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Adverse Childhood Experiences Indirectly Affect Child Telomere Length through Self-
Regulation

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of
Philosophy at Virginia Commonwealth University.

by

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Abstract

ADVERSE CHILDHOOD EXPERIENCES INDIRECTLY AFFECT CHILD TELOMERE LENGTH THROUGH SELF-REGULATION

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A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University.

Virginia Commonwealth University, 2019

Major Director: Wendy Kliewer, Ph.D.
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The goals of present study were: (a) to examine associations between adverse childhood experiences (ACEs) and telomere length during childhood using ACE composite scores both with and without “new” adversities (i.e., parental death and poverty), and (b) to determine if ACEs indirectly affect telomere length through children’s self-regulatory abilities (i.e., effortful control and self-control). The analytic sample consisted of national data from teachers, biological parents, and their children ($N = 2,527$; $M_{age} = 9.35$, $SD = .36$ years; 52% male; 45% Black). Results from linear regression analyses revealed a statistically significant main effect of updated (but not traditional) ACEs on child telomere length, controlling for hypothesized covariates, although the additional amount of variance explained by ACEs was negligible. Results from mediation analyses revealed an indirect effect of ACEs on child telomere length through self-control, assessed via a teacher-reported Social Skills Rating System, but not effortful control. While longitudinal studies are needed to strengthen claims of causation, the present study clarifies the association between ACEs and telomere length during middle childhood, and identifies a pathway from ACEs to changes in telomere length that should be explored further.

Adverse Childhood Experiences Indirectly Affect Child Telomere Length through Self-Regulation

The most recent report from the National Survey of Children's Health revealed that nearly half (46%) of all children in the United States experience at least one adverse childhood experience (ACE) prior to age 17 (Bethell, Davis, Gombojav, Stumbo, & Powers, 2017¹). This amounts to approximately 35 million youth who experience some form of adversity (e.g., economic hardship, maltreatment) that places them at risk for numerous short- and long-term physical and psychological health problems. Negative outcomes that occur at an increased rate among individuals exposed to childhood adversity include (but are not limited to): obesity, cardiovascular disease, sleep-related problems, depression, and anxiety (for a review, see Kalmakis & Chandler, 2015). Children exposed to ACEs also exhibit less visible health effects, such as increased inflammation and dysregulated cortisol levels (e.g., Miller, Chen, & Parker, 2011). Given the consistent, robust link between ACEs and health, understanding the biology underlying this association is critical, as it informs our knowledge of disease progression and consequently how to intervene to improve health.

Prevalence and Characteristics of Adverse Childhood Experiences

Traditional studies of childhood adversity examined the unique association between individual stressors and various developmental outcomes throughout the lifespan (e.g., childhood sexual abuse; Irish, Kobayashi, & Delahanty, 2010). While individual adversities are important to assess, a major limitation of this approach is that many children are exposed to multiple adversities that have both short- and long-term implications for development (e.g., McLaughlin & Sheridan, 2016). Recent prevalence rates from the National Survey of Children's Health revealed that 21.7% of all children in the United States – roughly 16 million individuals – report

¹While exposure to multiple adversities early in life is common across the globe, the simultaneous assessment of multiple ACEs is less common outside of the United States, and current ACE measures fail to adequately assess culturally relevant stressors (e.g., Quinn et al., 2018).

experiencing at least two ACEs prior to age 17 (Bethell et al., 2017). These experiences include events such as parental death or divorce, witnessing interpersonal violence in the home, and living with someone who is dealing with alcohol or drug problems. Moreover, exposure to childhood adversity varies as a function of age, race/ethnicity, and socioeconomic status (SES). For example, 12.1% of children age 0-5 report experiencing at least two ACEs, compared to 22.6% of children age 6-11, and 29.9% of children age 12-17. Approximately 19% of White children report experiencing at least two ACEs prior to age 17, compared to 33.8% of Black children and 21.9% of Hispanic children. Lastly, 34.7% of children below the poverty line report at least two ACEs before age 17, compared to 17.2% and 9.2% of children at or above the poverty line, respectively (Bethell et al., 2017). These prevalence rates emphasize the dynamic nature of childhood adversity, and the demographic and social factors that affect exposure to adverse experiences.

While prevalence rates are informative, it is important to determine which experiences should be included in a composite measure of childhood adversity. This is because the selection of events informs how researchers think about the developmental outcomes associated with adversity and mechanisms through which those outcomes occur. In their pioneering work linking childhood adversity to adult health, Felitti and colleagues (1998) developed the Adverse Childhood Experiences (ACE) Study. In their study, the authors queried adults about childhood adversities across two domains: childhood abuse and household dysfunction. Participants responded to multiple questions in each of the following areas: physical, sexual, and psychological abuse, parental substance use and mental illness, domestic violence, and other criminal behavior by a parent (i.e., incarceration). Latter data collection periods also included measures of physical and emotional neglect by a parent. Results from the study revealed that

25% of the sample (~ 1,000 individuals) reported experiencing at least two ACEs prior to age 18. Furthermore, individuals who had experienced four or more ACEs had a four- to 12-fold increase in risk for alcoholism, drug abuse, and depression, as well as a two- to four-fold increase in poor self-rated health compared to individuals reporting no ACEs. This study provided the first concise measure of childhood adversity that demonstrated efficacy in illuminating the association between early adversity and later health. While innovative, the selection of events included in this measure often is ignored, potentially excluding meaningful adversities. Since the publication of the original groundbreaking article, scholars have identified additional childhood adversities that are linked to health throughout the lifespan and may enhance the traditional ACEs questionnaire, namely parental death and poverty.

The primary motivation for including parental death and poverty in a score of childhood adversity is their consistent, robust associations with health and development. For example, recent evidence from a nation-wide sample revealed that paternal death was associated with significant decreases in telomere length – a biological marker of aging – among children, and that this effect was larger compared to paternal loss due to incarceration and separation/divorce (Mitchell et al., 2017). Moreover, a review by Miller, Chen, and Parker (2011) highlighted the consistent, positive association between poverty during childhood and various diseases of aging (e.g., cardiovascular disease) decades later in life. Given the association between these two adversities and development, as well as their frequent occurrence in current assessments of ACEs (e.g., Bethell et al., 2017), it is important to understand how they extend the traditional ACE index. Thus, the first goal of the current study was to examine the association between ACEs and telomere length during childhood using indexes with and without these two adversities in order to clarify their role in a composite score of ACEs.

Adverse Childhood Experiences and Health Outcomes

Since the publication of the original ACE Study, several investigations using this data revealed links between ACEs and negative outcomes prior to adulthood (e.g., teen pregnancy, adolescent alcohol use; Dube et al., 2006; Hillis et al., 2004). For example, Dube and colleagues found that – aside from physical neglect – each individual ACE was associated with an increased likelihood of using alcohol during adolescence. Moreover, these participants were more likely to initiate alcohol use prior to age 14. Another study by Hillis and colleagues (2004) found that teen pregnancy occurred in 16% of women (~960 individuals) exposed to at least one ACE, and that this rate increased as the number of ACEs increased. In regard to adult health outcomes, a recent meta-analysis by Hughes and colleagues (2017) found that ACEs were associated with a wide range of health outcomes; individuals exposed to at least four ACEs were at increased risk for negative health outcomes compared to those who did not experience any adversity. Specifically, participants with at least four ACEs were most at risk for substance use problems, sexual risk taking, and self-inflicted violence (i.e., odd ratios were greater than three). These results highlight both the short- and long-term health consequences of ACEs, and areas of focus for intervention efforts.

While many studies using the original ACE data focus on adult outcomes, separate studies of cumulative risk, which often use most of the original ACEs, also reveal robust links between childhood adversities and a plethora of childhood outcomes such as poor academic achievement, internalizing symptoms, externalizing problems, drug use, and risky sexual behavior (for a review, see Evans, Li, & Whipple, 2013). As an example, Larson and colleagues (2008) tested the association between various social risk factors and global (i.e., physical, socioemotional) health among children from birth to 17 years. Independent associations revealed

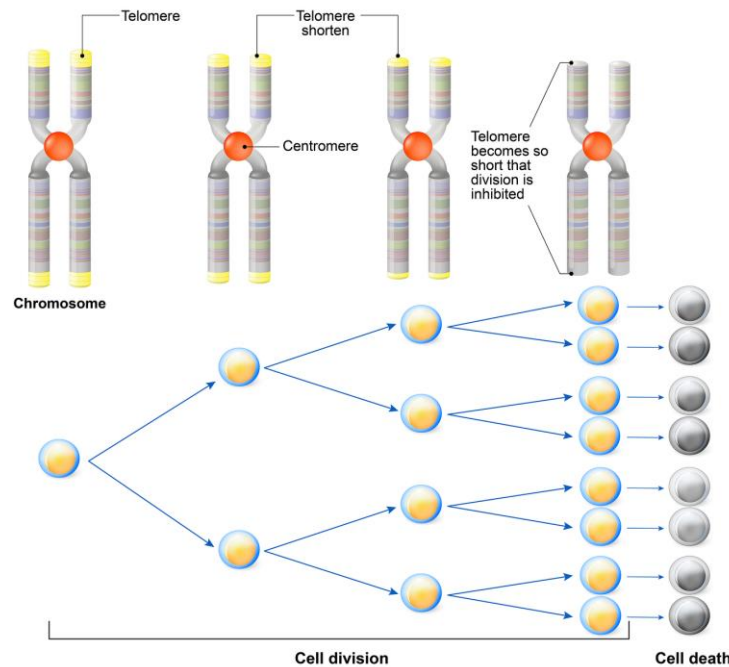
that minority status (i.e., Black or Hispanic race/ethnicity), low family income, low household education, and unsafe neighborhoods were associated with an increased likelihood of poorer ratings of overall child health. Similarly, residing in a single-parent household, poor maternal mental health and family conflict were associated with an increased likelihood of poorer ratings of the child's socioemotional health. Similar associations were noted when these variables were combined, with a particularly strong association for individuals of minority status and those with low income and education levels. These results highlight the global impact of childhood adversity on child health, and the unique risk placed upon children of minority status, those living in single-parent households, and households with conflict and/or a parent with mental health issues.

Telomeres as a Biological Indicator of Adversity and Health

While the link between ACEs and health is robust, the biological pathway(s) underlying this association often are not explicitly tested. Over the past decade, however, various models have been proposed to explain how childhood adversity “gets under the skin” to impact health outcomes throughout the lifespan (e.g., Miller et al., 2011; Repetti, Taylor, & Seeman, 2002). One biological factor that has received much attention is telomeres. Telomeres are protein-bound DNA structures located at the ends of chromosomes (in humans, they are comprised of multiple repeats of the sequence: TTAGGG; Blackburn, 2005; see Figure 1). Their primary function is to prevent the ends of chromosomes from being recognized as a DNA break(s), thereby allowing for stabilization of the chromosomes (Blackburn, Greider, & Szostak, 2006). However, during each somatic cell division, telomeres shorten by 30-200 base pairs because DNA polymerase is unable to fully replicate the 3' end of the DNA strand (Starkweather et al., 2014). This phenomenon is referred to as the “end replication problem” and leads to a decline in telomere

length over time. Telomeres are therefore viewed as a biological marker of aging.

Figure 1. *Schematic Representation of Telomere Structure and Attrition Process*



Note. Image credit: Nanalyze.

Telomere length has been proposed as an intermediary biological marker since it is linked to both adversity and health outcomes throughout the lifespan. For example, Hanssen, Schutte, Malouff, and Epel (2017) conducted a meta-analysis of 27 studies and over 16,000 participants, finding a small but significant association (i.e., $r = -.08$) between childhood psychosocial stressors (e.g., maltreatment, family violence) and telomere length. Effect sizes tended to be larger when studies used categorical indicators of stress (as opposed to levels of a stressor), when the time between the stressor(s) and telomere measurements was shorter, and when quantitative polymerase chain reaction (qPCR) was used to assay telomere data (compared to the Southern blot method); however, these differences did not remain statistically significant after Bonferroni correction to adjust for alpha inflation. The authors found no statistically significant differences

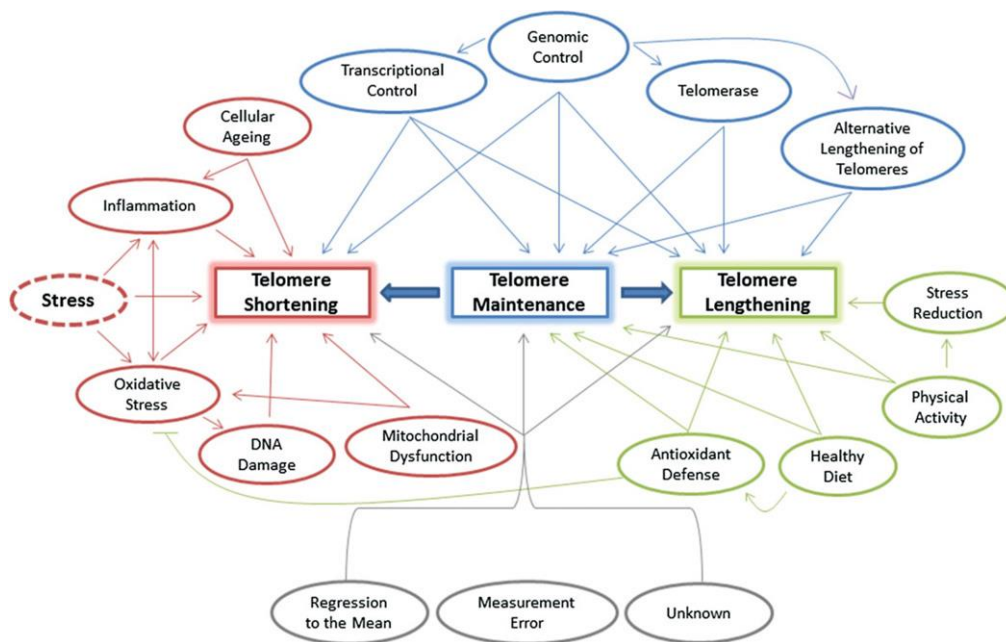
across studies on other key variables such as age, sex, and use of retrospective assessments of adversity. Another qualitative review by Oliveira and colleagues (2016) revealed similar results, finding that chronic stressors (e.g., poverty) were consistently, inversely associated with telomere length throughout adolescence and into adulthood.

In regard to physical health outcomes, telomere length has been linked to a variety of health problems such as cancer (e.g., Ma et al., 2011), hypertension (Yang et al., 2009), and all-cause mortality (e.g., Cawthon, Smith, O'Brien, Sivatchenko, & Kerber, 2003). A meta-analysis by Haycock and colleagues (2014) revealed that declines in telomere length were both prospectively and retrospectively associated with an increased risk of cardiovascular disease later in life. Specifically, when comparing the shortest and longest third of telomere length, the relative risk for coronary heart disease was 1.54 across all studies, 1.40 in prospective studies, and 1.80 in retrospective studies. These empirical studies and meta-analyses illuminate the independent associations between childhood adversity and telomere length, and telomere length and health; moreover, they illustrate a potential biological pathway from adversity to poor health. To date, the majority of the literature has relied on testing independent links between ACEs, telomere length, and health, while few examine these associations simultaneously or consider mechanisms underlying these associations (for exceptions, see Shalev, 2012; Shalev et al., 2013).

Mechanisms of telomere attrition. While evidence linking ACEs and telomere length exists, few developmental researchers have tested mechanisms of action that underlie telomere attrition. Factors currently known to directly affect telomere length include genetic regulation, epigenetic modification, and transcriptional control (Shalev, 2012). A review by Shalev (2012) explored potential mechanisms through which stress influences the rate of telomere attrition in humans. Stress affects telomere dynamics in several different ways, but two molecular processes

that have received much attention, to date, are inflammation and oxidative stress (see Figure 2). Briefly, when an individual appraises a situation as stressful and the relevant systems (e.g., ANS, HPA axis) are activated, inflammation occurs via the release of immune cells. In addition, levels of reactive oxygen species (ROS) increase. Both of these processes are beneficial in the short-term, but chronic activation is detrimental and associated with decreases in telomere length over time (e.g., Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Epel et al., 2004; von Zglinicki, 2002). To date, no studies have examined the simultaneous associations between ACEs, inflammation and/or oxidative stress, and telomere length in children². This is, in part, due to the lack of longitudinal data on children, and the difficulty in prospectively measuring inflammation and oxidative stress (e.g., high cost to collect these data).

Figure 2. *Schematic Representation of Factors Affecting Telomere Length*



Note. Image credit: Shalev (2012)

An alternative to directly examining molecular processes of telomere attrition is to test behavioral or cognitive factors that indicate stress reactivity and subsequent physiological and biological functioning. The primary benefit of this approach is that it is non-invasive and

²Epel and colleagues (2004) found that, among a sample of healthy, premenopausal women aged 20-50 years old, higher stress among a group of female caregivers was associated with greater levels of oxidative stress and shortened telomere length.

therefore allows researchers to collect data from a wider range of participants at a lower cost. In addition, the identification of a behavioral or cognitive factor that indicates biological changes allows clinicians and interventionists to intervene and improve health rather than overlooking biological consequences that are not immediately visible but have a noticeable effect on health and development. One such construct is self-regulation, which is closely tied to both environmental and biological factors (for a review, see Bridgett et al., 2015).

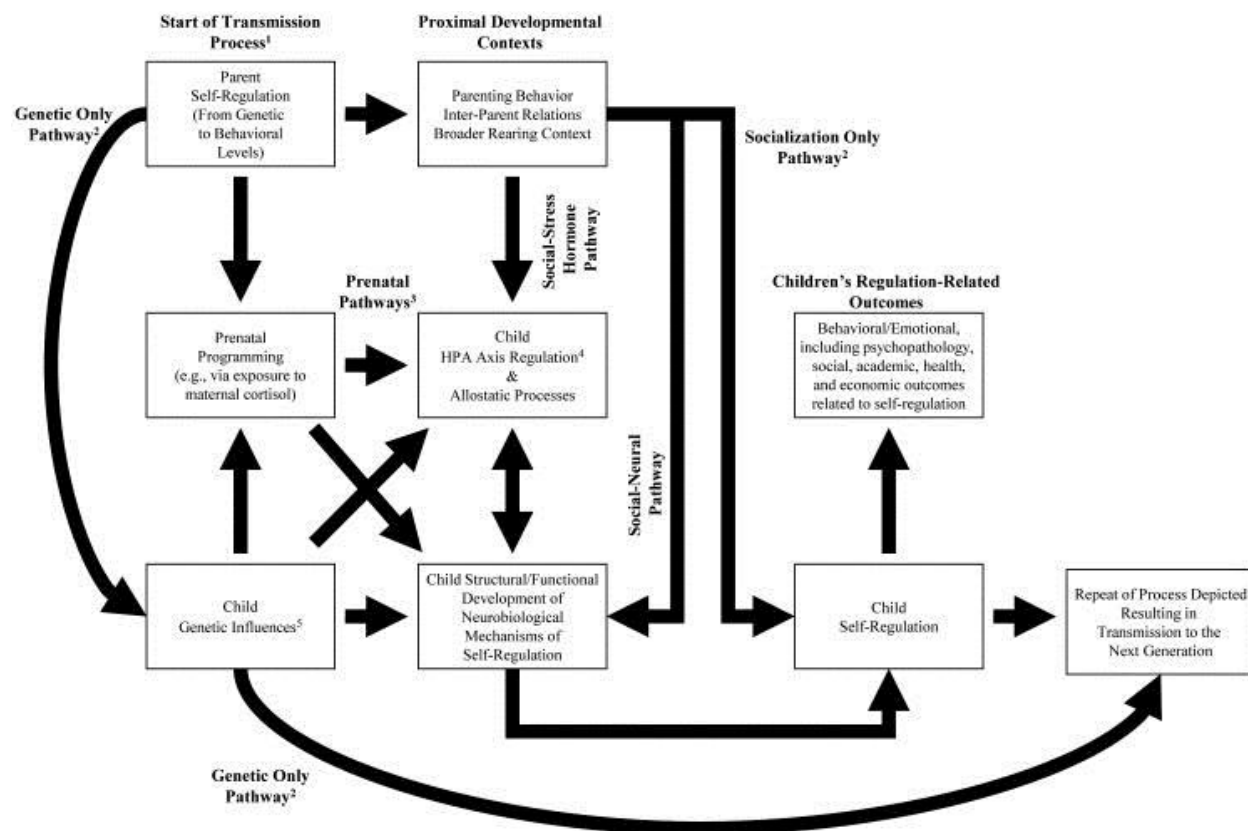
Self-Regulation and Child Development

It is well known that the ability to self-regulate is necessary for healthy development (for a review, see Murray, Rosanbalm, Christopoulos, & Hamoudi, 2015). There is a plethora of empirical evidence demonstrating the robust effect of self-regulation on a range of outcomes including overall physical health (e.g., Hampson et al., 2016), mental health (e.g., depression; Lengua, 2003), and social and behavioral issues (e.g., substance use; deBlois & Kubzansky, 2016). Furthermore, these effects remain after adjusting for key sociodemographic factors such as IQ and SES (Raver, Carter, McCoy, Roy, Ursache, & Friedman, 2012). Given the robust association between self-regulation and development, it is necessary to understand the etiology and structure of self-regulation to clarify how childhood adversity affects self-regulation and subsequent biological factors (i.e., telomere length).

As noted by Bridgett and colleagues (2015), the etiology of self-regulation is multifaceted, resulting from the complex interplay between genetic factors, prenatal programming (e.g., exposure to maternal cortisol), and proximal developmental contexts (e.g., parent-child relations). While this model focuses on the intergenerational transmission of self-regulation (see Figure 3), it provides a basis for understanding how self-regulation develops, and how parent-related stressors (e.g., substance use) can still be conceptualized as ACEs, having a

direct (or indirect) impact on children's self-regulatory abilities.

Figure 3. *Conceptual Model of the Intergenerational Transmission of Self-Regulation*



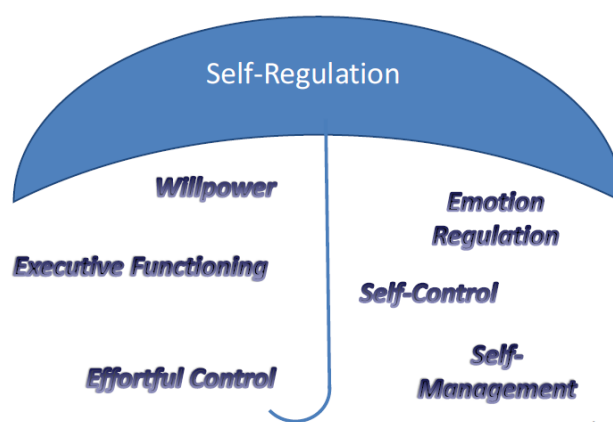
Note. Image credit: Bridgett et al. (2015).

Self-regulation can be defined as, “the act of managing cognition and emotion to enable goal-directed actions such as organizing behavior, controlling impulses, and solving problems constructively” (Murray et al., 2015, p. 5). This definition takes an applied perspective on self-regulation, which allows researchers to operationalize self-regulation in a way that is more readily applicable to interventionists. Indeed, empirical evidence suggests that self-regulation is malleable, making it a valuable target for intervention (e.g., Blair & Diamond, 2008; Piquero, Jennings, & Farrington, 2010). There is much debate, however, regarding what components are included in the construct of “self regulation” and should thus serve as targets for intervention (for

a comprehensive review of these constructs, see Nigg, 2017).

Traditionally, self-regulation is conceptualized as consisting of bottom-up and top-down processes (Bridgett et al., 2015; Nigg, 2017). Bottom-up processes consist of automatic, stimulus-driven responses like reflexes, whereas top-down processes consist of slower, more deliberate processes like working memory. While the literature to date focuses on top-down processes (e.g., self-control, emotion regulation), bottom-up processes often are targets of intervention (e.g., associative learning). For the purpose of the current study, terms associated with an applied definition of self-regulation were used for clarity and consistency. Figure 4 presents a graphical representation of terms commonly associated with self-regulation within an applied framework.

Figure 4. *Self-Regulation Terms*



Note. Image credit: Murray et al. (2015)

As can be seen in Figure 4, there are both top-down and bottom-up components that contribute to self-regulation, and these constructs cover a wide range of behaviors from impulse control to more complex behaviors requiring adaptation to situational demands. It is important to note that all terms under the umbrella coincide with an applied definition in that they contribute to goal-directed behaviors that can be targeted for intervention. The current study assessed self-

regulation via measures of effortful control and self-control during middle childhood. Effortful control often is equated with cognitive control (Nigg, 2017), which can be defined as, “a set of superordinate functions that encode and maintain a representation of the current task...marshaling to that task subordinate functions including working, semantic, and episodic memory, perceptual attention and action selection and inhibition” (Botvinick & Braver, 2015, p. 85). Self-control can be defined as the ability to avoid impulsive actions, and controlling one’s emotions in the service of controlling behavior (Diamond, 2013). Both of these terms have been used extensively in the executive function and broader self-regulation literature (e.g., Nigg, 2017), and reflect key components of self-regulation during middle childhood.

During middle childhood, self-regulation is characterized by the use of various cognitive strategies (e.g., internal speech) to control behavior, generate more precise appraisal of social situations, and handle emotions “on the fly,” which sets the stage for problem-solving skills (Murray et al., 2015). However, self-regulation often is overlooked during middle childhood since this period of development is seen as a period of latency (e.g., Raffaelli, Crockett, & Shen, 2010). Murray and colleagues (2015) point out that the development of self-regulation does reach a momentary plateau during ages 6-10 years; however, there is empirical evidence that self-regulation is malleable during this period (e.g., Raver, McCoy, & Lowenstein, 2013). The current study assessed self-regulation during middle childhood because the self-regulatory skills established during this developmental period are vital for healthy development. That is, fostering a child’s ability to control behavior and stay on task, manage emotions on their own, and navigate stressful situations has clear implications for the development of healthy coping strategies and overall responses to stress during adolescence and beyond.

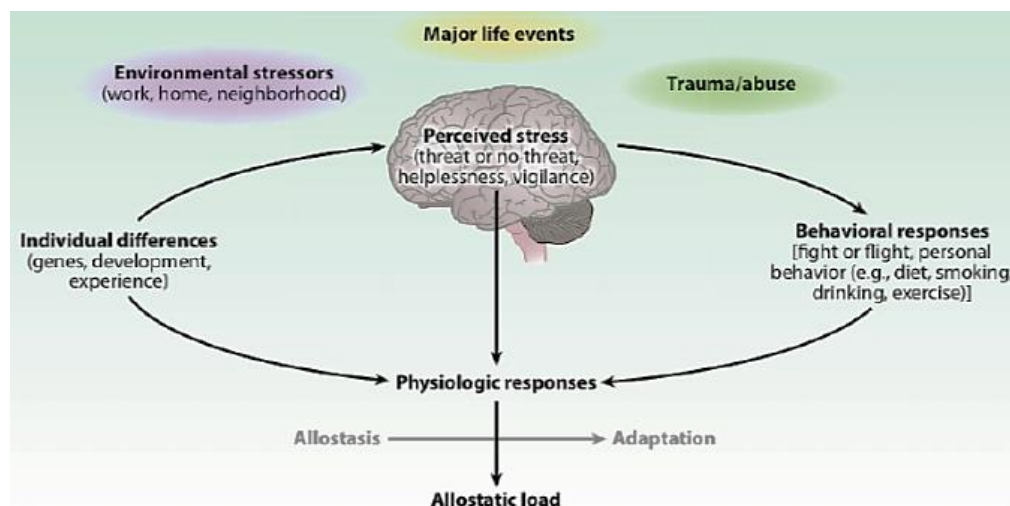
Childhood Adversity, Self-Regulation, and Telomeres

Understanding the role of self-regulation in the context of childhood adversity and telomere length is crucial because this knowledge can inform intervention efforts for children exposed to adversity that go beyond the standard approach of preventing exposure. To date, no studies have examined the association between self-regulation, childhood adversity, and telomere length simultaneously. Furthermore, investigators typically conceptualize self-regulation as a moderator in the association between adversity and developmental outcomes (e.g., Lengua & Sandler, 1996), but theoretical and empirical evidence point to a potential mediating role of self-regulation in the context of adversity and biological functioning (e.g., Lupien, McEwen, Gunnar, & Heim, 2009). The second goal of this study was to test whether self-regulation serves as a mediator through which ACEs indirectly affect telomere length during middle childhood.

Self-regulation as a mediator. Based on models of stress and disease (e.g., McEwen & Stellar, 1993) and the development of self-regulation (e.g., Bridgett et al., 2015), it is plausible to hypothesize that self-regulation serves a mediating role between childhood adversity and telomere length. The concept of allostatic load provides one framework for understanding the intermediary role of self-regulation. According to Sterling and Eyer (1988) the human body and its systems (e.g., immune, metabolic, HPA axis) have an operating range the body fluctuates within, and these systems can adjust to a new steady state when presented with a challenge; this is referred to as allostasis. This concept remains pivotal to understanding how the body can adapt to acute stressors; however, it neglects the long-term wear-and-tear that the body undergoes with prolonged exposure to stress (e.g., maltreatment, poverty). Building upon this work, McEwen and Stellar (1993) postulated that chronic stress results in long-term adjustments of these allostatic systems, which leads to wear and tear on the body, and ultimately, disease. Termed

allostatic load, this concept is crucial to understanding how one's biology and behavior are impacted by chronic stress.

Figure 5. *Conceptual Model of Allostatic Load*



Note. Image credit: medium.com

Allostatic load results from behavioral and biological responses to stress that are dependent on individual differences such as genetic predispositions, social context and status, gender, and developmental history (see Figure 5). When an individual is exposed to a stimulus, these factors influence how an individual processes the event and ultimately (does or does not) experiences stress. If the stimulus induces stress, various allostatic systems are activated and chronic activation of these systems leads to marked changes in these systems, and more importantly, the brain (McEwen & Stellar, 1993). For example, the hippocampus – a region in the brain's limbic system linked to ANS activity, memory, and emotion – is involved in the stress response, primarily serving as an inhibitor to shut off the HPA axis stress response (Jacobson & Sapolsky, 1991). A plethora of evidence over the last two decades demonstrates that chronic stress has a negative impact on the hippocampus, in part through excess secretion of glucocorticoids in response to stress (Lupien, Juster, Raymond, & Marin, 2018; Sapolsky, Krey,

& McEwen, 1986). Subsequent research finds that these effects on the hippocampus are associated with significant decreases in cognitive and affective regulation (for a review, see Lupien et al., 2009). Thus, if an individual is exposed to numerous stressors during childhood it is likely that their ability to self-regulate is inhibited via dysregulation of the HPA axis.

While the concept of allostatic load is useful for thinking about stressors directly experienced by the child, it is less useful for conceptualizing the effects of indirect stressors such as parent substance use. That is, the child does not experience substance use problems directly, but the effects of parental use (e.g., low-quality caregiving) have an impact on the development of self-regulation for the child. For example, using Bridgett and colleagues' (2015) framework, various behaviors by parents can directly impact the development of children's self-regulatory skills. In their review, the authors point out that inter-parental relations are consistently associated with children's self-regulation, including effortful control (e.g., Gustafsson, Cox, & Blair, 2012). These results provide ancillary evidence for ACEs both directly and indirectly experienced by the child influencing the development and functioning of self-regulation. Since middle childhood is a developmental period when self-regulatory skills begin to flourish, stunting of these processes may inhibit an individual's ability to properly self-regulate, and ultimately contribute to negative biological outcomes (e.g., decreased telomere length).

Statement of the Problem

While it is known that ACEs are associated with telomere length throughout the lifespan, and that telomere length is a marker for later health outcomes, there is a need to understand (a) how the addition of other ACEs (i.e., parental death, poverty) impact this association and (b) behavioral and cognitive factors that affect telomere length in children. Self-regulation is one factor associated with adversity and developmental outcomes; however, its association with

telomere length remains unknown. While self-regulation often is conceptualized as a protective factor, it is plausible that self-regulation operates as a mediator of the association between adversity and telomere length. Since self-regulation is a strong target for intervention (Murray et al., 2015) and ACEs have become a focal topic for researchers and clinicians over the past two decades, the present study sought to advance our current understanding on the biological consequences associated with ACEs and inform future prevention and intervention efforts.

Present Study

The present study was conducted using data from the nine-year follow-up wave of the Fragile Families and Child Wellbeing Study (for an overview of the study sample and design, see Reichman, Teitler, Garfinkel, & McLanahan, 2001). The study follows a cohort of approximately 4,700 children and their primary caregiver(s), many of whom (~3,600) were unwed at the time of birth. The overarching goals of the study were to better understand the conditions and capabilities of new unwed parents, the nature of their relationship, what factors bring unwed parents together, and how social policies (e.g., welfare reform, child support) impact these families. A wealth of data were collected that also address childhood experiences, adjustment across various domains of functioning, and biological indicators of health. The current study used data on (parent-reported) childhood adversity, (child- and teacher-reported) self-regulation, and telomere length to better understand the association between ACEs and telomere length, and pathways through which telomere attrition occurs. This study contributes to the extant literature on adversity and telomere length in several meaningful ways. First, it uses a nation-wide sample of children to assess the cumulative influence of childhood adversity on telomere length during middle childhood, a period often overlooked by researchers. Second, it is

the first study to test a behavioral/cognitive factor through which adversity indirectly influences telomere length in children. Third, it integrates additional ACEs into the original ACE index in order to advance the current understanding of ACE measures. In summary, this study advances our understanding of how ACEs and self-regulation simultaneously influence telomere length and provides valuable information for researchers, clinicians, and interventionists hoping to improve childhood health in the face of adversity.

Aims and Hypotheses

Based on the empirical literature and theories linking ACEs, self-regulation, and telomere length, the present study had two aims:

Aim 1

Examine if childhood adversity, measured via a composite of adverse experiences³, is associated with telomere length at age 9, adjusting for hypothesized covariates.

Hypothesis 1. Child adversity will be inversely associated with global telomere length (i.e., telomere length across all chromosomes), adjusting for hypothesized covariates.⁴ Thus, youth with higher numbers of ACEs will evidence shorter telomeres, on average, once covariates are considered.

Aim 2

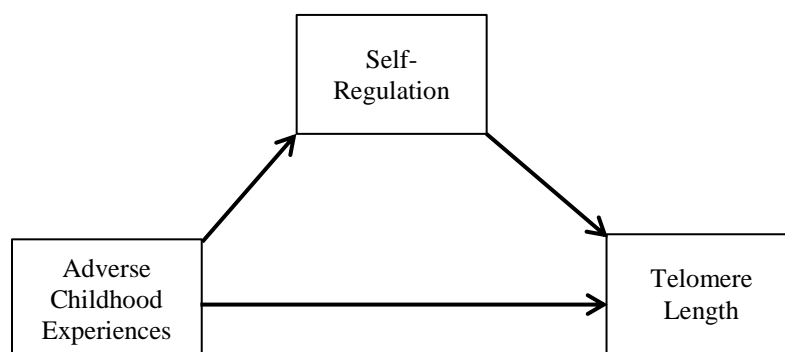
Examine if ACEs indirectly affect child telomere length through self-regulation⁵, measured via effortful control and self-control (see Figure 6).

³This association will be tested using both the “traditional” ACE index, and an updated ACE index including parental death and poverty.

⁴Covariates included: biological mother telomere length, child race, and child body mass index. Child gender was added to the mediation models assessing self-regulation (see Aim 2).

⁵While effortful control and self-control are hypothesized to be subsumed within the higher order construct of self-regulation, theoretical and empirical evidence suggests that they are distinct constructs. Therefore, all analyses pertaining to self-regulation will consist of separate models for effortful control and self-control.

Figure 6. Hypothesized Mediation Model for Study Aim 2



Hypothesis 1. Adverse childhood experiences will be indirectly associated with telomere length through changes in self-regulation, after accounting for hypothesized covariates. Thus, it is expected that youth with higher ACE levels will evidence more difficulties with self-regulation, which in turn will be associated with shorter telomere length.

Method

Participant Ascertainment and Overall Study Design

The present study used a subsample of children, their biological parent(s), and teachers from the nine-year follow-up wave of the Fragile Families and Child Wellbeing Study. Data collection for this wave was conducted from August 2007 through April 2010. Approximately 77% ($n = 3,630$) of primary caregivers, 76% ($n = 3,515$) of biological mothers, and 59% ($n = 2,652$) of biological fathers who were eligible for interviews completed interviews during this wave. Seventy-two percent ($n = 3,391$) of all eligible participants participated in the home visit. Data collection consisted of three components. First, biological parent surveys were completed using computer-assisted telephone interviewing. Second, home visits were scheduled and children completed a 20-minute interview using Computer-Assisted Personal Interview (CAPI) technology while the biological parent(s) completed a self-administered questionnaire. Saliva samples also were collected from biological mothers and the child participant (i.e., focal child)

during the home visit. Third, consent and contact information was obtained from teachers, and hard-copy interviews were mailed to the child's teachers. The analytic sample for the study was selected based on 2,527 children, ranging in age from 8-10 years ($M_{\text{age}} = 9.35$, $SD = .36$ years; 52% male) who had valid data for telomere length at the nine-year follow-up wave. The sample was ethnically diverse, with approximately 45% of children being categorized as Black ($n = 1,144$), 23% as Hispanic ($n = 569$), 16% as White ($n = 402$), and 10% as bi-racial ($n = 259$).⁶ Six percent of children ($n = 153$) were not able to be categorized into a racial category based on the available data.⁷ Fifteen percent of mothers had a college degree, while 41% had some college experience; 21% of mothers had a high school degree or equivalent training (e.g., GED) and 23% had less than a high school education. Similarly, 17% of fathers had a college degree, 35% had some college experience, 29% had a high school degree or equivalent training, and 19% had less than a high school education. Median household income was \$30,000 and \$40,000 for mothers and fathers, respectively. Data used for the current study consisted of biological parent reports of childhood adversities (e.g., poverty, maltreatment), child reports of self-regulation (i.e., effortful control), teacher reports of self-regulation (i.e., self-control), and child and biological maternal telomere length.

Adverse Childhood Experiences Measures

The ACEs assessed in the current study were selected based on those experiences included in the original ACE Study (cf, Felitti et al., 1998) and other adversities known to affect development (i.e., poverty, parental death). Table 1 provides an overview of the ACEs included in the present study. The original ACE Study separated childhood adversity into two domains: childhood abuse and household dysfunction. Most of the original ACEs could be assessed using the Fragile Families and Child Wellbeing Study data, except sexual abuse and suicide attempt by

⁶A portion of bi-racial couples ($n = 150$) can be classified as "majority-minority" couples (i.e., one White parent and one Black or Hispanic parent).

⁷A portion of parents ($n = 166$) were categorized as an 'other' race. Since the races included in this category are unknown, these individuals were recoded as missing.

a family member living in the household. Data were available for neglect, but the reliability for the measure was low ($\alpha = .55$), so these data were not used in analyses. Lastly, the current study differs from the original ACE Study in that all ACEs presented here are parent-reported (as opposed to child-report). For a comparison between the two measures, Appendix A provides the questions included in the original ACE Study.

Table 1. *Adverse Childhood Experiences Included in the Present Study*

Construct	Item(s)	Response Options	Original ACE?
Household Dysfunction			
Substance use			
Alcohol use	“In the past 12 months, was there ever a time when your drinking or being hung over interfered with your work at school, or a job, or at home?”	0 = No 1 = Yes	Yes
Drug use	“In the past 12 months, was there ever a time when your use of drugs interfered with your work at school, or a job, or at home?”	0 = No 1 = Yes	Yes
Mental illness			
Depression	Past-year diagnosis of a Major Depressive Episode?	0 = No 1 = Yes	Yes
Parental Loss*			
Incarceration	Mother/Father ever incarcerated?		Yes
Separation/Divorce	Mother/Father separated/divorced?	0 = No 1 = Yes	Yes
Death	Mother/Father deceased?		No
Domestic Violence*	“Father hurt you in front of child.”	0 = No 1 = Yes	Yes
Childhood Abuse			
Psychological [†]	“Called him/her dumb or lazy or some other name like that.”	0 = Never 1 = Once 2 = 3-5 times 3 = 6-10 times 5 = 20+ times 6 = Yes (lifetime)	Yes
Physical [†]	“Shook him/her.”		Yes
Poverty			
Economic Hardship [†]	“In the past 12 months, did you borrow money from friends or family to help you pay bills?”	0 = No 1 = Yes	No

Note. Construct categories are based on the original ACE Study. All measures are parent-report. “Original ACE” refers to whether the same construct was included in the original ACE Study. *Denotes lifetime reports, whereas all others refer to the past year. †Denotes that the construct is a multi-item measure and one sample item is presented.

Substance use problems. Biological parent alcohol and drug use was assessed using a subset of self-report items derived from the Composite International Diagnostic Interview Short-Form (CIDI-SF; Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998; see Appendix B). Alcohol use was measured via three items assessing the frequency of alcohol use (two items) and if alcohol use interfered with daily activities (one item) in the past year. In the current study, the interference item served as the indicator of alcohol use problems. The interference item asked, “In the past twelve months, was there ever a time when your drinking or being hung over interfered with your work at school, or a job, or at home?” Responses were coded as either ‘yes’ (1) or ‘no’ (0). Drug use was measured via 11 items assessing the use of nine individual drugs in the past year (nine items), the frequency of use of all drugs in the past year (one item), and the use of drugs interfering with work at school, a job, or at home in the past year (one item). The present study only used the interference item as the indicator of parent drug use problems. Responses to the interference item were coded as ‘yes’ (1) or ‘no’ (0).

Mental illness. Occurrence of a Major Depressive Episode (MDE) within the past year was assessed using a subset of the MDE questions from the CIDI-SF. Specifically, biological parents responded to 15 items about feelings of dysphoria or anhedonia that lasted for at least two weeks during the past year. If parents reported these feelings, additional questions regarding specific aspects of MDE were asked (e.g., feeling tired, trouble sleeping). Responses (i.e., yes/no) to these items were then used to determine the probability that an individual would be counted as a “case” (i.e., positively diagnosed with MDE in the past year; for details on scoring, see Kessler et al., 1998). Both liberal (Kessler et al., 1998) and conservative (Walters, Kessler, Nelson, & Mroczek, 2002) cut-offs for a positive diagnosis of MDE were previously calculated for the Fragile Families and Child Wellbeing Study. The primary difference between these cut-

offs is the conservative cut-off requires two-week depressive symptoms to last “most of the day” as opposed to “over at least half the day” for the liberal cut-off. The liberal cut-off were used in this study because symptoms occurring “over at least half the day” likely have a meaningful impact on caregiving and child development. Response options for this item were ‘yes’ (1) and ‘no’ (0).

Parental loss. Loss of a parent was assessed via three separate parent-report items that queried if loss of a biological mother or father ever occurred due to incarceration⁸, separation/divorce, or death. Each item was coded as ‘yes’ (1) or ‘no’ (0).

Domestic violence. Domestic violence perpetrated against the mother by either the biological father or current partner was assessed via two items that queried whether the mother ever (a) got into a physical fight with the father/partner in front of the child, and (b) if the father/partner physically hurt the mother in front of the child. Both items were coded as ‘yes’ (1) or ‘no’ (0). If the mother responded ‘yes’ to either item it was counted towards the child’s total ACE score.

Childhood abuse. Past year physical and psychological abuse was assessed using a subset of items from the Parent Child Conflicts Tactics Scale (CTSPC; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998; see Appendix C). The primary biological caregiver responded to four items about physical abuse (e.g., “shook him/her”) and four items about psychological abuse (e.g., “called him/her dumb or lazy or some other name like that”).⁹ All items were coded on a scale of 0 (never) to 5 (20 or more times). Similar to the original ACE Study, physical and psychological abuse were dichotomized. Endorsement of any of these experiences was coded as ‘yes’ (1) and no endorsement was coded as ‘no’ (0).

⁸The item assessing mother’s incarceration only asked about incarceration within the past four years (i.e., since the child was approximately five years old).

⁹“Spanked him/her on the bottom with your bare hand” and “Threatened to spank or hit him/her but did not actually do it” were removed from the physical and psychological abuse questionnaires, respectively due to their benign nature and representation of physical *discipline* rather than physical *abuse*.

Poverty. Poverty was assessed via biological parent-report of 10 items derived from the Survey of Income and Program Participation (SIPP; Bauman, 1998) and Social Indicators Survey (SIS; Social Indicators Survey Center, 1999; see Appendix D). Items on this questionnaire queried biological parents about resource availability in the past year (e.g., “In the past 12 months, did you borrow money from friends or family to help pay bills?”). Items responses were coded as either ‘yes’ (1) or ‘no’ (0). If a parent indicated that any of the 10 experiences occurred, the child received a ‘yes’ (1) for exposure to poverty in their ACE score.

Self-Regulation Measures

Effortful control. Effortful control was assessed via a child-report measure of task perseverance (see Appendix E). The five items used in this scale were modeled after the perseverance scale from the Child Development Supplement of the Panel Study of Income Dynamics (PSID-CDS-II and III; Child Development Supplement: Panel Study of Income Dynamics, 2007). A sample item from this measure is, “I stay with a task until I solve it.” Responses ranged on a scale from 0 (never) to 3 (often). Due to low frequency counts for the ‘never’ response option within the analytic sample it was combined with the ‘rarely’ response option. Updated response options ranged on a scale from 0 (never/rarely) to 2 (often). Reliability for this scale was acceptable ($\alpha = .73$). As described below, factor scores were derived from a confirmatory factor analysis (CFA) of this construct and used in subsequent analytic models.

Self-control. Self-control was assessed via the self-control subscale of the teacher-reported Social Skills Rating System (SSRS; Gresham & Elliott, 1990; see Appendix E). This 10-item scale assesses a child’s ability to manage their behaviors and emotions in a variety of challenging situations. A sample item from this measure is, “Controls temper in conflict situations with peers.” Response options range on a scale from 0 (never) to 3 (very often).

Reliability for this scale was good ($\alpha = .95$). Similar to effortful control, factor scores were derived from a CFA and used in subsequent analytic models.

Outcome Measure

Telomere length. Telomere data were obtained from children and their biological mother during the home visit stage of data collection using the Oragene[®] DNA Self-Collection Kit. Complete information on DNA data collection, storage, processing and quality control can be found elsewhere (<https://fragilefamilies.princeton.edu/restricted/genetic>). Telomere length was determined using a modified qPCR method that allows for the absolute measurement of telomere length (in kilobases per telomere), as described by O’Callaghan and Fenech (2011). Briefly, an 84-mer double-stranded oligonucleotide containing the sequence ‘TTAGGG’ was used to create a standard curve for telomere quantity, and a 79-mer double-stranded oligonucleotide containing a sequence from the *36B4* gene was used to create a standard curve for the reference gene. Telomere length was calculated by dividing the telomere quantity by the reference gene quantity. The telomere length/telomere ratio was then determined by dividing this value by 92. Each sample was assayed twice using qPCR, once using primers to amplify telomeric sequences and a second time using primers to amplify *36B4* sequences. All samples were measured in triplicate and the results averaged.

Covariates

Several covariates were included in all relevant analyses due to their known association with exposure to ACEs, self-regulation, and/or telomere length. These included: child body mass index (BMI; Starkweather et al., 2014), race and gender (Bethell et al., 2017; Murray et al., 2015), and biological mother’s telomere length (Slagboom, Droog, & Boomsma, 1994).

Analytic Strategy

All analyses were run using R version 3.5.2 (R Core Team, 2018). Prior to all analyses, data distributions were examined for normality and outliers. Both mother and child telomere length were skewed and kurtotic, so log-transformations were applied to these variables to ensure all assumptions of linear regression were met for the inferential analyses in Aim 1. No more than 5% data were missing on any variable included in the inferential analyses.¹⁰ Best practice suggests that parameter estimates are not biased – and missing data imputation is not necessary – when less than 5% of data are missing (Bennett, 2001; Schafer, 1999); therefore, listwise deletion was used for all analyses. Since Aim 2 used a path analytic framework, models were evaluated for goodness-of-fit. Following the recommendations of Hu and Bentler (1999), several indices were used to evaluate model fit, including comparative fit index (CFI; Bentler, 1992), root mean square error of approximation (RMSEA; Browne & Cudeck, 1993), and standardized root mean square residual (SRMR; Hu & Bentler, 1999). Models with a CFI value at or above 0.90, a RMSEA value at or below 0.05 (Jackson, Gillaspay, & Purc-Stephenson, 2009), and a SRMR values at or below 0.08 (Hu & Bentler, 1999) were considered to have good fit. All statistical analyses used a *p*-value of 0.05 and effect sizes (i.e., R^2) were reported for all models.

Factor Structure of Effortful Control and Self-Control

Effortful control and self-control were first modeled as latent constructs, in part to test their factor structure across key demographic variables, namely gender and race. Using methods outlined by Putnick and Bornstein (2016), measurement invariance across gender and race was tested for both self-regulation variables. First, a configural model was run to examine if factor loadings appeared to vary significantly (i.e., absolute difference > .30) across groups. If they did not differ, a second “metric invariance” model was run where factor loadings were fixed across

¹⁰Using the ‘LittleMCAR’ function from the R package *BaylorEdPsych*, missing data were determined to not be missing completely at random (MCAR; $p < .05$); therefore, imputation of missing values using full information maximum likelihood estimation (FIML) was not possible. Data are assumed to be missing at random (MAR).

groups. Lastly, a “scalar invariance” model was run where item intercepts were fixed across groups. All nested models were compared; if there was a decrease in model fit of at least .01 for key model fit indices (i.e., CFA, TLI, RMSEA, SRMR), then the poorer fitting model was determined to have significantly worse fit and the requirement of invariance was not met. As can be seen in Tables 2 through 5, multiple group models for gender and race were fully invariant, which is consistent with previous research (e.g., Walthall, Konold, & Pinata, 2005). Additional multiple group models were tested to examine if the factor structure varied as a function of minority-minority and majority-minority bi-racial children. As can be seen in Tables 6 and 7, these models also demonstrated full invariance. Based on these results, factor scores were derived from the configural model using the ‘lavPredict’ function from the R package *lavaan* (Rosseel, 2012) and used in relevant analyses.

Construction of Adverse Childhood Experiences Index

Prior to forming the composite ACE variables, bivariate correlations were run between all ACEs to determine if there was a pattern of association between the constructs. However, in order to draw comparative conclusions to previous ACEs research (e.g., Felitti et al., 1998), the construction of the ACE indexes consisted of summing the total number of ACEs each child experienced. Each ACE was dichotomized with a possible score of ‘0’ or ‘1’ indicating any exposure to the adversity. The range of possible scores was 0-8 for the “traditional” ACE index, and 0-10 for the updated ACE index that included parental death and poverty as ACEs.

Inferential Analyses

Aim 1. The first aim, which was to examine the associations between a composite measure of ACEs and child telomere length, adjusting for hypothesized covariates, was tested using ordinary least squares regression. Two sets of models were run, one using the traditional

ACE index and another using the updated ACE index. The dependent variable in the models was child telomere length and the primary predictor was the ACE index; covariates included maternal telomere length, child race, and child BMI. First, a model was run only with covariates included and then a second model was run with the ACE variable included. Since the covariates remained identical across models and only the ACE variable changed, three models were run to test these hypotheses. The overall change in R^2 between the covariate model and the model with each ACE variable was calculated to determine if each ACE index explained a significant amount of variance in child telomere length above and beyond the hypothesized covariates. Lastly, assumptions of linear regression are presented in Appendix F for the latter model that included all predictors.

Aim 2. The second aim, which was to examine the indirect association between ACEs and telomere length through self-regulation, was tested using path analysis. Two separate models were run; one using the traditional ACE index, and another using the updated ACE index. In addition, self-control and effortful control were assessed in separate models, for a total of four models tested for Aim 2. Variables included in the analysis were similar to Aim 1, but child gender was added as a predictor of each self-regulation variable since previous research suggests gender differences in these constructs (e.g., Murray et al., 2015). To ensure stability of the estimates, 5,000 bootstrap draws were taken for the standard errors in each model, and bias-corrected, bootstrapped confidence intervals were computed for all parameter estimates.

Results

Attrition Analyses

Prior to the inferential analyses, children who had valid telomere data ($N = 2,527$) were compared to those children whose families either refused to provide telomere data, or to families

whose data were not collected for another unspecified reason ($n = 444$). These two groups were compared on ACEs, both self-regulation variables, and each covariate included in the inferential analyses, using independent samples t -tests, chi-square tests, and Poisson regression analyses (for count outcomes) as appropriate. Results from independent samples t -tests revealed that children who provided telomere data had higher levels of self-control compared to those who did not provide telomere data, $t(317.62) = -2.16, p < .05$. Chi-square analyses revealed that more Black and Hispanic children had telomere data than expected, $\chi^2(3) = 30.86, p < .001$. No other differences were detected among these groups.

There also was a large portion of teachers ($n = 915$) who did not participate in wave nine data collection, which led to a smaller sample size ($n = 1,612$) for analyses using the self-control variable. To ensure that teacher dropout did not bias findings during this wave, additional attrition analyses were conducted to examine if children with teacher-reported self-control data differed from those without these data on ACEs, effortful control, gender, age, BMI, and race/ethnicity. Results from chi-square analyses revealed that teachers of Black students tended not to respond during wave nine data collection compared to other races/ethnicities, $\chi^2(3) = 36.10, p < .001$. No other group differences were detected.

Descriptive Statistics and Correlations between Study Variables

Table 8 provides descriptive statistics for the core study variables. Based on the constructed ACE indices, children experienced anywhere from zero to six traditional ACEs (median = 1) and zero to seven updated ACEs (median = 2). Table 9 provides the percentage of children exposed to different amounts of ACEs. While a majority (~72%) of children experienced anywhere from one to three ACEs (irrespective of index), 5.7% experienced at least four traditional ACEs and 15.6% experienced at least four updated ACEs.

Table 8. *Descriptive Statistics for Core Study Variables*

Variable	<i>N</i>	Mean (<i>SD</i>)	Range	Skew / Kurtosis
Traditional ACE	2,523	1.50 (1.17)	0 – 6	0.70 / 0.27
Updated ACE	2,523	2.12 (1.39)	0 – 7	0.44 / -0.22
Effortful Control	2,446	-0.02 (0.50)	-1.63 – 0.67	-0.31 / -0.57
Self-Control	1,612	-0.01 (0.83)	-2.57 – 1.47	-0.15 / -0.38
Child TL	2,527	8.09 (2.72)	3.00 – 20.91	1.09 / 1.59

Note. Range refers to the range of values observed across the data, not all possible values. Effortful control and self-control are represented as factors scores and are thus interpreted similarly to z-scores. TL = telomere length.

Table 9. *Percentage of Children Exposed to Different Number of Adverse Childhood Experiences*

Traditional ACE	Zero	One	Two	Three	Four	Five	Six	-
% Exposed	20.8%	34.4%	26.1%	12.7%	4.4%	1.1%	.2%	-
Updated ACE	Zero	One	Two	Three	Four	Five	Six	Seven
% Exposed	11.8%	24.3%	26.3%	21.6%	10.2%	4.3%	1.0%	.1%

Note. *N* = 2,523. Maximum number of possible experiences was eight and 10 for traditional and updated ACEs, respectively.

Table 10 provides information regarding the percentage of children exposed to each type of ACE, whether the ACE source was the mother or father, as well as how many children had both parents encounter substance use issues, mental health problems, incarceration, or economic hardship. As can be seen in Table 10, the majority of children (61.3%) experienced some form of economic hardship. Approximately half (50.8%) of children had either parent be incarcerated and 44.1% experienced psychological abuse from their primary caregiver (e.g., been told they were stupid/dumb/lazy), while 13.1% of children experienced some form of physical abuse (e.g., being shook by their primary caregiver). Lastly, few children were in a situation where both parents encountered substance use problems, mental health issues, incarceration, or economic

hardship. It is possible that both parents were perpetrators of physical and psychological abuse aimed at the child, but only the primary caregiver provided data on these items.

Table 10. *Percentage of Children Exposed to each Adverse Childhood Experience*

Adverse Childhood Experience	Mother	Father	Either	Both
Alcohol Use	7.5% (n = 370)	8.3% (n = 673)	9.1% (n = 906)	.1% (n = 906)
Drug Use	7.6% (n = 222)	8.2% (n = 279)	8.3% (n = 468)	.2% (n = 906)
Major Depressive Episode	17% (n = 2,446)	14% (n = 1,776)	24.8% (n = 2,499)	1.7% (n = 2,499)
Parent Incarceration [†]	32.3% (n = 65)	50.5% (n = 2,502)	50.8% (n = 2,502)	.6% (n = 2,502)
Parent Separation/Divorce	-	-	27.7% (n = 990)	-
Parent Death	.2% (n = 1,770)	1.3% (n = 2,445)	1.5% (n = 2,493)	-
Domestic Violence	6.1% (n = 2,149)	-	-	-
Psychological Abuse	-	-	44.1% (n = 2,395)	-
Physical Abuse	-	-	13.1% (n = 2,288)	-
Poverty	54.9% (n = 2,453)	56.5% (n = 859)	61.3% (n = 2,497)	12.1% (n = 2,497)

Note. N = 2,527; sample sizes vary according to whether the mother, father, or both parents provided data for each ACE. Participants missed data collection opportunities for various reasons, so denominators for the proportions will vary by cell. [†]The only available data on mother incarceration asked about the past four years, whereas father incarceration data refers to lifetime incarceration.

Tables 11 and 12 provide zero-order correlations between ACEs and the core study variables, respectively. As can be seen in Table 11, most ACEs were correlated with one another in the expected direction; however, alcohol and drug use were only correlated with parent incarceration and diagnosis of a MDE, while MDE was correlated with all ACEs. Parent incarceration was correlated with each ACE, except psychological abuse. In regard to

correlations between core study variables in Table 12, the traditional ACE measure and the updated ACE measure were highly correlated. Moreover, both ACE indexes were inversely correlated with effortful control and self-control and child telomere length, although the correlation between traditional ACEs and child telomere length was not significant. Lastly, effortful control and self-control were both positively correlated with child telomere length, although the magnitude of these associations was small.

Table 11. *Zero-Order Correlations Between Adverse Childhood Experiences*

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Alcohol Use	-								
2. Drug Use	.22***	-							
3. Incarceration	.08*	.03	-						
4. MDE	.12***	.11*	.14***	-					
5. Poverty	.06	.04	.25***	.21***	-				
6. Physical Abuse	.04	-.02	.09***	.08***	.09***	-			
7. Psychological Abuse	.03	.05	.03	.07***	.05*	.30***	-		
8. Divorce	.07	.01	.32***	.11***	.25***	.07*	-.03	-	
9. Dom. Violence	.06	.06	.06*	.10***	.08***	.06**	.05*	.14***	-

Note. Parental death is excluded since there would be no other parent data available for a correlation. All p-values are two-tailed.

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 12. *Zero-Order Correlations Between Study Variables*

	1.	2.	3.	4.	5.	6.	7.	8.
1. Trad. ACE	-							
2. Updated ACE	.94***	-						
3. Eff. Control	-.05**	-.05**	-					
4. Self-Control	-.15***	-.17***	.13***	-				
5. Child TL	-.03	-.05**	.04*	.08**	-			
6. Mom TL	.01	.001	.02	.06*	.28***	-		
7. Gender	.04*	.02	-.10***	-.19***	-.03	.004	-	
8. BMI	-.01	.01	.01	-.03	.01	.06*	-.07***	-

Note. TL = telomere length; Gender was coded as 0 = female and 1 = male. All p-values are two-tailed.

* $p < .05$, ** $p < .01$, *** $p < .001$

Aim 1

Hypothesis 1. Sequential regression analyses were run to test whether ACEs (traditional and updated) were associated with child telomere length, adjusting for hypothesized covariates. The first model, which included covariates only, was significant, $F(5, 1974) = 39.37, p < .001$, explaining 9% of the variance in child telomere length. Of the covariates in the model, biological mother telomere length was uniquely and positively associated with child telomere length ($b = .31, p < .001$). The next model that included traditional ACEs as a predictor also was significant, $F(6, 1973) = 33.52, p < .001$, but including traditional ACEs in the model did not explain a significant amount of variance in child telomere length above and beyond the hypothesized covariates, $\Delta F(1) = 2.36, p > .05, \Delta R^2 = .001$. Traditional ACEs were not uniquely associated with child telomere length in this model, ($b = -.01, p > .05$), but maternal telomere length remained a significant covariate ($b = .31, p < .001$). No other associations were detected in the model (see Table 13 for model results).

Table 13. Results from Multiple Regression Analyses Using Traditional ACEs

Predictor	Model 1		Model 2	
	<i>b</i> (<i>se</i>)	<i>p</i>	<i>b</i> (<i>se</i>)	<i>p</i>
Mother TL	.31 (.02)	< .001*	.31 (.02)	< .001*
Child BMI	-.0004 (.002)	.79	-.0004 (.002)	.79
White	.03 (.03)	.28	.03 (.03)	.33
Hispanic	-.002 (.02)	.94	-.001 (.03)	.81
Black	-.04 (.02)	.10	-.04 (.02)	.10
Traditional ACE	-	-	-.01 (.01)	.06

Note. All estimates are unstandardized. *Denotes a statistically significant estimate.

The next model that used the updated ACE index also was significant, $F(6, 1973) = 34.11, p < .001$, and including the updated ACE index in the model explained a significant amount of variance in child telomere length above and beyond the hypothesized covariates,

$\Delta F(1) = 5.56, p < .05, \Delta R^2 = .003$. Updated ACEs in this model was significantly and negatively associated with child telomere length ($b = -.01, p < .05$), such that each additional ACE was associated with a 1% decrease in child telomere length. Maternal telomere length also was significantly associated with child telomere length ($b = .31, p < .001$). No other associations were detected in the model (see Table 14 for model results). As can be seen in the paneled figures in Appendix F, all assumptions of linear regression were met for both of the hypothesized models including the ACE indices (i.e., linearity, normality of residuals, homoscedasticity, and statistical independence of residuals). While the updated ACE index was significantly associated with child telomere length above and beyond the hypothesized covariates, taking the change of effect size into account (.003), it appears that this amount additional variance is not practically meaningful.

Table 14. *Results from Multiple Regression Analyses Using Updated ACEs*

Predictor	Model 1		Model 2	
	<i>b</i> (<i>se</i>)	<i>p</i>	<i>b</i> (<i>se</i>)	<i>p</i>
Mother TL	.31 (.02)	< .001*	.31 (.02)	< .001*
Child BMI	-.0004 (.002)	.79	-.0004 (.002)	.80
White	.03 (.03)	.28	.02 (.03)	.40
Hispanic	-.002 (.02)	.94	-.01 (.03)	.71
Black	-.04 (.02)	.10	-.04 (.02)	.10
Updated ACE	-	-	-.01 (.01)	.01*

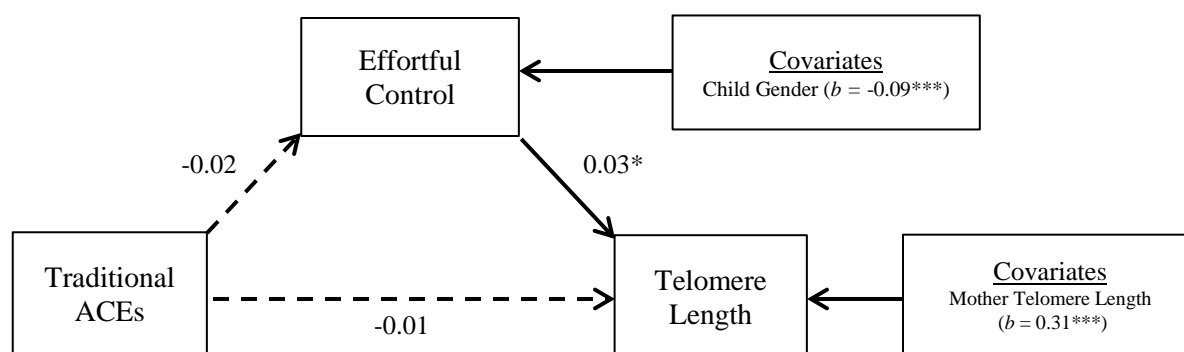
Note. All estimates are unstandardized. *Denotes a statistically significant estimate.

Aim 2

Hypothesis 1. Path analysis was used to test the second study aim that ACEs would be indirectly associated with child telomere length through self-regulation, adjusting for relevant covariates. The first model used effortful control as the mediator and the traditional ACE index to predict child telomere length. The model fit the data well ($\chi^2(3) = 1.66, p > .05$; CFI = 1.00; RMSEA = 0.00, 90% CI [0.00-0.03]; SRMR = .004), with predictors explaining 1% of the

variance in effortful control and 9% of the variance in child telomere length. As can be seen in Figure 7, traditional ACEs were not associated with child telomere length ($b = -.01, p > .05$) or effortful control ($b = -.02, p > .05$). There was a statistically significant association, however, between effortful control and child telomere length ($b = .03, p < .05$), such that a one unit increase in effortful control was associated with a 3% increase in child telomere length. Neither the total effect nor the indirect effect was statistically significant in this model. Regarding covariates, there was a statistically significant positive association between maternal telomere length and child telomere length ($b = .31, p < .001$), and a statistically significant association between gender and effortful control ($b = -.09, p < .001$), such that girls had higher levels of effortful control than boys.

Figure 7. Mediation Model with Traditional ACEs and Effortful Control



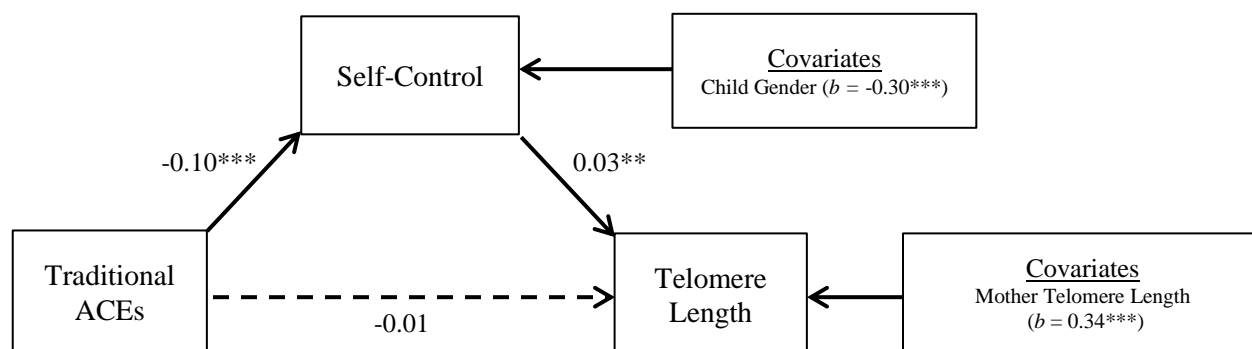
All estimates are unstandardized. Dashed lines indicate non-significant pathways. Neither the indirect effect ($b < .001, p > 0.05$) nor the total effect ($b = -0.01, p > 0.05$) was statistically significant.

* $p < .05$ ** $p < .01$ *** $p < .001$

The second model, which substituted effortful control for self-control also fit the data well ($\chi^2 (3) = 5.01, p > .05$; CFI = .99; RMSEA = 0.02, 90% CI [0.00-0.06]; SRMR = .01), with predictors explaining 11% of the variance in self-control and 12% of the variance in child telomere length. As can be seen in Figure 8, there was a statistically significant association between traditional ACEs and self-control ($b = -.10, p < .001$), such that with each additional

ACE, there was a .10 standard deviation decrease in self-control. There also was a statistically significant association between self-control and child telomere length ($b = .03, p < .05$), such that a one unit increase in self-control was associated with a 3% increase in child telomere length. While neither the direct effect of traditional ACEs, nor the total effect was significant, there was a significant indirect effect of traditional ACEs on child telomere length through self-control ($b = -.003, p < .05$). Regarding covariates, girls had higher levels of self-control compared to boys ($b = -.30, p < .001$), and Black children had lower levels of self-control compared to White and Hispanic children ($b = -.34, p < .001$). Lastly, there was a statistically significant positive association between maternal telomere length and child telomere length ($b = .34, p < .001$).

Figure 8. Mediation Model with Traditional ACEs and Self-Control



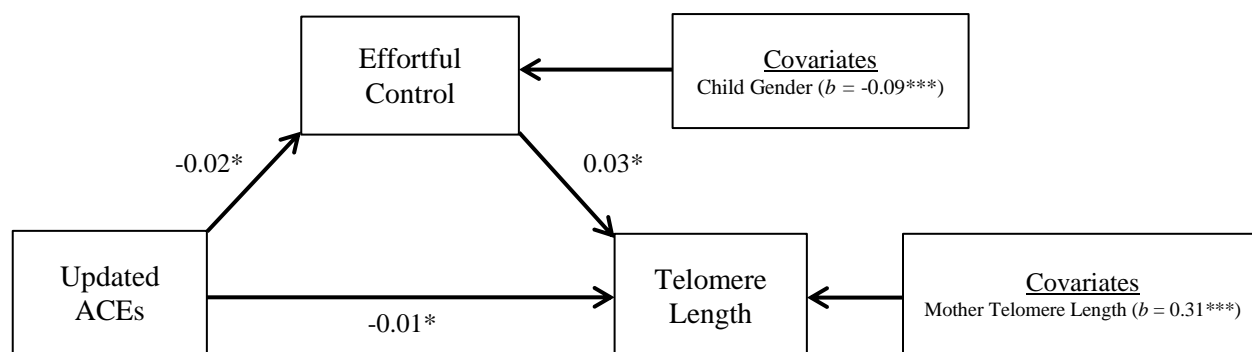
All estimates are unstandardized. Dashed lines indicate non-significant pathways. The indirect effect was statistically significant, $b = -.003, p < .05$ [95% CI = $-.01 - .001$], but the total effect was not statistically significant ($b = -.01, p > .05$).

* $p < .05$ ** $p < .01$ *** $p < .001$

The next two models used the updated ACE variable, and tested separate models for effortful and self-control. The first of these models, using effortful control as the mediator, fit the data well ($\chi^2(3) = 1.70, p > .05$; CFI = 1.00; RMSEA = 0.00, 90% CI [0.00-0.03]; SRMR = .004), with predictors explaining 1% of the variance in effortful control and 10% of the variance in child telomere length. As can be seen in Figure 9, there was a statistically significant

association between the updated ACEs and child telomere length ($b = -.01, p < .05$), such that with each additional ACE there was a 1% decrease in child telomere length. In addition, there was a statistically significant association between updated ACEs and effortful control ($b = -.02, p < .05$), such that with each additional ACE there was a .02 standard deviation decrease in effortful control. There also was a significant association between effortful control and child telomere length ($b = .03, p < .05$), such that a one unit increase in effortful control was associated with a 3% increase in child telomere length. The total effect on child telomere length ($b = -.01, p < .05$) was significant, but there was not a statistically significant indirect effect. Regarding covariates, girls had higher levels of self-control compared to boys ($b = -.09, p < .001$), and there was a statistically significant association between maternal telomere length and child telomere length ($b = .31, p < .001$).

Figure 9. Mediation Model with Updated ACEs and Effortful Control



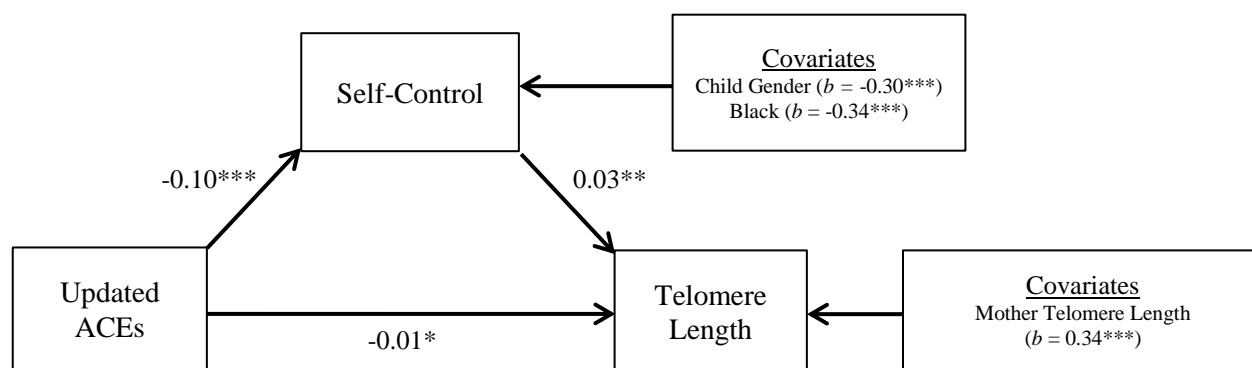
All estimates are unstandardized. Dashed lines indicate non-significant pathways. While the indirect effect ($b < .001, p > 0.05$) was not statistically significant, the total effect was statistically significant, $b = -0.01, p < 0.05$ [95% CI = $-.02 - -.002$].

* $p < .05$ ** $p < .01$ *** $p < .001$

The final model, which substituted effortful control with self-control, fit the data well ($\chi^2(3) = 4.78, p > .05$; CFI = .99; RMSEA = 0.02, 90% CI [0.00-0.06]; SRMR = .01), with predictors explaining 12% of the variance in self-control and 12% of the variance in child telomere length. As can be seen in Figure 10, the direct effect of the updated ACEs on child

telomere length was statistically significant ($b = -.01, p < .05$), such that each additional ACE was associated with a 1% decrease in child telomere length. The association between ACEs and self-control also was significant ($b = -.10, p < .001$), such that with each additional ACE, there was a .10 standard deviation decrease in self-control. There also was a statistically significant association between self-control and child telomere length ($b = .03, p < .05$), such that a one unit increase in self-control was associated with a 3% increase in child telomere length. There also was a statistically significant indirect effect of the updated ACEs on child telomere length through self-control ($b = -.003, p < .05$). The total effect was significant too ($b = .01, p < .05$). Regarding covariates, girls had higher levels of self-control compared to boys ($b = -.30, p < .001$), and Black children had lower levels of self-control compared to White and Hispanic children ($b = -.34, p < .001$). Lastly, there was a statistically significant association between maternal telomere length and child telomere length ($b = .34, p < .001$).

Figure 10. Mediation Model with Updated ACEs and Self-Control



All estimates are unstandardized. Dashed lines indicate non-significant pathways. The indirect effect was statistically significant, $b = -.003, p < .05$ [95% CI = $-.01 - .001$], as was the total effect, $b = -.01, p < .05$ [95% CI = $-.03 - .001$].

* $p < .05$ ** $p < .01$ *** $p < .001$

Discussion

While the association between ACEs and telomere length has been established, the selection of events included in an ACE index often is overlooked; moreover, behavioral

mechanisms of telomere attrition are not well known. The present study sought to (a) test the association between ACEs and telomere length during childhood, using the traditional ACE index and an updated index including two meaningful childhood adversities (i.e., parental death, poverty), and (b) identify a novel pathway (i.e., self-regulation) through which ACEs may contribute to telomere attrition. In doing so, researchers can better understand the association between ACEs and telomere length during childhood, but also isolate a strong target for intervening to promote healthy development for those who face childhood adversity. While the present findings did not support a meaningful, unique association between ACEs and child telomere length above and beyond the influence of hypothesized covariates, results from mediation analyses provide preliminary support for an indirect association between ACEs and child telomere length through the self-control component of self-regulation. While promising, interpretation of these findings must be considered in light of small effect sizes and an inability to establish causality given the cross-sectional nature of the data. Nonetheless, these results advance our understanding of the ACEs measure and pathways of telomere attrition, while raising important questions about the measurement of ACEs and ways to promote healthy development in the context of childhood adversity.

Adverse Childhood Experiences and Child Telomere Length

The first study aim and hypothesis, which stated that ACEs would be associated with child telomere length, was partially supported. The model using the traditional ACE index did not explain a significant amount of variance in child telomere length above and beyond the covariates, nor was the independent association between the ACE index and child telomere length significant. Although the updated ACE index was independently associated with child telomere length and explained a significant amount of variance above and beyond the covariates,

this difference in variance accounted for was negligible (i.e., .3%). While the lack of evidence for a unique association between traditional ACEs and child telomere length was unexpected, it is possible that having parent-report data for certain ACEs affected these findings. For example, parents may be hesitant to report their own use of abusive behaviors (e.g., “calling your child stupid/lazy/dumb,” “shaking your child”); collecting child-reported data could have revealed higher rates of exposure to physical and/or psychological abuse. Alternatively, the measures of experiences like physical and psychological abuse may not accurately capture abusive behavior that is detrimental to a child at the molecular level. Although the Conflict Tactics Scale often is used to assess physically abusive behaviors (e.g., “shaking your child”), more benign items also are included (e.g., “shouted or yelled at your child”). Given the high proportion of Black families in the current sample, two items related to spanking were removed. This was, in part, to account for the culturally normative practice of spanking among Black families that is not linked to negative adjustment in youth (e.g., Whaley, 2000). While appropriate, it may be the case that additional items need to be excluded (e.g., “shouted or yelled at your child”) or additional items need to be included that assess more severe forms of abuse (e.g., “hit your child so hard you left a bruise”).

While the finding that the updated ACE index was independently associated with child telomere length was promising, these results are tempered by the limited variance explained by this variable. While similar limitations exist for this model compared to the model using the traditional ACE index, there are several reasons why a larger difference between the two indices was not detected. First, only 1.5% ($n = 37$) of children lost a parent, with a majority of these children having lost their father. Given the limited proportion of individuals, it is possible that a stronger effect of parental loss due to death was not able to be detected. Second, while a plethora

of research supports an independent association between poverty and biological functioning (for a review, see Miller et al., 2011), the influence of poverty in the context of other variables often is not considered, nor is the measurement of poverty consistent across studies, which contributes to heterogeneity in association tests and effect sizes. The present study took a unique approach to measuring poverty by using specific, individual indicators of economic hardship (e.g., trouble paying rent), and only requiring endorsement of a single hardship to be categorized in the ‘yes’ category for this ACE. This was done primarily to maintain a consistent measurement model with the traditional ACE index. Moreover, these specific indicators have the advantage of tapping into resource availability that point towards a broader impoverished environment, but one could argue that they do lack a more direct, global assessment of poverty (e.g., composite of parent education and income) that may elicit a larger effect.

The challenges in measurement related to poverty/economic hardship speak to a broader measurement challenge when using the ACEs framework as it currently stands. One of the primary reasons that the ACEs questionnaire (as well as many other indices of cumulative risk) is so popular is its ease of measurement, particularly in the primary care setting. It is much easier for a physician, clinician, or researcher to administer a 10-item questionnaire with binary (i.e., yes/no) responses than to collect data on frequency, severity, or timing of childhood adversities. Even though there are consistent links between ACEs and health when using binary indicators (for a review, see Kalmakis & Chandler, 2015), it is unclear, for example, how having two versus one parent with a substance use problem exacerbates this association. To date, most studies of cumulative risk focus on the number of ACEs as opposed to the type or severity (Evans et al., 2013; McLaughlin & Sheridan, 2016). McLaughlin & Sheridan (2016) argue, however, that failing to take into account these contextual factors may obscure associations and

investigations into mechanisms, particularly because it is difficult to understand which ACE(s) are driving associations, and their subsequent mechanism(s) of action.

While the present data did not permit a thorough test of frequency of severity of each ACE, nor does previous research suggest that this information adds to the robustness of research findings (cf, Hanssen et al., 2017), researchers can (and should) explore these factors further. The majority of studies examining the association between ACEs (as a composite or individually) and telomere length and/or health outcomes often use preexisting measures of childhood maltreatment (e.g., Conflict Tactics Scale) and/or crude measures of frequency (e.g., never, once, more than once; Mason, Prescott, Tworoger, DeVivo, & Richd-Edwards, 2015). The current study attempted to address this issue by requiring parents to indicate any physically or psychologically abusive behavior occurring at least six to 10 times in order to categorize the child as having experienced physical or psychological abuse. While helpful, this method still lacks the ability to assess more physically and/or psychologically abusive behaviors, as well as the perceived impact from the child's perspective. Future work should consider creating augmented versions of the original ACE questionnaire that better measure this information. For example, an improved measure of ACEs could directly ask children the perceived impact of these experiences. Alternatively, McLaughlin and Sheridan (2016) suggest that researchers conceptualize childhood adversity along dimensions of deprivation and threat, as these factors underlie many ACEs (e.g., neglect, abuse, and poverty), are linked to biological processes (some that are tied to self-regulation), and would elucidate mechanisms linking childhood adversity to various biological and health outcomes.

Self-Regulation as a Mediator of ACEs and Telomere Length

There was partial support for the second study aim and hypothesis, which stated that ACEs would indirectly affect child telomere length through self-regulation, operationalized here as effortful control and self-control, separately. In the models using self-control as the mediator, both ACE indices were inversely associated with self-control, such that exposure to more ACEs was associated with significant decreases in self-control. Moreover, both models revealed significant indirect effects of ACEs on child telomere length through self-control. These results can be viewed through two lenses: child-focused ACEs and parent-focused ACEs. For the child-focused ACEs (physical and psychological maltreatment), McEwen and Stellar's (1993) theory of allostatic load dovetails nicely with these findings. Specifically, the results support a model whereby children who experience adversity directed at them may develop a dysregulated stress response due to either (a) repeated exposure to abuse or (b) fear of exposure to future abuse (i.e., increased threat vigilance). This, in turn, can affect biological factors susceptible to the physiological demands of chronic stress, namely telomere length. While self-regulation is only a putative indicator of these underlying processes driving telomere attrition, research linking dysregulated HPA functioning to healthy functioning emphasize the impact of HPA axis dysregulation on emotional reactivity (e.g., Lupien et al., 2009). Pairing these findings with physiological data (e.g., cortisol production) would aid in confirming this hypothesis.

It also is important to note that the self-control measure used in this study was emotion-focused (e.g., "controls temper in conflict situations with peers"). Felitti and colleagues (1998) attempted to select items that focused on the household, and more specifically on the parent-child relationship. Given the interpersonal nature of some ACEs (e.g., physical abuse) and implications for caregiving with others (e.g., parent substance use), it is clear how these

experiences can influence the emotional development of the child. For example, by thinking of the findings in terms of parent-focused ACEs, these results align well with Bridgett et al.'s (2015) model of the intergenerational transmission of self-regulation. Specifically, parents provide a rearing context for their children where their behaviors and experiences have an effect on the child's development, including self-regulatory skills. In the context of the present findings, parental behaviors like substance use, or mental illnesses like maternal depression likely directly affect the quality of care given to the child. For example, Li, Riis, Ghazarian, and Johnson (2018) sampled mothers and their five-year old children and found that maternal depressive symptoms were significantly, inversely associated with children's cognitive self-regulation (a construct comprised of effortful control). Additional research suggests that maternal depression can negatively affect children's self-regulatory abilities through hostile or withdrawn parenting behaviors (Canadian Pediatric Society, 2004). In the context of poverty, households experiencing economic hardship tend to be more chaotic and disorganized, and/or parents have less time to spend with their children. This lack of availability by parents can place children at risk because it is more difficult to engage in parent-child interactions that foster healthy development of self-regulatory skills (Blair, 2010). Given that children in the current sample are at an age where they are beginning to develop relationships outside the household context, experiences like parent substance use and maltreatment likely prime them for how they approach social situations with their peers.

The models testing effortful control as a mediator did not support the second study aim and hypothesis. While there was a significant, positive association between effortful control and child telomere length, only the updated ACE index was significantly associated with effortful control; moreover, there was no indirect effect of ACEs on child telomere length through

effortful control. A likely reason for these null findings is the measurement of effortful control, which was derived from a measure of task perseverance. While most studies use delay gratification tasks to assess effortful control (e.g., Dich, Doan, & Evans, 2015; Lengua & Sandler, 1996), the present study operationalized effortful control via a measure of task perseverance. While similar to effortful control, task perseverance can be confounded by the child's motivation to finish tasks. For example, a child may indicate that they do not stay with tasks until they are solved, but this could be for a variety of reasons (e.g., playing with friends; lack of interest) that are not indicative of deficits in self-regulatory skills. A more nuanced measurement and/or operationalization of effortful control will help to clarify the role of effortful control in the association between ACEs and child telomere length.

Strengths and Limitations

The present study had several strengths that add valuable information to the literature on ACEs, self-regulation, and telomere length. The primary strength of the study was the inclusion of a novel pathway to explain how ACEs can indirectly affect telomere length in children. Further, self-regulation is a malleable construct that is a popular and relatively easy target for intervention, so the current findings support further exploration of self-regulation as an indirect pathway through which ACEs can impact telomere length. An additional strength of the study was exploring these associations in middle childhood. While this period of development often is overlooked as a period of latency, the present findings show that self-regulation is indeed affected by ACEs in middle childhood (~ages 5-10 years) and associated with development at the molecular level.

The study also benefitted from a large sample, which provided adequate statistical power to allow for measurement invariance testing of the self-regulation constructs and detection of

small effects, which is common in the telomere literature. Moreover, the current study was able to closely replicate the traditional ACE questionnaire, while adding two additional ACEs to elucidate their role in the predictive power of the ACE index. While results did not suggest there was much unique information added with these constructs, the current ACEs questionnaire relied on parent-report, whereas much of the original work relied on retrospective reports by adults on their childhood. Detecting significant results with parent-report data provides support for the influence of these experiences, even when removing the child's perception of the experiences. Lastly, while the ACE measure was entirely parent-report, reports of effortful control and self-control were provided by children and teachers, respectively, eliminating any source bias in the significant findings.

While the present study's strengths advance our understanding of the interrelations between ACEs, self-regulation and child telomere length, it is not without limitations. A primary limitation of the present study is the at-risk sample. While the sample was collected across 20 major U.S. cities, the sample is comprised of many parents who were unwed at birth, and many whom made less than \$40,000 per year in 2010. Given these characteristics, it is possible that these findings are not generalizable to the U.S. (or global) population. Another limitation of the study was the over-reliance on parent-report measures, as well as their measurement, of ACEs. While certain ACEs (e.g., parent substance use) made sense to be parent-report, additional information (e.g., arrest records) could be used to provide a "check" to this information. Moreover, the original ACE measure assessed lifetime exposure to adversity, whereas the current study used a mixture of lifetime and past-year measures of exposure to adversity. While telomere length can change over shorter periods of time (e.g., 1 year), previous research often used retrospective reports that assessed telomere length decades after initial exposure to adversity.

While this approach is subject to confounding by many different factors, it does limit the comparability with the present study.

The present study also was limited in its measurement of effortful control. Even though a measure of task perseverance can be indicative of one's ability to cognitively focus on a task, there is the possible confounding of motivation, which could have biased the findings in models using effortful control as the marker of self-regulation. It also is important to point out that while there was a statistically significant association between the updated ACEs and telomere length, this index accounted for less than 1% additional variance to child telomere length. In addition, the indirect effects through self-control were minimal. In light of this, the findings for Aim 1 should be interpreted as not supporting the hypothesized association, and the findings for the Aim 2 should be viewed as preliminary. The sample size allowed for the detection of small differences in the sample, and while beneficial, it is important to note that these statistical differences may not carry much practical and/or clinical significance given the small effect sizes. Lastly, the cross-sectional nature of the data limits our ability to infer causal relationships with the mediation model.¹¹ Although the pathway from ACEs to telomere length through self-regulation is logical, it is possible that deficits in a child's self-regulatory abilities confer risk for parents engaging in harmful parenting practices. For example, if a child has poor self-control, a parent may be more inclined to use physically and psychologically abrasive parenting practices (e.g., hitting the child, screaming at the child). A longitudinal design with prior measures of self-regulation and telomere length would allow for stronger conclusions regarding the results from the mediation analyses.

¹¹Given the cross-sectional nature of the data and general difficulty of detecting "true" mediation in causal modeling, Hayes (2017) suggests researchers use the term "indirect effect analysis."

Future Directions

The present study provides insight into the behavioral, cognitive, and emotional processes driving telomere attrition in children, while also providing a nuanced examination of the ACEs questionnaire; however, there are questions that remain and should be addressed in future work. The most pressing need in the literature is to add contextual information to the ACEs questionnaire that assesses the timing and frequency of events. Although the advantage of the ACEs measure as it stands is the robust associations detected with quickly administered (and scored) binary indicators, most efforts, to date, examining the timing and frequency of ACEs are crude and do not provide an adequate test of how these factors influence the impact of specific ACEs. As McLaughlin and Sheridan (2016) state, there is a need to understand what each ACE affects (e.g., resource availability, threat vigilance) and use this information to better understand pathways to negative health outcomes. Moreover, understanding the timing of ACEs can allow us to detect sensitive periods that may place children and/or adolescents at unique risk for certain ACEs. For example, maternal emotion regulation is particularly problematic for the development of emotion regulation during early childhood, which in turn is linked to internalizing and externalizing problems during middle childhood (Crespo, Trentacosta, Aikins, & Wargo-Aikins, 2017). Future work should consider creating thorough measures of ACEs to improve the predictive validity of various outcomes.

Another direction for future research is to explore (simultaneously) physiological indicators of stress (e.g., cortisol production, oxidative stress) that may relate to telomere length in order to verify if self-regulation is an acceptable putative indicator of telomere attrition. While the current findings support this model, the addition of physiological measures will provide more biological data to confirm this hypothesis. Future work also would benefit from consideration of

moderators of these associations. To date, few studies consider moderators of the association between childhood adversity and telomere length (e.g., Sosnowski et al., 2019), and identification of these factors (e.g., social support) is key to understanding how to mitigate the negative impact of ACEs that have already occurred. In a similar vein, it is important to remember that telomeres can lengthen. Much, if not all, research, to date, has explored mechanisms of telomere shortening; however, as Shalev (2012) points out, many factors (e.g., physical activity, diet) can contribute to lengthening of telomeres. It is necessary for future studies to take a strength-based approach to telomeres to understand factors that lead to lengthening and slow biological aging in the face of adversity. Lastly, these results need to be interpreted in the context of the bigger picture of health outcomes. Many researchers have focused on identifying correlations between ACEs, telomere length, and specific health outcomes independently. Given the consistent links between these constructs, it is now imperative that these data be used to predict health outcomes simultaneously or longitudinally. This will require longitudinal data with telomeres, which are scarce, but will prove invaluable when building towards a model of how ACEs “gets under the skin” to affect health outcomes throughout the lifespan.

Conclusion

The present study sought to identify a novel marker that is indicative of the underlying physiological processes driving telomere attrition, while providing an augmented view of the traditional ACE questionnaire. While findings did not support a meaningful, unique association between ACEs and child telomere length, they shed light on issues related to measurement of ACEs. Results also partially supported the indirect effect of both ACE indices on child telomere length, but only through the self-control component of self-regulation. While longitudinal data

are needed to explore causal relations with health outcomes, and additional information is needed to improve our understanding of ACEs, the present study takes an important step in this direction and provides preliminary evidence for a unique pathway for how ACEs “get under the skin.”

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Table 2. Tests of Measurement Invariance by Gender for Effortful Control

Model	χ^2 (df)	CFI	TLI	RMSEA (90% CI)	SRMR	Model comp	$\Delta\chi^2$ (Δdf)	ΔCFI	ΔTLI	$\Delta RMSEA$	$\Delta SRMR$	Decision
M0: CFA for original scale	52.65 (5)***	.98	.95	.06 (.05-.08)	.04	-	-	-	-	-	-	-
M1: Configural Invariance	63.41 (10)***	.97	.94	.07 (.05-.08)	.04	-	-	-	-	-	-	Accept
M2: Metric Invariance	66.59 (14)***	.97	.96	.06 (.04-.07)	.05	M1	3.18 (4)	0	.02	.01	.01	Accept
M3: Scalar Invariance	83.21 (18)***	.97	.96	.05 (.04-.07)	.05	M2	16.62 (4)**	0	0	.01	0	Accept

Note. $N = 2,446$; female = 1,182; male = 1,264.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$.

Table 3. Tests of Measurement Invariance by Gender for Self-Control

Model	χ^2 (df)	CFI	TLI	RMSEA (90% CI)	SRMR	Model comp	$\Delta\chi^2$ (Δdf)	ΔCFI	ΔTLI	$\Delta RMSEA$	$\Delta SRMR$	Decision
M0: CFA for original scale	667.01 (35)***	.99	.99	.11 (.09-.11)	.03	-	-	-	-	-	-	-
M1: Configural Invariance	694.87 (70)***	.99	.99	.11 (.09-.11)	.03	-	-	-	-	-	-	Accept
M2: Metric Invariance	606.86 (79)***	.99	.99	.09 (.08-.10)	.04	M1	88.01 (9)***	0	0	.01	.01	Accept
M3: Scalar Invariance	706.69 (98)***	.99	.99	.09 (.08-.09)	.03	M2	99.83 (19)***	0	0	0	.01	Accept

Note. $N = 1,612$; female = 778; male = 834.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$.

Table 4. Tests of Measurement Invariance by Race for Self-Control

Model	χ^2 (df)	CFI	TLI	RMSEA (90% CI)	SRMR	Model comp	$\Delta\chi^2$ (Δdf)	ΔCFI	ΔTLI	$\Delta RMSEA$	$\Delta SRMR$	Decision
M0: CFA for original scale	667.01 (35)***	.99	.99	.11 (.09-.11)	.03	-	-	-	-	-	-	-
M1: Configural Invariance	689.18 (140)***	.99	.99	.10 (.09-.11)	.03	-	-	-	-	-	-	Accept
M2: Metric Invariance	498.21 (167)***	.99	.99	.07 (.06-.08)	.04	M1	190.97 (27)***	0	0	.03	.01	Accept
M3: Scalar Invariance	714.88 (224)***	.99	.99	.08 (.07-.08)	.03	M2	216.67 (57)***	0	0	.01	.01	Accept

Note. $N = 1,510$; White = 308; Black = 702; Hispanic = 339; Multiracial = 161.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$.

Table 5. Tests of Measurement Invariance by Race for Effortful Control

Model	χ^2 (df)	CFI	TLI	RMSEA (90% CI)	SRMR	Model comp	$\Delta\chi^2$ (Δdf)	ΔCFI	ΔTLI	$\Delta RMSEA$	$\Delta SRMR$	Decision
M0: CFA for original scale	52.65 (5)***	.98	.95	.06 (.05-.08)	.04	-	-	-	-	-	-	-
M1: Configural Invariance	54.89 (20)***	.98	.96	.05 (.04-.07)	.04	-	-	-	-	-	-	Accept
M2: Metric Invariance	69.14 (32)***	.98	.97	.05 (.03-.06)	.05	M1	14.25 (12)	0	.01	0	.01	Accept
M3: Scalar Invariance	113.30 (44)***	.96	.97	.05 (.04-.06)	.05	M2	44.16 (12)***	.02	0	0	0	Accept

Note. $N = 2,295$; White = 385; Black = 1,111; Hispanic = 549; Multiracial = 250.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$.

Table 6. Tests of Measurement Invariance by Bi-racial Groups for Self-Control

Model	χ^2 (df)	CFI	TLI	RMSEA (90% CI)	SRMR	Model comp	$\Delta\chi^2$ (Δdf)	ΔCFI	ΔTLI	$\Delta RMSEA$	$\Delta SRMR$	Decision
M0: CFA for original scale	667.01 (35)***	.99	.99	.11 (.09-.11)	.03	-	-	-	-	-	-	-
M1: Configural Invariance	507.51 (70)***	.99	.99	.10 (.09-.11)	.03	-	-	-	-	-	-	Accept
M2: Metric Invariance	380.39 (79)***	.99	.99	.08 (.07-.09)	.03	M1	127.12 (9)***	0	0	.02	0	Accept
M3: Scalar Invariance	444.23 (98)***	.99	.99	.08 (.07-.08)	.03	M2	63.84 (19)***	0	0	0	0	Accept

Note. $N = 1,202$; Minority-Minority couples = 1,102; White-Minority couples = 100.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$.

Table 7. Tests of Measurement Invariance by Bi-racial for Effortful Control

Model	χ^2 (df)	CFI	TLI	RMSEA (90% CI)	SRMR	Model comp	$\Delta\chi^2$ (Δdf)	ΔCFI	ΔTLI	$\Delta RMSEA$	$\Delta SRMR$	Decision
M0: CFA for original scale	52.65 (5)***	.98	.95	.06 (.05-.08)	.04	-	-	-	-	-	-	-
M1: Configural Invariance	45.74 (10)***	.98	.95	.06 (.04-.08)	.05	-	-	-	-	-	-	Accept
M2: Metric Invariance	48.45 (14)***	.98	.97	.05 (.04-.07)	.05	M1	2.71 (9)	0	.02	.01	0	Accept
M3: Scalar Invariance	55.99 (18)***	.97	.97	.05 (.03-.06)	.05	M2	7.54 (4)	.01	0	0	0	Accept

Note. $N = 1,910$; Minority-Minority couples = 1,767; White-Minority couples = 143.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$.

Appendix A

Original Adverse Childhood Experiences Questionnaire

RESPONSE FORMAT:

1 = Yes

0 = No

Childhood Abuse**Psychological***(Did a parent or other adult in the household...)*

Often or very often swear at, insult, or put you down?

Often or very often act in a way that made you afraid that you would be physically hurt?

Physical*(Did a parent or other adult in the household...)*

Often or very often push, grab, shove, or slap you?

Often or very often hit you so hard that you had marks or were injured?

Sexual*(Did an adult or person at least 5 years older ever...)*

Touch or fondle you in a sexual way?

Have you touch their body in a sexual way?

Attempt oral, anal, or vaginal intercourse with you?

Actually have oral, anal, or vaginal intercourse with you?

Household Dysfunction**Substance abuse**

Live with anyone who was a problem drinker or alcoholic?

Live with anyone who used street drugs?

Mental illness

Was a household member depressed or mentally ill?

Did a household member attempt suicide?

Mother treated violently*Was your mother (or stepmother)*

Sometimes, often, or very often pushed, grabbed, slapped, or had something thrown at her?

Sometimes, often, or very often kicked, bitten, hit with a fist, or hit with something hard?

Ever repeatedly hit over at least a few minutes?

Ever threatened with, or hurt by, a knife or gun?

Criminal behavior in household

Did a household member go to prison?

Appendix B

Parental Substance Use Questions

Alcohol Use

1. In the past twelve months, was there ever a time when your drinking or being hung over interfered with your work at school, a job, or at home?
 - a. Yes
 - b. No

2. In the past twelve months, how often did you have four or more drinks in one day? Was it...
 - a. Every day or almost every day
 - b. A few times a week
 - c. A few times a month
 - d. About once a month, or
 - e. Less than once a month?

3. What is the largest number of drinks you had in any single day during the past twelve months?
 - a. None
 - b. 1-3
 - c. 4-10
 - d. 11-20
 - e. More than 20

Drug use

1. During the past twelve months did you use...
 - a. Sedatives, including either barbiturates or sleeping pills on your own?
 - b. Tranquilizers or “nerve pills” on your own?
 - c. Amphetamines or other stimulants on your own?
 - d. Analgesics or other prescription painkillers on you own?
 - e. Inhalants that you sniff or breathe to get high or to feel good?
 - f. Marijuana or hashish?
 - g. Cocaine or crack or freebase?
 - h. LSD or other hallucinogens?
 - i. Heroin?

2. In the past twelve months, how often did you use any of those drugs? Was it...
 - a. Every day or almost every day
 - b. A few times a week
 - c. A few times a month
 - d. About once a month, or
 - e. Less than once a month?

3. In the past twelve months did you use of any of those drugs ever interfere with your work at school, a job, or at home?
 - a. Yes
 - b. No

Appendix C

Child Maltreatment and Neglect Questions

RESPONSE FORMAT:

- 0 = Never
- 1 = Once
- 2 = 3-5 times
- 3 = 6-10 times
- 4 = 11-20 times
- 5 = More than 20 times
- 6 = Yes but not in the past year

Neglect

1. Had to leave your child home alone, even when you thought some adult should be with him/her.
2. Were so caught up with your own problems that you were not able to show or tell your child that you loved him/her.
3. Were not able to make sure your child got the food he/she needed.
4. Were not able to make sure your child got to a doctor or hospital when he/she needed it.
5. Were so drunk or high that you had a problem taking care of your child.

Physical Assault

1. Spanked him/her on the bottom with your bare hand.
2. Hit him/her on the bottom with something like a belt, hairbrush, a stick or some other hard object.
3. Slapped him/her on the hand, arm or leg.
4. Pinched him/her.
5. Shook him/her.

Psychological Aggression

1. Shouted, yelled, or screamed at him/her.
2. Threatened to spank or hit him/her but did not actually do it.
3. Swore or cursed at him/her.
4. Called him/her dumb or lazy or some other name like that.
5. Said you would send him/her away or kick him/her out of the house.

Appendix D

Economic Hardship Questions

RESPONSE FORMAT:

1 = Yes

0 = No

1. In the past twelve months, did you do any of the following because there wasn't enough money?
 - a. In the past twelve months, did you receive free food or meals?
 - b. (In the past twelve months,) Were you ever hungry, but didn't eat because you couldn't afford enough food}?
 - c. (In the past twelve months,) Did you not pay the full amount of rent or mortgage payments?
 - d. (In the past twelve months,) Were you evicted from your home or apartment for not paying the rent or mortgage?
 - e. (In the past twelve months,) Did you not pay the full amount of a gas, oil, or electricity bill?
 - f. (In the past twelve months,) Was your gas or electric service ever turned off, or the heating oil company did not deliver oil, because there wasn't enough money to pay the bills
 - g. (In the past twelve months,) Did you borrow money from friends or family to help pay bills?
 - h. (In the past twelve months,) Did you move in with other people even for a little while because of financial problems
 - i. (In the past twelve months,) Did you stay at a shelter, in an abandoned building, an automobile or any other place not meant for regular housing, even for one night?
 - j. (In the past twelve months,) Was there anyone in your household who needed to see a doctor or go to the hospital but couldn't go because of the cost?

Appendix E

Self-Regulation Questions

Effortful Control

RESPONSE FORMAT:

- 0 = Never
- 1 = Rarely
- 2 = Sometimes
- 3 = Often

1. I stay with a task until I solve it.
2. Even when I task is difficult, I want to solve it anyway.
3. I keep my things orderly.
4. I try to do my best on all my work.
5. When I start something, I follow it through to the end.

Self-Control

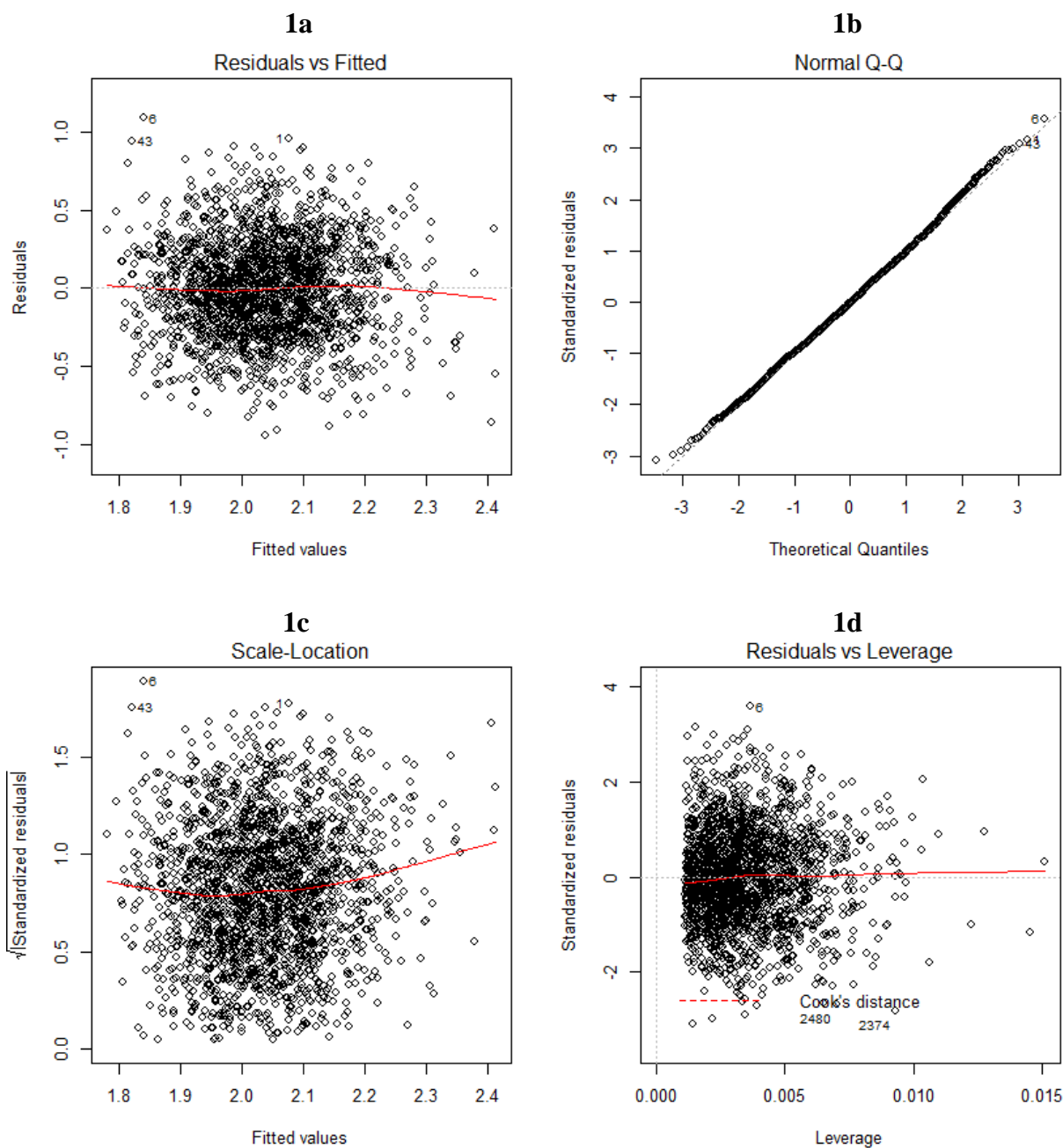
RESPONSE FORMAT:

- 0 = Never
- 1 = Sometimes
- 2 = Often
- 3 = Very often

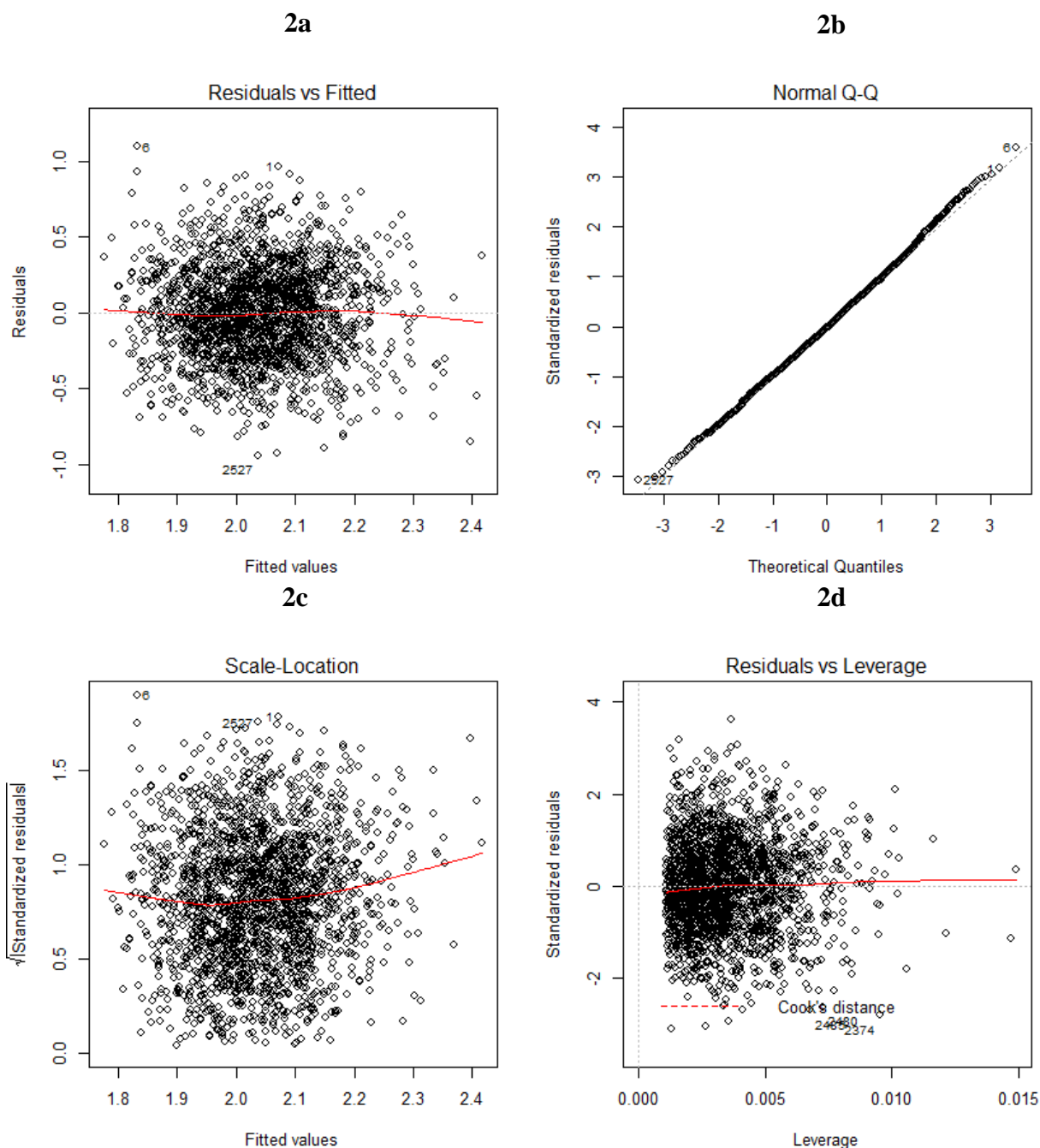
1. Controls temper in conflict situations with peers.
2. Compromises in conflict situations by changing own ideas to reach agreement.
3. Responds appropriately to peer pressure.
4. Responds appropriately to teasing by peers.
5. Controls temper in conflict situations with adults.
6. Receives criticism well.
7. Accepts peers' ideas for group activities.
8. Cooperates with peers without prompting.
9. Responds appropriately when pushed or hit by other children.
10. Gets along with people who are different.

Appendix F

Assumption Checking for Multiple Linear Regression Models



Note. Test of model assumptions for the linear model assessing the association between traditional ACEs and child telomere length, adjusting for hypothesized covariates. Graph 1a demonstrates no violation of the assumption of linearity (i.e., straight red line); graph 1b demonstrates a normal distribution of residuals (i.e., minimal deviation from the diagonal); graph 1c demonstrates homoscedasticity of residuals (i.e., no pattern to the residuals); and graph 1d demonstrates no significant impact of specific cases (i.e., no cases far beyond Cook's distance).



Note. Test of model assumptions for the linear model assessing the association between updated ACEs and child telomere length, adjusting for hypothesized covariates. Graph 2a demonstrates no violation of the assumption of linearity (i.e., straight red line); graph 2b demonstrates a normal distribution of residuals (i.e., minimal deviation from the diagonal); graph 2c demonstrates homoscedasticity of residuals (i.e., no pattern to the residuals); and graph 2d demonstrates no significant impact of specific cases (i.e., no cases far beyond Cook's distance).