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Non-supportive Parenting Affects Telomere Length in Young Adulthood Among African Americans: Mediation through Substance Use

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Abstract

Telomere length (TL) is an indicator of age related changes at the cellular level associated with heightened mortality risk. The effect of non-supportive parenting (NSP) during late adolescence and young adulthood on TL 5 years later was examined in a sample of N = 183 young adult African Americans to determine if effects of NSP on TL were mediated by substance use. Results indicated that the effect of caregiver reported NSP on diminished TL was mediated by escalation of drinking and smoking in young adulthood, even after controlling effects of socioeconomic status risk, gender, BMI, young adult stress, and intervention status. Results suggest that prevention of NSP may influence later physical health consequences by influencing substance use trajectory.

Keywords

health promotion; parent-child relations; stress; psychological; telomere shortening

Supportive parenting may be important for adolescent and young adult health in the context of difficult economic and social circumstances such as those confronting African American youth raised in impoverished rural counties in the southeast. In these areas African American youth face a range of stressors and lack of resources while residing in one of the most economically disadvantaged areas in the United States. In this context, lack of family support and the quality of parent-child relationships may contribute to chronic diseases later in life (Shonkoff, Boyce, & McEwen, 2009), in part, by increasing health-compromising behaviors, such as substance use. One potential mechanism of interest regarding long-term health effects is telomere shortening, a process that may be responsive to chronic stress.

Telomeres are complex protein caps that maintain chromosome integrity during replication (von Zglinicki, 2002), preventing loss of genetic material at the end of the chromsome. On average, TL becomes progressively shorter at each cell division until cell division ceases. For this reason, telomere attrition is thought by some to influence cell senescence. In addition, reduction in TL is associated with a number of specific diseases of aging such as

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cardiovascular disease, cancer, stroke, diabetes, and autoimmune disease (e.g., Ma et al., 2011; Wu et al., 2011; Yang et al., 2009), and is also a prospective risk-related bio-marker for cancer, hypertension, and all-cause mortality (Cawthon, Smith, O'Brien, Sivatchenko, & Kerber, 2003). A number of psychosocial factors have been shown to be associated with TL, e.g., cumulative exposure to stress (Lin, Epel, & Blackburn, 2012), childhood adversity (Kananen, et al., 2010; O'Donoven et al., 2011), strain associated with caregiving (Kiecolt-Glaser et al., 2011), perceived overall stress (Bauer, Jeckel, & Luz, 2009), and exposure to violence (Shalev et al., 2013).

One avenue by which NSP may become associated with TL is by increasing the risk for development of substance use among adolescents (Brody, Flor, Hollett-Wright, McCoy, 1998), with numerous studies indicating that problematic parenting is a significant predictor of substance use across early to late adolescence (Ryzin Fosco, & Dishion, 2012; Piko & Balazs, 2012). Rapid increase in substance use across early adolescence, in turn, predicts development of substance abuse disorders, poor psychosocial functioning, and poor mental health outcomes (Centers for Disease Control and Prevention, 2000). There is also evidence that substance use is associated with shortened telomeres, with TL being nearly halved in alcohol abusers compared with controls (Pavanello, et al, 2011). Likewise, cigarette and alcohol consumption negatively affected TL in a large, representative sample (Strandberg et al., 2012).

In keeping with the prior literature suggesting the likely effect of NSP on TL and the particular relevance of indirect pathways through substance use, we tested two hypotheses: 1) that among adolescents who, at age 17, received highly nonsupportive parenting, there would be a significant impact on TL, leading to shorter TL at age 22; 2) that the effect of NSP on TL would be mediated by its effect on rapid escalation in substance use during young adulthood.

Method

Participants

Participants in the current sample were drawn from the AIM trial (see Brody Yu et al., 2012 for a full description) which included 367 African American youths who, at the beginning of the study, were in high school. The median family income of $2,012 per month was representative of the sampled population (Boatright, 2005) who can be described as working poor. Of the 367 youths who participated in the AIM trial, 183 agreed to provide blood and saliva for biological assessments. These 183 participants (treatment group = 96; control group = 87) constituted the sample in the present study. Primary caregivers 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. The study was approved by the University of Georgia IRB.

To evaluate the equivalence of the baseline assessments of the study variables for participants who did not provide saliva or blood data compared to those who provided both, a series of F-tests were executed contrasting those with biological data (N = 183) to all

others in the original data set (n = 184). Table 1 summarizes these comparisons and presents the means for each study variable. No significant differences were observed.

Those participants in the AIM prevention program received a family-based, skills-training group intervention for rural African American preadolescents (Brody et al., 2004) consisting of six consecutive weekly meetings. African American group leaders presented the prevention curriculum. The parents' curriculum was designed to promote provision of emotional and instrumental support, as well as facilitate problem-focused coping, and occupational and educational mentoring. Effects of program participation are controlled in all analyses and direct effects on TL are more fully described in Brody et al. (2014).

Data Collection Procedures

Data on parenting, substance use, and demographic characteristics were collected at baseline in participants' homes using a standardized protocol when youths were 17 (*M* = 17.70, *SD* = 0.72). The follow-up assessments of substance use were obtained 7 months after baseline, when most of the youths were 18, and again when participants were 19 and 20. TL was measured when youths were 22 (*M* = 21.82, *SD* = 1.15), i.e., five years after baseline, at which time BMI was also assessed. All assessments were conducted by pairs of African American field researchers who worked separately with the primary caregiver and the target youth. Interviews were conducted privately, with no other family members present to enhance the validity of self-report of potentially sensitive information.

Measures

In addition to the primary study measures of nonsupportive parenting, substance use and TL, other baseline variables (gender, intervention status, socio-economic risk) were assessed and were statistically controlled because they were found to be associated with shorter TL in prior research (Price et al., 2013). We also controlled variables assessed in young adulthood (BMI at age 22 and young adult stress assessed at baseline) that might provide alternative pathways to individual differences in TL.

Smoking and alcohol use—Two items were used to assess past-month smoking and alcohol use at ages 17, 18, 19, and 20. 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. In keeping with standard practice, items were summed to form a frequency scale of substance use (Brody, et al., 2012).

Non-supportive parenting (NSP)—The non-supportive parenting index was derived from baseline measures of parent-child conflict and parent support. Parent-child conflict was measured using two scales: the Ineffective Arguing Inventory (IAI; Kurdek, 1994) on which they rated from 0 (*disagree strongly*) to 4 (*agree strongly*) statements about their conflicts with their children; $\alpha = .79$ (e.g. “You and your child's arguments are left hanging and unsettled) and the Arguing subscale from the Discussion Quality Scale (DQS; Brody et al., 1998), on which parents reported from 1 (*never*) to 4 (*always*), how frequently they and their children argued about a range of topics; $\alpha = .76$. The sum of the IAI and DQS provided an

indicator of overall conflict in the parent-child relationship. Parental support was measured based on parents' report of Emotional Support on the Carver Support Scale (CSS; Carver, Scheier, & Weintraub, 1989); α = .79, and a 5 item subscale from the Family Support Inventory (FSI; Wills, Vaccaro, & McNamara, 1992); α = .80. The sum of the CSS and FSI provided an indicator of parental support. Parent-child conflict and parental support scores were standardized, and parent support was subtracted from parent-child conflict to yield a single index of Non-supportive parenting, with higher values indicative of high parent-child conflict and low levels of emotional support (i.e., high NSP).

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.

Socioeconomic status risk index—Six dichotomous variables were summed to form a socioeconomic risk index that has been found to forecast biomarkers of stress in African American adolescents (Brody, Yu, Chen, Miller, et al., 2013). 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. Thus, higher scores indicate greater risk.

Life Stress was assessed at baseline using a 12-item checklist of negative stressful events (Ge, Conger, Lorenz, & Simons, 1994) to indicate whether or not a given event had occurred during the previous 6 months. The items on the checklist focused on serious events that the youth experienced directly, for example, having a serious accident, death of a friend or family member, failing in school, getting in trouble with the police, or being the victim of a violent crime. The maximum possible score of 12 would corresponded to a participant responding that he/she had experienced all of the negative life events on the list in the past six months. Eighty percent of respondents reported they had experienced at least one of the negative events (range 0 – 8, M = 1.678, SD = 1.47).

Telomere length—Certified phlebotomists went to each participant's home to draw a blood sample and collect information on height and weight for the calculation of BMI. Following our standard protocol, mononuclear (e.g. lymphocyte) cell pellets were generated using Ficoll separation (see Philibert, Beach, & Brody, 2012) and 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. The resulting ratios were normalized to the geometric mean of a set of three internal LC DNA standards plated 8 times on each plate. Additional laboratory methodological detail follows.

Forty ng of LC DNA were placed robotically into 384-well optical PCR plates and then amplified using a set of primers specific for either telomeric sequence or a single-copy-number standard gene (albumin) using a protocol slightly modified from Cawthon (2009).

The primer sequence is TGTTAGGTATCCCTATCCCTATCCCTATCCCTATCCCTAACA (forward) and ACACTAAGGTTTGGGTTTGGGTTTGGGTTTGGGTTAGTGT (reverse). For the single-copy-number standard gene, albumin, the primers are forward GCCCGGCCCGCCGCGCCCGTCCCGCC GGAAAAGCATGGTCGCCTGTT and reverse CGGCGGCGGGCGGCGCGGGCTGGGCG GAAATGCTGCACAGAATCCTTG. 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) was used to supply the buffer, DNA polymerase, and deoxynucleotides.

Results

Plan of Analysis

We tested hypothesized relationships in two steps. First, we examined direct effects between non-supportive parenting and telomere length using a standard regression framework and using control variables assessed at baseline and in young adulthood. Second, we examined hypothesized mediation through substance use, distinguishing initiation from escalation, by utilizing a two-part latent growth model (Muthen, 2001). This strategy decomposed change in substance use over young adulthood into a) a binary variable that indexed use vs. non-use in the preceding measurement period and b) a continuous variable that represented the frequency of substance use given that some had taken place. Use vs. non-use was analyzed using a random effects logistic latent growth model with the log odds of use regressed on predictors. For those reporting any use, level of use was analyzed as a latent growth model with intercept and slope regressed on predictors and with non-use treated as missing for purposes of the analysis of change in level of use.

Descriptive statistics—At baseline assessment, participants were 17.70 years old and 19.6% indicated having ever smoked cigarettes and 52.2% indicated having ever tried alcohol. The average participant scored 1.94 on the socioeconomic risk index, indicating that, on average, the sample was experiencing two indicators of poverty, with a range from 0 to 5) Indeed, 65.3 % of the sample was below 150% of the poverty level. Range of normalized TL for targets in the sample was −3.108 to 1.889. The average TL value did (not) differ as a function of gender for univariate or multivariate analysis, with mean normalized value for males = −.127 and mean for females = −.081.

Results of the initial regression analyses are presented in Figure 1. The hypothesized negative association of greater NSP with shorter TL was significant (β = −.150, p = .034). There were no significant effects attributable to sex or level of SES-related risk. There was a direct effect of intervention on TL (β = .195, p = .007), in the direction of intervention being associated with greater TL, an effect that has been further examined elsewhere (see Brody et al., 2014), demonstrating that intervention has beneficial effects on family dynamics. In addition, there was a significant effect of BMI (β = −.155, p = .030), but no significant effect of young adult stress.

To examine the hypothesized mediation effect, we entered substance use into the model, using the previously described two-stage approach to better characterize initiation vs. escalation. Substance use experience increased substantially across the ages examined. Percent reporting any use in the past month increased from 23% at age 17, to 27.3% at 18, 36.1% at 19, and 45.4% at twenty, and at the same time some individuals were escalating their level of past month use. Of primary theoretical interest was whether the significant effect of NPS on TL would become non-significant when substance use was entered into the model. As can be seen in figure 2, the mediational model fit the data well, chi-square (df = 3) = 2.111, p = .550. Turning to the paths in the model, the previously significant effect of NSP on TL became non-significant when substance use was entered into the model (going from β = −.150, p = .034 to β = −.013, p = .880). As hypothesized, there was a significant indirect effect = −.098, p = .027 that accounted for 91.59% of the association between NSP and TL. The significant indirect effect was due to the significant association of greater NSP with greater substance use escalation (β = .299, p = .005) and the significant association of greater substance use escalation with shorter TL (β = −.460, p = .000). There was no significant mediation related to substance use initiation. For the control variables there was no significant effect of BMI, young adult stress, or SES risk on substance use escalation, precluding these from being alternative sources of indirect influence on TL.

Discussion

Telomere length (TL) in young adulthood provides a biological marker that forecasts health effects in later years, allowing for examination of the role of non-supportive parenting (NSP) in creating a foundation for future health outcomes. Partially supporting our original hypotheses, we found that (1) caregiver-reported high levels of NSP were associated with diminished TL, and (2) that the effect of NSP on TL was fully mediated by its impact on substance use escalation. Below we elaborate implications and discuss limitations.

First, we found that the significant effect of NSP on TL was mediated through its effect on substance use escalation. Importantly, the two-stage approach used to characterize the effects allowed us to disaggregate impact on age of onset from level of escalation once onset had occurred. It was only the latter that was significantly associated with telomere length and with NSP, suggesting that it is rapid escalation in substance use in young adulthood that is associated with shorter TL, not simply initiation or experimentation. Accordingly, these results suggest the potential benefit of efficacious secondary prevention efforts focused on prevention of escalation of use among youth who have already tried alcohol and cigarette smoking (Brody, Yu, et al., 2012). A secondary prevention model would attempt to detect early substance use and prevent both rapid escalation and subsequent health consequences (Beach et al., 2014).

The findings taken as a whole suggest the value of early intervention to reduce NSP as a means of reducing both direct and indirect effects on long-term health outcomes, particularly to the extent these are forecast by TL. Indeed, direct intervention effects on TL were detected, even after including controls for other sources of influence and salient sources of stress, suggesting the potential value of intervention in interrupting the effects of NSP on later health outcomes (Brody et al., 2014). Likewise, direct effects of BMI on TL were

observed, indicating that obesity may also exert a negative effect on TL over time and may be an appropriate target of early intervention.

Several limitations of the present study should be noted. First, because we did not have repeated measures of TL we were not able to assess change in TL over time, raising the possibility that unmeasured individual differences accounted for observed effects. In particular, it is possible that events prior to the assessment of parenting at age 17 may have influenced telomere length as well as parenting at baseline, giving rise to the observed pattern. Although there were no observed differences between those providing biological data and those not providing biological data, because a large proportion of the sample did not contribute biological samples, unknown selection bias may have been introduced. Second, because recent meta-analyses suggest high heritability for telomere length (Broer et al., 2013), additive genetic effects may need to be better controlled in future examinations. Third, because parenting was not assessed until late in high school, the current study may have missed some important early effects of NSP. Fourth, it would be useful to test generalization to urban African Americans and to families of other ethnicities living in either rural or urban communities. Finally, the sample size is relatively small and so replication in a larger sample would be useful. Nevertheless, the current analyses suggest that one of the ways in which parenting processes are linked to TL is through their effect on substance use escalation, suggesting the potential value of early prevention as well as the potential value of secondary prevention efforts.

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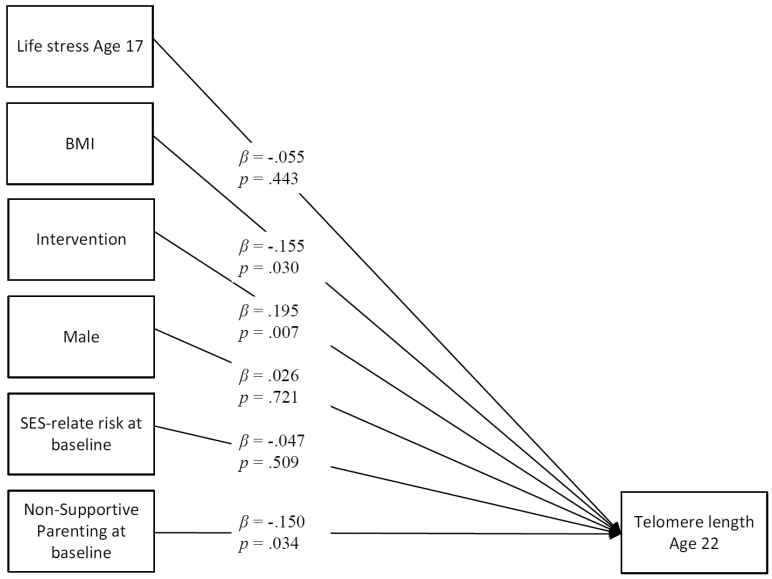

Figure 1.

A model of the impact of non-supportive parenting at baseline on telomere length at age 22, with socioeconomic-related risk, life stress, BMI, and gender controlled. *N* = 183 (males = 65; females = 118).

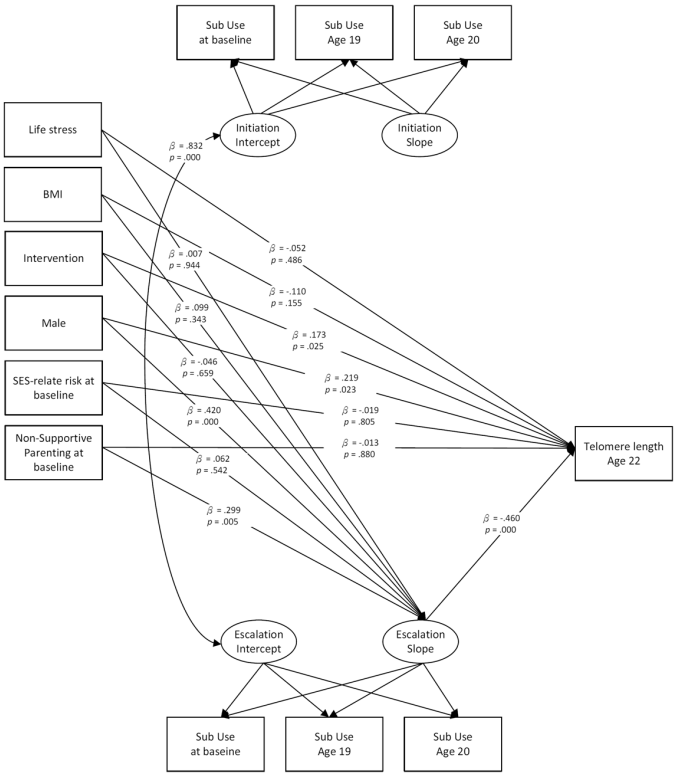

Figure 2.

Chi-square = 2.111, *df* = 3, *p* = .550. A mediation model of the effect of non-supportive parenting at baseline, growth of substance use from age 18 to 20, and telomere length at age 22 with effects of socioeconomic-related risk and gender controlled. Significant indirect pathway from NSP to TL through Escalation Slope = −.098, *p* = .027, accounting for 91.59% of the association between NSP and TL. *N* = 183 (males = 65; females = 118).

Table 1

Pretest Equivalence of Experimental Conditions for Participants Who Did or Did Not Provide Biological Data

	With Biological Data				Without Biological Data					
	Intervention (*n* = 97)		Control (*n* = 86)		Intervention (*n* = 90)		Control (*n* = 94)			
Variables at Pretest	***M***	***SD***	***M***	***SD***	***M***	***SD***	***M***	***SD***	***F* (1,363)**	***p***
Gender (male)	.29	.46	.44	.50	.46	.50	.46	.50	2.192	.140
Family socioeconomic risk	1.91	1.27	1.94	1.38	2.18	1.39	2.05	1.54	.300	.584
Life stress	2.41	1.63	2.06	1.48	2.23	1.60	2.18	1.74	.796	.373
Substance use (past month)	.30	.68	.34	.70	.26	.57	.37	.80	.294	.588
Smoking (past month)	.08	.34	.12	.45	.08	.34	.19	.57	.767	.382
Alcohol use (past month)	.22	.48	.22	.45	.18	.41	.18	.42	.000	.988
Non-supportive parenting	−.07	2.00	−.23	1.83	.30	2.04	.04	1.81	.050	.824
Emotional support (FSI)	22.32	3.13	22.67	2.97	22.41	3.13	22.37	2.84	.389	.533
Social support (CSS)	16.99	2.68	17.17	3.00	17.32	3.05	16.46	3.08	2.890	.090
Ineffective arguing (DAI)	13.94	5.35	13.40	5.10	14.13	5.01	14.48	4.83	.701	.403
Discussion quality (DQS)	2.04	2.26	2.03	2.49	2.58	3.20	2.60	3.00	.001	.975

Note. FSI = Family Support Inventory. CSS = Carver Support Scale. IAI = Ineffective Arguing Inventory. DQS = Discussion Quality Scale.