling with replacement to build a sampling distribution. Gender and family SES were controlled in the analysis. Intervention status was significantly associated with changes in anger from pretest to posttest and TL at age 22, with AIM participants reporting smaller increases in anger and evincing longer TL than did participants in the control condition. These changes are depicted in Fig. 2. The significant negative coefficient between changes in anger and TL indicated that, the more a participant's anger increased from pretest to posttest, the shorter his or her TL was at age 22. Changes in anger significantly mediated the group effect on TL, such that the group effect was significantly reduced after accounting for changes in anger (indirect effect estimates = 0.116, BCA = 0.003–0.343 with 5,000 bootstrapping), which reduced the group effect to a nonsignificant relation (see Fig. 3). These analyses were re-executed using a latent difference score model. The results using this approach were identical to those of the previous analyses.

Discussion

On the basis of risky family theory (Repetti et al. 2002) and resilience theory (Rutter 2005), a protective-stabilizing hypothesis was tested about prevention effects at age 17 on TL at age 22 among rural African Americans. The results indicated that (a) caregiver-reported high levels of nonsupportive parenting were associated with diminished TL, and (b) young adults experiencing these risks who were assigned to the control condition evidenced shorter TL than did young adults assigned to the AIM condition who were exposed to the same parenting risk. This demonstration of program-induced maintenance of TL is important from a theoretical viewpoint because it shows that the developmental progression from a parenting risk factor to TL is not immutable. From a public health perspective, the results suggest that developmentally appropriate interventions designed to enhance supportive parenting practices can buffer the effects of receipt of nonsupportive parenting on TL.

Given the importance of family support, we maintain that these prevention efforts should be family-centered, focusing particularly on protective relationship processes and on the enhancement of emotion-regulatory and self-regulatory skills in adolescents. Helping parents to develop the ability to use emotional support, instrumental assistance, and communication about potential areas of concern promotes security, reduces hypervigilance, and encourages a positive sense of self that enables young adults to cope effectively with both daily hassles and self-imposed pressures that can take a silent toll on their biological health (Brody et al. 2013c). Additional components of such prevention programming could equip youths at different ages with developmentally appropriate (a) stress-coping skills and cognitive-behavioral management skills (Antoni et al. 2001), (b) mindfulness training that can help youths to relax and focus on the present (Bishop 2002), and (c) interventions that help children and adolescents to build and to access prosocial support networks.

We tested an exploratory hypothesis that reductions in anger would serve as a mediator connecting participation in AIM or the control condition with TL for adolescents who received high levels of nonsupportive parenting. This hypothesis was based on research suggesting that growing up with large doses of nonsupportive parenting does not lead to positive physical and mental health outcomes (Repetti et al. 2002). Its most prominent legacy concerns problems with emotion regulation, particularly with respect to elevated levels of anger and its expression (Simons et al. 2011). Research indicates that youths who received nonsupportive parenting over time become more likely to maintain a heightened state of vigilance for signs of anger and to respond with anger to incidents in which anger is expressed (Cicchetti and Rogosch 2009). Episodic and chronic anger states are hypothesized to trigger reactions from the HPA and SNS systems, which result in frequent releases of cortisol and catecholamines (Chorpita and Barlow 1998). These stress physiology pathways have been hypothesized and found to contribute to TL (Aviv 2008; Blackburn 2005). It is plausible that exposure to AIM may have dampened biological stress responses and inflammatory responses that have implications for TL. Future research should examine prevention-induced reductions in biological stress responses and, in turn, TL. Such a suggestion is not without precedent. A recent experimental evaluation of an intervention for children experiencing caregiver disruptions in foster care indicated that changes in the caregiving environment had ameliorative effects on catecholamine levels (Fisher et al. 2007).

Several limitations of the present study should be noted and addressed in future research. Despite ruling out potential threats to internal validity by demonstrating group equivalence on baseline factors and using relevant variables as statistical controls, this study is quasi-experimental. Although informative, this design can miss unmeasured direct and mediated effects. Clearly, assessing TL at baseline would allow a true experimental test of the study hypothesis. Other limitations should also be addressed in future research. First, additional measurements of nonsupportive parenting and anger across time would have strengthened the study by allowing assessments of the associations of their continuities or discontinuities with TL. Second, parents were the sole reporters of unsupportive parenting. Having youth as well as parent reports would strengthen the measurement of this variable. Third, data on psychosocial outcomes were not obtained at the age 22 assessment, when the telomere data were gathered. In future research, data should be gathered that will allow the examination of contemporaneous relations among TL, substance abuse, externalizing problems, and internalizing problems. Finally, it is not known whether the results of this study are generalizable to urban African Americans or to families of other ethnicities living in either rural or urban communities. Nevertheless, this analysis is one of the first to examine the ways in which nonsupportive parenting processes are linked to TL and to suggest that family-centered preventive intervention may interrupt this process.

Acknowledgments

This research was supported by Award Number P30DA027827 from the National Institute on Drug Abuse.

References

- Aiken, LS.; West, SG. Multiple regression: Testing and interpreting interactions. Thousand Oaks: Sage; 1991.
- Antoni MH, Lehman JM, Klibourn KM, Boyers AE, Culver JL, Alferi SM, et al. Cognitive-behavioral stress management intervention decreases the prevalence of depression and enhances benefit finding among women under treatment for early-stage breast cancer. *Health Psychology*. 2001; 20:20–32.10.1037/0278-6133.20.1.20 [PubMed: 11199062]
- Aviv A. The epidemiology of human telomeres: Faults and promises. *Journals of Gerontology: Series A Biological Sciences and Medical Sciences*. 2008; 63A:979–983. Retrieved from <http://biomedgerontology.oxfordjournals.org/content/63/9/979.abstract>.
- Bauer ME, Jeckel CMM, Luz C. The role of stress factors during aging of the immune system. *Annals of the New York Academy of Sciences*. 2009; 1153:139–152.10.1111/j.1749-6632.2008.03966.x [PubMed: 19236337]
- Bishop SR. What do we really know about mindfulness-based stress reduction? *Psychosomatic Medicine*. 2002; 64:71–83. Retrieved from <http://www.psychosomaticmedicine.org/content/64/1/71.abstract>. [PubMed: 11818588]
- Blackburn EH. Telomeres and telomerase: Their mechanisms of action and the effects of altering their functions. *FEBS Letters*. 2005; 579:859–862.10.1016/j.febslet.2004.11.036 [PubMed: 15680963]
- Boatright, SR. The Georgia county guide. 24. Athens: Center for Agribusiness and Economic Development; 2005.
- Brody GH, Flor DL, Hollett-Wright N, McCoy JK. Children's development of alcohol use norms: Contributions of parent and sibling norms, children's temperaments, and parent-child discussions. *Journal of Family Psychology*. 1998; 12:209–219.10.1037/0893-3200.12.2.209
- Brody GH, Murry VM, Gerrard M, Gibbons FX, Molgaard V, McNair LD, et al. The Strong African American Families program: Translating research into prevention programming. *Child Development*. 2004; 75:900–917.10.1111/j.1467-8624.2004.00713.x [PubMed: 15144493]
- Brody GH, Chen Y-f, Kogan SM. A cascade model connecting life stress to risk behavior among rural African American emerging adults. *Development and Psychopathology*. 2010a; 22:667–678.10.1017/S0954579410000350 [PubMed: 20576186]
- Brody GH, Chen Y-f, Kogan SM, Smith K, Brown AC. Buffering effects of a family-based intervention for African American emerging adults. *Journal of Marriage and Family*. 2010b; 72:1426–1435.10.1111/j.1741-3737.2010.00774.x [PubMed: 20976130]
- Brody GH, Chen Y-f, Yu T, Beach SRH, Kogan SM, Simons RL, et al. Life stress, the dopamine receptor gene, and emerging adult drug use trajectories: A longitudinal, multilevel, mediated moderation analysis. *Development and Psychopathology*. 2012a; 24:941–951.10.1017/S0954579412000466 [PubMed: 22781864]
- Brody, GH.; Kogan, SM.; Grange, CM. Translating longitudinal, developmental research with rural African American families into prevention programs for rural African American youth. In: King, RB.; Maholmes, V., editors. *The Oxford handbook of poverty and child development*. New York: Oxford University Press-USA; 2012b. p. 553-570.
- Brody GH, Yu T, Chen Y-f, Kogan SM, Smith K. The Adults in the Making program: Long-term protective stabilizing effects on alcohol use and substance use problems for rural African American emerging adults. *Journal of Consulting and Clinical Psychology*. 2012c; 80:17–28.10.1037/a0026592 [PubMed: 22182263]
- Brody GH, Yu T, Beach SRH, Kogan SM, Windle M, Philibert RA. Harsh parenting and adolescent health: A longitudinal analysis with genetic moderation. *Health Psychology*. 2013a Advance online publication. 10.1037/a0032686
- Brody GH, Yu T, Chen E, Miller GE, Kogan SM, Beach SRH. Is resilience only skin deep? Rural African Americans' preadolescent socioeconomic status-related risk and competence and age 19 psychological adjustment and allostatic load. *Psychological Science*. 2013b; 24:1285–1293.10.1177/0956797612471954 [PubMed: 23722980]
- Brody GH, Yu T, Chen Y-f, Kogan SM, Evans GW, Beach SRH, et al. Cumulative socioeconomic status risk, allostatic load, and adjustment: A prospective latent profile analysis with contextual

- and genetic protective factors. *Developmental Psychology*. 2013c; 49:913–927.10.1037/a0028847 [PubMed: 22709130]
- Carver CS, Scheier MF, Weintraub JK. Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology*. 1989; 56:267–283.10.1037/0022-3514.56.2.267 [PubMed: 2926629]
- Cawthon RM. Telomere length measurement by a novel monochrome multiplex quantitative PCR method. *Nucleic Acids Research*. 2009; 37:Article e21.10.1093/nar/gkn1027
- Cawthon RM, Smith KR, O'Brien E, Sivatchenko A, Kerber RA. Association between telomere length in blood and mortality in people aged 60 years or older. *Lancet*. 2003; 361:393–395. Retrieved from <http://proxy-remote.galib.uga.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=cmedm&AN=12573379&site=ehost-live>. [PubMed: 12573379]
- Chorpita BF, Barlow DH. The development of anxiety: The role of control in the early environment. *Psychological Bulletin*. 1998; 124:3–21.10.1037/0033-2909.124.1.3 [PubMed: 9670819]
- Cicchetti, D.; Rogosch, FA. Adaptive coping under conditions of extreme stress: Multilevel influences on the determinants of resilience in maltreated children. In: Skinner, EA.; Zimmer-Gembeck, MJ., editors. *New directions for child and adolescent development*: No. 124. Coping and the development of regulation. San Francisco: Jossey-Bass; 2009. p. 47–59.
- Epel ES. Telomeres in a life-span perspective: A new “psychobiomarker”? *Current Directions in Psychological Science*. 2009; 18:6–10.10.1111/j.1467-8721.2009.01596.x
- Epel ES, Blackburn EH, Lin J, Dhabhar FS, Adler NE, Morrow JD, et al. Accelerated telomere shortening in response to life stress. *Proceedings of the National Academy of Sciences of the USA*. 2004; 101:17312–17315.10.1073/pnas.0407162101 [PubMed: 15574496]
- Fisher PA, Stoolmiller M, Gunnar MR, Burraston BO. Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. *Psychoneuroendocrinology*. 2007; 32:892–905.10.1016/j.psyneuen.2007.06.008 [PubMed: 17656028]
- Fredrickson BL, Maynard KE, Helms MJ, Haney TL, Siegler IC, Barefoot JC. Hostility predicts magnitude and duration of blood pressure response to anger. *Journal of Behavioral Medicine*. 2000; 23:229–243.10.1023/A:1005596208324 [PubMed: 10863676]
- Glass D, Parts L, Knowles D, Aviv A, Spector TD. No correlation between childhood maltreatment and telomere length. *Biological Psychiatry*. 2010; 68:e21–e22.10.1016/j.biopsych.2010.02.026 [PubMed: 20678755]
- Kananen L, Surakka I, Pirkola S, Suvisaari J, Lönnqvist J, Peltonen L, et al. Childhood adversities are associated with shorter telomere length at adult age both in individuals with an anxiety disorder and controls. *PLoS ONE*. 2010; 5:Article e10826.10.1371/journal.pone.0010826
- Kiecolt-Glaser JK, Gouin JP, Weng NP, Malarkey WB, Beversdorf DQ, Glaser R. Childhood adversity heightens the impact of later-life caregiving stress on telomere length and inflammation. *Psychosomatic Medicine*. 2011; 73:16–22.10.1097/PSY.0b013e31820573b6 [PubMed: 21148804]
- Kurdek LA. Conflict resolution styles in gay, lesbian, heterosexual nonparent, and heterosexual parent couples. *Journal of Marriage and the Family*. 1994; 56:705–722.10.2307/352880
- Luthar, SS. Resilience in development: A synthesis of research across five decades. In: Cicchetti, D.; Cohen, DJ., editors. *Developmental psychopathology*: Vol. 3. Risk, disorder, and adaptation. 2. Hoboken: Wiley; 2006. p. 739–795.
- Ma H, Zhou Z, Wei S, Liu Z, Pooley KA, Dunning AM, et al. Shortened telomere length is associated with increased risk of cancer: A meta-analysis. *PLoS ONE*. 2011; 6:Article e20466.10.1371/journal.pone.0020466
- McEwen BS. Protective and damaging effects of stress mediators. *New England Journal of Medicine*. 1998; 338:171–179.10.1056/NEJM199801153380307 [PubMed: 9428819]
- Monaghan P, Haussmann MF. Do telomere dynamics link lifestyle and lifespan? *Trends in Ecology and Evolution*. 2006; 21:47–53.10.1016/j.tree.2005.11.007 [PubMed: 16701469]
- O'Donovan A, Epel E, Lin J, Wolkowitz O, Cohen B, Maguen S, et al. Childhood trauma associated with short leukocyte telomere length in posttraumatic stress disorder. *Biological Psychiatry*. 2011; 70:465–471.10.1016/j.biopsych.2011.01.035 [PubMed: 21489410]

- Philibert RA, Beach SRH, Brody GH. Demethylation of the aryl hydrocarbon receptor repressor as a biomarker for nascent smokers. *Epigenetics*. 2012; 7:1331–1338.10.4161/epi.22520 [PubMed: 23070629]
- Preacher KJ, Hayes AF. SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments and Computers*. 2004; 36:717–731.10.3758/BF03206553
- Price LH, Kao HT, Burgers DE, Carpenter LL, Tyrka AR. Telomeres and early-life stress: An overview. *Biological Psychiatry*. 2013; 73:15–23.10.1016/j.biopsych.2012.06.025 [PubMed: 22831981]
- Repetti RL, Taylor SE, Seeman TE. Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*. 2002; 128:330–336.10.1037/0033-2909.128.2.330 [PubMed: 11931522]
- Rutter ML. Environmentally mediated risks for psychopathology: Research strategies and findings. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2005; 44:3–18.10.1097/01.chi.0000145374.45992.c9 [PubMed: 15608539]
- Shalev I, Moffitt TE, Sugden K, Williams B, Houts RM, Danese A, et al. Exposure to violence during childhood is associated with telomere erosion from 5 to 10 years of age: A longitudinal study. *Molecular Psychiatry*. 2013; 18:576–581.10.1038/mp.2012.32 [PubMed: 22525489]
- Shen BJ, Countryman AJ, Spiro A III, Niaura R. The prospective contribution of hostility characteristics to high fasting glucose levels: The moderating role of marital status. *Diabetes Care*. 2008; 31:1293–1298.10.2337/dc07-1945 [PubMed: 18460671]
- Shonkoff JP, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *Journal of the American Medical Association*. 2009; 301:2252–2259.10.1001/jama.2009.754 [PubMed: 19491187]
- Simons RL, Lei MK, Beach SRH, Brody GH, Philibert RA, Gibbons FX. Social environment, genes, and aggression: Evidence supporting the differential susceptibility perspective. *American Sociological Review*. 2011; 76:883–912.10.1177/0003122411427580
- Spielberger, CD.; Jacobs, G.; Russell, S.; Crane, RS. Assessment of anger: The state-trait anger scale. In: Butcher, JN.; Spielberger, CD., editors. *Advances in personality assessment*. Hillsdale: Erlbaum; 1983. p. 159-187.
- Tyrka AR, Price LH, Gelernter J, Schepker C, Anderson GM, Carpenter LL. Interaction of childhood maltreatment with the corticotropin-releasing hormone receptor gene: Effects on hypothalamic-pituitary-adrenal axis reactivity. *Biological Psychiatry*. 2009; 66:681–685.10.1016/j.biopsych.2009.05.012 [PubMed: 19596121]
- Weidner G, Sexton G, McLellarn R, Connor SL, Matarazzo JD. The role of type A behavior and hostility in an evaluation of plasma lipids in adult women and men. *Psychosomatic Medicine*. 1987; 49:136–145. Retrieved from <http://www.psychosomaticmedicine.org/content/49/2/136>. [PubMed: 3575602]
- Wills TA, Vaccaro D, McNamara G. The role of life events, family support, and competence in adolescent substance use: A test of vulnerability and protective factors. *American Journal of Community Psychology*. 1992; 20:349–374.10.1007/BF00937914 [PubMed: 1415032]
- Wu X, Amos CI, Zhu Y, Zhao H, Grossman BH, Shay JW, et al. Telomere dysfunction: A potential cancer predisposition factor. *Journal of the National Cancer Institute*. 2003; 95:1211–1218.10.1093/jnci/djg011 [PubMed: 12928346]
- Yang Z, Huang X, Jiang H, Zhang Y, Liu H, Qin C, et al. Short telomeres and prognosis of hypertension in a Chinese population. *Hypertension*. 2009; 53:639–645.10.1161/HYPERTENSIONAHA.108.123752 [PubMed: 19255364]

Fig. 1.

The contribution of nonsupportive parenting to youths' telomere length by intervention status. *Numbers* in parentheses refer to simple slopes for control group and intervention group. ** $p < 0.01$

Fig. 2. Means of **a** change in anger from pretest to posttest and **b** telomere length at age 22 for the control ($n=28$) and intervention ($n=37$) groups. Error bars= ± 1 standard error

Fig. 3.

A mediational model of intervention status, change in anger from pretest to posttest, and telomere length at age 22 with socioeconomic-related risk and gender controlled.

Unstandardized coefficients are presented. $n=65$. $*p<0.05$

Table 1

Pretest equivalence of experimental condition for participants who did or did not provide telomere data

Variables at pretest	With telomere data			Without telomere data			$F_{1,363}$	p
	<u>Intervention ($n=114$)</u>			<u>Control ($n=102$)</u>				
	M	SD		M	SD			
Gender (male)	0.32	0.47		0.44	0.50		0.46	0.50
Family socioeconomic risk	1.90	1.32		1.97	1.42		2.04	1.52
Life stress	2.32	1.58		2.16	1.52		2.08	1.75
Smoking (past month)	0.06	0.13		0.07	0.13		0.05	0.12
Alcohol use (past month)	0.02	0.08		0.03	0.10		0.05	0.15
Nonsupportive parenting	-0.12	2.95		-0.26	3.01		0.42	3.03
Emotional support (FSI)	22.39	3.07		22.71	2.92		22.26	2.87
Social support (CSS)	17.17	2.62		17.20	3.02		16.28	3.05
Ineffective arguing (IAI)	13.91	5.29		13.72	5.04		14.27	4.90
Discussion quality (DQS)	2.15	2.45		2.20	2.66		2.50	2.93
Anger	31.99	10.50		32.29	10.69		30.38	11.72

FSI family support inventory, CSS Carver support scale, IAI ineffective arguing inventory, DQS discussion quality scale

Table 2

Nonsupportive parenting and intervention status as predictors of telomere length

Predictors	Telomere length (age 22) (n=216)					
	Model 1			Model 2		
	B	SE	β	B	SE	β
1. Gender, male	-0.135	0.119	-0.088	-0.121	0.118	-0.079
2. Family socioeconomic risk (age 17)	-0.002	0.038	-0.003	-0.003	0.038	-0.005
3. Life stress (age 17)	-0.027	0.034	-0.056	-0.040	0.034	-0.083
4. Smoking (age 17)	0.051	0.606	0.006	0.170	0.600	0.021
5. Alcohol use (age 17)	0.010	0.419	0.002	0.054	0.414	0.009
6. Blood pressure (age 22)	-0.008	0.006	-0.104	-0.010	0.006	-0.128
7. Body mass index (age 22)	0.024	0.034	0.062	0.023	0.033	0.059
8. Nonsupportive parenting (age 17)	-0.034	0.017	-0.139*	-0.074	0.024	-0.301**
9. Intervention (AIM)	-	-	-	0.171	0.103	0.114
10. Nonsupportive parenting $\times$ intervention	-	-	-	0.074	0.033	0.216*

AIM Adults in the Making intervention program

* $p<.05$,
** $p<.01$