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Childhood Personality, Betrayal Trauma, and Leukocyte Telomere Length in Adulthood: A Lifespan Perspective on Conscientiousness and Betrayal Traumas as Predictors of a Biomarker of Cellular Aging

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Abstract

Conscientiousness is associated with longevity. As such, identifying the biological pathways linking personality to mortality is important. This study employs longitudinal data spanning >40 years to test prospective associations with Leukocyte Telomere Length (LTL), a potential marker of cellular aging. Because telomeres shorten over time, and are sensitive to oxidative stress, shorter LTL may reflect cumulative damage associated with negative health behaviors and past stressful events. We investigated childhood conscientiousness as a protective factor, expecting an association with longer LTL in adulthood, possibly reflecting slower LTL shortening. Potential lifespan pathways involving childhood trauma, smoking behaviors, and Body Mass Index (BMI) were explored. Childhood conscientiousness showed a small raw association with LTL (r = .08, p = .04), although this effect did not persist when controlling for age and sex. Despite this lack of a direct effect on LTL, we detected an indirect effect operating jointly through BMI and smoking. Higher rates of childhood betrayal trauma were associated with shorter LTL. Contrary to our hypothesis that conscientiousness would buffer this effect, we found evidence for an interaction with childhood betrayal traumas where the association between childhood betrayal traumas and LTL was larger for those higher on conscientiousness in childhood.

Keywords

Childhood traumatic experiences; childhood conscientiousness; personality-health mechanisms; brief betrayal-trauma survey; leukocyte telomere length; personality health hypothesis

Introduction

When the association between conscientiousness and mortality was first described using lifespan data in the Terman sample (Friedman et al., 1993), a new field focused on explaining possible mechanisms for the association between personality and mortality emerged. In the intervening years, the association between conscientiousness and mortality has been replicated many times over, and is now well established (Friedman & Kern, 2014). While mortality studies have provided the impetus to focus research on personality and health processes, objective health outcomes observable decades before mortality are needed in order to move the field forward. If conscientiousness is causally related to mortality, there must be mechanisms to explain this association, and these mechanisms must be observable in biological systems and corresponding health states. We refer to this development as the personality health hypothesis. Support for this hypothesis was recently demonstrated by Hampson, Edmonds, Goldberg, Dubanoski, and Hillier (2015) using data from the Hawaii Personality and Health Cohort. Childhood conscientiousness was associated with adult health status as assessed using an index of seven objective markers of health in a sample with average age of 51 years. This association was mediated through cumulative health behaviors modeled over the lifespan. Although conscientiousness is moderately stable over 40 years (r = .25; Edmonds, Goldberg, Hampson, & Barckley, 2013) in this sample, the association between the childhood trait and adult physical health was not mediated by adult conscientiousness, showing an independent association with adult health.

One challenge for research on personality and health is the selection and implementation of valid measures of physical health. While self-reported health is widely used due to its low cost, face validity, and predictive validity with respect to mortality (Idler & Benyamini 1997; Ganna & Ingelsson, 2015), these measures provide little in the way of explanatory biological pathways. Indices that sum objective markers of health have the advantages of aggregating small effects across multiple biomarkers, and of representing health as a multivariate construct (e.g., allostatic load; McEwan & Seeman, 1999; metabolic syndrome; Alberti, Zimmet, & Shaw, 2005). Despite these advantages, broad indices of physical health require complex assessments, increasing the cost and complexity of data collection. Aggregates may also obscure specific pathways and mechanisms implicated in health processes. Differences in global health indices employed across studies additionally complicate replication. For example, work in the Dunedin study linking childhood self-control to adult health (Moffitt et al. 2011) assessed adult health in young adults, and perhaps because of this, used an index of five objective health markers distinct from those used by Hampson et al. (2015).

Mortality is an unequivocal and reliable outcome and because of this mortality studies are regarded by many as the gold standard in personality health research (Friedman, Kern, Hampson, & Duckworth, 2014). However researchers cannot rely on mortality studies to elucidate pathways within a time frame that is sufficiently rapid for the research to provide a public benefit. In order to accelerate the pace of research on the personality health hypothesis, it is therefore necessary to identify and test novel objective markers of health. Ideally, these biomarkers could be assessed easily, operate as surrogate endpoints for mortality in relatively healthy samples, and be readily implemented in future replications.

Telomere length is a promising biomarker that may suit these aims well. Telomeres are repetitive nucleoprotein sequences that cap the ends of linear chromosomes, preventing chromosome ends from fraying, and preventing end to end fusion (Blackburn, 2000). With each cell division, telomeres shorten (Blackburn, 2001). Telomeres are also sensitive to damage from oxidative stress and inflammation (O'Donovan et al., 2011). When telomeres are reduced in length below a critical threshold, this can trigger cell senescence and cell death, which contribute to aging (Harley, Vaziri, Counter, & Allsopp, 1992). This has led some to view telomere length as a metric for biological age. The best evidence to date shows age-associated declines in LTL across the lifespan, with a possible leveling off or slight increase appearing past age 75 (Lapham et al., 2015). Leukocyte telomere length (LTL) can be assayed from cells in peripheral blood, and in many studies is prospectively associated with mortality (Epel et al., 2009; Fitzpatrick et al., 2011; Weisher et al 2013; Needham et al., 2015; Rode, Nordestgaard, & Bojesen, 2015). However, associations with mortality have not been consistently observed (Mather, Jorm, Parslow, & Christensen, 2011; Svensson et al., 2014). Because of this, LTL is perhaps best characterized as a marker of cumulative cellular insults (Der et al., 2012).

Many health behaviors and environmental factors associated with health are also associated with LTL. Both refraining from smoking and maintaining a healthy weight are associated with having longer LTL in adulthood (Strandberg et al., 2011; Valdes et al., 2005). Indicators of socioeconomic status are associated with LTL, such that those with higher levels of educational attainment tend to have longer LTL (Adler et al., 2013). Consistent with work showing associations between adverse experiences in childhood and poorer physical health in adulthood (Edwards, Holden, Felitti, & Anda, 2003; Felitti et al., 1998), stressful and traumatic experiences in childhood have also been linked to shorter LTL in multiple studies (Tyrka et al., 2010; O'Donovan, 2011; Shalev et al., 2013). However, it should be noted that associations between traumatic experiences in childhood and LTL have not been supported across the literature (Mason, Prescott, Tworoger, DeVivo, & Rich-Edwards, 2015). The source of the observed inconsistency in results is unclear, although this may stem from broad differences in how childhood traumatic and stressful events are conceptualized and measured. Positioned at the nexus of social, behavioral, and biological indicators of health, LTL has been referred to as a psychobiomarker (Epel, 2009), and offers the potential to operate as an integrative outcome across multiple domains. However, this promising biomarker has received limited attention from lifespan research on personality and health.

Very few studies have considered associations between dispositional traits and LTL. Following evidence that stressful life events are associated with shorter LTL (Tyrka, et al., 2010), O'Donovan et al. (2012) tested for an association between neuroticism and shorter LTL cross-sectionally in adulthood, but found no association. Recent work using data from the Helsinki Birth Cohort Study found no cross-sectional association between Big Five traits and LTL in elderly individuals, although follow up analyses did show a dis-ordinal interaction of agreeableness by gender (Savolainen, Eriksson, Kajantie, Pesonen & Räikkönen, 2015). One study has shown a cross-sectional association between higher conscientiousness and longer LTL in a sample of young adult Japanese students (Sadahiro et al, 2015). Similar to much of the existing research on LTL, these studies all tested associations cross-sectionally at one time point.

Conscientiousness has been linked to many of the same outcomes associated with longer LTL, as individuals higher on conscientiousness are less likely to smoke, more likely to maintain healthy weight, and more likely to be physically active (Bogg & Roberts, 2004). In pilot work drawing from the Hawaii Personality and Health Cohort, we demonstrated preliminary evidence that childhood personality is prospectively associated with LTL in adulthood (Edmonds, Côté, & Hampson, *2015*). The pilot data consisted of an extreme groups design including the 30 female participants with the highest scores on conscientiousness in childhood, and the 30 female participants with the lowest scores on this trait[1]. Our pilot results provided preliminary evidence suggesting that women ranked higher in conscientiousness by their teachers in childhood are more likely to have longer LTL 40 years alter in adulthood. However, the small sample size employed did not allow for formal tests of mediation, or sophisticated modeling.

Operating as a cumulative marker of health, and offering the potential to integrate health related factors across many domains, LTL is a promising outcome for models relating personality to health. This is especially true for lifespan models relating childhood personality to physical health decades later. Evidence supports the notion that childhood personality can influence cumulative health behaviors decades later, and that these effects are both important for health outcomes, and may operate independently from adult personality (Hampson, Edmonds, Goldberg, Dubanoski, & Hillier, 2013). While clearly important, health behavior mechanisms only provide a partial account of the effects of conscientiousness on health outcomes (Turiano, Chapman, Gruenewald, & Mroczek, 2015).

A more complete account of the pathways linking personality to health must also consider pathways outside of health behavior mechanisms. Traumas can be conceptualized as events that entail intense fear, social betrayal, or a combination of these (Freyd, Klest, & Allard, 2005). Traumatic events in childhood have been linked to physical health outcomes later in life (Dong et al., 2004) and to premature mortality (Brown et al., 2009). Betrayal traumas are those perpetrated by people or institutions on whom a person is dependent. As such, betrayal traumas in children often involve damage to the trust that underlies primary caregiving relationships (Freyd, 1996). Research relating personality to health has rarely considered childhood traumatic events and their impact on health. In contrast to low betrayal traumas, betrayal traumas in particular are expected to be especially impactful for mental and physical health (Edwards, Freyd, Dube, Anda, & Felitti, 2012; Freyd, 1996; Goldsmith, Freyd, & DePrince, 2012). While measures used in previous work relating childhood trauma to LTL often include items describing betrayal trauma, retrospective reports of betrayal traumas in childhood have not been explicitly tested as correlates of LTL. Pathways linking childhood traumas to physical health outcomes, and those linking personality traits to adult health, may have additive effects or multiplicative effects. As such, traumatic events represent an important pathway or moderator contributing to understanding associations between personality and health outcomes. In the context of the personality health hypothesis, childhood conscientiousness may provide a resource for resilience, buffering associations between childhood trauma and LTL, although this hypothesis has never been tested.

[1]Although these same participants (n = 58) appear in the current sample, the LTL data used here involved new assays for LTL using those participants' stored dried blood samples.

In the present study, we used LTL assessed at average age 51 as an outcome in lifespan models designed to test the personality health hypothesis. Conscientiousness assessed in childhood at average age 10 was hypothesized to be associated with longer LTL 40 years later in adulthood. Lifespan health behaviors including smoking, and healthy weight (BMI) were expected to mediate this effect. This represents the first test of LTL as an outcome in a lifespan model of personality, heath behaviors, and health using childhood assessments of personality. Replicating and extending previous work relating exposure to childhood traumatic events to shorter LTL, we tested for an association between childhood traumas and shorter LTL in adulthood. We employed a measure of childhood trauma that distinguishes between high and low betrayal traumas. To our knowledge this is the first test of an association between high betrayal traumas in childhood and LTL in adulthood. We hypothesized that higher rates of traumas in childhood, and betrayal trauma in particular, would be associated with shorter LTL in adulthood. Because childhood conscientiousness is related to lower rates of health damaging behaviors, and those higher in childhood traumas may be more likely to engage in these (Anda et al., 1999), childhood conscientiousness may operate as a resource to buffer the effects of trauma in childhood. We additionally hypothesized that for those higher on childhood conscientiousness, associations between trauma and LTL would be smaller in magnitude.

Following work showing that socioeconomic status is associated with LTL (Adler et al., 2013; Surtees, 2012), we assessed parental home ownership as an indicator of SES of origin. In addition, children of older fathers have previously been shown to have slightly longer LTL (Kimura et al., 2008), and women tend to have slightly longer LTL when compared to men of the same age (Gardner et al., 2014). LTL has been found to vary across cultural groups (Diez-Roux, 2009; Côté et al., 2012). In our pilot work (Edmonds, Côté, & Hampson, 2015), we found evidence of differences across cultural groups, such that Native Hawaiians had the shortest LTL. Accordingly, we controlled for cultural group membership using the three groups endorsed by the largest number of participants; Japanese American, Native Hawaiian/Part Native Hawaiian, and Caucasian. In sum, our models controlled for chronological age, sex, parental home ownership, paternal age at conception, and cultural group membership.

Method

Participants

Over 2,000 children in elementary schools in Hawaii in the 1960's were comprehensively assessed on their personality characteristics by their teachers. Since 1998, 1,942 (84%) of 2,321 members of this cohort have been located (Hampson et al., 2001). Of the 1,904 located and available for recruitment, 1,396 (73%) agreed to participate in further studies.

For the present report, the sample (n = 699) comprised 366 women and 333 men who provided responses on the Brief Betrayal and Trauma Survey (BBTS; Goldberg & Freyd, 2006), and for whom dried blood spots were collected at the clinic exam. This subsample included a higher proportion of women (52%) compared to those in the adult sample who were not included in this study (44%), but was more comparable to the original child cohort (47% girls). Compared to the rest of the adult sample, the subsample included a lower

proportions of Caucasians (16% vs. 22%), somewhat fewer Native Hawaiians and part Hawaiians (20% vs. 25%), somewhat more Japanese Americans (35% vs. 29%), and more of other ethnicities (29% vs. 24%). As children, this subsample was viewed by teachers as somewhat more conscientious (measured in standard scores) than the rest of the adult sample (M = .12, SD = .99 vs. M = .00, SD = .99, $t(1380) = 2.15$, p = .03, 95% CI [.01, .22], d = .11), and they had somewhat higher levels of educational attainment on a nine-point scale (M = 6.92, SD = 1.76 vs. M = 6.56, SD = 1.99, $t(1300) = 3.41$, p = .001, 95% CI [.151, .561], d = .19). The mean age for both women and men at the time of their clinical examination was 51 years.

Measures

Timeline—Teacher assessments of child personality for this sample were obtained in 1965 or 1967 when participants were in grades 1, 2, 5 or 6 (M age = 10 years; minimum 8 years, maximum 13 years). The adult clinical examinations were conducted between 2003 and 2011 (M age = 51 years; minimum 45 years, maximum 60 years). The average interval between the child assessment and clinic visit was 41 years (minimum 36 years, maximum 51 years).Questionnaire (Q) data used in this report were drawn from surveys sent to participants over a 14-year period to complete at home and return, with one resend for non-responders: Q1 in November 1999 (1,350 returns); Q2 in August, 2002 (936 returns); Q3 in March, 2005 (801 returns); Q4 in May, 2007 (857 returns); Q5 in June, 2008 (848 returns); Q6 in June, 2013 (849 returns). The analyses reported here use data drawn from the clinic assessment, Q1, Q3, Q4, Q5, and Q6. Nearly all participants completed Q1 (95%) and Q3 (93%) prior to their clinic visit. Most participants (72%) completed Q4 after their clinic visit, with 90% doing so within three years of their clinic visit. Most participants completed Q5 (88%) and Q6 (99%) after the clinic visit, within 5.17 and 9.75 years respectively.

Childhood Big Five personality traits—Teachers provided personality assessments for all of the students in their classrooms. Assessments were conducted at the end of the school year, and each child was assessed on only one occasion. The number of attributes differed slightly between schools and ranged from 43–49, with a common core of 39 items. Definitions of each attribute, developed by focus groups of teachers, were provided (e.g., “Persevering: Keeps at his/her work until it is completed; sees a job through despite difficulties, painstaking and thorough”). For each item, teachers used a rating form with the full item at the top of the page, and a series of boxes designed to approximate a quasi-normal distribution. Teachers rank-ordered all the children in their classroom by entering their names into boxes, thus placing them in this distribution. This method of assessment resembles a Q-sort, except that students were ranked for each item, rather than items being sorted for each target. Because students’ personality attributes were fit in a fixed distribution within each classroom, personality assessments were effectively standardized within classroom having a mean of zero. This has the advantage of controlling for and reducing some sources of systematic rater bias across teachers, and also effectively controls for mean level differences associated with age in childhood. The Big Five personality traits were recovered in analyses of the original childhood cohort, and orthogonal factor scores for each of the Big Five (Extraversion, Agreeableness, Conscientiousness, Emotional Stability, and Openness or Intellect/Imagination) were derived for each participant using all available

items in each sample (see Goldberg, 2001, for further details). Alpha reliabilities range from .60 to .75 (Edmonds et al., 2013). The child Big Five factor scores have demonstrated predictive validity with respect to adult personality (Hampson & Goldberg, 2006) and consequential outcomes in adulthood (Hampson et al., 2015; Edmonds et al, 2015; Woods & Hampson, 2010).

Big Five personality in adulthood—Self-ratings of adult personality were collected as part of Q1. Participants completed the 44-item Big Five Inventory (BFI; John & Srivastava, 1999) using a 5-point scale, ranging from 1 (*very inaccurate*) to 5 (*very accurate*). Alpha reliabilities ranged from .79 to .84 (Edmonds et al., 2013).

Trauma in childhood and adulthood—The BBTS was administered in Q5. It described traumatic events in behavioral terms, and for each event the participants indicated whether they had experienced it before age 12 (child), between ages 12 and 17 (teen), and at age 18 and older (adult). Five items described a high-betrayal trauma perpetrated by "someone very close to you" that either happened to the self, or was witnessed by the self as it happened to another (e.g., "Personally witnessed someone with whom you were very close deliberately attack one of your family members so severely as to result in marks, bruises, blood, broken bones, or broken teeth"). Seven items described low-betrayal trauma perpetrated by "someone with whom you were not close" that either happened to the self, or was witnessed by the self as it happened to another (e.g., "Personally witnessed someone with whom you were not so close deliberately attack a member of your family so severely as to result in marks, bruises, blood, broken bones, or broken teeth"). Response options were "Yes," "No," "Don't know/can't remember," and "Prefer not to respond." "Yes" responses were summed separately for high and low betrayal items to form two scales, high-betrayal trauma and low-betrayal trauma, for each of the three ages (child, teen, and adult). For the present analyses, only the high-betrayal and low-betrayal scales for childhood (before the age of 12) were used.

Lifespan health behaviors—Lifetime smoking history was assessed by items from Q1 and Q4 that used standard survey questions to ask about current and past smoking histories (e.g., when they started smoking, how much they smoked, when they stopped smoking). Smoking items assessed on Q4 had the advantage of allowing for cumulative lifetime estimates of pack years. This measure captured exposure to cigarette smoking across the lifespan among both current and ex-smokers. A pack year, derived from participants' smoking histories, was defined as the equivalent of smoking one pack a day for one year. Items from Q1 and Q4 were recoded to create a five item scale (0 = ≤ 100 cigarettes, 1 = previously smoked < 8 pack-years, 2 = previously smoked ≥ 8 pack-years, 3 = currently smoke in last year ≤ ½ pack a day, 4 = currently smoke in last year > 1/2 pack a day). BMI (kg/m^2) was assessed objectively during the clinic assessment. Physical inactivity over the lifespan was assessed on Q3 asking participants to check the time periods when they "engaged in no exercise and were quite inactive," and to check the time periods when they "engaged in sports or were otherwise quite physically active." For each question, they checked as many of the following 10 periods as applied: before entering school, during elementary school, during junior high, during high school, from age 18–25, from age 26–30,

from age 31–35, from age 36–40, from age 41–45, and from age 46 onward. The sum of periods of physical inactivity minus the sum of periods of activity was calculated to create a cumulative physical inactivity score (maximum score = 10).

Adult physiological dysregulation—A composite measure of dysregulation was formed from cardiovascular and metabolic biomarkers: systolic and diastolic blood pressure (means of two assessments), total cholesterol/HDL ratio, HDL (reversed), fasting triglycerides, fasting blood glucose, and urine protein (log transformed), obesity (clinically measured BMI), and medication for cholesterol and/or blood pressure (Hampson, Goldberg, Vogt, Hillier, & Dubanoski, 2009). A total dysregulation score was derived by z-scoring each measure across men and women and summing them, with lower scores indicating better health.

Leukocyte telomere length—During the same clinic visit where the components of dysregulation were assessed, venous blood was collected from an arm draw or, for a portion of the sample from whom a venous arm draw could not be completed (n = 40), via finger prick. Blood was blotted onto paper filter (Whatman/ GE Healthcare Life Sciences; FTA Micro-cards cat# WB120210) cards designed to preserve dried blood and prevent DNA degradation. As per the manufacturer's instructions, these were air dried, after which the dried blood spots were placed in plastic bags and stored in the dark, at room temperature, within larger bags containing a desiccant. To avoid error from batch effects, all dried blood spots for the current study were shipped at controlled room temperature in one batch to the lab where they were assayed. Assays were performed randomly and blindly to all participant data. DNA was extracted from six 2mm punches from each dried blood spot using QIA Amp kit on a QIA Cube (QIAGEN). Ultimately, all undiluted DNA extracts were assayed in duplicate twice, with two different kits, using a monochromatic multiplex qPCR method adapted from that developed by Cawthon (2009), as described in Zanet et al. (2013), resulting in four LTL estimates for each participant. For both kits, the standard curve was built from a serial dilution of human genomic DNA pooled from healthy donors. All primers were at a final concentration of 0.9 μm. Two internal controls (one DNA from a human cell line, one from pooled genomic DNA) were included in each run and used for quality control. All samples were amplified successfully and met our quality control criteria. The assay produces a count for telomeric DNA (T), and for a specific single copy nuclear gene DNA (S), in our assay the human albumin gene. Typically, the average ratio (T/S) of duplicate assays is used as a measure of relative LTL.

The first set of duplicate measures was obtained using the SYBR Select kit, (ABI, now merged with Invitrogen) and the standard curve was prepared from pooled buffycoat DNA. All runs had a PCR efficiency between 91 and 98%. The inter-run coefficient of variation (CV) was <5%. The second set was assayed using the SYBR Green 1 Master kit (Roche) with EDTA added to the master mix to a final concentration of 1.2 mM. All runs had a PCR efficiency between 95 and 99%. The inter-run coefficient of variation (CV) was <3.5%.

Rather than using the average of each set of duplicate measures, we used data from each of the four runs. Using the four available T/S ratios, LTL was modeled as a latent variable and these four estimates served as indicators. This has the advantage of using all available data

on each sample, directly estimating error, and extracting maximum error free variance. Using this model, factor scores were saved to generate error free estimates of LTL. These resulting scores are standardized factor scores, with lower scores indicate shorter LTL. Previous evidence has indicated that LTL assays performed on blood taken from a finger prick can upwardly bias LTL assays in comparison to those performed on blood taken from an arm draw (Zanet et al. 2012). We tested for evidence of this by comparing LTL across finger prick and venous arm draw samples. We found no mean level difference ($t(697) = .88$, $p = .38$, $d = .09$) across these groups, and elected to include both finger prick and arm draw samples in our analysis.

Control variables—These included participant age, sex, parental home ownership in childhood, and self-selected cultural identity. Parental home ownership across the participant's lifetime was assessed on Q3. Participants indicated whether or not their parents owned their own home at each of 10 periods across the participant's life from before they went to school to after age 46. The number of periods was summed to give a measure of extent of parental home ownership up to age 12. Paternal age at participant's birth was derived from parental age, assessed in Q6. Cultural identity was assessed as part of Q1. Participants were asked to select their own cultural identity from 12 options. They were instructed to select the one cultural group that they most identified with. For the present analyses, dummy codes were generated for the three cultural groups endorsed by participants in our sample: Japanese American, Native Hawaiian/part Native Hawaiian, and Caucasian. These were used in all models to control for differences across cultural groups.

Analysis Plan

Prior to running more complex analysis, we tested raw correlations between study variables and LTL, partial correlations controlling for age and sex, and tested for cultural group differences. Following this, separate path models where constructed to test our two main hypotheses. First, we tested health behaviors as mediators of childhood conscientiousness on LTL. Tests of mediation were evaluated by estimating the indirect effects using bootstrapping. Second, we tested the interaction of childhood conscientiousness and childhood high betrayal trauma with respect to LTL. Finally, we tested the degree to which observed effects operated independently by testing a full path model including both mediation tests, and the interaction term.

Results

Sample characteristics for all study variables are presented in Table 1. The convergence between LTL latent scores and the two raw LTL assays were high. These along with raw correlations for age, sex, and childhood conscientiousness are reported in Table 2. We next tested correlations of the extracted factor scores for LTL with all of the Big Five in childhood, health behaviors, childhood traumas, and our set of control variables. As a comparison, we additionally tested for associations with physiological dysregulation. Raw correlations are reported in Table 3. At the zero order level, childhood conscientiousness was correlated with LTL such that those rated as more conscientious in childhood showed longer LTL 40 years later in adulthood ($r = .08$, $p = .04$). No other trait in childhood was associated

with LTL. None of the Big Five in adulthood showed any association with LTL. BMI was negatively correlated with LTL, although smoking and physical activity were not. High-betrayal traumas in childhood showed a significant negative correlation with LTL in adulthood. Low-betrayal traumas did not. Dysregulation showed a negative association with LTL ($r = -.09$, $p = .02$) such that those with worse health had shorter LTL. We tested for differences in LTL across the three cultural groups endorsed by the largest number of participants in our sample: Japanese American, Caucasian, and Native Hawaiian/Part Hawaiian. A one-way ANOVA showed no significant differences across cultural groups in adult LTL, $F(2, 572) = 1.646$, $p = .194$. With respect to other control variables, participants who were older, male, and those with younger fathers at conception tended to have shorter LTL.

We next evaluated whether the associations evidenced between study variables and LTL at the univariate level would be changed when controlling for age and sex. Table 3 reports partial correlations controlling for these. The effect of conscientiousness was completely attenuated ($r_{part} = .00$, $p = .91$). Effect sizes for paternal home ownership ($r_{part} = .10$, $p = .03$) and smoking ($r_{part} = -.09$, $p = .04$) increased slightly in magnitude when controlling for age and sex while the association between LTL and dysregulation was completely attenuated ($r_{part} = -.01$, $p = .90$). Other associations remained largely unchanged.

Because mediation can occur even in absence of a direct effect (Rucker, Preacher, Tormala & Petty, 2011), we proceeded to test for a significant indirect path from conscientiousness on LTL via health behaviors known to be associated with LTL. Physical activity was not included as a mediator because it did not show any association with LTL, and thus did not satisfy the basic conditions necessary for mediation. At the raw level, smoking was also not associated with LTL at the zero order level, but did show an association with LTL after controlling for age and gender. Childhood conscientiousness was associated with both smoking ($r = -.16$, $p < .01$) and BMI ($r = -.22$, $p < .01$). No other childhood Big Five traits were significant predictors of BMI or smoking, and thus did not satisfy the basic conditions for testing mediation. BMI and smoking were tested as multiple mediators of childhood conscientiousness and adult LTL. Results from the full model are reported in Table 4. In order to test the presence of an indirect effect of child conscientiousness on LTL, we created a path model in Mplus (Figure 1) which included both BMI and smoking as mediators while controlling for age, sex, paternal age, parental home ownership, cultural group membership, and adult conscientiousness. Missing data across variables were accounted for using full information maximum likelihood estimation. With the exception of the data imputation previously described for BMI, no other data imputation was employed. Estimates for specific and total indirect effects, along with 95% bias corrected confidence intervals, were generated using bootstrapped distributions in Mplus. Higher lifetime BMI ($\beta = -.08$, $p = .02$), remained a significant predictor of shorter LTL, although lifetime smoking did not ($\beta = -.07$, $p = .06$). Childhood conscientiousness showed no direct effect on LTL. Smoking did not show a significant indirect effect on LTL (indirect effect = .11, $p = .106$, 95% CI [−.002, .024]). In the case of BMI the indirect effect for childhood conscientiousness on adult LTL appeared significant based on the estimated p-value, while the 95% confidence interval on the indirect effect included zero (indirect effect = .12, $p = .049$, 95% CI [.00, .023]).

However, the total indirect effect on LTL through both BMI and smoking (indirect effect = .023, $p = .01$, 95% CI [.006, .041]) was significant.

Based on results in Table 3, childhood traumas, and betrayal traumas in particular are related to LTL. We tested the degree to which childhood conscientiousness might operate as a buffer, such that those higher in conscientiousness in childhood might show a reduced association between childhood betrayal traumas and LTL in adulthood. We tested for the hypothesized interaction using path modeling in Mplus. Table 5 reports the results from a path model predicting LTL from childhood conscientiousness, number of self-reported betrayal traumas, and their interaction term, along with age, sex, parental home ownership, and paternal age at birth. The interaction between childhood betrayal traumas and LTL was significant, although not in the hypothesized direction ($\beta = -.09$, $p = .01$). Individuals who were ranked higher on conscientiousness in childhood by their teachers showed a stronger association between the number of high-betrayal traumas experienced in childhood and shorter LTL in adulthood 40 years later. Figure 2 presents this interaction by estimating this effect while centering child conscientiousness one standard deviation above, and one standard deviation below the mean on conscientiousness. For those individuals highest on conscientiousness in childhood, the association between number of high-betrayal traumas in childhood and LTL in adulthood was estimated at $\beta = .18$, $p < .001$. For those one standard deviation below the mean in conscientiousness, this effect was non-significant, $\beta = .01$, $p = .899$.

We next modeled a full path model, including both the mediated health behavior pathway, and the interaction reported above. This allowed us to evaluate the degree to which the observed interaction operated independently from the mediation effect described above. Age, sex, parental home ownership, paternal age at conception, adult conscientiousness and cultural group membership were included as control variables. Standardized indirect effects along with 95% bias corrected confidence intervals were estimated using bootstrapping. In the full model, the magnitudes of both the interaction and the mediation effects were estimated at similar levels to what had been observed in the simpler models, indicating that these effects operate on LTL as independent pathways. Results of the full model are reported in Table 6.

Discussion

We found evidence that higher childhood conscientiousness was associated with lower rates of smoking, and healthier cumulative BMI over the following 40 years. In addition, having more high-betrayal traumas in childhood was associated with shorter adult LTL while low-betrayal traumas showed no association with LTL. These findings are consistent with previous work demonstrating that stressful childhood events (Tyrka et al., 2010; O'Donovan et al., 2011; Shalev et al., 2013), as well as smoking, and BMI (Strandberg et al., 2011; Valdes et al., 2005) are associated with LTL. The association between high-betrayal traumas was on par with the observed effects for both BMI and smoking. We additionally found that childhood conscientiousness showed a small indirect effect via smoking and BMI on LTL 40 years later in adulthood. The mediation path estimate is necessarily smaller in magnitude than effects observed for smoking and BMI. In contrast to our hypothesized model,

childhood conscientiousness did not exhibit a substantial direct effect on LTL 40 years later. When testing for an interaction between childhood conscientiousness and LTL, we observed a significant interaction between childhood conscientiousness and high-betrayal traumas. However this was not the buffering effect we hypothesized. Rather than reducing the association between high-betrayal traumas and LTL, higher levels of conscientiousness in childhood were associated with an increased effect where higher numbers of high-betrayal traumas were associated with shorter LTL 40 years later. For those high in conscientiousness, the observed effect size for high-betrayal traumas was larger than that observed for BMI or smoking. For those highest in conscientiousness and reporting the most betrayal-traumas, our model predicts their LTL at roughly .85 standard deviations bellow the mean for the sample. Based on the raw regression coefficient for age predicting LTL in our sample ($\beta = .15$), this represents an average of roughly 5.7 years of LTL shortening. While large in apparent magnitude, it is important to note that this estimate may not generalize to other samples.

To our knowledge this is the first time betrayal traumas in childhood have been related to LTL. Previously these effects had not been examined in the context of lifespan personality-health models. Consistent with the personality health hypothesis, childhood conscientiousness operated though smoking and BMI, such that individuals higher on childhood conscientiousness were more likely to exhibit beneficial health behaviors, showing an observable association with LTL in adulthood. This piece of our final model replicated previous work (Hampson et al., 2015) in this same sample testing physiological dysregulation as a health outcome. In contrast to those models, the association with conscientiousness via the observed indirect path was considerably smaller. Additionally, there was no direct effect from childhood contentiousness to LTL, in contrast to models using dysregulation as the outcome. It is interesting to note that in the current sample, dysregulation had a small zero order association with LTL, and this was completely attenuated when controlling age and sex. In as much LTL and dysregulation both act as summary measures of overall health, we might expect them to operate similarly. However, as they appear largely unrelated, these cannot be viewed as interchangeable outcomes, and may have additive effects with respect to overall health.

Our hypothesis that childhood conscientiousness would be protective was founded on the notion that conscientiousness would provide resources that would be beneficial in the face of severe trauma. Because betrayal traumas are perpetrated by agents upon whom children are dependent, betrayal traumas are more likely than other stressful events to overwhelm coping resources. Children have little autonomy to escape or correct the conditions that expose them to betrayal traumas. In response to stressful life events, individuals higher on conscientiousness are more likely to engage in problem-focused coping (Connor-Smith & Flachsbart, 2007). When applied in response to an uncontrollable stressor, problem-focused coping can be unproductive, and can lead to exhaustion of resources and negative outcomes (Compas et al., 2001). Betrayal traumas by their very nature are unlikely to be resolved through problem-focused coping. Because this effect appeared to be independent of health behaviors, it may represent a negative direct physiological consequence (shorter LTL) of applying problem-focused coping in response to events that were out of children's control, and the experience of stress resulting from this. In support of this possible pathway, recent

research has found traumatic stress to be associated with inflammation (O'Donovan, Neylan, Metzler, & Cohen, 2012), which in turn is associated with shorter LTL (O'Donovan et al., 2011). Additionally, higher rates of epigenetic aging have been found in young adults in disadvantaged environments, and this effect was stronger for those higher in self-control (Miller, Yu, Chen & Brody, 2015). While the current study differed from that work in many ways, both studies demonstrate similar outcomes in individuals coping with uncontrollable stressors. In both cases, these results parallel research relating control orientations normally viewed as beneficial to negative outcomes in situations of low control (Lachman, Neupert, & Agrigoroaei, 2011; Heidemeier, & Göritz, 2013).

Conscientiousness has many beneficial effects associated with health outcomes, operating through multiple pathways, and at multiple points across the lifespan. The summation of these multiple pathways likely accounts for the now well-established and consistent result that higher conscientiousness is associated with greater longevity (Kern & Friedman, 2008). The negative association observed for those individuals with greater number of high-betrayal traumas represents a rare instance where high conscientiousness is associated with a health outcome in a detrimental way. When viewed in the large context of the personality health hypothesis, an interaction of this nature, to the degree that it has consequences for longevity, has the potential to suppress the observed association between conscientiousness and longevity. If this is the case, estimates of the degree to which conscientiousness is associated with longevity may be underestimated as long as they do not account for rates of childhood traumas.

In these data, adult Big Five traits showed no association with LTL. This replicates the recent work of Savolainen et al., (2015), and the null effect for adult neuroticism reported by O'Donovan et al. (2012). Our results however are not consistent with those reported by Sadahiro et al. (2015). This may have resulted from the differences in age in our sample, (*M* age = 51) versus that employed by Sadahiro et al. (*M* age = 23). Ideally, future studies will test cross sectional associations between Big Five traits, and LTL will be tested in multiple samples at a variety of ages. Overall, the lack of effects associated with adult traits supports the importance of childhood personality and conscientiousness in particular for understanding associations between personality and health.

In addition to physical health benefits, higher conscientiousness is associated with beneficial outcomes in many domains of life, including higher rates of education, career attainment, marital stability, and income (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007; Shiner & Masten, 2012). Increases in conscientiousness associated with educational environments are viewed as an important developmental pathway leading to many beneficial outcomes in adulthood, and there is increasing interest in educational interventions aimed at increasing conscientiousness in childhood (Heckman & Kautz, 2012). We consider research on early environments that support positive trait development to be especially valuable. If the interaction observed here can be replicated across samples, this would suggest that interventions targeted at increasing conscientiousness in children could have negative consequences for children exposed to high-betrayal traumas. Consistent with developmental perspectives calling for a broad perspective on the causes and outcomes of developmental

processes (Conti & Heckman, 2013), interventions to increase conscientiousness may need to consider trauma exposures and trait development jointly.

The current study has specific limitations. Our measures of trauma are retrospective, and do not provide specific details such as the duration or frequency of specific types of traumatic events. Ideally, this information could be used to test more detailed hypotheses about the pathways and mechanisms linking childhood betrayal trauma to LTL. Similarly, having only one assessment of LTL in adulthood, we cannot test whether the observed shortening in LTL occurred in childhood proximal in time to the traumas reported on, or if we are observing the result of a cascade of effects following childhood traumas accumulating over decades. Childhood is a time of relatively rapid shortening in LTL (Côté et al., 2012) and childhood trauma exposure may be an important environmental factor accounting for individual differences in rates of LTL change. Lacking more data points, it is similarly impossible to determine whether increases in BMI preceded LTL, or if it is the reverse. Investigating such mechanisms should be a goal of future work. Our assessments of smoking and physical activity were also retrospective. This may have reduced the accuracy of these assessments, which would have attenuated our observed effects. The timing of questionnaires in adulthood also varied with respect to the clinic visit. Average age of quitting in the sample was 33 years of age, with nearly all (99%) of former smokers reporting quitting prior to age 39. Clinic assessments were not conducted until much later. But, the questionnaire assessing smoking was assessed at different times with respect to the clinic across participants. This introduces the possibility of error in estimating smoking quantity for current smokers proximal to their blood samples being collected. These sources of error may have attenuated the estimated association between smoking and LTL.

The final model we present provides a rich and complex picture. The mediation path via health behaviors highlights long lasting benefits associated with childhood conscientiousness with respect to LTL, an objective marker of cellular aging. This effect is tempered somewhat by the observed negative effect exerted by the interaction of conscientiousness and trauma on LTL. As noted above, the health behavior mediation path replicates previous work in the Hawaii cohort using both self-rated health (Hampson, Goldberg, Vogt, & Dubanoski, 2006) and dysregulation as health outcomes (Hampson et al., 2015). After running these models, we tested the interaction of childhood conscientiousness and childhood high-betrayal trauma with respect to dysregulation. In this case, the interaction was not significant. This suggests that dysregulation and LTL are subject to different mechanisms, and that each registers specific health processes, thus indicating that there is much we can still learn by incorporating specific objective health outcomes into lifespan models of personality and health.

In the context of the personality health hypothesis, this work highlights the need to assess physical health using multiple objective markers. Health is complex, and no single biomarker is likely to provide a comprehensive view of overall health status. Our results support the notion that health behaviors link childhood personality and adult health. They also highlight the importance of including other health relevant factors, such as childhood trauma, in lifespan models of personality and health. As an outcome, LTL may operate as

integrative biomarker that may provide novel biological pathways in support of the personality health hypothesis.

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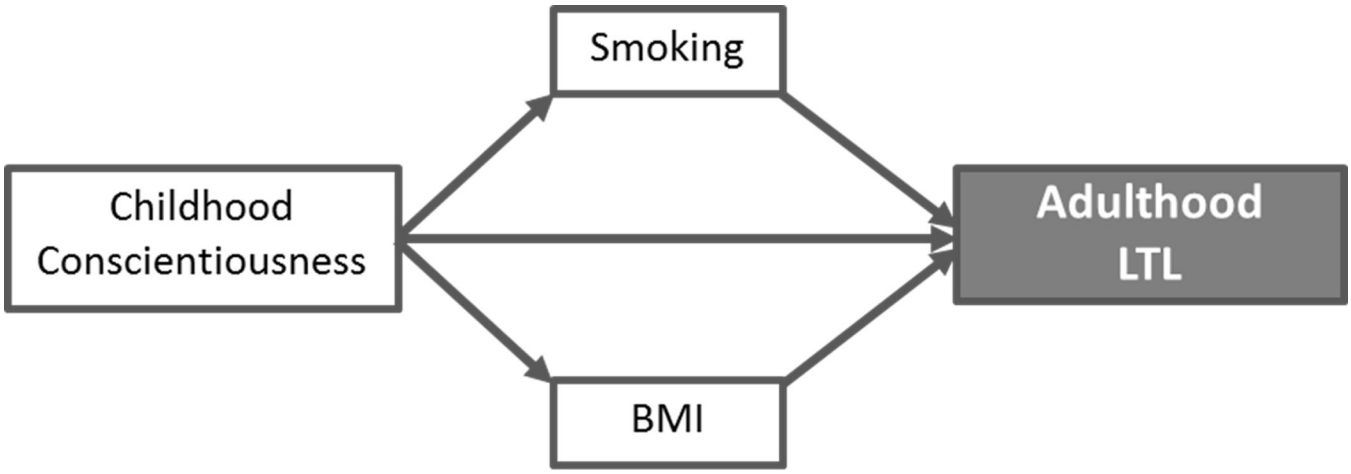

figure 1.

Path model showing BMI and Smoking as multiple mediators linking childhood conscientiousness and LTL assessed in adulthood. Indirect effects and bias-corrected 95% confidence intervals for each mediator and the total indirect effect were estimated using bootstrapped resampling in Mplus.

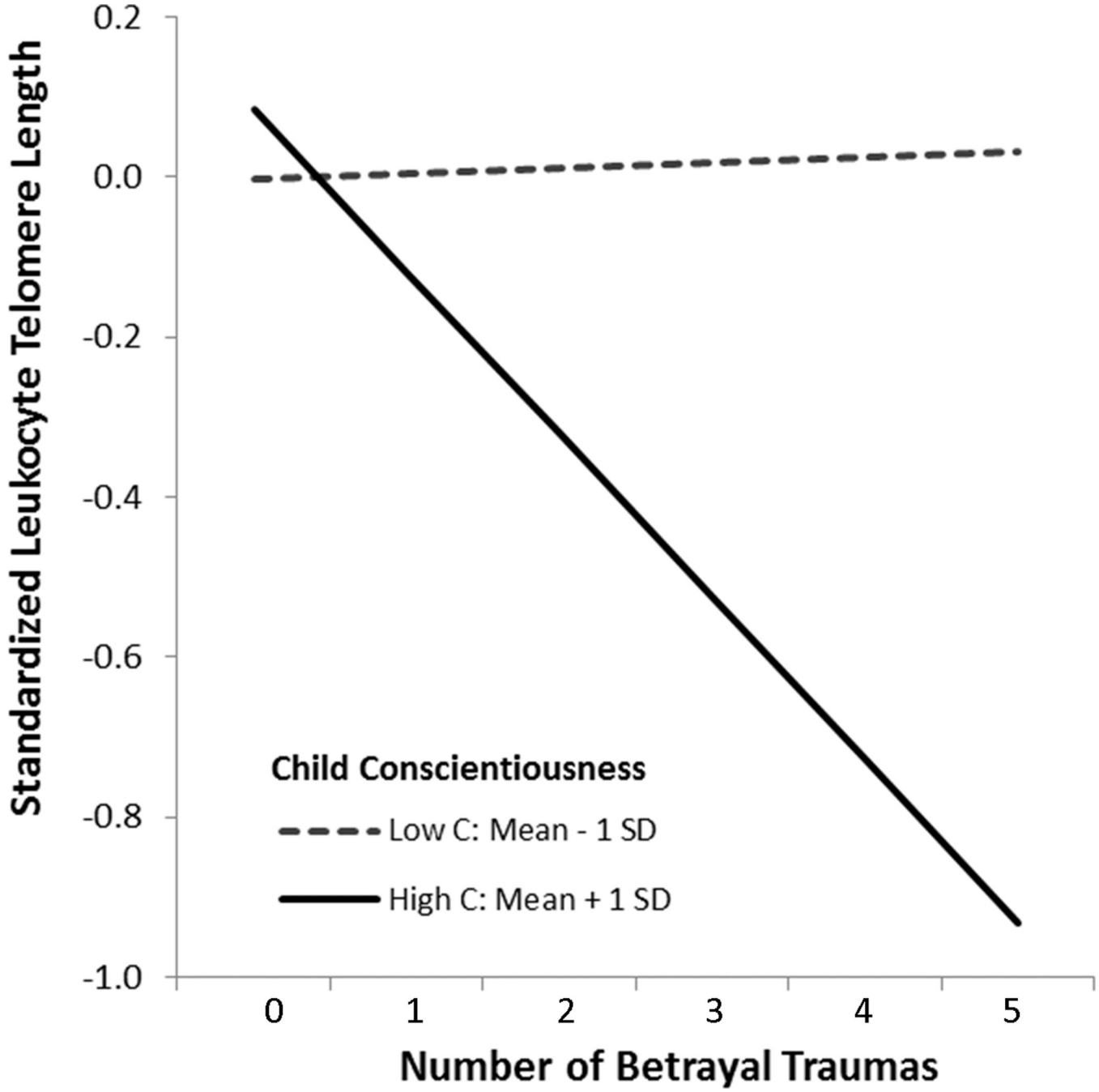

figure 2.

Interaction between child conscientiousness and betrayal traumas predicting LTL in adulthood. Effects were estimated using path modeling in Mplus using maximum likelihood estimation. The association between leukocyte telomere length (LTL) and childhood betrayal traumas is shown at two levels of childhood conscientiousness. These effects were estimated by re-centering childhood conscientiousness at 1 standard deviation above and 1 standard deviation below the mean. Control variables include age, sex, parental home ownership, paternal age at birth, adult conscientiousness, and cultural identity. Leukocyte telomere

length is relative LTL, measured as a ratio of telomere DNA copy number to a single gene copy number (T/S), and LTL scores are standardized factor scores.

Table 1

Sample characteristics.

Characteristic	Frequency (%) or Mean (SD)
Cultural Identity	
European American	109 (16%)
Japanese American	248 (35%)
Hawaiian American	133 (20%)
Other	196 (27%)
Missing	13 (2%)
Sex (female)	366 (52%)
Age at Adult Assessment (years)	51 (2.86)
Physiological Dysregulation[1]	−.19 (.49)
Parental Home Ownership[2]	1.04 (.81)
BMI	29.02 (6.77)
Smoking[3]	1.05 (1.36)
Physical Activity[4]	3.64 (3.55)
Adult Conscientiousness[5]	−.06 (.99)
Child Conscientiousness	.12 (.99)
LTL Roche[6]	9.22 (2.73)
LTL Invitrogen	9.84 (.69)
Betrayal Trauma	.42 (.89)
None	386 (55.2 %)
One	76 (10.9%)
Two	25 (3.6%)
Three	15 (2.1%)
Four	8 (1.1%)
Five or more	2 (0.3%)

[1] Physiological Dysregulation reported in z scores; higher scores indicate poorer health

[2] Parental home ownership reported for two stages in childhood prior to age 12

[3] Smoking was coded into five categories (0–4) based on life-time smoking history where 0 = ≤ 100 cigarettes, 1 = previously smoked < 8 Pack-years, 2 = previously smoked ≥ 8 pack years, 3 = current smoker at ≤ ½ pack a day, 4 = current smoker > ½ pack a day

[4] Physical activity is a weighted sum of three items assessing number of instances of mild, moderate, and strenuous exercise in a typical week over the previous year

[5] Adult conscientiousness measured using BFI

[6] Leukocyte telomere length is relative LTL, measured as a ratio of telomere DNA copy number to a single gene copy number (T/S)

Table 2

Correlations between Raw and Latent Leukocyte Telomere Length (LTL), Age, Gender, and Child Conscientiousness

	LTL Latent Score	LTL via Roche	LTL via Invitrogen
Age	**−.43**	**−.56**	**−.15**
Sex	**−.14**	**−.11**	**−.14**
Child Conscientiousness	**.08**	.07	.07
LTL Latent Score	-	**.89**	**.83**

Note. n = 699

Leukocyte telomere length is relative LTL, measured as a ratio of telomere DNA copy number to a single gene copy number (T/S)

Correlations in bold are significant at the $p < .05$ level

Table 3

Correlations Between Leukocyte Telomere Length (LTL) and Study Variables

	r	p	r_{part}	p	n
Background Variables					
Age	−.**43**	.00			699
Sex	−.**14**	.00			699
Paternal age at birth	.**13**	.01	.**13**	.01	398
Paternal home ownership	−.08	.10	.**10**	.03	480
Japanese American	.**10**	.01	.03	.47	699
Caucasian	−.01	.80	.06	.12	699
Hawaiian/Part Hawaiian	−.24	.52	.00	.92	699
Health Behaviors					
Smoking	−.07	.12	−.**09**	.04	516
BMI	−.**12**	.01	−.**10**	.02	510
Physical activity	−.05	.25	.01	.90	480
Childhood Personality					
Extraversion	−.07	.05	−.04	.29	699
Agreeableness	.05	.21	.01	.78	699
Conscientiousness	−.**08**	.04	.00	.91	699
Neuroticism	.02	.67	.04	.33	699
Intellect/Openness	.00	.94	.01	.78	699
Adult BFI					
Extraversion	−.03	.46	−.05	.23	661
Agreeableness	.04	.28	.01	.85	661
Conscientiousness	−.01	.88	−.02	.56	661
Neuroticism	.02	.60	.04	.30	661
Intellect/Openness	−.01	.87	.05	.20	661
Childhood Trauma					
High-Betrayal	−.**09**	.04	−.**10**	.02	511
Low-Betrayal	−.05	.24	−.07	.13	511
Physical Health					

	r	p	r_{part}	p	n
Dysreg	−.**09**	.02	−.01	.90	658

Note. Partial correlations (r_{part}) controlling for age and sex. BMI = Body Mass Index (kg/m^2). High-betrayal= childhood high-betrayal traumas. Low-betrayal = childhood high-betrayal traumas. Dysreg = physiological dysregulation. Smoking was coded into five categories (0–4) based on life-time smoking history where 0 = ≤ 100 cigarettes, 1 = previously smoked < 8 Pack-years, 2 = previously smoked ≥ 8 pack years, 3 = current smoker at ≤ ½ pack a day, 4 = current smoker > ½ pack a day. Paternal age is estimated using father's age at birth. Parental home indexes home ownership in childhood before age 12. High-betrayal = childhood high-betrayal traumas; perpetrated by a person the child was dependent upon. Low-betrayal = childhood low-betrayal traumas.

Effect sizes in bold are significant at the $p < .05$ level.

Table 4

Model Results Testing Direct and Indirect Standardized Effects for Paths from Childhood Conscientiousness on LTL via BMI and Smoking.

	β	*p*	95% CI
Control variables			
Age	−.**432**	<.001	[−.492, −.373]
Sex	−.**101**	.002	[−.166, −.029]
Paternal age	.**109**	.047	[.004, .204]
Parental home	.031	.318	[−.056, .115]
Adult C	−.029	.445	[−.102, .040]
Japanese American	.119	.184	[−.065, .279]
Caucasian	.**220**	.024	[.017, .394]
Hawaiian/Part Hawaiian	.**202**	.049	[.010, .403]
Direct effects			
Child C	−.002	.954	[−.082, .074]
BMI	−.**081**	.018	[−.149, −.018]
Smoking	−.072	.058	[−.109, −.001]
Indirect effects			
Child C→BMI→LTL	.**012**	.049	[.000, .024]
Child C→Smoking→LTL	.011	.093	[−.002, .025]
Total indirect effect			
C→LTL	.**023**	.010	[.006, .041]

Note. Paternal age is estimated using father's age at birth. Parental home indexes home ownership in childhood before age 12. Adult C = BFI adult conscientiousness. LTL = Relative leukocyte telomere length. Child C = childhood conscientiousness. BMI = Body Mass Index (kg/m^2). Trauma = Childhood high-betrayal traumas. Effects reported are raw effects estimated with standardized variables. Dichotomous variables (sex and cultural dummy codes) are not standardized. Bias corrected 95% CIs for standardized indirect effects estimated using bootstrap.

Effect sizes in bold are significant at the $p < .05$ level.

Table 5

Model Results Testing the Interaction of Childhood High-Betrayal Traumas and Childhood Conscientiousness on LTL.

	β	*p*	95% CI
Control variables			
Age	−.**428**	<.001	[−.488, −.368]
Sex	−.**111**	.002	[−.177, −.040]
Paternal age	.**103**	.045	[.002, .195]
Parental home	.031	.462	[−.052, .114]
Adult C	−.025	.499	[−.101, .042]
Japanese American	.131	.143	[−.052, .296]
Caucasian	.**229**	.019	[.026, .406]
Hawaiian/Part Hawaiian	.164	.110	[−.036, .371]
Main effects and interaction			
Child C	−.009	.748	[−.070, .088]
Trauma	−.**086**	.030	[−.169, −.011]
Trauma X Child C	−.**093**	.006	[−.161, −.020]

Note. Paternal age is estimated using father's age at birth. Parental home indexes home ownership in childhood before age 12. Adult C = BFI adult conscientiousness. LTL = Relative leukocyte telomere length. Child C = childhood conscientiousness. Trauma = Childhood high-betrayal traumas. Effects reported are raw effects estimated with standardized variables. Dichotomous variables (sex and cultural dummy codes) are not standardized. Bias corrected 95% CIs for standardized indirect effects estimated using bootstrap.

Effect sizes in bold are significant at the $p < .05$ level.

Table 6

Model Results Testing Direct and Indirect Standardized Effects for Paths from Childhood Conscientiousness on LTL via BMI and Smoking, and the Interaction of Childhood Betrayal Traumas and Childhood Conscientiousness.

	β	*p*	95% CI
Control variables			
Age	−.**430**	<.001	[−.489, −.370]
Sex	−.**103**	.001	[−.169, −.035]
Paternal age	.**103**	.046	[−.003, .195]
Parental home	.027	.515	[−.056, .111]
Adult C	−.030	.430	[−.102, .040]
Japanese American	.113	.207	[−.065, .280]
Caucasian	.**220**	.024	[−.014, .395]
Hawaiian/Part Hawaiian	.**207**	.046	[.012, .410]
Direct effects			
Child C	−.025	.509	[−.084, .072]
Trauma	−.078	.050	[−.159, −.001]
Trauma X Child C	−.**080**	.022	[−.147, −.005]
BMI	−.**070**	.042	[−.138, −.006]
Smoking	−.064	.093	[−102, .005]
Specific indirect effects			
C→BMI→LTL	.**010**	.082	[.001, .024]
C→Smoking→LTL	.010	.133	[.000, .027]
Total indirect effect			
C→LTL	.**021**	.021	[.006, .042]

Note. Paternal age is estimated using father's age at birth. Paternal home indexes home ownership in childhood. LTL = Relative leukocyte telomere length. Child C = childhood conscientiousness. BMI = Body Mass Index (kg/m^2). Trauma = Childhood betrayal traumas. Effects reported are raw effects estimated with standardized variables. Dichotomous variables (sex and cultural dummy codes) are not standardized. Bias corrected 95% CIs for standardized indirect effects estimated using bootstrap.

Effect sizes in bold are significant at the $p < .05$ level.