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Attachment insecurity and the biological embedding of reproductive strategies: Investigating the role of cellular aging

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ABSTRACT

Evolutionary-developmental psychologists have posited that individuals who grow up in stressful rearing circumstances follow faster life history strategies, thereby increasing their chances of reproduction. This preregistered study tested this stress-acceleration hypothesis in a low-risk longitudinal sample of 193 Dutch mother-child dyads, by investigating whether infant-mother attachment insecurity at 12 months of age predicted earlier pubertal onset and more callous-unemotional traits, aggression and risk-taking about a decade later. Also evaluated were the possible mediating roles of two biomarkers of accelerated aging (i.e., telomere length, epigenetic aging) at age 6. Structural equation modelling revealed no effects of attachment insecurity on biomarkers, pubertal timing or behavior. These null findings suggest that the explanatory value of evolutionary-developmental thinking might be restricted to high-risk samples, though unexplored variation in susceptibility to environmental influences might also explain the null findings.

1. Introduction

Early-life stress affects physical, behavioral and psychological functioning later in life (Repetti et al., 2002). According to evolutionary-developmental theorizing, stressful rearing circumstances accelerate pubertal development, while fostering antisocial and risky behavior, all considered reflective of a fast reproductive/life-history strategy (Belsky et al., 1991; Belsky, 2012, 2019; Del Giudice et al., 2015; Ellis et al., 2009). The current, preregistered, longitudinal study tested this multi-step hypothesis in a Dutch low-risk community sample, by investigating whether infant-mother attachment insecurity, as an indicator of early life stress, predicted earlier pubertal onset and more callous-unemotional traits, aggression and risk-taking about a decade later. Furthermore, this inquiry evaluated whether two biomarkers of accelerated aging (i.e., telomere length and epigenetic aging), also known to be related to antecedent stressful conditions, mediated the hypothesized links of attachment insecurity with accelerated pubertal and antisocial and risky behavioral development.

1.1. Attachment insecurity and child development: an evolutionary point of view

Infants are born with a survival-promoting instinct to form affectional bonds, called attachments, to caregivers (Bowlby, 1982).

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Observational and experimental evidence indicates that the quality of these bonds is influenced by the rearing circumstances, most notably the caregiver's sensitivity (Ainsworth et al., 1978; Fearon & Belsky, 2016). Enduringly compromised caregiver sensitivity fosters insecure attachment, a relatively stable affectional bond, that can contribute to heightened infant stress (Ainsworth et al., 1978; Bowlby, 1982; De Wolff & Van IJzendoorn, 1997) and difficulties in socio-emotional development (DeKlyen & Greenberg, 2008; Fearon et al., 2010; Groh et al., 2012). As such, compromised sensitive care and subsequent attachment insecurity are considered indexes of potential stressful early experiences within the Early Life Stress model (Loman & Gunnar, 2010).

Traditionally, sequelae of attachment insecurity have been viewed as evidence of maladaptive development. However, some evolutionary (life-history) thinkers have challenged this view, regarding many of these sequelae as adaptive responses to the external circumstances in the service of longer-term reproductive goals (Belsky et al., 1991; Ellis et al., 2009; Del Giudice et al., 2015). According to these accounts, early rearing circumstances disclose prospective information about future risks and uncertainties, including the trustworthiness of others (Belsky et al., 1991) and risk of premature mortality (Chisholm, 1993). Stress early in life might thus be an indication of risk of death before reproduction, resulting in accelerated sexual maturation, and poor future prospects, fostering a more antisocial and risky behavioral orientation (Belsky et al., 1991; Wolf et al., 2007). In line with these theoretical claims, research chronicles associations between attachment insecurity and aggression (see for reviews DeKlyen & Greenberg, 2008; Fearon et al., 2010), and – to a lesser extent – callous-unemotional traits (see for a review Van Der Zouwen et al., 2018) and risk-taking (Delker et al., 2018; Fuertes et al., 2017; Kobak et al., 2009).

A unique prediction of this evolutionary-developmental view is that, in addition to influencing psychological and behavioral development, early-life stress will accelerate development, thus resulting in puberty occurring earlier than would otherwise be the case (Belsky et al., 1991; Chisholm, 1993). These developmental effects would have evolved to increase the chances of children surviving to reproductive age and passing on their genes (Belsky et al., 1991; Belsky, 1997; Draper & Harpending, 1982; Ellis et al., 2009; Del Giudice et al., 2015). Although such "fast" reproductive strategies may prove detrimental to health and well-being in the long run, such costs would be discounted by natural selection given the primacy placed on reproductive success (Belsky et al., 1991; Belsky, 2012, 2019). Notably, there is some evidence that stressful family circumstances in the first five years of life, including insensitive parenting (Hartman et al., 2015) and infant-mother attachment insecurity (Belsky et al., 2010; Sung et al., 2016), are associated with earlier pubertal development in the case of females (Belsky, 2012; Belsky et al., 2010; Ellis, 2004; for review of counterevidence, see Sear et al., 2019).

Importantly, most current evidence in line with evolutionary-developmental theorizing comes from high-risk samples in which various forms of adversity are relatively prevalent (e.g., Delker et al., 2018; Kobak et al., 2009; Simpson et al., 2012; Sung et al., 2016; Van Der Zouwen et al., 2018). It remains to be determined whether similar effects would emerge in studies of low-risk populations, i.e., populations with minimal forms of adversity. Herein we address this gap in the literature and prospectively investigate whether infant-mother attachment insecurity is related to earlier pubertal onset and more callous-unemotional traits, risk-taking, and aggression in a Dutch low-risk community sample.

1.2. Sex-specific effects

Certain effects of early life stress on reproductive strategies may be stronger in one of the two biological sexes, due to differences in sex-selection pressures (Hämäläinen et al., 2018). Antisocial and risky behavior are thought to be more common and adaptive for males (Cale & Lilienfeld, 2002; Del Giudice, 2009; Ellis et al., 2012; Eme & Kavanaugh, 1995; Fearon et al., 2010), but the relation of these behavioral traits to early life stress remains unclear as some work documents stronger associations for females (Lehmann et al., 2018; Munson et al., 2001). Furthermore, theory and evidence suggest that the accelerating effect of early life stress may be restricted to females (Belsky, 2012; Ellis, 2004), possibly as a consequence of a more pronounced trade-off in females between early puberty and other fitness-relevant returns such as bodily growth and long-term health (Charalampopoulos et al., 2014; Ellis, 2004; James et al., 2012). Notably, however, several recent studies document links between early life stress and accelerated pubertal development even in males (Gur et al., 2019; Lian et al., 2018; Sun et al., 2017). In consequence, we focus on effects of attachment insecurity on pubertal development and behavior in both males and females, and explore possible differences between the sexes.

1.3. Biological embedding

When considering the effects of early-life stress on development, questions about biological processes of influence arise: Through what physiological mechanisms might stress come to regulate the timing of puberty? Recently, it has been proposed that cellular aging could be one biological process through which reproductive/life-history strategies become embedded (Belsky, 2019; Belsky & Shalev, 2016; Shalev & Belsky, 2016). There are two non-mutually exclusive theoretical lines of thought that explain how life history strategies could become embedded through somatic deterioration, including cellular aging. According to a first line of thought (the *external* prediction model), stress, especially in early life, accelerates cellular aging (Belsky, 2019; Price et al., 2013; Shalev, 2012), which in turn might serve as an internalized cue about future external circumstances (Belsky, 2019; Belsky & Shalev, 2016; Shalev & Belsky, 2016; Nettle et al., 2013). According to a second line of thought (the *internal* prediction model), cellular aging could predict the body's own longevity, and thereby trigger acceleration of development as an adaptive response to internal states, irrespective of whether cellular aging predicts future external circumstances (Bateson & Nettle, 2018; Nettle et al., 2013; Rickard et al., 2014). Indeed, a substantial body of evidence links accelerated cellular aging to compromised mental and physical health in adulthood, as well as early mortality (e.g., Arbeev et al., 2020; Bakaysa et al., 2007; Chen et al., 2016; D'Mello et al., 2015; Gillis et al., 2019; Han et al., 2018; Lindqvist et al., 2015; Ma et al., 2011; Marioni et al., 2015; Perna et al., 2016; Rode et al., 2015; Smith et al., 2019; Suarez et al., 2018). Furthermore, several reviews on animal studies suggest a link between cellular aging and growth and reproduction, signifying its potential to affect life-history trade-offs (Monaghan & Ozanne, 2018; Parrott & Bertucci, 2019; Sudyka, 2019). Nevertheless, it remains unclear whether cellular aging plays a causal role in these outcomes or is merely a correlate of other influential processes such as changes in HPA axis activity (Bateson & Nettle, 2018; Belsky & Shalev, 2016; Shalev & Belsky, 2016). It also remains unknown whether accelerated aging may forecast—and perhaps causally influence—pubertal development or behavior during early adolescence. Notably, a few recent studies suggest that this could be the case (Beijers, Daehn, et al., 2020; Binder et al., 2018; Koss et al., 2020; Suarez et al., 2018; Wojcicki et al., 2015). Herein we extend this emerging body of work by investigating whether there are indirect effects of attachment insecurity on future child functioning via two biomarkers that are independently associated with aging (D. Belsky et al., 2018; Marioni et al., 2016; Vetter et al., 2019): telomere length and epigenetic methylation of select genes (from here on referred to as "epigenetic aging").

Telomeres are protective DNA-protein sequences at the end of chromosomes that shorten with each cell division, and thus with increasing age (Bojesen, 2013; López-Otín et al., 2013). Across the lifespan, environmental factors such as (early life) stress become predictive of telomere shortening (Beijers, Hartman, et al., 2020; Pepper et al., 2018; Price et al., 2013; Shalev, Entringer, et al., 2013). Once

telomeres reach a critical length, they no longer maintain chromosomal integrity and cellular senescence (cessation of cell division) occurs (Bojesen, 2013). Cellular senescence, in turn, is considered as one of the hallmarks of aging and is linked to aging-related decline in system integrity (Baker et al., 2011; López-Otín et al., 2013), possibly as a consequence of senescence-induced inflammation and oxidative stress (Coppé et al., 2010; Freund et al., 2010; Rodier et al., 2009).

Epigenetic age estimates are derived from methylation of genomic DNA at select positions across the genome (typically CpG dinucleotides). Indices of epigenetic age correlate strongly and positively with chronological age (Horvath & Raj, 2018; Jones et al., 2015). Like telomere length, epigenetic age appears to be affected by stress (Gassen et al., 2017; Zannas et al., 2015) and is related to health. Indeed, studies link stress early in life with accelerated epigenetic aging in children (Jovanovic et al., 2017; Sumner et al., 2019), while epigenetic age acceleration in adults increases risk for morbidity and mortality (Chen et al., 2016). The most widely used measure of epigenetic aging is Horvath's multi-tissue epigenetic clock (Horvath, 2013). As previous studies revealed this index to have limited accuracy in children (Simpkin et al., 2016; Simpkin et al., 2017; Tollenaar et al., 2019), the current research relies on the newly developed Pediatric-Buccal-Epigenetic (PedBE) clock, which measures epigenetic aging in children with greater accuracy (McEwen et al., 2019).

1.4. Current study

Within a healthy, low-risk, Dutch community sample, we evaluated a longitudinal, developmental model of a fast reproductive/life-history strategy, as depicted in Fig. 1. The model includes the following components and sub-hypotheses: (1) Greater infant-mother attachment insecurity at 12 months of age forecasts (a) earlier pubertal onset and (b) more (i) callous-unemotional traits, (ii) aggression, and (iii) risk-taking about a decade later. (2) Greater infant-mother attachment insecurity predicts (a) shorter telomere length and/or (b) accelerated epigenetic aging at 6 years of age. (3) Shorter telomere length and/or accelerated epigenetic aging forecasts earlier (a) pubertal onset and (b) more (i) callous-unemotional traits, (ii) aggression, and (iii) risk-taking.

Collectively, then, the integrated model being tested stipulates that there are indirect effects of infant-mother attachment insecurity on pubertal onset and callous-unemotional traits, aggression, and risk-taking via telomere length and/or epigenetic aging. Full mediation was not expected, as attachment insecurity is also likely to affect behavioral development via its impact on other mediating processes not considered herein (e.g., cognitive representations of relationships; internal working models, Bowlby, 1982). In addition to the confirmatory hypotheses expressed above, this study explored sex-specific effects. An effect of attachment insecurity on pubertal onset in females was expected (Belsky, 2012; Ellis, 2004), but in the case of males this was an open question. The effect of sex on the relationship between attachment insecurity and behavior was also investigated in an exploratory manner.

2. Methods

Following current recommendations about research practices (e.g., Wagenmakers et al., 2012), the sample size, measures, confirmatory and exploratory hypotheses, and statistical analyses of this study were pre-registered at AsPredicted: https://aspredicted.org/gt6mi.pdf. Data are available for replication purposes upon request.

2.1. Participants

This study made use of existing longitudinal data of 193 healthy, low-risk, Dutch mother-infant dyads from the BIBO (Basal Influences on Child Development) project (see also Beijers et al., 2011). BIBO protocols were approved by the ethical committee of the Faculty of Social Sciences of the Radboud University (#ECG300107), following the Declaration of Helsinki. All mothers provided written consent. Pregnant women were recruited through midwife practices in and around Nijmegen, the Netherlands. Inclusion criteria were: an uncomplicated, singleton pregnancy with term delivery, no drug use during pregnancy, no physical and/or mental health problems, and a 5-minute infant Apgar score of ≥ 7. Furthermore, fluency in the Dutch language was required. Table 1 provides an overview of the demographic characteristics of the 193 dyads. Only those dyads with a score for attachment (in)security were included in the current study, resulting in a final sample of 185 dyads.

2.2. Procedures

Infant-mother attachment was assessed at the beginning of a lab visit at 12 months of age. During a school visit at age 6, child buccal cheek swab samples were collected and subsequently DNA was extracted, in order to measure telomere length and epigenetic age. Pubertal development was investigated with self-reports at ages 10, 11, 12.5, and 14 years. The first measurement round for pubertal development was part of a set of hard-copy questionnaires that were filled in by the child independently during a home visit, but in the presence of the researcher who could answer questions. Subsequent rounds took place at home (age

Table 1
Overview of the demographic characteristics.

	M	*SD*	Range
Maternal age at delivery	32.46	1.52	21.10–42.90
Mother born in the Netherlands	95,8%		
Maternal marital status (% living with partner)	97.9%		
Maternal educational level	3.8%		
- Primary education	20.4%		
- Secondary education			
- College or university	75.8%		
Infant sex (girls)	47.2%		
First born child	41.0%		
Infant birth weight (g)	3616.97	465.32	2645.0–4730.0
Apgar score at 5 min	9.66	0.63	7.0–10.0

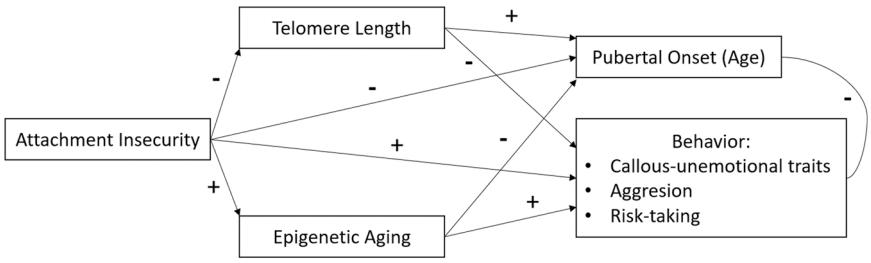

Fig. 1. Hypothesized model linking attachment insecurity, (age of) pubertal onset, and antisocial and risky behavior, partially mediated by two biomarkers of cellular aging.

11), online (age 12.5) and during a home visit (age 14), following similar procedures as described before. Risk-taking was tested with a behavioral task during the home visit at age 10. The child independently performed the task, but the researcher was available for questions. At age 12.5, callous-unemotional traits were measured with a self-report questionnaire during a fMRI lab visit, and aggressive behavior was measured with an online mother-report questionnaire.

2.3. Measures

2.3.1. Infant-mother attachment insecurity

Infant-mother attachment insecurity was assessed with the Strange Situation procedure (Ainsworth et al., 1978), a classic, reliable, and valid test of attachment (Solomon & George, 2008). This procedure consists of a series of repeated interactions, separations, and reunions with the mother and a stranger in an unfamiliar laboratory environment, which is supposed to activate the attachment system. Qualified staff at the Institute of Child Development, University of Minnesota, blind to all other information on the children and their families, coded the tapes of the infants' interactive behavior and rated their levels of proximity-seeking, contact maintaining, resistance, and avoidance. A very small number of these ratings ($n = 5$) (0.65%) were missing and imputed using the expectation-maximization algorithm (Dempster et al., 1977). Subsequently, these ratings were used in the computation of a continuous score for attachment insecurity, following Van IJzendoorn and Kroonenberg's (1990) adaptation of the Richters et al. (1988) algorithm. Positive values were converted into negative values and vice versa. Thus, higher continuous scores reflect greater attachment insecurity. An alternative, dichotomous operationalization of attachment insecurity, based on the observers' categorization of the infants' attachment behavior as secure or insecure (avoidant, resistant, or disorganized), was examined in the sensitivity analyses. Additionally, we examined a dummy variable for disorganized versus organized (secure, avoidant, or resistant) attachment in the sensitivity analyses. Interobserver reliability was very good (Cohen's κ of 0.82, intraclass correlation of 0.86).

2.3.2. Pubertal onset

2.3.2.1. Tanner pubertal onset . Tanner pubertal onset was measured in terms of Tanner stages (Marshall & Tanner, 1969, 1970), which track the physical development of primary and secondary sex characteristics (i.e., breasts or male genitals, and pubic hair) on a scale from 1 (pre-pubertal) to 5 (completed maturation). Children were presented with sex-appropriate schematic drawings (Lee, 2001) and descriptions of the five Tanner stages, and indicated for each sex characteristic which Tanner stage most closely resembled their own physical development. The age of measurement (10, 11, 12.5 years) at which a child first reported to have reached stage 2 or higher on at least one of the Tanner sex characteristics was used to index the timing of pubertal onset (similar to the approach used by Belsky et al., 2010). Although physician ratings are generally considered the gold standard for assessment of sex characteristics, self-reports have acceptable validity (Coleman & Coleman, 2002; Dorn et al., 1990; Duke et al., 1980; Ellis, 2004). Due to missing values (7.7%)–and 'reversals' whereby 31 children reported to be more advanced in pubertal development at earlier than at later measurement rounds–pre-processing was required. It was decided to only use data from children who participated in at least two of the three measurement rounds, in order to guarantee reliability. Missing data were imputed using the expectation-maximization algorithm, after it was established that they could be assumed to be completely at random, Little's MCAR test: $\chi 2$ (21) = 12,352, $p = .930$. If the inconsistencies in the reversal cases comprised a difference of 2 or more stages, or if the participant reversed back to stage 1 (no puberty), the unreliable, earlier timepoint for that respective sex characteristic was disregarded. Complete removal of reversal cases was examined in the sensitivity analyses. Additionally, when data from the measurement round at age 14 years became available, we performed yet another approach to determine age of pubertal onset. We started at the latest measurement round to determine when pubertal onset occurred, and only turned to the previous measurement round if the child reported to be in stage 2 or higher. Using this alternative approach resulted in a similar number of inconsistencies in the data (i.e., 30 inconsistencies for 27 participants) and the same estimates of age at pubertal onset. Girls reached puberty at an average age of 10.80 ($SD = 0.92$) years, while boys reached puberty at an average age of 10.96 ($SD = 1.14$) years, $t(151) = 0.904$, $p = .368$.

2.3.2.2. Menarche. For girls, pubertal onset was additionally measured with self-reports on menarche. At age 10 and age 11, the first two pubertal measurement occasions, none of the girls (0%) had yet experienced menarche; by the third measurement at age 12.5 years, 21 of the 66 girls (31.8%) who reported on menarche had experienced it. Because 68.2% experienced their first menstruation at later ages, a dichotomous variable was created to indicate whether girls had experienced their first menstruation by the age of 12.5 years or not, in accordance with the preregistration. With the data stemming from the measurement round at age 14, a continuous variable based on self-reported age at menarche was created and investigated in additional primary analyses. By this age, 57 of 65 girls (87.7%) who reported on menarche had experienced it.

2.3.3. Callous-unemotional traits

Callous-unemotional traits were measured with the 24-item Youth Self-Report version of the Inventory for Callous Unemotional Traits (ICU) (Frick, 2004). The ICU has three subscales: callousness, uncaring, and unemotional. All items were scored on a four-point Likert scale ranging from 0 (not at all) to 3 (definitely true). With an average score of 21.39 ($SD = 6.40$), boys tended to score higher than girls ($M = 19.30$, $SD = 5.67$; $t(95) = -1.691$, $p = 0.094$). Previous studies demonstrated that the ICU has acceptable internal consistency and external validity (Cardinale & Marsh, 2020; Essau et al., 2006; Roose et al., 2010). In the current sample, Omega was 0.75, indicating that the internal consistency of the scale was good (McDonald, 1999).

2.3.4. Aggression

Aggression was measured with mother report on the subscale "Aggressive Behavior" of the Child Behavior Checklist for school aged children (CBCL/6–18) (Achenbach & Rescorla, 2001). This checklist is part of the Achenbach System of Empirically Based Assessment (ASEBA). The subscale "Aggressive Behavior" measures behavior such as fighting, arguing, bullying, and bragging. It consists of 20 items, which are scored on a three-point Likert scale (0 =absent, 1 = occurs sometimes, 2 =occurs often). For both boys and girls the average score was 3.61 ($SD = 3.63$). The CBCL is widely used in both clinical and non-clinical situations and has proven to be of adequate validity and reliability (Achenbach & Rescorla, 2001). Omega for the "Aggressive Behavior" subscale in this sample was 0.89, indicating that the internal consistency of the scale was good (McDonald, 1999).

2.3.5. Risk-taking

Risk-taking was measured with an adapted Youth Version of the Balloon Analogue Risk Task (BART-Y), a widely used and validated behavioral measure of risk-taking (Lejuez et al., 2007). In this computerized task, participants earn points by pumping air into 15 depicted balloons, one at a time, with unknown probability of the balloon bursting. If participants stopped pumping, they collected the points that they had accumulated up to that point. However, if the balloon burst before they decided to stop, all points for that trial were lost. The children were told by the researcher that, if they collected enough points, they would earn a 5 euro voucher. After the end of the BART-Y, all children received this voucher.

Risk-taking propensity was calculated as the average number of pumps on unexploded balloons, in accordance with the guidelines for the use of the BART-Y (Lejuez et al., 2002; Lejuez et al., 2007). In order to deal with some recurring issues in the data, we decided that: (1) if participants ($n = 26/148$) collected zero points from an unexploded balloon (which can be fairly assumed to have happened due to an accidental double click on the stop button), these points were not used to calculate their personal average; and (2) data from at least 8 (more than half) of the balloons were required in order to receive a final score for risk-taking (96.6%). Boys had an average risk-taking score of 15.14 ($SD = 6.66$), while girls had an average score of 14.52 ($SD = 6.15$); this difference was not significant, $t(141) = -0.566$, $p = 0.572$. An alternative operationalization of risk-taking was examined in the sensitivity analyses. This operationalization included zero points from non-burst trials in the personal risk-taking average, and calculated scores only for participants who pumped all 15 balloons.

2.3.6. Biological markers of accelerated aging

2.3.6.1. Telomere length. For 148 participants, DNA was extracted from buccal epithelial cells collected at age 6 ($M = 6$ years and 20 days, $SD =$ 67 days) using QIAamp DNA Mini Kit (Qiagen, Germany), and quantified using Quant-iT PicoGreen reagent (Thermo Fisher Scientific). DNA was stored at − 80 ℃ until telomere length and DNA methylation assays. Telomere length was determined using a quantitative PCR protocol adapted from Cawthon (2002). Telomere length was expressed as a ratio of telomere content (T) to a single-copy housekeeping gene (S). The single copy gene used in the assay was 36B4. The T/S ratio was calculated using the formula $T/S = (\frac{E_T^{Cq_T}}{E_S^{Cq_S}})^{-1}$, where $E_{T/S}$ is the efficiency of exponential amplification for reactions targeting the telomere or single-copy gene respectively, and $Cq_{T/S}$ is the PCR cycle at which the sample crosses a critical threshold for detection of the telomere and 36B4 reactions. Detailed descriptions of sample handling and processing, as well as details regarding qPCR assay and quality control are summarized in the supplemental Table S1 in accordance with guidelines recommended by the Telomere Research Network (https://osf.io/9pzst/). To account for age differences at the time of data (buccal cell) collection, this study's index of telomere length reflects the standardized residuals derived from regressing telomere length at the 6-year measurement occasion on the child's precise chronological age in months at the moment of data collection (this approach was previously used by Beijers, Daehn, et al., 2020; Beijers, Hartman, et al., 2020; Beijers et al., in press). Positive residuals indicate longer than expected telomere length, and thus slower aging; negative residuals indicate shorter than expected telomere length, and therefore accelerated aging.

2.3.6.2. Epigenetic aging. The DNA that was extracted from buccal epithelial cells collected at age 6 was used to determine epigenetic age. Genome-wide DNA methylation was described using the Infinium EPIC array. Signal extraction from raw image files, quality control and preprocessing steps were performed using the Minfi package in R (Aryee et al., 2014). Epigenetic age was calculated using the newly developed Pediatric-Buccal-Epigenetic (PedBE) clock (McEwen et al., 2019). Epigenetic aging was operationalized as the residuals from a linear model regressing PedBE-derived estimates of epigenetic age on chronological age in months at the moment of data collection. A positive value for epigenetic aging indicates higher than expected epigenetic age, and thus accelerated aging, whereas a negative value for epigenetic aging indicates lower than expected epigenetic age, and therefore slower aging.

2.6. Data analyses

2.6.1. Data preparation

In the final dataset, all variables were checked for violations of normality by testing skewness and kurtosis. Univariate outliers (defined as cases >3 SD above or below the mean) ($n = 6$) were winsorized (i.e., replaced with the mean ± 3 SD). A robust estimator (MLR) was used to deal with non-normality. Correlations between all study variables and child sex, maternal age-at-delivery, and buccal cell count (percentage of buccal cells in the swab sample) were evaluated to check whether the latter could act as confounding factors. Since no such links were established, the analyses proceeded without consideration of confounding factors.

In the final dataset comprising 185 children, the following data were missing due to participants skipping a measurement round (e.g., the school visit at age 6 because of reluctance towards school involvement; the home visit at age 10 due to lack of time and/or scheduling difficulties; the fMRI round at age 12.5 due to wearing braces and/or scheduling difficulties): Tanner pubertal onset ($n = 32$), callous-unemotional traits ($n = 88$), risk-taking ($n = 42$), aggression ($n = 37$), telomere length ($n = 41$), and epigenetic aging ($n = 43$). The BaylorEdPsych package (Beaujean, 2012) in R (R Core Team, 2018) was used to ascertain that missingness could be assumed to be completely at random, Little's MCAR test: $\chi 2$ (91) = 77.804, $p = 0.836$. Full information maximum likelihood (FIML) was used to deal with the missingness.

2.6.2. Statistical analysis

Preliminary analyses were conducted to provide descriptive statistics (mean, SD, and bivariate Pearson correlations). To test the hypothesized model, we constructed a structural equation model with the lavaan package (Rosseel, 2012) in R (R Core Team, 2018). This model consisted of direct and indirect paths from attachment insecurity (as IV) to pubertal onset and callous-unemotional traits, aggression, and risk-taking (as DVs), via telomere length and epigenetic aging (as mediators). Initially, callous-unemotional traits, aggression, and risk-taking were expected to form a latent construct reflective of a fast reproductive/life-history strategy (i.e., antisocial and risky behavior). In the literature, life history traits are regularly assumed to cluster together (Belsky et al., 1991; Ellis et al., 2009; Figueredo et al., 2007), though this view has been challenged both conceptually and empirically (Frankenhuis & Nettle, 2020; Sear, 2020). However, because the behavioral traits were not inter-correlated, each was treated in the model as a separate dependent variable. According to the rule of thumb that there should be at least 5 cases per parameter (Bentler & Chou, 1987), our model with 24 parameters was sufficiently powered with a sample size of 185 participants. Following Kline's (2005) recommendations, we evaluated model fit on the basis of 4 global fit indices: the Chi-square ($\chi 2$), Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), and Standardized Root Mean Square Residual (SRMR). The main model was created for both males and females and included Tanner-stage measurement of pubertal onset at age 10, 11 and 12.5. A second model, with 9 parameters, focused exclusively on females using the dichotomous menarche variable at age 12.5 as pubertal onset indicator. Because Tanner data from the measurement round at age 14 recently became available, we conducted additional non-preregistered primary analyses including these data. To test sex-specific effects, the main model was subsequently tested for males and females separately in a multiple group comparison. Post-hoc sensitivity analyses followed these preregistered analyses. Finally, non-preregistered Bayes factors for the null versus the alternative hypotheses were calculated.

3. Results

3.1. Preliminary analyses

Descriptive statistics and bivariate Pearson correlations between study variables are presented in Table 2. (Separate estimates for males and females are available in supplemental Tables S2 and S3.) Bivariate correlations were calculated with the *psych* package in R (Revelle,

Table 2
Descriptive Statistics and Bivariate Correlations between Study Variables.

	M	*SD*	1	2	3	4	5	6	7
1. Child sex									
2. Attachment insecurity[a]	-0.56	2.43	.06						
3. Tanner pubertal onset (age)	10.88	1.04	.07	.04					
4. Callous-unemotional traits	20.41	6.16	.17	.03	-.05				
5. Risk-taking	14.86	6.42	.05	-.15	-.04	.01			
6. Aggression	3.71	4.03	.00	-.01	-.12	.00	.05		
7. Telomere length[b]	0.00	1.00	.06	-.07	.05	-.02	.00	-.08	
8. PedBE Epigenetic aging[b]	-0.01	0.68	-.15	-.06	-.10	-.24 *	.02	-.02	.15

Note * indicates $p < 0.05$.
[a] Higher (more positive) values reflect greater attachment insecurity and lower (more negative) values reflect greater attachment security.
[b] refers to residualized scores, corrected for age at buccal swab collection.

2018), using pairwise deletion of missing values. Most study variables proved to be unrelated to each other. The only significant correlation indicated that accelerated epigenetic aging was related to less callous-unemotional traits.

3.2. Primary analyses

Tanner-Stage Model: A structural equation model was created to test whether attachment insecurity was predictive of pubertal onset, callous-unemotional traits, aggression, and risk-taking, with telomere length and epigenetic aging operating as mediating variables. Fig. 2 presents a visual overview of this model, including the standardized estimates.

While the fit of the model was adequate ($\chi 2$ (4) = 3.326, p = .505; CFI = 1.000; RMSEA = .000; SRMR = .029), attachment insecurity did not directly predict pubertal onset, nor any of the behavioral variables or either of the two biomarkers of accelerated aging, telomere length and epigenetic aging. These biomarkers also did not predict the outcome variables. In consequence, there were no significant indirect effects of attachment insecurity on pubertal onset or behavioral development via the accelerated-aging biomarkers. The model explained only 0.5% of the variance in telomere length, 0.3% of the variance in epigenetic aging, 1.5% of variance in pubertal onset, 6.0% of the variance in callous-unemotional traits, 2.1% of the variance in risk-taking, and 0.9% of variance in aggression. Parameter estimates and bootstrapped confidence intervals are presented in Table 3.

Menarche-Model. A structural model for girls only (n = 85) tested direct and indirect effects of attachment insecurity on menarche, with telomere length and epigenetic aging as mediating variables. The fit of the model was adequate according to the global fit indices. As in the prior model, no significant direct or indirect effects emerged. Table 4 shows the parameter estimates and bootstrapped confidence intervals for this model. The model explained 8.1% of the variance in menarche.

Following preregistration of the primary analyses including Tanner data at ages 10, 11 and 12.5, Tanner data at age 14 became available, enabling a repletion of the primary analyses including these data. Results for the main model were no different than previously reported, results for the menarche model were also similar, though a significant relation between menarche age and telomere length in opposite direction to the hypothesis appeared (see supplementary Table S4 for results of the model with age at menarche as outcome variable).

3.3. Preregistered exploratory, sex-difference analysis

To test for sex-specific effects, the main model was subsequently tested for males and females separately (see Table 5 for the estimates). A Satorra and Bentler (2001) scale corrected chi-square difference test was performed using ANOVA to evaluate whether the grouped (freely estimated) model was significantly better than the constrained model in which parameters were equal across the sexes. This test was not significant, $\chi 2$ (23) = 33.482, p = .07, implying that the model for males was not different from that for females.

3.4. Preregistered and post-hoc sensitivity analyses

Following the preregistration, a sensitivity analysis with and without outliers was performed. This analysis largely produced null results, although a few significant results appeared in the opposite direction of the hypotheses (see supplemental Tables S5, S6, S7 and S8). In addition to the preregistered analyses, post-hoc sensitivity analyses were performed to investigate the robustness of our null results when using

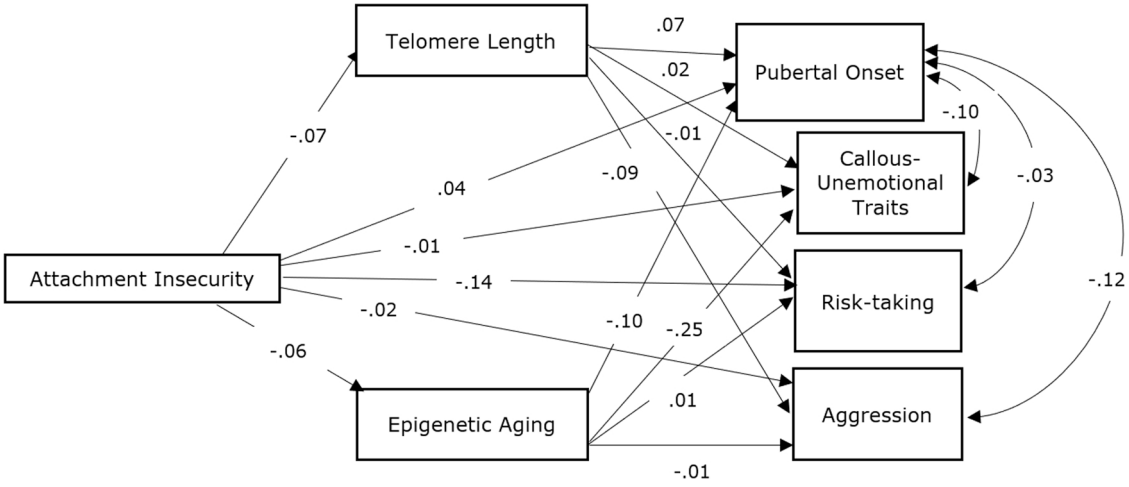

Fig. 2. Final SEM model of associations between attachment insecurity (as IV) and Tanner pubertal onset and child behaviors (as DV's), partially mediated via telomere length and epigenetic aging. Standardized estimates are presented.

Table 3
Parameter Estimates and Bootstrapped 95% Confidence Intervals for the Main Model.

	B	*SE*	Lower CI	Upper CI	ß
Regression paths					
Telomere length					
AI (a1)	-.029	.033	-.093	.035	-.074
Epigenetic aging					
AI (a2)	-.016	.023	-.061	.029	-.058
Tanner pubertal onset					
AI (c1)	.016	.034	-.049	.082	.038
TL (b1)	.071	.103	-.130	.272	.065
EA (b2)	-.151	.134	-.414	.113	-.097
Callous-unemotional traits					
AI (c2)	-.018	.283	-.573	.536	-.007
TL (b3)	.108	.687	-1.239	1.454	.017
EA (b4)	-2.229	1.139	-4.460	.003	-.245
Risk-taking					
AI (c3)	-.377	.208	-.785	.030	-.143
TL (b5)	-.088	.612	-1.287	1.110	-.013
EA (b6)	.099	.913	-1.691	1.889	.010
Aggression					
AI (c4)	-.024	.135	-.288	.239	-.016
TL (b7)	-.350	.377	-1.089	.388	-.092
EA (b8)	-.038	.452	-.924	.848	-.007
Covariances					
Tanner pubertal onset					
CU	-.593	.652	-1.871	.686	-.096
RT	-.191	.555	-1.279	.897	-.029
AGG	-.431	.259	-.939	.078	-.116
Indirect effects					
Mediator: Telomere length					
AI→ TL→ TPO (a1 x b1)	-.002	.003	-.008	.004	-.005
AI→ TL→ CU (a1x b3)	-.003	.020	-.042	.036	-.001
AI→ TL→ RT (a1x b5)	.003	.018	-.034	.039	.001
AI→ TL→ AGG (a1x b7)	.010	.015	-.019	.039	.007
Mediator: Epigenetic aging					
AI→ EA→ TPO (a2 x b2)	.002	.004	-.006	.011	.006
AI→ EA→ CU (a2x b4)	.036	.053	-.067	.139	.014
AI→ EA→ RT (a2x b6)	-.002	.014	-.029	.026	-.001
AI→ EA→ AGG (a2x b8)	.001	.007	-.014	.015	.000

Note. AI = attachment insecurity; TL = telomere length; EA = epigenetic aging; TPO = Tanner pubertal onset; CU = callous-unemotional traits; RT = risk-taking; AGG = aggression.

Table 4
Parameter Estimates and Bootstrapped 95% Confidence Intervals for the Menarche Model.

	B	*SE*	Lower CI	Upper CI	ß
Regression paths					
Telomere length					
AI (a1)	-.019	.050	-.117	.079	-.043
Epigenetic aging					
AI (a2)	.001	.031	-.060	.061	.002
Menarche					
AI (c1)	-.013	.022	-.057	.031	-.069
TL (b1)	.111	.071	-.027	.250	.252
EA (b2)	-.077	.091	-.256	.102	-.104
Indirect effects					
AI→ TL → Menarche (a1 x b1)	-.002	.006	-.014	.010	-.011
AI→ EA → Menarche (a2 x b2)	-.000	.002	-.005	.005	-.000

Note. $\chi 2$ (1) = 0.846, p = .358; CFI = 1.000; RMSEA = .000; SRMR = .034. AI = attachment insecurity; TL = telomere length; EA = epigenetic aging; Menarche (0 = never had menstruation; 1 = had experienced menstruation).

alternative parameterizations of select variables. Results proved similar for a dichotomous instead of continuous measure of attachment insecurity (Table S9), and a dichotomous variable comparing disorganized versus organized attachment (Table S10), with a few significant results in opposite direction of the hypotheses. Furthermore, results were

Table 5
Unstandardized Model Estimates and Bootstrapped 95% Confidence Intervals for Males and Females.

	Males		Females	
	B	95% CI	*B*	95% CI
Regression paths				
Telomere length				
AI	-.043	[-.123, .036]	-.020	[-.119, .079]
Epigenetic aging				
AI	-.029	[-.094, .035]	.003	[-.057, .063]
Tanner pubertal onset				
AI	.042	[-.057, .140]	-.015	[-.099, .069]
TL	.158	[-.184, .500]	.009	[-.226, .244]
EA	-.252	[-.613, .109]	.002	[-.326, .331]
Callous-unemotional traits				
AI	.288	[-.646, 1.222]	-.286	[-802, .230]
TL	.550	[-1.822, 2.921]	.075	[-1.607, 1.757]
EA	-.341	[-3.509, 2.828]	-3.963 *	[-6.222, -1.704]
Risk-taking				
AI	-.357	[-.901, .186]	-.473	[-1.023, .078]
TL	-.580	[-2.173, 1.013]	.299	[-1.535, 2.134]
EA	-1.036	[-3.312, 1.241]	2.195	[-.542, 4.932]
Aggression				
AI	-.180	[-.534, .173]	.154	[-.276, .585]
TL	-.1031	[-2.107, .045]	-.017	[-0.904, .939]
EA	.444	[-.490, 1.378]	-.646	[-2.126, .835]
Covariances				
Tanner pubertal onset				
CU	-.701	[-2.498, 1.096]	-.296	[-1.666, 1.074]
RT	-1.691 *	[-3.348, -.035]	1.705 *	[.561, 2.849]
AGG	-.259	[-.896, .377]	-.606	[-1.436, .224]

Note. AI = attachment insecurity; TL = telomere length; EA = epigenetic aging; CU = callous-unemotional traits; RT = risk-taking; AGG = aggression. * indicates significance. Fit indices for the freely estimated model: $\chi 2(8)$ = 4.699, p = .789; CFI = 1.000; RMSEA = .000; SRMR = .035. Fit indices for the constrained model: $\chi 2(31)$ = 37.637, p = .191; CFI = .000; RMSEA = .048; SRMR = .098.

similar when deleting reversal cases of pubertal measurement (Table S11), and when including all collected points (including zero points) in the personal risk-taking average for participants who pumped all 15 balloons (Table S12). To summarize, none of these variations yielded support for the hypotheses. The null results of the primary analyses thus appear robust.

3.5. Bayes factors for the null versus the alternative hypotheses

In order to quantify the support for our null findings, we performed a Bayesian evaluation of the null hypothesis versus the alternative hypothesis for each parameter in our structural equation model, using the R package *bain* (Gu et al., 2019; Van Lissa et al., 2021). Results from these analyses can be found in Supplementary Table S13 and S14. To summarize, the Bayes factors quantifying the relative support for the null hypotheses versus the alternative hypotheses range from 4.103 to 40.977 (for the Main Model M = 16.192, SD = 11.185; for the Menarche Model M = 17.237, SD = 9.537), meaning that the data are 4.103–40.977 times more likely under the null hypotheses than the alternative hypotheses. Therefore, we conclude that there is substantial to strong support for the null findings in our data (Kass & Raftery, 1995).

4. Discussion

Inspired by evolutionary/life-history thinking (Belsky, 2012; Belsky et al., 1991; Del Giudice et al., 2015; Ellis et al., 2009), this study evaluated whether infant-mother attachment insecurity, as a reflection of stressful rearing circumstances in early life, predicted earlier pubertal onset and more antisocial and risky behavior later in life in a low-risk community sample. Moreover, this study sought to extend prior evolutionary-developmental work by determining whether, as has been more recently hypothesized, cellular aging might function as a mechanism by which development in the service of reproductive goals is biologically embedded (Belsky, 2019; Belsky & Shalev, 2016; Rickard et al., 2014; Shalev & Belsky, 2016). Indisputably, all theory-derived predictions failed to receive empirical support in this inquiry; and this was so irrespective of whether alternative parameterizations of core constructs were considered (e.g., continuous vs. categorical attachment measure of attachment). Attachment insecurity at 12 months of age did not predict pubertal onset or callous-unemotional traits, aggression, and risk-taking behavior about a decade later. Additionally, attachment insecurity did not predict either telomere length or epigenetic age, nor did these biomarkers of accelerated aging predict pubertal onset or behavioral functioning. In consequence, no indirect effects of attachment insecurity via these biomarkers emerged.

Given the evolutionary-developmental theoretical foundation of the hypotheses, the question arises how these null findings can be explained. But before considering several possibilities, as well as the limits of this inquiry, it is critical to recall the fundamental adage when it comes to embracing the null: Absence of evidence is not evidence of absence. Now turning to possible explanations of our null results, the fact of the matter is that empirical evidence regarding the effects of rearing circumstances on pubertal development and antisocial and risky behavior is mixed (for reviews see Sear, 2020; Sear et al., 2019). Nevertheless, several studies have documented associations between stressful rearing circumstances and pubertal development and behavioral traits like those considered herein (e.g., Belsky et al., 2010; Moffitt et al., 1992; Richardson et al., 2020; Sheppard et al., 2014; Simpson et al., 2012). Therefore, it would be misguided to conclude that the null findings represent an indisputable falsification of the theory. Rather, they seem to point to a restriction on the theory's applicability.

One possibility is that the theory may not apply to attachment insecurity, despite the latter being a reflection of psychosocial stress (Belsky et al., 1991; Belsky & Fearon, 2008; Bowlby, 1982; Ainsworth et al., 1978). In fact, only a few studies have investigated the relation between attachment insecurity and pubertal development (Belsky et al., 2010; Sung et al., 2016), or the relation between attachment insecurity and callous-unemotional traits, and risk-taking (Delker et al., 2018; Van Der Zouwen et al., 2018). It is therefore possible that attachment insecurity may not be predictive of reproductive strategies and the associated behavioral orientation, despite theory suggesting otherwise (Belsky, 1997; Belsky et al., 1991). On the other hand, there is considerable evidence linking early attachment with later aggression (for a review, see Fearon et al., 2010), leading to the expectation that this relation would prove detectable in the present inquiry. Perhaps one could question whether attachment insecurity is an appropriate marker of early life stress in low-risk samples. In a low-risk sample, attachment insecurity might reflect less and qualitatively different environmental stressors than attachment insecurity in a high-risk sample (i.e., environmental stressors that might lead to compromised caregiving in low-risk samples may be of a different kind than those in high-risk samples) (Belsky & Fearon, 2008). For that reason, it could be that attachment insecurity is predictive of life history trajectories in high-risk, but not in low-risk samples.

Even if one were to assume that attachment insecurity reflects similar experiences of stress across low-risk and high-risk samples, presumably the most likely explanation for the null findings lies in the study sample. While 40% of children were insecurely attached and 60% securely attached, comparable to levels reported in other studies (Cassidy & Shaver, 2016), this otherwise healthy, low-risk, Dutch community sample was largely characterized by high socioeconomic status (SES) (measured by maternal educational levels) and stable marital relationships. Moreover, the sample came from a society with a strong social safety net, one providing decent health care and financial support to all families no matter their status. Therefore, the sample was mostly free from a range of major stressors, other than attachment insecurity. In light of theoretical accounts that emphasize the impact of cumulative risk on development (Evans et al., 2013), it is conceivable that the overall risk, and thereby the overall stress levels, in this sample were too low to accelerate life history strategies.

Important to appreciate is that attachment insecurity is thought to give rise to long-term developmental effects mostly in interaction with other psychosocial stressors in the rearing environment (Belsky & Fearon, 2002; DeKlyen & Greenberg, 2008; Erickson et al., 1985; Kobak et al., 2005). Indeed, the strength of the association between attachment insecurity and aggression and risk-taking is higher in low SES samples (Delker et al., 2018; Fearon et al., 2010) and the review by Van Der Zouwen et al. (2018) reported no relation between attachment insecurity and callous unemotional traits in community samples. Moreover, an investigation by Sung and colleagues (2016) revealed that attachment insecurity predicted accelerated pubertal development only under conditions of early life socioeconomic harshness. The low-risk nature of the current sample may thus have obscured (interactive) effects of attachment insecurity that might emerge in other, less privileged samples.

Also important to consider is the differential-susceptibility hypothesis, which stipulates that individuals vary in the degree to which they are influenced by developmental experiences, including for temperamental or genetic reasons (Belsky & Pluess, 2009; Boyce & Ellis, 2005; Pluess & Belsky, 2011). This possibility is consistent with studies that show that effects of early life adversity on pubertal development vary as a function of stress physiology (Ellis et al., 2011) and genotype (Hartman et al., 2015; Manuck et al., 2011), as well as with evidence that effects of rearing circumstances on telomere erosion depend on levels of prenatal stress (Beijers, Hartman, et al., 2020). The relation between attachment insecurity and child development may thus be more complex than is expressed by the absence or presence of a main effect—of attachment insecurity—as evaluated herein.

As there were no main effects of attachment insecurity on pubertal onset or child behavior, it is not surprising that no indirect effects via biomarkers of aging emerged. However, it is still noteworthy that the biomarkers themselves were not predictive of pubertal onset or behavioral development, especially given some limited evidence that points in this direction (Beijers, Daehn, et al., 2020; Binder et al., 2018; Koss et al., 2020; Wojcicki et al., 2015). Further, telomere length and epigenetic age also proved to be unrelated to each other. We believe this is the first study to address this issue in the case of children, but research with adults has previously shown that correlations between different epigenetic aging clocks and telomere length tend to be low, indicating that each biomarker might reflect distinct aspects of the aging process (D. Belsky et al., 2018).

Our reliance on buccal cells as a non-invasive method for obtaining DNA might raise the question whether results would have been different had we obtained DNA from different cell types, especially because the exact pathways from stress to changes in buccal telomere length or epigenetic aging remain unclear. In this regard, it is important to consider that prior research indicates that telomere length is highly correlated across tissues, suggesting tissue-independence (Daniali et al., 2013; Demanelis et al., 2020; Gadalla et al., 2010; Lin et al., 2019). Moreover, several studies show that stress-induced alterations can be detected in buccal cell DNA (Drury et al., 2012; Essex et al., 2013; McGill et al., 2022; Non et al., 2016; Shalev, Moffitt, et al., 2013). It should be noted, though, that telomere length is genetically influenced and therefore not a "pure" indicator of the effect of stress on biological aging (Broer et al., 2013; Hjelmborg et al., 2015). It would thus have been

preferable to have telomere measurements at birth so that the study could have focused on the effects of stress, as indexed via attachment insecurity, on *change* in telomere length (i.e., telomere erosion) in the early years.

4.1. Strengths and limitations

Methodologically, this study had several strengths, including its longitudinal design, repeated measurements of pubertal development, inclusion of two biomarkers of aging of which one was the newly developed and highly accurate PedBE-clock, strong theoretical foundation of the predictions made, pre-registration of hypotheses and analyses, and consideration of alternative parameterizations of core constructs in sensitivity analyses.

Despite these multiple strengths, there are also limitations. One concerns sample size. Although the rule of thumb (Bentler & Chou, 1987) indicated that a sample size of 185 participants was sufficient for our model, some authors recommend more stringent sample size criteria for structural equation models (e.g., Schreiber et al., 2006). Moreover, the relatively large number of missing and imperfect measurements might have compromised statistical power; a larger sample size would have been preferable, especially for sex-stratified analyses.

Pubertal onset measurements were limited to the timing of measurement occasions at 10, 11 and 12.5 (and 14) years. Since prior work has detected accelerating effects in the range of 3–6 months, some effects could have gone undetected due to imprecision in measurement (Belsky et al., 2010). Furthermore, the measurement of pubertal onset was limited by the use of self-reports. Prior research suggests that child self-reports are a reliable alternative to physician ratings of pubertal development (Coleman & Coleman, 2002; Dorn et al., 1990; Duke et al., 1980). Nevertheless, the current study revealed some reliability problems of children's self-reports, as a non-negligible number of children reported to regress back to earlier stages of pubertal development over time. This might reflect the tendency for younger children to overestimate their pubertal development (Schlossberger et al., 1992). Physician ratings would have been preferable, but less feasible in a longitudinal study. Future studies might also consider assessing puberty through steroid hormone concentrations in saliva or hair (Grotzinger et al., 2018). More frequent self-report measurement rounds could also offer a solution to both of the problems mentioned above, by making the outcomes less dependent on a single data point in time.

The behavioral measures also had their limitations. For the measurement of callous-unemotional traits we relied exclusively on self-report, the behavioral task for risk-taking did not cover risk-taking in context, and for aggression we relied exclusively on mother-report. The fact that we used different measurement methods (observational tasks, child report, maternal report) for the different behavioral traits, might explain the lack of intercorrelation amongst these traits. Note, however, that as previously indicated covariance between life history traits is under debate (Frankenhuis & Nettle, 2020; Sear, 2020), and even the first evolutionary-developmental theory (Belsky et al., 1991) stipulates that experiences throughout the life span may influence and deflect trajectories, which could also result in lack of clustering (Belsky, 1991; Del Giudice & Belsky, 2011). Moreover, the measurement of attachment was limited to the relationship with only one attachment figure (the mother; in most cases the primary caregiver). Not to be forgotten in terms of limitations is the low-risk nature of the sample, which prevents generalization of these findings. All these considerations should warn against premature embracement of the null.

5. Conclusion

To our knowledge this is the first prospective study testing an evolutionary, life-history model of accelerated aging in the service of reproductive goals—by evaluating the proposition that an indicator of early life stress, attachment insecurity, would predict biomarkers of accelerated aging (telomeres, epigenetic age) and, thereby, pubertal onset and behavioral development. Clearly, no support for the model of the developmental origins of a fast reproductive strategy emerged in this inquiry. Indeed, not a single component link of the integrated model depicted in Fig. 2 garnered empirical support. Given sample and measurement limitations, including the focus on a low-risk sample, it would seem premature—at this point—to discard the theory based on our null results. Future testing of diverse samples, with alternative measurements that consider variation to susceptibility to environmental influence, should enable us to better understand the implications of our findings. Finally, future investigation of other physiological processes, such as cortisol regulation and inflammation, may shed new lights on the biological embedding of life history strategies.

Conflicts of interest

The authors report no conflicts of interest.

Data Availability

Data are available for replication purposes upon request.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.biopsycho.2022.108446.

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