

THE ROLE OF OPTIMISM, CONNECTEDNESS, AND NEIGHBORHOOD COLLECTIVE
EFFICACY AS MODERATORS OF HARSH PARENTING ON TELOMERE ATTRITION

by

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(Under the Direction of Kalsea Koss)

ABSTRACT

Research to date has examined telomere length in relation to adverse childhood events but has rarely examined these associations longitudinally. Furthermore, few studies have examined whether protective factors act as a buffer to offset this effect. Using data from the Future Families and Child Wellbeing Study, the current study examines optimism, social connectedness, and neighborhood collective efficacy at 15 as potential protective factors against the effects of harsh parenting on telomere attrition during adolescence. This study examines cumulative exposure to harsh parenting across childhood (ages 3, 5, and 9). Telomere length at 15 and attrition from ages 9 and 15 are also examined. Results show optimism, connectedness, and neighborhood collective efficacy are protective for certain minority and male youth. Implications of these findings provide evidence for a need for safer, more cohesive neighborhood environments for Black and male youth, as well as a need for social connection for Hispanic youth.

INDEX WORDS: Telomere length; Telomere attrition; Harsh parenting; Optimism; Social connectedness; Neighborhood collective efficacy

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CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

Early life adversity broadly refers to the negative experiences that one may face during childhood. Forms of early life adversity include, but are not limited to, childhood maltreatment, parental separation, parental loss, or low socioeconomic status (Elwenspoek et al., 2017).

Adversity is detrimental to health across the lifespan and frequently is associated with higher risk for disease in adulthood (Elwenspoek et al., 2017). Researchers have often focused on aspects of early life adversity and psychosocial stressors throughout development to examine how these experiences affect biological processes from childhood to adulthood (Rentscher, Carroll, & Mitchell, 2020), including investigations for how early adversity may affect biological weathering. Biological weathering refers to the phenomenon in which individuals age “faster” than one’s chronological age. (Geronimus et al., 2006). Biological weathering is associated with a number of chronic illnesses such as diabetes, heart disease, high blood pressure, obesity, and irritable bowel syndrome (Blackburn, 2005).

One common way researchers have operationalized biological weathering is through the measurement of telomere length. Telomeres are the DNA sequences at the ends of chromosomes (TTAAGG) that protect from loss of genomic material during replication (Blackburn, Greider, & Szostak, 2006; Blackburn, Epel, & Lin, 2015). When DNA replication occurs, chromosomes duplicate, but often with a loss of genomic material (Moore, 2011). Because of this phenomenon, telomeres naturally shorten with every cycle of DNA replication. Consequently, telomere length, while a correlate of chronological age, may also reflect biological weathering or

biological aging. Biological age, in contrast to chronological age, captures the advanced aging of individuals who may appear to “age faster” than their chronological age (Ferrucci et al., 2020).

Aging is inherently longitudinal, and requires longitudinal examination. To date, few studies have examined longitudinal change in telomere length during childhood and adolescence (Gaydosch et al., 2021). Telomere attrition is the longitudinal loss of telomere length over time (Rentscher, Carroll, & Mitchell, 2020). However, change in telomere length may also not *only* include shortening, as studies have found that repeatedly measured telomeres may reflect lengthening (Gaydosch et al., 2021). Thus, it is important to investigate how childhood adversity influences both telomere length and change in telomere length over time.

1.1 Childhood Adversity and Telomere Length

A myriad of childhood adverse experiences is associated with shorter telomeres including experiences of poverty, childhood sexual abuse, and parental loss (Mitchell et al., 2017; Sosnowski et al., 2019). Further, a systematic review of adverse childhood experiences (ACEs) and telomere length showed that individuals who experienced a higher number of ACEs have shorter telomeres (Lang et al., 2020). A separate meta-analysis demonstrated that early life adversity in the form of abuse, neglect, socioeconomic disadvantage, and other adverse experiences were associated with shortened telomeres (Ridout et al 2018). However, the majority of this research focuses on cross-sectional telomere length rather than longitudinal change. Research on telomere attrition is scarce (Gaydosch et al., 2021). One study that did focus on telomere attrition found that exposure to violence during childhood is associated with increased telomere attrition from ages 5 to 10 (Shalev et al., 2013). Despite this exception, we know little about telomere attrition and other forms of childhood adversity.

1.1.1 Harsh Parenting and Telomere Length

A specific form of adversity is harsh parenting (Ethier, Couture, & Lacharite, 2004). Harsh parenting is a term used to reflect both parenting that lacks warmth and positive engagement between the parent and child, as well as the presence of harmful parenting (Repetti, Taylor, & Seeman, 2002). Neglect and aggression are two aspects of harsh parenting frequently examined. Neglect refers to parents failing to meet their child's emotional or physical needs; while aggression includes harm or the threat of harm directed toward the child (Stahmer et al., 2008). A substantial body of research demonstrates that harsh parenting is associated with poor health, anxiety, depression, and drug use (Repetti, Taylor & Seeman, 2002).

Both emotional and physical neglect have been associated with shorter telomeres later in life (Tyrka et al., 2010). Furthermore, harsh parenting, in the form of physical and emotional aggression, has been associated with shorter telomeres later in development compared to individuals who experienced no aggression (Mason, 2015). Moreover, a meta-analysis of 54 studies examining the association between child maltreatment and telomeres found that experiences of abuse and threat were associated with shorter telomeres, but experiences of neglect were not (Colich et al., 2020). It is important to note that these studies have not examined telomere attrition. Thus, the association between harsh parenting and telomere attrition (longitudinal shortening over time) remains unclear.

1.2 Resilience

Despite exposure to adversity, not all youth will demonstrate shortened telomere length or telomere attrition. It is necessary to examine the factors that account for this heterogeneity and identify those factors that contribute to resilience. However, little research on telomere length has examined the protective factors that lessen the impact of adversity on telomere length and attrition (Masten & Garmezy, 1985). Indeed, research demonstrates that positive parenting

practices buffer against adverse experiences and children's shorter telomeres. For example, parental responsiveness significantly moderated the association between early life stress and telomere length such that greater parental responsiveness was associated with longer telomeres in children that experienced higher levels of early life adversity (Asok et al., 2013). A recent meta-analysis found that positive parenting was associated with buffering against the effects of child maltreatment on shorter telomeres cross-sectionally for children (Colich et al 2020). Research also shows an indirect positive effect of prevention programs in ameliorating telomere shortening over longer periods of time (Beach et al., 2014).

In addition to buffering effects, some ecological factors may serve as *promotive* factors which demonstrate beneficial main effects on telomere length regardless of risk status. For example, children's positive mood and parents' marital affection predict longer telomeres in children (Robles et al., 2016), suggesting that positive family environments predict better outcomes for telomere length for all children, regardless of presence of risk. Both protective and promotive factors may reside in different ecological levels (Bronfenbrenner & Morris, 1998). As such, in the present study we will examine protective and promotive factors that are situated within the individual, within social relational contexts, and within community contexts.

1.3 Protective and Promotive Factors

1.3.1 Optimism

Optimism is the belief that the outcomes of a given experience or event will be positive (Carver, Scheier, & Segerstrom, 2010). People who have a more optimistic view on life are less susceptible to poor mental health outcomes, and in turn have reduced rates of depression, fatigue, and anxiety (Chida & Steptoe, 2008). Individuals with higher levels of optimism are significantly less likely to endorse feelings of stress and are more likely to seek and obtain social support

(Dougall, 2001). Optimism also serves as a protective factor for individuals experiencing adversity. For example, Gillham & Reivich (2004) found that helping adolescents think more optimistically led to a decrease in already present depressive symptoms. Further, optimism has been found to serve as a promotive factor such that higher levels of optimism are associated with longer telomeres in adults (Schutte, Palanisamy, & McFarlane, 2016). While protective effects of optimism against the effects of risk on shorter telomeres has yet to be investigated, existing research suggests that optimism may be protective against an adverse experience such as harsh parenting.

1.3.2 Social Connectedness

Social connectedness is the positive perception of how connected individuals are with peers, family members, or other individuals (Lee & Robbins, 1998). Connectedness and positive relationships with peers and friends can offset the risk of early life stress (Dumont & Provost, 1999). Research has found that children at pre-pubertal ages who endorse higher levels of connectedness with peers, school, neighborhood, family, and friends were better adjusted in adolescence as evidenced by higher confidence, life satisfaction, positive affect, and aspirations (Jose, Ryan, & Pryor, 2012). Further, research using the Future Families Child and Well-Being Study shows that social connectedness moderated associations between profiles of ACEs and depressive symptoms in adolescents (Sosnowski, Musci, & Johnson, 2021), such that higher levels of connectedness ameliorated the effects of ACEs on later depressive symptoms. Social connectedness also predicts longer telomeres in adults (Sosnowski et al., 2019). To our knowledge, the utilization of social connectedness as a protective factor for the effects of adverse childhood experiences on telomere length has only been examined for adults (Sosnowski et al., 2019). In a study on adults, Stein and colleagues (2018) found that a lack of social support

predicted shorter telomere length. Additionally, studies have found that social connectedness can offset the risk of childhood adversity effects on telomere length in perinatal women (Mitchell et al., 2018). Yet, research on adolescents in this area is lacking. Existing research reviewed suggests social support could protect against telomere attrition among individuals experiencing harsh parenting.

1.3.3 Neighborhood Collective Efficacy

Collective efficacy refers to the level of cohesion and trust within a neighborhood (Sampson, Raudenbush, & Earls, 1998). Individuals who reside in neighborhoods higher in collective efficacy report more adaptive behaviors and traits such as higher self-esteem (Wang & Fowler, 2019). Further, neighborhood collective efficacy has been found to protect children against maladaptive outcomes after exposure to adversity. Yonas et al. (2010) found that neighborhood collective efficacy protected individuals from the development of externalizing behaviors when they had experienced abuse and neglect. To our knowledge, higher levels of collective efficacy have not yet been investigated as a protective factor for telomere length; however, studies have found low neighborhood cohesion and lack of perceived safety are related to shorter telomeres (Thierry, 2020). Thus, research on neighborhood collective efficacy as a protective factor against shorter telomeres is lacking.

1.4 The Current Study

Although research demonstrates the effects of childhood adversity on biological weathering as indicated by telomere length, little research has examined the role of protective factors that may offset this risk. Thus, I investigate optimism, social connectedness, and neighborhood collective efficacy as moderators of this association. Individuals being more optimistic, having better social connections with others, and strong ties within their

neighborhoods may provide protective effects, off-setting higher levels of risk due to harsh parenting throughout childhood. The present study includes two aims. First, we investigate the extent to which harsh parenting during childhood is associated with shorter telomere length at age 15 as well as attrition from ages 9 to 15. Second, we examine whether optimism, social connectedness, and collective neighborhood efficacy serve as protective factors to attenuate the association between harsh parenting and (a) shorter telomere length and (b) telomere attrition. We hypothesize that higher levels of harsh parenting will result in shorter telomeres, as well as higher rates of telomere attrition. We also hypothesize that optimism, social connectedness, and collective neighborhood efficacy will significantly moderate the association of maltreatment and telomere length, such that higher levels of optimism, connectedness, and collective efficacy will attenuate the influence of harsh parenting on telomere length at age 15 and telomere attrition from ages 9 to 15.

CHAPTER 2

METHOD

2.1 Participants

Participants are from the Future Families and Child Wellbeing Study (FFCWS), which is a stratified, multistage sample of 4898 children born in large U.S. cities (with populations over 200,000) between 1998 and 2000. Births to unmarried mothers were oversampled by a 3 to 1 ratio, which resulted in the inclusion of a large number of Black, Hispanic, and low-income families. Mothers and fathers were interviewed shortly after birth and follow-up interviews were conducted when children were approximately ages 1, 3, 5, 9, and 15. Sample demographics at baseline (birth) are presented in Table 1. Unmarried parents accounted for 75.75% of participants and married parents accounted for 24.23%. The sex of the focal children was 52.18% male and 47.80% female. Average income at baseline was \$31,994 (SD =\$31,567.20). The ethnic/racial breakdown of the sample was 47.49% Black, 27.28% Hispanic, 21.03% White, and 3.96% reported other racial/ethnic backgrounds. Biological mothers accounted for 91.10% of primary caregivers (PCG), biological fathers accounted for 4.28% and 4.62% of PCGs reported other relationships with the child (e.g., grandparents). Sample descriptive statistics at age 9 are reported in Table 1. At age 9, youth's age in the study averaged 9.30 years (SD =.40). At age 15, youth's age in the study averaged 15.63 years (SD =.71). Saliva samples were collected for 86.79% of participants that participated in age 9 data collection. Participant demographics are shown in Table 1.

2.2 Procedures

2.2.1 *Telomere Length*

Saliva samples were collected at age 9 and age 15 in the study. A more detailed description of the telomere length collection and assay method can be found in Mitchell et al. (2017) and Gaydosh et al. (2021). Briefly, relative telomere length was calculated with the utilization of a quantitative real-time polymerase chain reaction (qPCR) approach which includes an oligomer standard approach. The relative telomere length was determined by the ratio of telomere copy repeats to a single copy reference gene (36B4). In order to create an absolute measure of telomere length as opposed to a relative measure, an 84mer oligomer standard TTAGGG was utilized to create a standard curve. A different standard curve was created to identify single-copy genes with a 79mer, and the same reference gene (36B4). With the new standard curve, a total telomere length per diploid genome was calculated. Length per chromosome was calculated by dividing telomere length per genome by 92 (number of telomeres per diploid genome). The final number was mean scored. The mean telomere length for this sample at age 9 was 9.90 kb (SD=3.30 kb). Telomere length was collected utilizing the same approach at age 9 and age 15. More information on telomere data for this sample at age 15 can be found in Gaydosh et al. (2021).

2.3 Measures

2.3.1 Harsh Parenting

The Conflict Tactics Scale (CTS) was used to measure harsh parenting. This questionnaire included 14 items adapted from the parent-child version of the CTS (Straus et al., 1998). Primary caregivers completed the CTS questionnaire when their child was ages 3, 5, and 9 (waves 3-5) to assess their use of physical assault (5 items), psychological aggression (5 items), and neglect (5 items). Physical assault items referred to how often the parent resorts to physical aggression and discipline (e.g. shaking their child in the last year, spanking their child). Psychological aggression items referred to the parent's tendency to react harshly towards the child without using physical force (e.g. threatening to spank or hit their child but not actually doing it, shouting, yelling, or screaming at him/her). Neglect items referred to the extent that a parent cannot meet their child's needs (e.g. getting too high or drunk to take care of their child, not able to make sure their child got the food they needed). Caregivers rated how often they used these strategies. Response options were a) once, b) twice, c) 3-5 times, d) 6-10 times, e) 11-20 times, f) more than 20 times, g) not in the past year, but it happened before, or h) this has never happened before. Responses g and h were both scored as zero. Each subscale was measured separately with a calculated mean score. Higher scores on the CTS scale reflects more harsh parenting and a cumulative harsh parenting from waves 3-5 was calculated to reflect harsh parenting score experienced throughout childhood. This scale demonstrates high reliability (Jouriles, 1997). Internal reliability of the harsh parenting scales for the full sample at age 3 were $\alpha = .94$, at age 5 were $\alpha = .99$, and at age 9 were $\alpha = .99$, respectively.

2.3.2 Optimism

Optimism was measured using a 4-item child-response scale at age 15 that refers to the individual's hopefulness about the future. Optimism was measured as a subscale of the positive adolescent functioning scale of engagement, perseverance, optimism, connectedness, and happiness (EPOCH; Kern et al., 2016). Children rate how much they agreed with the statements regarding their level of optimism (e.g. I am optimistic about my future). Responses were scored on a Likert scale that ranged from 1 (*strongly agree*) to 5 (*strongly disagree*). Answers were reverse coded for higher scores to indicate higher optimism. Psychometric properties demonstrate this scale has adequate reliability and validity (Kern et al., 2016). Internal reliability in this study was $\alpha = .61$.

2.3.3 Social Connectedness

Social connectedness refers to the sense that the individual has healthy and fulfilling relationships with other people, provoking a feeling of being loved, valued, or supported. Connectedness was measured as a subscale of the EPOCH scale (Kern et al., 2016). Example items include "when something good happens to me" and "I have people who I like to share good news with". Connectedness was reported by the child at age 15 on a 4-item Likert scale that ranged from 1 (strongly agree) to 5 (strongly disagree) during the past four weeks. Answers were reverse coded to signify a higher score meaning higher connectedness. Psychometric properties demonstrate this scale has adequate reliability and validity (Kern et al., 2016). Internal reliability for this scale was $\alpha = .56$.

2.3.4 Neighborhood Collective Efficacy

Neighborhood collective efficacy was a 9-item parent-response scale reported at age 15. The measure for neighborhood collective efficacy included two sets of items. This scale was modeled after the measures created by Sampson, Raudenbush, and Earls (1997). The first set referred to informal social control of neighborhood, and the second set of items referred to the amount of trust and cohesion in the individual's neighborhood. Informal social control generally refers to the ability of a group of people to regulate members of the group in compliance with their own set of expectations, rather than receiving formal, or forced control (Sampson, Raudenbush, & Earls, 1997). Level of trust and cohesion in the individual's neighborhood refers to a group's members ability to trust that other members will intervene to uphold a common harmony, or create an environment of solidarity (Sampson, Raudenbush, & Earls, 1997). Questions examined cohesion (i.e. "people around here are willing to help their neighbors") and perceived safety (i.e. "a fight broke out in front of the house or building"). Responses were administered on a Likert scale that ranged from 1 (very unlikely) to 4 (very likely). This scale has been found to have adequate reliability (Sampson, Raudenbush, & Earls, 1997). Internal reliability in this sample was $\alpha = .76$.

2.3.5 Covariates

Covariates included child sex, chronological age at the year 15 assessment, race and ethnicity, parental marital status and income at baseline, and pubertal development at age 9. Mothers reported pubertal development using the Pubertal Development Scale (PDS; Petersen et al., 1988). The PDS is a 4-point scale (5 items) with response options no, yes barely, yes definitely, and development is complete. This measure items included sex-specific bodily changes occurring during puberty such as growth spurts, gonadal development, and adrenal

hormone signals (body hair and skin changes). PDS scores were assessed and then converted into Tanner stages in accordance with Shirtcliff, Dahl, & Pollak (2009) reflecting gonadal and adrenal hormone development, as early pubertal development has been found to be associated with shorter telomeres for adolescent girls (Koss et al., 2020).

2.4 Data Analytic Plan

Regression analyses were conducted in Mplus 8.0 (Muthén and Muthén 1998-2017) to determine the effect of harsh parenting on telomere length and attrition. Optimism, connectedness, and neighborhood collective efficacy were tested as main effects (e.g., promotive factors) on telomere outcomes and as moderators of the link between harsh parenting and telomere outcomes (e.g., protective factors). Then, analyses were stratified by child sex and race/ethnicity. Child sex, chronological age at year 15, race and ethnicity, parental marital status, income at baseline, and pubertal development at age 9 were used as covariates when analyzing the full sample. Chronological age at year 15, race and ethnicity, parental marital status, income at baseline, and pubertal development at age 9 were used as covariates when stratifying by child sex. Finally, child sex, chronological age at year 15, parental marital status, income at baseline, and pubertal development at age 9 were used as covariates when stratifying by race and ethnicity.

CHAPTER 3

RESULTS

3.1 Descriptive Statistics

Means, standard deviations, and ranges for all study variables are displayed in Table 2. A one-way between-subjects analysis of variance (ANOVA) was performed to assess whether the study variables differed by child sex and race/ethnicity (see Table 3). There were no significant differences among boys and girls in telomere length at age 15, telomere attrition, optimism, and connectedness. However, there were significant differences between boys and girls in harsh parenting and neighborhood collective efficacy. Boys ($M = .92, SD = .55$) experienced higher levels of harsh parenting than girls ($M = .82, SD = .53; F(4221) = 35.73, p < .001$). Boys ($M = 2.91, SD = .62$) also reported higher levels than girls ($M = 2.87; SD = .63$) in neighborhood collective efficacy ($F(3415) = 3.76, p = .05$). A one-way between subjects ANOVA was conducted to examine the differences among racial/ethnic groups in TL at age 15, telomere attrition, harsh parenting, neighborhood collective efficacy, optimism, and connectedness (See Table 4). Significant differences were not observed among groups in TL at age 15, telomere attrition, and connectedness. However, there were significant racial/ethnic differences in harsh parenting, neighborhood collective efficacy, and optimism. Black youth ($M = 1.01, SD = .55$) were exposed to higher levels of harsh parenting than White youth ($M = .77, SD = .44$), Hispanic youth ($M = .76, SD = .50$), and youth from other racial and ethnic backgrounds ($M = .77, SD = .54; F(3164) = 40.69, p < .001$). Black youth ($M = 3.48, SD = .47$) also reported higher levels of optimism than White ($M = 3.29, SD = .53$), Hispanic ($M = 3.38, SD = .50$), and youth from other

racial and ethnic backgrounds ($M = 3.32$, $SD = .46$; $F(3262) = 21.09$, $p < .001$). Lastly, White youth had significantly higher levels of neighborhood collective efficacy ($M = 2.99$, $SD = .62$) than Black youth ($M = 2.87$, $SD = .64$; $F(3245) = 5.13$, $p < .001$). Bivariate correlations are displayed in Table 3. Cumulative harsh parenting was correlated across multiple time points (ages 3, 5, and 9), suggesting that harsh parenting maintained relatively stable rank order throughout childhood. Cumulative harsh parenting was also correlated with marital status at baseline, child sex, neighborhood collective efficacy, optimism, and connectedness such that cumulative harsh parenting was higher when parents were unmarried, when children were male, and when children lived in neighborhoods with low collective efficacy. Cumulative harsh parenting was negatively correlated with optimism and connectedness. Telomere length at age 15 was not significantly correlated with any of the study variables; however, telomere attrition was strongly correlated with income at baseline such that lower family income was associated with more telomere attrition ($r = -.083$, $p < .01$). All three moderators were strongly correlated with harsh parenting at each time point such that higher harsh parenting was associated with lower neighborhood collective efficacy, optimism, and connectedness.

3.2 Harsh Parenting and Telomere Length at 15

Hierarchical regression analyses were performed to determine the main effects of cumulative harsh parenting on telomere length at age 15. Two-way interactions were then computed between harsh parenting and each of the three study moderators: optimism, connectedness, and neighborhood collective efficacy. The first step included each covariate (child sex, family income, parental marital status, race/ethnicity, pubertal status, and child age) and a mean-centered predictor to examine the effects of cumulative harsh parenting. The second step included the addition of the main effect of the moderator for each analysis (optimism,

connectedness, and neighborhood collective efficacy). The third step added the interaction effect between cumulative harsh parenting and the moderator. Analyses were conducted separately for each moderator. Results are shown in Tables 6-14. Main effects of cumulative harsh parenting were not predictive of telomere length at age 15 (all $ps > .05$).

3.2.1 Optimism as a Moderator in the Full Sample

Results of harsh parenting predicting telomere length at age 15 with optimism as a moderator are displayed in Table 6. Results showed that the main effect of optimism was not predictive of telomere length at age 15 ($\beta = -.004, p = .88$). The interaction between optimism and cumulative harsh parenting was not significantly associated with telomere length at age 15 ($\beta = .04, p = .15$).

3.2.2 Connectedness as a Moderator in the Full Sample

Results of harsh parenting predicting telomere length at age 15 with connectedness as a moderator are displayed in Table 9. The results for the main effects of connectedness showed non-significant associations between social connectedness and telomere length at age 15 ($\beta = .01, p = .84$). The interaction between social connectedness and cumulative harsh parenting was also not significantly associated with telomere length at age 15 ($\beta = .28, p = .16$).

3.2.3 Neighborhood Collective Efficacy as a Moderator in the Full Sample

Results of harsh parenting predicting telomere length at age 15 with neighborhood collective efficacy as a moderator are reflected in Table 12. The main effects for neighborhood collective efficacy predicting telomere length at age 15 was not significant ($\beta = .00, p = .99$). The interaction between cumulative harsh parenting and neighborhood collective efficacy was not significantly associated with telomere length at age 15 ($\beta = .04, p = .20$).

3.2.4 Cumulative Harsh Parenting Predicting Telomere Length at 15 Stratified by Child Sex

Regression analyses were stratified by child sex to examine whether there were unique effects for boys and girls. Hierarchical regression models for predicting telomere length at age 15 for boys and girls are shown in Tables 7, 10, and 13. Results showed that when separated by sex, the effects of cumulative harsh parenting on telomere length at age 15 were not significant for boys or girls, respectively ($\beta = .01, p = .72$) ($\beta = -.03, p = .49$). There was also no significant moderation for either boys or girls for each two-way interaction (optimism, connectedness, and neighborhood collective efficacy).

3.2.5 Cumulative Harsh Parenting Predicting Telomere Length at 15 Stratified by Race/Ethnicity

The regression analyses were stratified by race/ethnicity to examine whether there were unique effects for Black, Hispanic, and White Youth.

3.2.5.1 Cumulative Harsh Parenting Predicting Telomere Length at 15 for White Youth

The hierarchical regression models for predicting telomere length at age 15 for White youth are shown in Tables 8, 11, and 14. Results showed that there were no main effects of cumulative harsh parenting on telomere length at age 15 for White youth ($\beta = .04., p = .55$). There was also no significant moderation for White youth for each two-way interaction (optimism, connectedness, or neighborhood collective efficacy).

3.2.5.2 Cumulative Harsh Parenting Predicting Telomere Length at 15 for Black Youth

The hierarchical regression models for predicting telomere length at age 15 for Black youth are shown in Tables 8, 11, and 14. There were no main effects of cumulative harsh parenting on telomere length at age 15 ($\beta = -.01., p = .56$) nor moderation by connectedness for Black youth ($\beta = -.01, p = .90$). However, the effect of harsh parenting on telomere length at age 15 was significantly moderated by optimism among Black youth ($\beta = .13, p < .01$) (See Table 8).

To probe interaction effects, simple slopes plots (see Figure 3A; Dawson 2014) and Johnson-Neyman plots (see Figure 3B; Johnson & Neyman, 1936) were used. In the Johnson-Neyman plots, the x-axis represents optimism, and the y-axis represents the unstandardized coefficient of the effects of harsh parenting on telomere length at 15. The Johnson-Neyman plot demonstrates the effect of harsh parenting on telomere length is significant at lower, but not higher levels of optimism. A simple slopes analysis revealed that for Black youth with low levels of optimism (plotted at a mean-centered value of -1.50), the effect of harsh parenting on telomere length at age 15 was significant ($t = -3.06, p < .01$) such that high harsh parenting was associated with shorter telomere length. For youth with higher levels of optimism (plotted at the mean), there was no effect of harsh parenting on telomere length ($t = -.83, p = .41$). Collectively, these findings are consistent with a buffering hypothesis. Lastly, there was significant moderation by neighborhood collective efficacy for Black youth ($\beta = .09, p < .05$) (See Table 14). The Johnson-Neyman plot reveals that the effect of harsh parenting on telomere length is significant at lower, but not higher levels of collective efficacy. A simple slopes analysis revealed that for Black youth with low levels of neighborhood collective efficacy (plotted at a mean-centered value of -1.00), the effect of harsh parenting on telomere length at age 15 was significant ($t = -2.01, p < .05$) such that high harsh parenting was associated with shorter telomere length. For Black youth with higher levels of neighborhood collective efficacy (plotted at the mean), there was no effect of harsh parenting on telomere length ($t = -.40, p = .69$). These results are also consistent with a buffering hypothesis.

3.2.5.3 Cumulative Harsh Parenting Predicting Telomere Length at 15 for Hispanic Youth

The hierarchical regression models for predicting telomere length at age 15 for Hispanic youth are shown in Tables 8, 11, and 14. Results showed that there were no main effects of cumulative harsh parenting on telomere length at age 15 for Hispanic youth ($\beta = .01, p = .56$). There was also no moderation by optimism ($\beta = -.05, p = .38$) nor neighborhood collective efficacy ($\beta = -.02, p = .64$) for Hispanic youth. There was significant moderation by connectedness for Hispanic youth ($\beta = -.10, p < .05$). The Johnson-Neyman plot reveals that the effect of harsh parenting on telomere length is significant at lower, but not higher levels of connectedness. A simple slopes analysis revealed that for Hispanic youth with low levels of connectedness (plotted at a mean-centered value of -2.00), the effects of harsh parenting on telomere length at age 15 were significant ($t = .29, p < .05$) such that high harsh parenting was associated with longer telomere length. For youth with higher levels of connectedness (plotted at a mean-centered value of $.20$), there was no effect of harsh parenting on telomere length ($t = -1.00, p = .32$).

3.3 Harsh Parenting and Telomere Attrition

Hierarchical regression analyses were performed to examine the main effect of harsh parenting on telomere change between ages 9 and 15. Two-way interactions were then entered into the analysis; interactions were computed between harsh parenting and the three moderators, optimism, connectedness, and neighborhood collective efficacy. The first step included each covariate (child sex, family income, parental marital status, race/ethnicity, pubertal status, and child age) and the mean-centered predictor to examine the main effect of cumulative harsh parenting. The second step included the same covariates, the main effect of harsh parenting, and the main effect of the moderator for each analysis (optimism, connectedness, and neighborhood

collective efficacy). The third step included the interaction between cumulative harsh parenting and each moderator. Results are shown in Tables 15-23. Main effects of cumulative harsh parenting were not predictive of telomere attrition ($\beta = -.03, p = .41$).

3.3.1 Optimism as a Moderator in the Full Sample

Results of harsh parenting predicting telomere attrition with optimism as a moderator are displayed in Table 15. Results showed that main effect of optimism was not predictive of telomere attrition ($\beta = -.01, p = .64$). The interaction between optimism and cumulative harsh parenting was not significantly associated with telomere attrition ($\beta = -.03, p = .29$).

3.3.2 Connectedness as a Moderator in the Full Sample

Results of harsh parenting predicting telomere attrition with optimism as a moderator are displayed in Table 18. There was no main effect of connectedness on telomere attrition ($\beta = -.02, p = .41$). The interaction between social connectedness and cumulative harsh parenting were also not significantly associated with telomere attrition ($\beta = .02, p = .46$).

3.3.3 Neighborhood Collective Efficacy as a Moderator in the Full Sample

Results of harsh parenting predicting telomere attrition with optimism as a moderator are displayed in Table 21. The main effect of neighborhood collective efficacy was not significant ($\beta = -.03, p = .41$). The interaction between cumulative harsh parenting and neighborhood collective efficacy was not significantly associated with telomere attrition ($\beta = -.02, p = .58$).

3.3.4 Cumulative Harsh Parenting Predicting Telomere Attrition Stratified by Child Sex

The hierarchical regression models for predicting telomere attrition stratified by child sex are shown in Tables 16, 19, and 22. Results showed that when separated by sex, the effects of cumulative harsh parenting on telomere attrition were not significant for boys or girls ($\beta = .02, p = .62$; $\beta = -.07, p = .12$, respectively). There was also no significant moderation for girls for each

two-way interaction (optimism, connectedness, and neighborhood collective efficacy). There was no moderation by optimism ($\beta = -.02, p = .49$) nor connectedness as a moderator ($\beta = .02, p = .62$) for boys. However, neighborhood collective efficacy significantly moderated the association between cumulative harsh parenting and telomere attrition for male youth ($\beta = -.08, p = <.05$). The Johnson-Neyman plot reveals that the effect of harsh parenting on telomere attrition is significant at lower, but not higher levels of collective efficacy. A simple slopes analysis revealed that for male youth with low levels of neighborhood collective efficacy (plotted at a mean-centered value of -1.00), the effect of harsh parenting on telomere attrition was significant ($t = 2.19, p < .05$) such that high harsh parenting was associated with higher telomere attrition. For youth with higher levels of neighborhood collective efficacy (plotted at the mean), there was no effect of harsh parenting on telomere attrition ($t = .52, p = .60$).

3.3.5 Cumulative Harsh Parenting Predicting Telomere Attrition Stratified by Race/Ethnicity

The regression analyses predicting telomere change from ages 9 to 15 were stratified by race/ethnicity to examine whether there were unique effects for Black, Hispanic, and White Youth.

3.3.5.1 Cumulative Harsh Parenting Predicting Telomere Attrition for White Youth

The hierarchical regression models predicting telomere attrition for White youth are shown in Tables 17, 20, and 23. Results showed that there were no main effects of cumulative harsh parenting on telomere attrition for White youth ($\beta = .03., p = .69$). There was also no significant moderation for White youth for each two-way interaction (optimism, connectedness, or neighborhood collective efficacy).

3.3.5.2 Cumulative Harsh Parenting Predicting Telomere Attrition for Black Youth

The hierarchical regression models predicting telomere attrition for Black youth are shown in Tables 17, 20, and 23. Results showed that there were no main effects of cumulative harsh parenting on telomere attrition for Black youth ($\beta = -.02, p = .21$). There was also no significant moderation by connectedness ($\beta = .02, p = .80$), neighborhood collective ($\beta = .03, p = .55$), nor optimism ($\beta = -.08, p = .09$) for Black youth (See Table 17).

3.3.5.3 Cumulative Harsh Parenting Predicting Telomere Attrition for Hispanic Youth

The hierarchical regression models predicting telomere attrition for Hispanic youth are shown in Tables 17, 20, and 23. Results showed that there were no main effects of cumulative harsh parenting on telomere attrition for Hispanic youth ($\beta = -.08, p = .15$). There was also no significant moderation by optimism ($\beta = .05, p = .32$) nor neighborhood collective efficacy ($\beta = .05, p = .30$) for Hispanic youth. Lastly, the effects of cumulative harsh parenting on telomere attrition were significantly moderated by connectedness for Hispanic youth ($\beta = .08, p = .10$) (See Table 20). The Johnson-Neyman plot revealed that the effects of harsh parenting on telomere attrition are significant at moderate levels of connectedness. A simple slopes analysis revealed that for Hispanic youth with low levels of connectedness (plotted at a mean-centered value of -1.50), the effect of harsh parenting on telomere attrition was significant ($t = -2.02, p < .05$) such that high harsh parenting was associated with less telomere attrition. For youth with higher levels of connectedness (plotted at the mean), there was no effect of harsh parenting on telomere attrition ($t = -1.34, p = .18$).

CHAPTER 4

DISCUSSION

The current study aimed to examine the impact of cumulative harsh parenting throughout childhood on telomere length at age 15 and telomere attrition from ages 9 to 15, as well as test the moderating role of optimism, connectedness, and neighborhood collective efficacy in these associations. Findings in this study were both consistent and inconsistent with prior research. For example, I found no evidence of direct effects of harsh parenting predicting telomere length during adolescence or attrition which is inconsistent with prior research (Tyrka et al., 2010). Our findings suggest that optimism, connectedness, and neighborhood collective efficacy may be uniquely protective for minority youth, which is consistent with prior research (Sosnowski, Musci, & Johnson, 2021) (Yonas et al., 2010). These findings suggest that minority youth may benefit uniquely from protective personality traits and strong social ties in interpersonal and community relationships.

Expanding on these findings, the current study's results showed that cumulative harsh parenting measured across ages 3, 5, and 9 years did not have independent effects on telomere length at age 15 when controlling for income at baseline, marital status, ethnicity, sex, pubertal status, and chronological age. These findings were contradictory to our first hypothesis. Although past research suggests harsh parenting may be negatively associated with telomere length (Tyrka et al., 2010), our prospective research throughout childhood does not support the effects of harsh parenting on telomere length. There are several differences between the present study and past research. First, Tyrka and colleagues (2010) included adults in their sample,

which is an important distinction to consider when doing research on biological aging relative to adolescent samples. Utilizing an adult sample may differ from utilizing an adolescent sample as there are little known demographic patterns for adolescents and telomere length over time (Gaydosh et al., 2021). A second consideration is retrospective versus prospective research with harsh parenting and telomere length. Retrospective reports of harsh parenting may be masked by the emotional perception of being parented harshly in the past (Brewin, Andrews, & Gotlib, 1993) and could lead to differences in how individuals report their parents' behavior toward them compared to concurrent reports of parents' behavior. Lastly, harsh parenting and maltreatment are not the same. Child maltreatment refers to abusive and neglectful parenting toward a child (Binggeli, Hart, & Brassard, 2001). Harsh parenting refers to a general lack of warmth and positive engagement between the parent and the child (Repetti, Taylor, & Seeman, 2002). Careful consideration of is needed when comparing overlapping yet distinct forms of child adversity.

Additionally, the sample utilized in this study contained participants who, on average, had telomeres that lengthened over time (Gaydosh et al., 2021). The findings in this sample challenge the typical assumption that telomeres only shorten as individuals age. On average, most longitudinal research conducted has found that telomere length shortens across adulthood (Bendix et al., 2014). However, there has been research that found telomere lengthening in a significant portion of their sample (Berghlund et al., 2016). Thus, the findings highlight a need for further investigation of longitudinal measurement of biological aging in the form of telomere attrition and lengthening as well as the environmental contexts that lead to each.

There also may be unique processes occurring during the pubertal transition that could mask associations with telomere length. Little is known about the developmental patterns of

telomere length through the life course and in particular during puberty (Gaydos et al., 2021). In this same sample, Koss and colleagues (2020) found that early pubertal development was associated with shorter telomeres for adolescent girls at age 9. Gaydos and colleagues (2021) find that a large portion of this sample demonstrates telomere lengthening; taken together these studies highlight the potential for complex associations during puberty. Estrogen, which is a gonadal hormone that increases during puberty, has been associated with increased telomerase activity (Bayne et al., 2008). This evidence may support unique demographic patterns during the pubertal development transition that may mask associations with the environment.

When the sample as a whole was considered, there were neither promotive or protective factors; however, moderation effects varied by racial and ethnic groups and child sex. Thus, context-specific investigation may be required in future work to ascertain which youth specifically may benefit from unique protective factors. There were not significant findings on the effect of harsh parenting on telomere length or telomere attrition for White youth. There were neither main effects (promotive) or interaction effects (protective). White youth may already be protected inherently by privilege that allows them easier access to resources that promote health. In fact, Mitchell and colleagues (2014) in the same sample of youth found that social determinants of health were indicative of telomere length for Black youth in this same sample, but not for White youth, which is consistent with the findings.

The effect of harsh parenting on telomere length at age 15 was moderated by optimism and neighborhood collective efficacy for Black youth. Results showed that telomere length was the shortest among Black youth that reported low levels of optimism and experienced high levels of harsh parenting. This is consistent with literature that suggests youth may have shorter telomeres when experiencing harsh parenting (Colich et al., 2020). Findings were consistent with

a buffering hypothesis. Black youth in particular, benefit from being more optimistic as they may experience unique struggles as an ethnic minority that may lead to the experience of chronically challenging and discriminatory environments (Gallow & Matthews). As such, Black youth may benefit more than their non-minority peers by having a more optimistic view on life. Oreskovic and Goodman (2011) found that specifically for Black youth, optimism offset the effects of cardiometabolic risk on telomere length in adolescents. These findings offer insight toward the buffering effect of optimism for Black youth when examining the effects of harsh parenting on telomere length.

Findings in the current study also support the moderating role of neighborhood collective efficacy for Black youth. This may suggest that high neighborhood collective efficacy plays a protective role in the loss of genomic material in the form of telomere length for Black youth. This is consistent with findings that neighborhood collective efficacy is especially protective for minority youth already at risk (Prince et al., 2019). Researchers have found that Black youth reside in neighborhoods characterized with higher rates of violence and mistrust at higher rates than their White peers (Sampson, Raudenbush, & Earls, 1998). As such, it may be especially important for Black youth to reside in environments that are high in cohesion, trust, and safety. This can provide benefit that extends over and above the risk of experiencing harsh parenting (Simons et al., 2005).

There was a significant interaction effect between cumulative harsh parenting and connectedness on telomere length at age 15 for Hispanic youth. Results showed that connectedness served as a protective buffer such that there was no effect of harsh parenting on telomere length at age 15 for Hispanic youth with high levels of connectedness. This is consistent with previous research demonstrating specific protective and promotive effects for

familism and familial connection for Hispanic youth (Perez and Cruess, 2014). Familism is best described as a strong attachment and identification between individual family members in Latinx families, as well as strong feelings and expectations of loyalty, dependability, and respect (Triandis et al., 1982). Among youth with low levels of connectedness, the direction of the association among harsh parenting and TL was counterintuitive to our hypothesis. This may be due to the nature of parenting in different ethnic contexts. Latinx families have greater prevalence rates of authoritarian parenting than do European American families (Steinberg, Dornbusch, & Brown, 1992). Authoritarian parenting can be characterized as parenting with lower levels of warmth and higher levels of control (Baumrind, 1991). Cultural expectations such as loyalty, dependability, and high reliance on one another exist in Latinx cultures toward authoritarian parenting that may be characterized as normative and less harsh than in White American families (Mogro-Wilson & Cifuentes, 2021). As such, Hispanic youth may benefit more from environments with higher levels of control that may be consistent with items in the self-report assessment of harsh parenting in this study. This could further explain why harsh parenting may not function the same way, or have the same effect, in Hispanic families than in White families. However, future research is needed to replicate this counterintuitive finding especially as authoritarian parenting may still be associated with poorer mental health in Hispanic youth. The larger body of research is not necessarily straightforward, with some findings that Hispanic youth fare better with more authoritarian parenting (Chen, 2022), and others finding that Hispanic youth fare worse when they experience authoritarian parenting (Calzada et al., 2017).

Connectedness also served as moderator in predicting telomere attrition among Hispanic youth. Telomere attrition was highest for those Hispanic youth who were low in connectedness

and low in harsh parenting. Research supports these findings suggesting that connectedness for Hispanic youth is a large reason for positive health outcomes when comparing Hispanic youth to other racial/ethnic youth (Perez & Cruess, 2014), and lack of connection could be a risk factor. Telomere attrition was lower for youth who experienced high levels of connectedness. Previous research supports this, as connectedness was found to be promotive for youth (Jose, Ryan, & Pryor, 2012), and protective for individuals already at-risk (Sosnowski et al., 2019). The findings for telomere attrition mirror the counterintuitive patterns found with telomere length in Hispanic youth. As stated earlier, harsh parenting may be operating differently in Hispanic families and for Hispanic youth (Steinberg, Dornbusch, & Brown, 1992), which could be contributing to the findings for harsh parenting being insignificant for Hispanic youth.

Neighborhood collective efficacy moderated the effect of harsh parenting on telomere attrition in adolescent boys. Loss of genomic material in the form of telomere attrition was highest for male youth that were low in neighborhood collective efficacy and high in harsh parenting. This is consistent with previous research that found low neighborhood safety as a risk factor for telomere length (Thierry, 2020). Telomeres shortened more for male participants high in collective efficacy when they experienced harsh parenting, suggesting that harsh parenting may have unique detrimental effects over and above the protective nature of living in a safe and cultivating neighborhood environment. This could be explained by research that found harsh parenting to be associated with negative outcomes for youth (Repetti, Taylor & Seeman, 2002). Higher neighborhood collective efficacy is associated with lower levels of delinquency, mistrust, and violence in youth, which may be especially protective for the biological aging of adolescent boys who are at higher risk for these behaviors. Neighborhood collective efficacy may be

especially beneficial for adolescent boys, as boys are typically known to have higher rates of delinquency and impulsive traits than adolescent girls (Kofler, Zhang, & Gao, 2022).

The present study is not without limitations including concerns regarding the measurements in the current study. Cumulative harsh parenting was reported by primary caregivers. As such, caregivers may be inclined to report lower levels of negative parenting due to social desirability. Furthermore, cumulative harsh parenting was measured longitudinally, which was a strength of the study. However, the measurement of harsh parenting was only assessed at ages 3, 5, and 9 years. A more complete measurement of harsh parenting throughout childhood could have strengthened this study. Specifically, measuring harsh parenting more frequently or prior to age 3. With 2 and 4 years between data collection time points, there may be harsh parenting experiences that are missed between points of measurement. Although findings in this study supported significant correlations for harsh parenting between different time points, segments of youth's childhoods between measurement could be missed, as the measure asked for retrospective harsh parenting "in the last year." Lastly, connectedness measured the extent to which youth had meaningful connections with others. This may have been too broad of a measure and future research should examine unique effects of connection with specific social relationships including connections with peers and within the family.

This study contains notable strengths. As stated earlier, harsh parenting was measured cumulatively, and incorporated data from three time points throughout different developmental stages of childhood. This captures a more complete picture of the parenting experience that children in this sample endured. In respect to the sample, Future Families and Child Wellbeing Study includes a diverse sample, which is beneficial for the generalizability of findings. This study also included multiple moderators, each at different proximity to the focal child. This is

important for ascertaining unique effects of micro vs macro level sources of influence, as protective and promotive factors may reside in different ecological levels (Bronfenbrenner & Morris, 1998).

4.1 Conclusion

The results in the current study highlight the importance of analyzing biological aging markers both at single time points as well as longitudinally. The interaction effects in this study were unique for telomere length measured at age 15 and telomere attrition measured longitudinally from age 9 to 15. There may be processes that occur during the pubertal transition that can be further elucidated with comparative research utilizing both as outcomes. In this specific sample, on average telomeres lengthened between ages 9 and ages 15 (Gaydosh et al., 2021). This may contribute to lack of simple, direct effects. Nonetheless, since aging is a longitudinal process, researchers should examine change of telomere length as well as telomere length at a single time point to ascertain processes that contribute to both.

The findings from the current study expand on existing literature on the effects of risk and protective factors contributing to telomere length. The study utilized data from a larger sample than current research available. Overall, findings were unique to minority youth highlighting protective factors may vary across ethnic and racial groups. Implications of these findings provide evidence for a need for safer, more cohesive neighborhood environments for Black and male youth, as well as a need for social connection in the form of connectedness for Hispanic youth. The current study elucidates the effects of harsh parenting on both telomere length in adolescence and telomere attrition, but specifically highlights the protective factors that exist in Hispanic, Black, and male youth.

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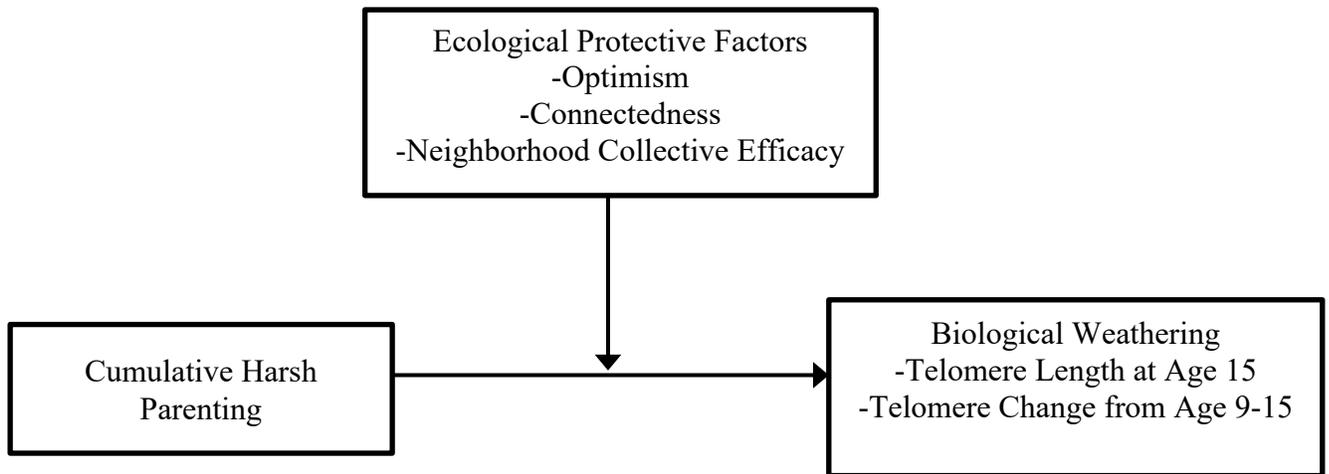
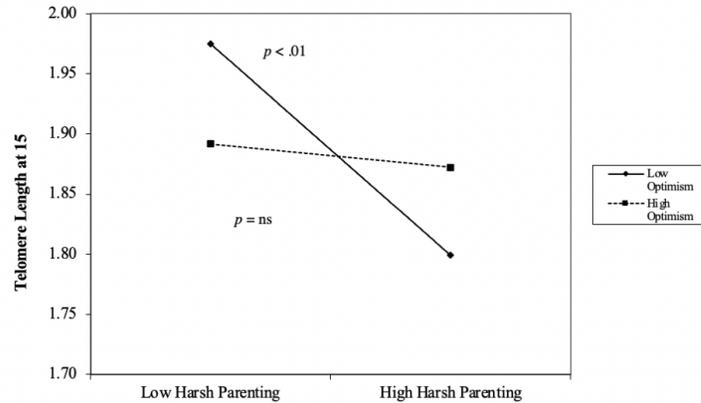


Figure 1. Proposed Conceptual Model.

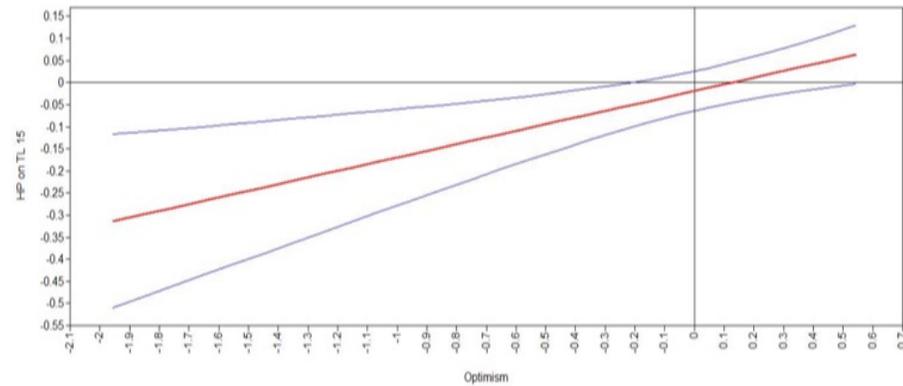
Figure 2

The Effect of Harsh Parenting on Telomere Length Moderated by Optimism among Black Youth

A



B

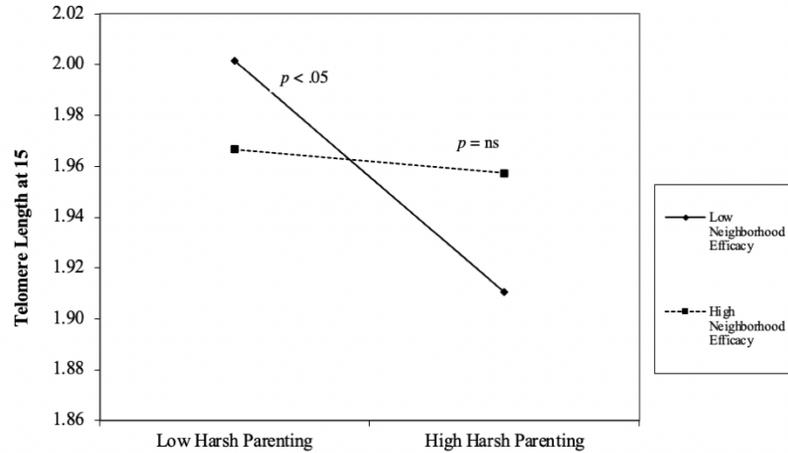


Note. Figure depicts the significant moderation of the harsh parenting-telomere length association by optimism among Black Youth. Panel 2A shows the simple slope plot for the association between harsh parenting (-/+ 1 SD for low and high values) and telomere length at age 15 at low and high levels of optimism (mean-centered value of -1.00, mean-centered value of 0.00, respectively). Panel 2B contains the Johnson-Neyman plot depicting the association between harsh parenting on telomere length at varying levels of optimism. The region of significance ranges from mean-centered values of -0.20 through the lower end of observed values.

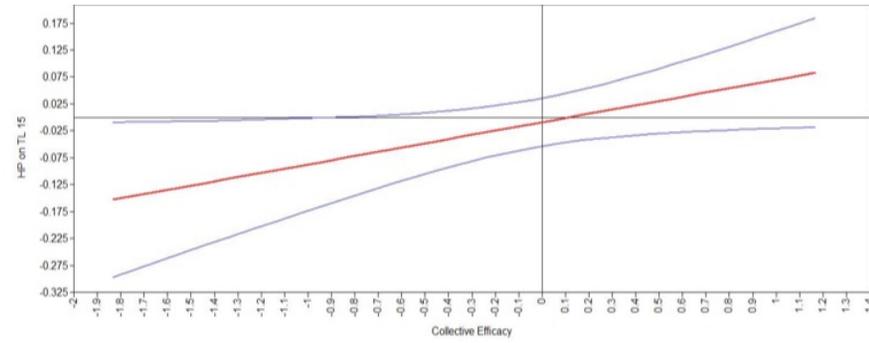
Figure 3

The Effect of Harsh Parenting on Telomere Length Moderated by Collective Efficacy among Black Youth

A



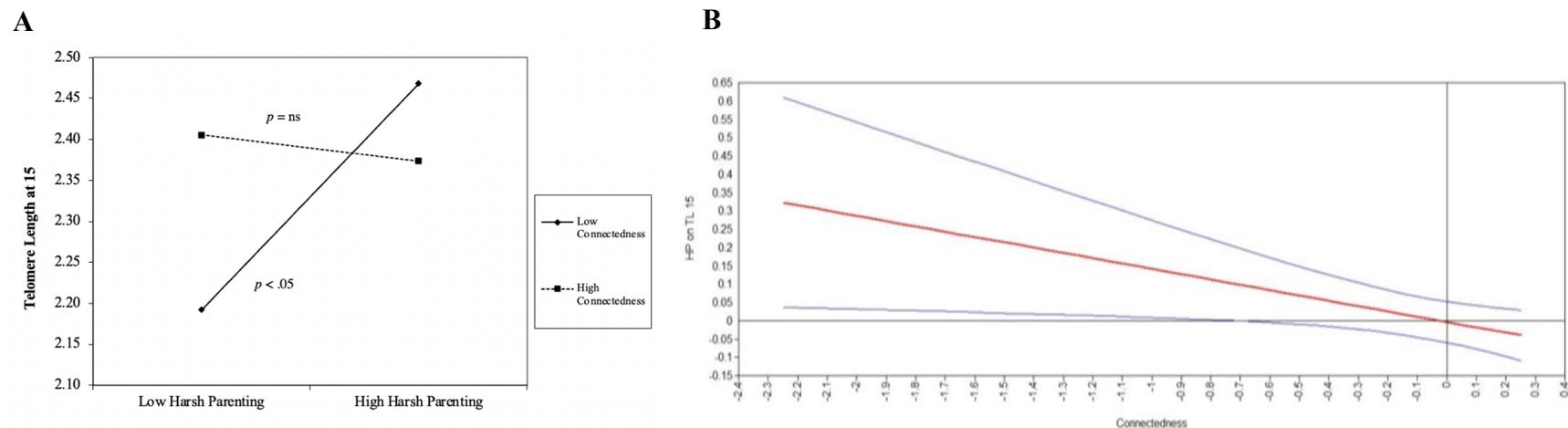
B



Note. Figure depicts the significant moderation of the harsh parenting-telomere length association by collective efficacy among Black Youth. Panel 3A shows the simple slope plot for the association between harsh parenting (-/+ 1 SD for low and high values) and telomere length at age 15 at low and high levels of collective efficacy (mean-centered value of -1.00, mean-centered value of 0.00, respectively). Panel 3B contains the Johnson-Neyman plot depicting the association between harsh parenting on telomere length at varying levels of collective efficacy. The region of significance ranges from mean-centered values of -0.90 through the lower end of observed values.

Figure 4

The Effect of Harsh Parenting on Telomere Length Moderated by Connectedness among Hispanic Youth

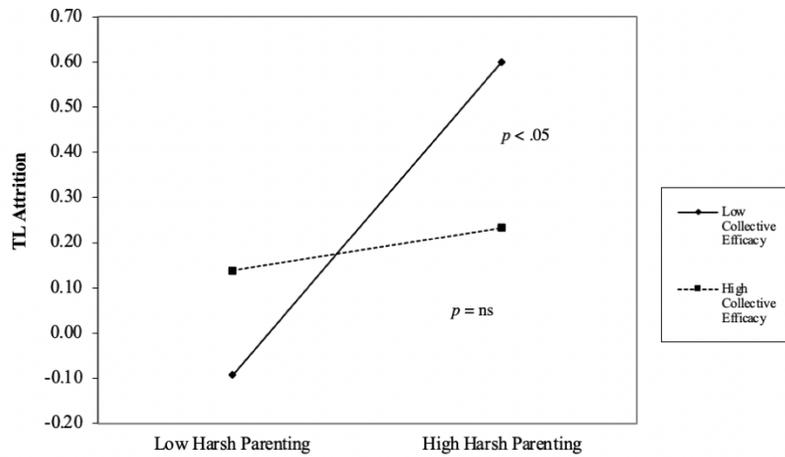


Note. Figure depicts the significant moderation of the harsh parenting–telomere length association by connectedness among Hispanic Youth. Panel 4A shows the simple slope plot for the association between harsh parenting (-/+ 1 SD for low and high values) and telomere length at age 15 at low and high levels of connectedness (mean-centered value of -2.00, mean-centered value of 0.20, respectively). Panel 4B contains the Johnson-Neyman plot depicting the association between harsh parenting on telomere length at varying levels of connectedness. The region of significance ranges from mean-centered values of -0.70 through the lower end of observed values.

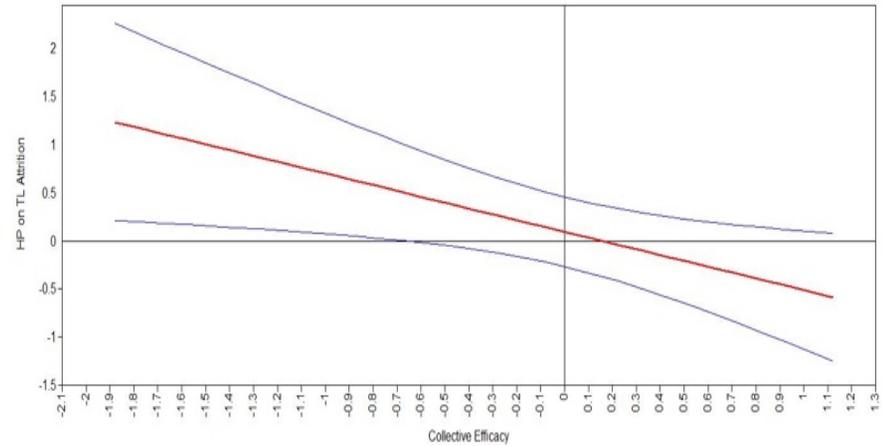
Figure 5

The Effect of Harsh Parenting on Telomere Attrition Moderated by Collective Efficacy among Male Youth

A



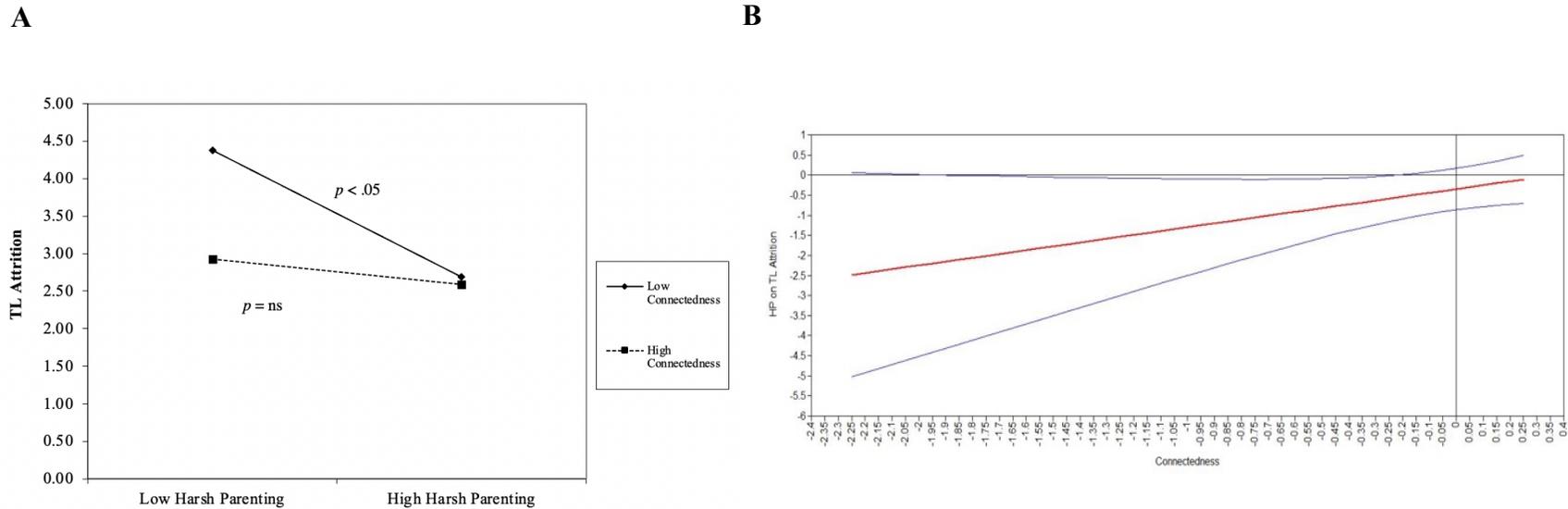
B



Note. Figure depicts the significant moderation of the harsh parenting-telomere length association by collective efficacy among Male Youth. Panel 5A shows the simple slope plot for the association between harsh parenting (-/+ 1 SD for low and high values) and telomere attrition at low and high levels of collective efficacy (mean-centered value of -1.00, mean-centered value of 0.00, respectively). Panel 5B contains the Johnson-Neyman plot depicting the association between harsh parenting on telomere attrition at varying levels of collective efficacy. The region of significance ranges from mean-centered values of -0.70 through the lower end of observed values.

Figure 6

The Effect of Harsh Parenting on Telomere Attrition Moderated by Connectedness among Hispanic Youth



Note. Figure depicts the significant moderation of the harsh parenting-telomere length association by connectedness among Hispanic Youth. Panel 6A shows the simple slope plot for the association between harsh parenting (-/+ 1 SD for low and high values) and telomere attrition at low and high levels of connectedness (mean-centered value of -1.50, mean-centered value of 0.00, respectively). Panel 6B contains the Johnson-Neyman plot depicting the association between harsh parenting on telomere attrition at varying levels of connectedness. The region of significance ranges from mean-centered values of -0.20 through the lower end of observed values (-1.95).

Table 1. Sample Demographics

	<i>M (SD) or N (%)</i>
Marital Status <i>N (%)</i>	
Married	1187 (24.23%)
Non-Married	3710 (75.75%)
Sex <i>N (%)</i>	
Female	2341 (47.80%)
Male	2556 (52.18%)
Youth Age at Wave 5 (Years) <i>M (SD)</i>	9.30 (.40)
Youth Age at Wave 6 (Years) <i>M (SD)</i>	15.63 (.71)
Family Income at Birth <i>M (SD)</i>	\$31994 (\$31567.20)
Race/Ethnicity <i>N (%)</i>	
White	1030 (21.08%)
Black	2326 (47.61%)
Hispanic	1336 (27.34%)
Other	194 (3.97%)
PCG <i>N (%)</i>	
Biological Mother	3828 (91.10%)
Biological Father	180 (4.28%)
Other <i>N (%)</i>	194 (4.61%)
Saliva Samples Collected <i>N (%)</i>	
Yes	2929 (86.79%)
No	446 (13.21%)

Table 2. Descriptive Statistics for Study Variables

Construct	<i>M (SD)</i>	Min-Max	<i>N</i>
TL Age 15 (ln)	2.08 (.27)	1.49, 2.85	1628
TL Attrition	-.18 (2.30)	-9.91, 9.43	1598
Harsh Parenting at Age 3	.96 (.63)	0.00, 3.53	3321
Harsh Parenting at Age 5	.96 (.61)	0.00, 3.33	2999
Harsh Parenting at Age 9	.76 (.62)	0.00, 3.53	3336
Cumulative Harsh Parenting	.87 (.54)	0.00, 3.53	4223
Optimism	3.41 (.50)	1.00, 4.00	3437
Connectedness	3.77 (.36)	1.00, 4.00	3437
Neighborhood Collective Efficacy	2.89 (.63)	1.00, 4.00	3416

Table 3. Analysis of Variance Results By Child Gender

Construct	Girls <i>M (SD)</i>	Boys <i>M (SD)</i>	<i>F</i>	<i>df</i>	<i>p</i>	<i>Pairwise Comparisons</i>
TL Age 15	2.09 (.26)	2.08 (.28)	0.89	1627	.35	n/a
TL Attrition	-.17 (2.31)	-.19 (2.28)	0.06	1597	.81	n/a
Harsh Parenting	.82 (.53)	.92 (.55)	35.73	4221	<.001***	Boys > Girls
NCE	2.87 (.63)	2.91 (.62)	3.76	3415	.05*	Boys > Girls
Optimism	3.39 (.50)	3.42 (.50)	3.13	3436	.07	n/a
Connectedness	3.78 (.36)	3.76 (.36)	2.18	3436	.14	n/a

Note. Significant pairwise comparisons are reported utilizing the Bonferonni posthoc comparison; *** $p < .001$; ** $p < .01$; * $p < .05$; NCE = Neighborhood Collective Efficacy.

Table 4. Analysis of Variance Results By Race and Ethnicity

Construct	White <i>M</i> (<i>SD</i>)	Black <i>M</i> (<i>SD</i>)	Hispanic <i>M</i> (<i>SD</i>)	<i>Other</i> <i>M</i> (<i>SD</i>)	<i>F</i>	<i>df</i>	<i>p</i>	<i>Pairwise Comparisons</i>
TL Age 15	2.08 (.26)	2.07 (.28)	2.09 (.27)	2.07 (.24)	.43	1534	.79	n/a
TL Attrition	-.22 (2.37)	-.14 (2.32)	-.08 (2.24)	-.33 (1.98)	.60	1506	.66	n/a
Harsh Parenting	.77 (.44)	1.01 (.55)	.76 (.50)	.77 (.54)	40.69	3164	<.001***	B>W; B>H; B>O
NCE	2.99 (.62)	2.87 (.64)	2.89 (.60)	2.90 (.64)	5.13	3245	<.001***	W>B
Optimism	3.29 (.53)	3.48 (.47)	3.38 (.50)	3.32 (.46)	21.09	3262	<.001***	B>W; H>W; B>H; B>O
Connectedness	3.79 (.35)	3.76 (.37)	3.78 (.35)	3.76 (.29)	1.10	3262	.36	n/a

Note. Significant pairwise comparisons are reported utilizing the Bonferonni posthoc comparison. W=White, B=Black, H=Hispanic, O=Other. *** $p < .001$; ** $p < .01$; * $p < .05$; NCE = Neighborhood Collective Efficacy.

Table 5. Correlation Table

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Youth Age at 15	1.00													
2. Marital Status	-.04*	1.00												
3. Income at Baseline	-.06**	.26**	1.00											
4. Pubertal Status (PDSS)	.04*	-.12**	-.02	1.00										
5. Child Sex	-.01	-.01	.00	.30**	1.00									
6. Harsh Parenting Age 3	.01	-.11**	-.03	.02	-.09**	1.00								
7. Harsh Parenting Age 5	.02	-.11**	-.04	.02	-.07**	.58**	1.00							
8. Harsh Parenting Age 9	.00	-.08**	-.03	.02	-.07**	.42**	.51**	1.00						
9. Cumulative Harsh Parenting	.00	-.11**	-.03	.03	-.09**	.85**	.87**	.83**	1.00					
10. Teen TL	.01	.00	-.03	-.02	.02	-.04	.04	-.01	.00	1.00				
11. TL Attrition	.03	.01	-.08**	.02	.01	.01	-.05	-.02	-.02	-.47**	1.00			
12. NCE	.01	.08**	.04*	-.05**	-.03	-.06**	-.05*	-.07**	-.07**	.00	-.03	1.00		
13. Optimism	.04*	-.01	-.01	.07**	-.03	-.02	-.05*	-.05**	-.05**	.01	-.01	.15**	1.00	
14. Connectedness	.00	.03	.04*	.02	.03	-.02	-.05*	-.06**	-.05**	.01	-.01	.20**	.41**	1.00

Note. * $p < .05$, ** $p < .01$, *** $p < .001$. Child Sex: 0=Male, 1=Female; Marital Status: 0=Unmarried, 1=Married; NCE = Neighborhood Collective Efficacy; *** $p < .001$; ** $p < .01$; * $p < .05$

Table 6. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Length at Age 15 with Optimism as Moderator

Construct	Full Sample											
	Model 1				Model 2				Model 3			
	β	B	SE	p	β	B	SE	p	β	B	SE	p
HP	.00	.00	.02	.88	-.01	.00	.02	.86	-.01	.00	.02	.86
Optimism					-.01	.00	.02	.77	-.01	-.01	.02	.69
HP X Optimism									.04	.05	.03	.15
R^2	.00				.00				.01			
N	1271				1270				1270			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, race/ethnicity, and child sex; HP=Harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 7. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Length at Age 15 with Optimism as a Moderator

Construct	Girls												Boys											
	Model 1				Model 2				Model 3				Model 1			Model 2			Model 3					
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
HP	-.03	-.02	.02	.49	-.03	-.01	.02	.53	-.03	-.02	.02	.45	.01	.01	.02	.72	.01	.01	.02	.80	.01	.01	.02	.74
Optimism					.03	.02	.02	.39	.03	.02	.02	.46					-.04	-.02	.02	.27	-.05	-.03	.02	.25
HP X Optimism									.05	.05	.04	.21									.04	.05	.05	.33
R^2	.01				.01				.02				.02			.02			.02					
<i>N</i>	625				625				625				646			645			645					

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and race/ethnicity. HP= Cumulative harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 9. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Length at Age 15 with Connectedness as a Moderator

Construct	Full Sample											
	Model 1				Model 2				Model 3			
	β	B	SE	p	β	B	SE	p	β	B	SE	p
HP	.00	.00	.02	.88	.00	.00	.02	.89	-.28	-.15	.11	.16
Connectedness					.01	.00	.02	.84	.00	.00	.02	.89
HP X Connectedness									.28	.04	.03	.16
R^2	.00				.00				.01			
N	1271				1270				1270			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, race/ethnicity, and child sex. HP= Cumulative harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 10. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Length at Age 15 with Connectedness as a Moderator

Construct	Girls												Boys											
	Model 1				Model 2				Model 3				Model 1				Model 2				Model 3			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
HP	-.03	-.02	.02	.49	-.03	-.01	.02	.51	-.03	-.01	.02	.53	.01	.01	.02	.72	.01	.01	.02	.74	.01	.00	.02	.87
Connectedness					.03	.02	.03	.38	.03	.02	.03	.37					-.02	-.02	.04	.65	-.02	-.01	.03	.67
HP X Connectedness									-.04	-.06	.06	.31									-.06	-.08	.06	.17
<i>R</i> ²	.01				.01				.01				.02				.02				.02			
<i>N</i>	625				625				625				646				645				645			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and race/ethnicity. HP= Cumulative harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 12. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Length at Age 15 with Neighborhood Collective Efficacy as a Moderator

Construct	Full Sample											
	Model 1				Model 2				Model 3			
	β	B	SE	p	β	B	SE	p	β	B	SE	p
HP	.00	.00	.02	.88	.00	.00	.02	.92	.00	.00	.02	.98
NCE					.00	.00	.01	.99	.00	.00	.01	.93
HP X NCE									.04	.03	.03	.20
R^2	.00				.00				.01			
N	1271				1268				1268			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, race/ethnicity, and child sex. HP= Cumulative harsh parenting; NCE = Neighborhood collective efficacy. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 13. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Length at Age 15 with Neighborhood Collective Efficacy as a Moderator

Construct	Girls												Boys											
	Model 1				Model 2				Model 3				Model 1				Model 2				Model 3			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
HP	-.03	-.02	.02	.49	-.03	-.02	.02	.49	-.02	-.01	.02	.61	.01	.01	.02	.72	.02	.01	.02	.67	.02	.01	.02	.67
NCE					-.02	-.01	.02	.66	-.01	-.01	.02	.73					.01	.00	.02	.82	.01	.00	.02	.81
HP X NCE									.06	.05	.04	.14									.02	.02	.04	.58
R^2	.01				.01				.02				.02				.02				.02			
<i>N</i>	625				625				625				646				643				643			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and race/ethnicity. HP= Cumulative harsh parenting; NCE = Neighborhood collective efficacy. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 14. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Length at Age 15 with Neighborhood Collective Efficacy as a Moderator

Construct	White												Black												Hispanic												
	Model 1				Model 2				Model 3				Model 1				Model 2				Model 3				Model 1				Model 2				Model 3				
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	
HP	.04	.02	.04	.55	.04	.02	.04	.51	.03	.02	.04	.57	-.02	-.01	.02	.56	-.02	-.01	.02	.61	-.02	-.01	.02	.69	.01	.00	.03	.92	.01	.00	.03	.92	.01	.00	.03	.91	
NCE					-.05	-.02	.02	.36	-.06	-.02	.02	.31					.01	.00	.02	.86	.01	.01	.02	.74					.01	.01	.02	.83	.01	.01	.03	.82	
HP X NCE																																					
<i>R</i> ²	.01				.01				.01				.01				.01				.01				.02				.02				.02				
<i>N</i>	483				280				280				549				548				548				341				340				340				

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and child sex. HP= Cumulative harsh parenting; NCE = Neighborhood collective efficacy. *** $p < .001$; ** $p < .01$; * $p < .05$; Numbers in bold were significant at $p < .05$.

Table 15. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Attrition with Optimism as a Moderator

Construct	Full Sample											
	Model 1				Model 2				Model 3			
	β	B	SE	p	β	B	SE	p	β	B	SE	p
HP	-.03	-.11	.14	.41	.00	.04	.14	.88	-.03	-.12	.14	.39
Optimism					-.01	-.06	.13	.64	-.01	-.05	.13	.69
HP X Optimism									-.03	-.27	.25	.29
R^2	.01				.01				.01			
N	1247				1246				1246			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, race/ethnicity, and child sex. HP= Cumulative harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 16. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Attrition with Optimism as a Moderator

Construct	Girls												Boys											
	Model 1				Model 2				Model 3				Model 1				Model 2				Model 3			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
HP	-.07	-.32	.20	.12	-.07	-.33	.20	.10	-.07	-.31	.20	.12	.02	.09	.19	.62	.02	.01	.19	.61	.02	.09	.19	.65
Optimism					-.04	-.17	.19	.37	-.03	-.16	.19	.42					.01	.04	.18	.80	.01	.05	.18	.79
HP X Optimism									-.03	-.31	.41	.45									-.02	-.22	.31	.49
R^2	.03				.03				.03				.00				.00				.00			
<i>N</i>	612				612				612				635				634				634			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and race/ethnicity. HP= Cumulative harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 17. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Attrition with Optimism as a Moderator

Construct	White												Black												Hispanic											
	Model 1				Model 2				Model 3				Model 1				Model 2				Model 3				Model 1				Model 2				Model 3			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
HP	.03	.14	.36	.69	.03	.15	.37	.68	.03	.15	.38	.68	-.02	-.01	.19	.71	-.02	-.09	.19	.65	-.02	-.07	.19	.74	-.08	-.39	.27	.15	-.09	-.40	.27	.13	-.08	-.39	.27	.15
Optimism					.03	.11	.25	.66	.03	.12	.27	.67					-.04	-.21	.23	.36	-.04	-.21	.23	.36					-.03	-.15	.22	.48	-.04	-.18	.22	.40
HP X Optimism																																				
<i>R</i> ²	.04				.04				.04				.02				.02				.02				.03				.03				.04			
<i>N</i>	275				275				275				539				539				539				333				332				332			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and child sex. HP= Cumulative harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 18. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Attrition with Connectedness as a Moderator

Construct	Full Sample											
	Model 1				Model 2				Model 3			
	β	B	SE	p	β	B	SE	p	β	B	SE	p
HP	-.03	-.11	.14	.41	0	0	0	.95	-.03	-.11	.14	.41
Connectedness					-.02	-.15	.18	.41	-.02	-.15	.18	.40
HP X Connectedness									.02	.29	.40	.46
R^2	.01				.01				.01			
N	1247				1246				1246			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, race/ethnicity, and child sex. HP= Cumulative harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 19. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Attrition with Connectedness as a Moderator

Construct	Girls												Boys											
	Model 1				Model 2				Model 3				Model 1				Model 2				Model 3			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
HP	-.07	-.32	.20	.12	-.07	-.32	.20	.12	-.07	-.33	.20	.10	.02	.09	.19	.62	.02	.08	.19	.66	.02	.10	.19	.61
Connectedness					-.01	-.08	.27	.78	-.02	-.09	.27	.73					-	-.21	.24	.39	-.03	-.21	.24	.38
HP X Connectedness									.04	.57	.68	.41									.02	.23	.47	.62
<i>R</i> ²	.03				.03				.03				.00				.00				.01			
N	612				612				612				635				634				634			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and race/ethnicity. HP= Cumulative harsh parenting. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 21. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Attrition with Neighborhood Collective Efficacy as a Moderator

Construct	Full Sample											
	Model 1				Model 2				Model 3			
	β	B	SE	p	β	B	SE	p	β	B	SE	p
HP	-.03	-.11	.14	.41	.01	.08	.10	.80	-.03	-.12	.14	.39
NCE					-.03	-.13	.10	.21	-.04	-.13	.10	.20
HP X NCE									-.02	-.13	.23	.58
R^2	.01				.01				.01			
N	1247				1244				1244			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, race/ethnicity, and child sex. HP= Cumulative harsh parenting; NCE = Neighborhood collective efficacy. *** $p < .001$; ** $p < .01$; * $p < .05$

Table 22. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Attrition with Neighborhood Collective Efficacy as a Moderator

Construct	Girls												Boys											
	Model 1				Model 2				Model 3				Model 1				Model 2				Model 3			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
HP	-.07	-.32	.20	.12	-.07	-.32	.2	.11	-.07	-.30	.20	.13	.02	.09	.19	.62	.02	.10	.19	.61	.02	.10	.18	.60
NCE					-.05	-.18	.15	.24	-.05	-.17	.15	.27					-.02	-.06	.13	.65	-.02	-.07	.13	.60
HP X NCE									.03	.26	.38	.48									-.08	-.61	.26	.02
<i>R</i> ²	.03				.03				.03				.00				.00				.01			
<i>N</i>	612				612				612				635				632				632			

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and race/ethnicity. HP= Cumulative harsh parenting; NCE = Neighborhood collective efficacy. *** *p*<.001; ** *p* < .01; * *p* < .05; Numbers in bold were significant at *p* < .05.

Table 23. Summary of Hierarchical Regression Analysis for the Effect of Cumulative Harsh Parenting on Telomere Attrition with Neighborhood Collective Efficacy as a Moderator

Construct	White												Black												Hispanic																							
	Model 1				Model 2				Model 3				Model 1				Model 2				Model 3				Model 1				Model 2				Model 3															
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>								
HP	.03	.14	.36	.69	.03	.15	.37	.69	.02	.14	.37	.70	-.02	-.07	.19	.71	-.02	-.07	.19	.72	-.02	-.07	.19	.69	-.08	-.39	.27	.15	-.08	-.38	.26	.15	-.08	-.39	.27	.15	-.08	-.39	.27	.15								
NCE					-.01	-.05	.19	.81	-.01	-.05	.19	.80					-.02	-.09	.16	.58	-.03	-.09	.16	.56					-.08	-.31	.20	.13	-.08	-.31	.20	.12	-.08	-.31	.20	.12	-.08	-.31	.20	.12				
HP X NCE									-.01	-.09	.56	.88									-.03	-.19	.32	.55									.05	.42	.41	.30	.05	.42	.41	.30	.05	.42	.41	.30	.05	.42	.41	.30
<i>R</i> ²	.04				.04				.04				.02				.02				.02				.03				.04				.04															
<i>N</i>	275				275				275				539				538				538				333				332				332															

Note: Control variables were included in analyses, but omitted in final tables for brevity. Control variables were youth age at 15, marital status, income at baseline, pubertal development status, and child sex. HP= Cumulative harsh parenting; NCE = Neighborhood collective efficacy. *** *p*<.001; ** *p* < .01; * *p* < .05