Health Psychology

ISSN: 0278-6133
https://doi.org/10.1037/hea0001576

Predictions From Early Adolescent Interpersonal Aggression to Accelerated Aging in Adulthood: Relational and Biological Mechanisms of Linkage

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Objective: This study examined early adolescent interpersonal aggression, subsequent conflict with parents, and aggression toward close peers as predictors of accelerated biological aging by age 30. ***Method:*** Participants (N = 123; 46 males and 75 females) were assessed repeatedly, along with parents and close friends, ages from 13 to 30. ***Results:*** Early adolescent interpersonal aggression was found to predict later accelerated aging even after accounting for adolescent gender, family income, prior health difficulties, and body shape ratings in adolescence. Path analyses suggested that the effects of early interpersonal aggression were potentially mediated via higher levels of father–adolescent conflict reported by fathers in adolescence and by aggressive behavior toward close peers as reported by those peers in early adulthood. Follow-up analyses suggested that these same factors also predicted adult body mass index scores after accounting for body shape in adolescence. ***Conclusions:*** Results are interpreted as evidence that social difficulties with lifelong health implications may be identified beginning in early adolescence, thus highlighting the potential importance of early interventions to address these difficulties.

Public Significance Statement
This study identifies lifelong health implications of social difficulties identified beginning in early adolescence. As such it highlights the potential importance of early interventions to address such difficulties and the potential long-term benefits of such interventions.

Keywords: aggression, aging, relationships, conflict, adolescence

Supplemental materials: https://doi.org/10.1037/hea0001576.supp

Relationship struggles in adulthood have been repeatedly linked to significant health risks up to and including early mortality (Holt-Lunstad et al., 2010; Siegman & Smith, 2013; Williams et al., 1988). Theoretically, the roots of these struggles could be observable as early as adolescence, as primary social relationships begin to take on adult-like characteristics and the interpersonal patterns established then often cascade into relationship qualities in adulthood (Oudekerk et al., 2015; Scholte & Van Aken, 2020). This study examined early adolescent interpersonal aggression as a predictor of accelerated biological aging in adulthood. It also sought to identify specific behavioral and physiological mechanisms that potentially account for early links to longer-term health outcomes so as to inform not only intervention approaches but also our broader understanding of the social determinants of health across the lifespan.

The study examined two comprehensive markers of adult biological aging to address the potential sequelae of early adolescent interpersonal aggression. These markers use algorithms to combine data from a broad range of physiological and blood chemistry indicators to yield estimates of biological age. The Klemera–Doubal method yields an estimated age at which the combination of measures observed in any individual would be considered approximately normal given population-wide data (Klemera & Doubal, 2006). Similarly, the PhenoAge method yields an

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This study was supported by grants from the National Institute of Child Health and Human Development and the National Institute of Mental Health (5R37HD058305-23, R01HD058305-16A1, and R01-MH58066).

Joseph P. Allen served as lead for conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, project administration, resources, supervision, and writing–original draft. Meghan A. Costello served in a supporting role for conceptualization, methodology, and writing–review & editing. Gabrielle L. Hunt served in a supporting role for conceptualization, funding acquisition, methodology, project administration, and writing–review & editing. Bert N. Uchino served in a supporting role for conceptualization, funding acquisition, methodology, and writing–review & editing. Karen Sugden served in a supporting role for conceptualization, data curation, formal analysis, methodology, and writing–review & editing.

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estimated age at which the risk of mortality is approximately equal to the mortality risk at that age in population-wide data (Liu et al., 2018). Importantly, both methods have been validated on large nationally representative samples and shown to not only be far superior to predictions obtained from any single biological indicator, including chronological age, but also to be better predictors of morbidity and mortality than existing epigenetic aging measures (Kwon & Belsky, 2021).

Several lines of research suggest the likely presence of links between interpersonal aggression and relationship conflict and long-term aging outcomes. The stress created by interpersonal aggression and conflict in relationships, particularly if chronic, has been identified as a risk factor for disturbances of both the digestive system and the hypothalamic–pituitary–adrenocortical axis with likely attendant impacts on health (Sapolsky, 2000; Sapolsky, 2004; Sapolsky et al., 2002). Social safety theory suggests that in evolutionary time, interpersonal conflict created a risk of bodily injury and led to upregulation of the immune system in preparation; when conflict and upregulation of the immune system are chronic, however, this leads to significant health problems (Slavich, 2020). Given that adolescents appear particularly sensitive to social stimuli, even at the neural level (Somerville, 2013), they would also appear likely to be particularly sensitive to the effects of social conflict that accompanies interpersonal aggression. In addition, if adolescent interpersonal aggression establishes a pattern of social relationship behavior that cascades forward into similar behavior in adult relationships (Oudekerk et al., 2015), it would suggest a likely pathway from such aggression to adult aging outcomes.

Though virtually no research has directly addressed the prediction of aging outcomes from adolescent interpersonal aggression, several lines of research suggest potential health effects of related behaviors in adulthood. In adulthood, conflict and violent behavior in family relationships have been linked to multiple markers of poor physical health (Kiecolt-Glaser et al., 1996; Pietromonaco & Collins, 2017; Robles et al., 2014). Hostility in adulthood has also been repeatedly linked to risk for future coronary artery disease (Chida & Steptoe, 2009) and to telomere shortening, which in turn has been linked to premature aging (Brydon et al., 2012; Watkins et al., 2016).

Similarly, child and adolescent hostility have been linked to several more focal physiological outcomes. Hostility in childhood has been concurrently associated with higher blood pressure and greater body mass index (BMI), both of which are known to predict future health risks (Grunbaum et al., 1997). High levels of adolescent conflict with parents have been found to predict higher levels of inflammation 20 years later (Ehrlich et al., 2024). Aggressive parent behavior toward their adolescents has also been found to predict higher adolescent levels of inflammation (Byrne et al., 2017).

In peer relationships, poor conflict resolution skills in adolescence and peer ratings of aggression in early adulthood have both been found to predict higher levels of adult inflammation (Allen et al., 2018). Similarly, difficulty negotiating disagreements with peers in ways that maintain an individual's autonomy and sense of connectedness during adolescence has been found to predict advanced epigenetic aging a decade later in adulthood (Allen et al., 2023). These predictions to discrete markers of physiological functioning raise the likelihood that adolescent interpersonal aggression would be linked to broader indicators of accelerated aging, although this premise has never been tested.

The likelihood of finding links between adolescent interpersonal aggression and global aging outcomes is further bolstered by evidence slightly later in development linking hostility in college-aged samples to future all-cause mortality (Siegler et al., 1992). Similarly, in childhood, constructs that are at least potentially related to interpersonal aggression (e.g., antisocial and rule-breaking behavior) have also been found to predict accelerated aging in adulthood (Langevin et al., 2022). Both of these research findings were notable for their use of comprehensive markers of health outcomes, an approach which is particularly valuable in capturing the combined effects of multiple physiological processes that may be influenced by social factors (Holt-Lunstad et al., 2010).

To the extent interpersonal aggression predicts accelerated aging, identifying potential mechanisms by which interpersonal aggression may lead to physiological changes becomes a crucial step. Although a great deal of work to date has focused on links to inflammation and telomere length, this study examined the prediction of future BMI, a relatively less explored potential outcome of relationship difficulties, but one that has itself been repeatedly linked to poor health outcomes (Nuttall, 2015). BMI is not included in the biological aging algorithms we use, given its high level of redundancy with included measures of actual function (e.g., C-reactive protein, glucose, and white blood cell count; Levine et al., 2018); yet it has been strongly linked to numerous physiological problems and is a frequent target of intervention efforts (Elovainio et al., 2011; Vicennati et al., 2009). Chronic stress, such as that which accompanies interpersonal aggression, appears likely to lead to obesity via its effects in hyperactivating the hypothalamic–pituitary–adrenocortical axis, reducing sleep quality, and leading to stress-induced eating (Tomiyama, 2019). In adolescence in particular, social experiences strongly influence the development of neurocognitive reward circuitry that has been implicated in the development of obesity (Blakemore & Mills, 2014; Reyes et al., 2024). Somewhat surprisingly, although 70% of obese adults were not obese as children or adolescents (Simmonds et al., 2016), research on what predicts adult BMI, especially after accounting for indices of adiposity in adolescence, has focused almost entirely on nutrition and activity levels (Viner & Cole, 2006). No work to our knowledge has examined relational factors as predictors of adult BMI after accounting for related measures in adolescence; identifying such predictors would obviously be important in highlighting both the need and a venue for early intervention.

This 70-year, multimethod, prospective study examined predictors of a broad marker of biological aging in adulthood that captures physical deterioration and health risk across multiple systems, including the cardiovascular, immune, and metabolic systems (Kwon & Belsky, 2021). It employed a diverse community sample to examine both direct and mediated pathways from adolescent interpersonal aggression to adult biological aging. Methodologically, one limit of most (though not all, see, e.g., Smith et al., 2008; Smith et al., 2007) research in this area has been reliance upon self-reports of interpersonal aggression and hostile behavior. Not only are such self-reports often unlikely to correspond to what others observe, but they also appear to be potentially less powerful as predictors of future health outcomes (Smith et al., 2008). In addition, reliance solely upon self-reports has made it difficult to disentangle the effects of interpersonal aggression as a subjective experience versus as a characteristic of relationships. This study utilized both self-reported interpersonal

aggression as well as reports from two types of external reporters (parents and close peers) to begin to address these issues. This study also controlled for important potential confounds that might predict rather than result from interpersonal aggression, including family poverty and education levels, preexisting health issues from childhood, and an indirect marker of adiposity in adolescence. Although the relative paucity of prior research on predictors of biological aging renders this study necessarily exploratory, it examined pathways from early interpersonal aggression to accelerated aging via five primary hypotheses:

1. Self-reported interpersonal aggression in early adolescence will predict accelerated aging by age 30, after accounting for demographic factors, a marker of adolescent adiposity, and childhood history of health problems.
2. High levels of conflict with mothers and with fathers across adolescence as reported by parents will predict adult accelerated aging after accounting for the covariates described earlier.
3. Poor conflict resolution skills with close friends in adolescence and high levels of conflictual and punitive behavior with close friends in early adulthood as reported by those friends will predict adult accelerated aging after accounting for the covariates described earlier.
4. Conflict with parents and peers will potentially play a mediating role in the relation of early adolescent interpersonal aggression to accelerated aging.
5. Predictors of accelerated aging will also predict adult BMI, even after accounting for body shape ratings that serve as a proxy measure of adiposity in adolescence.

Method

Participants

This report is drawn from a larger longitudinal investigation of adolescent social development in familial and peer contexts (Allen et al., 2006). The final sample of participants (N = 121 [46 males and 75 females]) was a subset of the original sample of 184 adolescents first assessed at age 13 and for whom biological aging data were able to be obtained at age 30 (M = 29.7, SD = 2.16). This reflected a 34% rate of total attrition across the 17 years of the study. The final sample was racially/ethnically and socioeconomically diverse: 66 (55%) adolescents identified themselves as White, 40 (33%) as Black/African American, 1 (1%) as Asian, 1 (1%) as Hispanic, 1 (1%) as American Indian, and 12 (10%) as from other or mixed racial/ethnic groups. Adolescents' parents reported a median annual family income in the $40,000–$59,999 range at the initial assessment, in line with the national median annual family income in the United States at that time of $42,000.

Adolescents were initially recruited from the seventh and eighth grades of a public middle school drawing from suburban and urban populations in the Southeastern United States. Students were recruited via an initial mailing to all parents of students in the school along with follow-up contact efforts at school lunches. Families of adolescents who indicated they were interested in the study were contacted by telephone. Of all students eligible for participation, 63% agreed to participate either as target participants or as peers providing collateral information. All participants provided informed assent before each interview session, and parents provided informed consent. Interviews took place in private offices within a university academic building.

Participants were first assessed annually over a 5-year period across adolescence from age 13.35 (SD = 0.64) to age 18 (SD = 1.04). BMI was first assessed via an observational procedure in adolescence and then obtained via direct measurement at ages 27.7 (SD = 0.99) and 28.6 (SD = 1.02). Extensive physical health assessments were obtained in person at age 29.7 (SD = 2.15).

Participants in adolescence also nominated the same-gender person they currently identified as "the peer to whom they were closest" to be included in the study. Participants could select a different person at each assessment, given that friendships change over time. In adolescence, close peers came in during a visit along with the target participant. After age 21, close friends could be of any gender and typically completed measures via mail or online survey. Close friends in adolescence reported that they had known participants for an average of 4.3–5.7 years (SD = 3.1–3.8) across the various assessment periods. From adolescent age 21–28, close peers reported having known participants for an average of 8.1–13.3 years (SD = 5.4–10.1). Parents provided data at adolescent ages 13, 16, and 18.

Procedure

In the initial introduction and throughout all sessions, confidentiality was assured to all study participants and adolescents were told that their parents would not be informed of any of the answers they provided. Informed assent was obtained from adolescents, and informed consent was obtained from adolescents' parents and from adult participants. Adolescent/adult participants, their parents, and their peers were all paid for participation.

Attrition Analyses

Attrition analyses compared the 121 participants in the final sample to the 63 who were excluded because they lacked biological aging data. Attrition analyses examining all baseline measures revealed only a slight difference in BMI at baseline assessment (those who did not have later biological aging data had slightly lower coded BMI).

Measures

Biological Aging (Age 30)

We quantified biological aging using two methods: the Klemera–Doubal method (KDM) (Klemera & Doubal, 2006) and PhenoAge (Levine et al., 2018). Briefly, these methods generate Biological Aging measures based on profiles of biomarkers linked to the aging process. The resultant estimates are generated through a three-step process: (1) selection of starting biomarkers and training of aging algorithms in NHANES III data, (2) testing performance against previously published estimates of Biological Aging in NHANES IV data, and (3) projecting the newly trained algorithms onto the data that is the focus of this study. These steps were carried out using the R package *BioAge* (version 0.1.0) following the suggested procedures outlined in Kwon and Belsky (2021) and at https://

github.com/dayoonkwon/BioAge. The list of biomarkers used to train biological aging was based on a modified set used by Kwon and Belsky (2021), namely, systolic blood pressure (mmHg), blood urea nitrogen (mg/dL), HbA1c (%), total cholesterol (mg/dL), total creatinine (μmol/L), serum glucose (mmol/L), red cell distribution width (%), albumin (g/L), alkaline phosphatase (U/L), mean cell volume (fL), C-reactive protein (mg/dL), and white blood cell count (1,000 cells/uL). Values for blood urea nitrogen, creatinine, alkaline phosphatase, C-reactive protein, and HbA1c were log-transformed before use to align with NHANES III/IV data. To assess age acceleration, chronological age was included as a covariate in all analyses.

When projected onto NHANES IV data for comparison and validation purposes, our newly calculated modified KDM and PhenoAge age measures were significantly correlated with chronological age ($r = .71$ and $r = .96$, respectively), and our newly calculated KDM and PhenoAge age advancement measures were significantly correlated with the originally reported KDM and PhenoAge age advancement measures ($r = .76$ and $r = .83$, respectively). Both our newly calculated KDM and PhenoAge age acceleration measures significantly predicted mortality in the NHANES IV data (Cox proportional hazard ratio, HR [95% CI] = 1.29 [1.25–1.33] and 1.50 [1.45–1.55], respectively), similar in magnitude to the originally reported measures (HR = 1.36 [1.20–1.55] and 1.46 [1.42–1.51]).

Interpersonal Aggression (Self-Reported; Ages 13–15)

Each year from age 13 to 15, target participants rated their interpersonal aggression using an 8-item shortened version of the interpersonal aggression subscale from the Child Behavior Checklist (Achenbach, 1991; Achenbach & Edelbrock, 1981). The short form used (Lizotte et al., 1992) has been shown to reliably predict delinquency similarly to the full scales. Participants rated themselves on items such as "destroys things belonging to others" and "I get in many fights." Scores for the 3 years were averaged together to yield the final scale, which had good internal consistency (Cronbach's $\alpha = .81$).

Conflict With Father and With Mother (Parent-Reported; Ages 13, 16, and 18)

At adolescent ages 13, 16, and 18, each parent reported on the total amount of conflict they had with their adolescent using a 39-item measure that assesses how often parents and teens have disagreed about four general areas: deviant behaviors, adolescent issues, household routines, and behavior toward others (Hetherington & Clingempeel, 1992). Results were aggregated across ages to derive the final measure of conflict with each parent. This measure had strong internal consistency (Cronbach's αs = .93 and .95 for mothers and fathers, respectively).

Conflict Resolution With Close Peer (Age 16)

This three-item scale from the Friendship Quality Questionnaire (Parker & Asher, 1993) utilizes a close-friend's report about the participant's ability to get over being mad, to resolve arguments quickly, and to make up easily after a fight and has previously been identified as a predictor of epigenetic aging (Allen et al., 2023). Internal consistency was good (Cronbach's $\alpha = .75$).

Conflict With Close Friend (Close-Friend Report; Ages 21–28)

The level of conflict with the closest friend as identified by the participant for each year was reported by that friend annually from participant ages 21–28 using the three-item Conflict subscale of the Network of Relationships Inventory (Furman & Buhrmester, 1985). For example, one item asks, "How much do you and this person argue with each other." Items were scored on a 5-point Likert scale, and internal consistency was excellent across assessments (Average Cronbach's $\alpha = .89$).

Punitive Behavior Toward Close Friend (Close-Friend Report; Ages 21–28)

Using the same approach as for conflict with a close friend described earlier, the level of punitive behavior displayed toward the closest friend was obtained from the three-item punishment subscale of the Network of Relationships Inventory (Furman & Buhrmester, 1985). For example, one item asks, "How much does this person punish you." Internal consistency was good across assessment waves (Average Cronbach's $\alpha = .79$).

History of Serious Childhood Illness

In the adult phase of the study, participants retrospectively reported the total number of a series of 43 distinct significant possible health problems first experienced before age 18 that led at some point to a hospitalization. Reported serious childhood illness was a significant predictor of self-reported adult health quality at age 25 ($r = .24$, $p = .006$) (Allen et al., 2015). This measure was thus treated as a baseline marker of health difficulties and used as a covariate in all analyses.

Adolescent Body Shape (Rated, Ages 13 and 14)

Adolescent body shape relative to height was coded on a 9-point scale using the Figure Rating Scales (Stunkard et al., 1983), with each point linked to a schematic drawing of an individual with a different BMI (Cheung et al., 2011). Ratings were made based on observation of the adolescent in 15 min of videotaped interaction by three independent raters. Interrater reliability was high for the average score of the three raters ($ICC = .94$ and .93 at ages 13 and 14, respectively) with significant stability across these 2 years ($r = .87$, $p < .001$). Prior research with adolescents found that this rating system yielded scores that were highly correlated with measured BMI (rs = .84–.87 across raters), suggesting the measure is an adequate proxy measure of BMI (Sherman et al., 1995).

Family Income in Adolescence; Household Income in Adulthood (Adolescent Ages 13 and 17; Adult Ages 27 and 28)

Income was calculated as a percentage of the federal poverty line (i.e., taking into account both income and household size). Averages were obtained for family of origin income (ages 13 and 17) and current household income in adulthood (ages 27 and 28).

Parent Education Level (Adolescent Age 13)

Both parents reported their highest level of education on a 1–9 scale ranging from eighth grade or less to a postgraduate degree.

The highest score of the two parents was used as an index of educational resources available to the adolescent.

Adult Education Level (Adult Age 29)

Using the same scale described earlier, participants reported their highest education level attained by age 29.

Adult BMI (Adult Ages 28 and 29)

Adult height (in m) and weight (in kg) with light clothing were assessed for each of two years and calculated using the standard formula BMI = weight/height^2, with results averaged across years to yield the final score.

Transparency and Openness

The study hypotheses and analysis plan were not preregistered. De-identified data and analysis code are available from the corresponding author upon reasonable request. Study materials are available upon request. This study followed APA Journal Article Reporting Standards (JARS).

Results

Preliminary Analyses

Means, standard deviations, and intercorrelations for primary variables used in the study are presented in Supplemental Table A. A small number of outliers were detected for measures of biological age (N = 3), adolescent interpersonal aggression (N = 2), and conflict with fathers (N = 1); these were winsorized to the next highest value within 3.5 SD of the mean. We also examined possible moderating effects of gender, racial/ethnic minority group membership, and adolescent family income on the relation of social relationship qualities to accelerated aging. Moderating effects were assessed by creating interaction terms based on the product of the centered main effect variables. No moderating effects were found.

Given the high degree of overlap of measures of household income in adolescence and adulthood, and parent education and participant education level as an adult, initial analyses entered family of origin income as a predictor variable, with subsequent analyses assessing whether any of the other variables significantly added to the prediction of biological age. These subsequent analyses revealed that after accounting for family of origin income, none of the other measured income or education measures added to the prediction of biological age; thus, these were not considered further.

Primary Analyses

For all primary analyses, linear regressions were conducted using SAS PROC CALIS (version 9.4, SAS Institute, Cary, NC). To best address any potential biases due to attrition in longitudinal analyses, full information maximum likelihood methods were used with analyses including all variables that were linked to future missing data (i.e., where data were not missing completely at random). Because these procedures have been found to yield the least biased estimates when all available data are used for longitudinal analyses (vs. listwise deletion of missing data), the entire original sample of 184 adolescents was utilized for these analyses.

Hypothesis 1: Self-reported interpersonal aggression in early adolescence will predict accelerated aging by age 30, after accounting for demographic factors, a marker of adolescent adiposity, and childhood history of health problems. In a hierarchical linear regression, participant's chronological age at the time of biological age assessment, their gender, and their baseline family income during adolescence were entered in Step 1, followed by history of childhood illness and ratings of adolescent body shape in Step 2. Adolescents' self-reported interpersonal aggression from ages 13 to 15 was entered in Step 3 to test the primary hypothesis. By examining predictors of biological age after accounting for chronological age, the result is a prediction of biological age acceleration (e.g., the extent to which a participant is biologically aging faster than their chronological age would suggest). As shown in Table 1, interpersonal aggression in adolescence significantly added to the prediction of both measures of

Table 1
Early Adolescent Interpersonal Aggression Predicting Age Acceleration at Chronological Age 30

	Biological age acceleration (at chronological age 30)					
	Klemera–Doubal method			PhenoAge method		
Predictors	β	ΔR^2	R^2	β	ΔR^2	R^2
Step I						
Chronological age	.03			.00		
Gender (1 = M; 2 = F)	−.07			−.27**		
Adolescent family income	−.09			.01		
Statistics for step		.034	.034		.055	.055
Step II						
History of childhood illness	−.05			−.01		
Body shape (ages 13–14)	.37***			.52***		
Statistics for step		.153***	.187***		.275***	.330***
Step III. Self-reports						
Interpersonal aggression (ages 13–15)	.19*			.22**		
Statistics for step		.027*	.214***		.027*	.357***

Note. β weights are from the final full model.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2
Parent-Observed Predictors of Age Acceleration at Chronological Age 30

	Biological age acceleration (at chronological age 30)					
	Klemera–Doubal method			PhenoAge method		
Predictors	β	ΔR^2	R^2	β	ΔR^2	R^2
Step I						
Chronological age	.06			.06		
Gender (1 = M; 2 = F)	−.03			−.22**		
Adolescent family income	−.07			.03		
Statistics for step		.034	.034		.055	.055
Step II						
History of childhood illness	−.07			−.04		
Body shape (ages 13−14)	.35***			.50***		
Statistics for step		.153***	.187***		.275***	.330***
Step III. Parent observed						
Conflict with father (ages 13–18)	.34**			.30**		
Conflict with mother (ages 13–18)	−.10			.10		
Statistics for step		.070*	.257***		.089***	.419***

Note. β weights are from the final full model.
* $p < .05$. ** $p < .01$. *** $p < .001$.

accelerated biological aging at age 30, even after accounting for the covariates described earlier. Scatterplots depicting these relationships are presented in Supplemental Figure A.

Hypothesis 2: High levels of conflict with mothers and with fathers across adolescence as reported by parents will predict accelerated aging after accounting for the covariates described earlier. Using the same approach described for Hypothesis 1, the role of adolescents' levels of conflict with both their fathers and their mothers (as reported by each of those parents, respectively) was assessed next. As shown in Table 2, the level of conflict with fathers, but not with mothers, significantly predicted faster biological aging at age 30, even after accounting for covariates.

Hypothesis 3: Poor conflict resolution skills with close friends in adolescence and high levels of conflictual and punitive behavior with close friends in early adulthood as reported by those friends will predict accelerated aging after accounting for the covariates described earlier. Using the same approach described for Hypothesis 1, the role of low levels of conflict resolution skill in adolescence and high levels of conflictual and punitive behavior with peers in adulthood, all as reported by the participant's closest friend at each period was assessed next. As shown in Table 3, punitive behavior toward peers across early adulthood (ages 21–28) was found predictive of faster biological aging at age 30 even after accounting for covariates for the Klemera–Doubal measure. Punitive behavior was not a significant predictor of the PhenoAge measure in initial analyses. This appeared, however, due to the collinearity of punitive peer behavior and the peer conflict measure, as when the latter measure was removed as a predictor in follow-up analyses, punitive behavior toward peers was predictive of accelerated aging ($\beta = .18$, $p = .02$).

Table 3
Peer-Observed Predictors of Age Acceleration at Chronological Age 30

	Biological age acceleration (at chronological age 30)					
	Klemera–Doubal method			PhenoAge method		
Predictors	β	ΔR^2	R^2	β	ΔR^2	R^2
Step I						
Chronological age	.04			.00		
Gender (1 = M; 2 = F)	−.01			−.24**		
Adolescent family income	−.03			−.01		
Statistics for step		.034	.034		.055	.055
Step II						
History of childhood illness	−.05			−.01		
Body shape (ages 13–14)	.38***			.56***		
Statistics for step		.153***	.187***		.275***	.330***
Step III. Peer observed						
Conflict resolution (age 16)	−.15			−.02		
Punitive behavior toward close friend	.30**			.16		
Conflictual behavior with close friend	−.17			.04		
Statistics for step		.061*	.248***		.016	.346***

Note. β weights are from the final full model.
* $p < .05$. ** $p < .01$. *** $p < .001$.

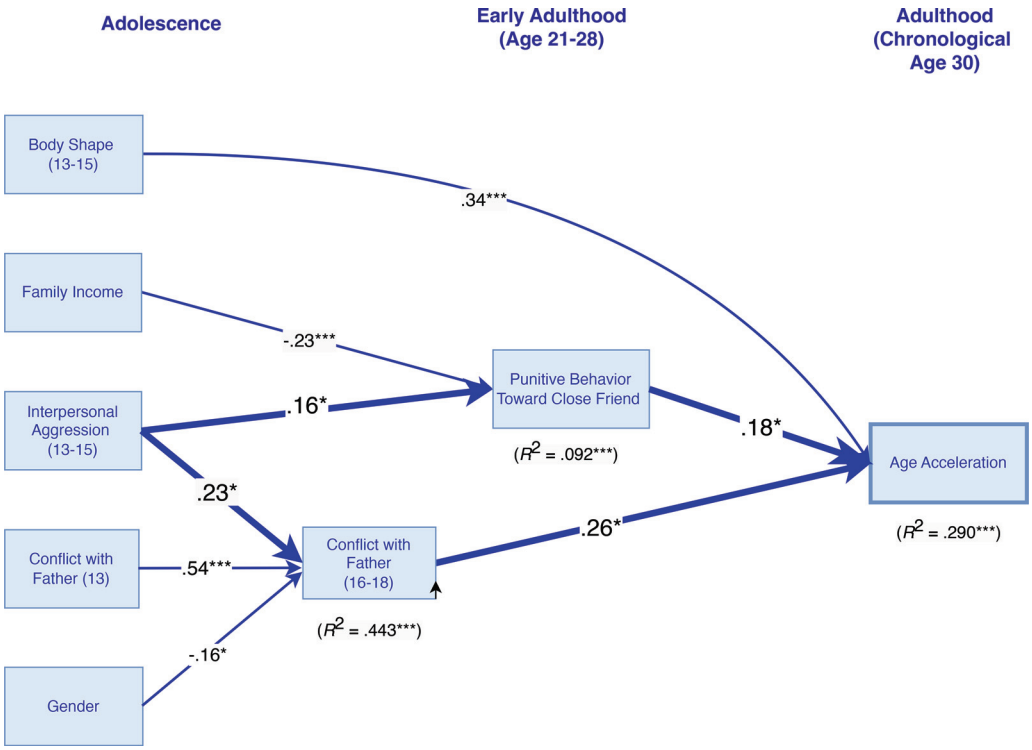
Hypothesis 4: Conflict with parents and peers will potentially play a mediating role in the relation of early adolescent interpersonal aggression to accelerated aging. A path model was used to test Hypothesis 4, considering the prediction of biological aging from the conjoint effects of the predictors identified as significant in Hypotheses 1–3. The model included paths capturing autoregressive effects for conflict with fathers and used lack of conflict resolution skills as a proxy for assessing autoregressive effects regarding punitive behavior toward close friends. All other paths suggested as significant by modification indices were also included. Given the similarities of the two models in the aforementioned analyses, results below were based on the Klemera–Doubal aging measure (the PhenoAge measure yielded highly similar results). The final model fit the data well, $\chi^2(13) = 18.2$, $p = .15$, GFI = 0.96, CFI = 0.96, RMSEA = 0.047, and results are presented in Figure 1. For clarity, the figure does not depict nonsignificant paths nor correlations among constructs assessed contemporaneously. These results suggested that interpersonal aggression in adolescence predicted future increases in levels of conflict with fathers, as well as future punitive behavior toward close friends. Conflict with fathers and punitive behavior toward close friends then each added unique variance to the prediction of accelerated biological aging after accounting for adolescent-era BMI. After accounting for conflict with fathers and punitive behavior toward peers, the direct effect of adolescent interpersonal aggression on biological aging became nonsignificant ($\beta = .07$, $p = .48$). Notably, high levels of conflict with fathers were also predicted by lower family income in adolescence. Punitive behavior toward close friends was also predicted by male gender.

Hypothesis 5: Predictors of accelerated aging will also predict adult BMI, even after accounting for body shape ratings in adolescence. A path model was used to examine whether the identified behavioral predictors of early aging might also predict adult BMI after accounting for a strongly related proxy measure of body shape in adolescence. This model included all of the aforementioned identified predictors of biological aging, using the same analytic approach described for Hypothesis 4. The final model fit the data well, $\chi^2(6) = 8.82$, $p = .18$, GFI = 0.98, CFI = 0.99, RMSEA = 0.051. Results were nearly identical to those seen in Figure 1, indicating that even accounting for adolescent adiposity, early interpersonal aggression predicted future conflict with fathers and punitive behavior with friends, which in turn predicted future BMI by age 28–29 (see Supplemental Figure B).

Discussion

This study found that adolescent interpersonal aggression beginning at age 13 was predictive of accelerated biological aging at chronological age 30 after accounting for adolescent gender and baseline measures of family income, body shape, and prior history of serious illness. There was also evidence that subsequent conflict in relationships with fathers and with peers potentially mediated this effect, and that these subsequent conflicts also predicted participants' greater adult BMI. Each of these

Figure 1
Predicting Accelerated Aging



Note. See the online article for the color version of this figure.
* $p < .05$. *** $p < .001$.

findings is discussed below along with consideration of the limitations of the study.

The long-term prediction of accelerated aging from adolescent interpersonal aggression is consistent with prior work on effects of hostility in adulthood, although focused more tightly on interpersonal behaviors as opposed to intrapsychic anger, cynicism, and hostility. In addition, although a great deal of work on the effects of hostility has focused on risk for cardiovascular disease, this study observed predictions from interpersonal aggression to far more comprehensive indicators of overall physiological functioning assessed via a wide range of biomarkers. These composite measures of aging have been found to be a better predictor of future illness and mortality than chronological age, current illness, epigenetic aging markers, or any other single risk factor (Belsky et al., 2020; Kwon & Belsky, 2021).

These findings are conceptually consistent with prior research findings predicting from antisocial and rule-breaking behavior in childhood to accelerated biological aging (Langevin et al., 2022). They also are consistent with and extend previous findings from the present sample linking poor social integration in adolescence to accelerated epigenetic aging by age 30 (Allen et al., 2023). They establish links not only to a stronger measure of overall biological aging but also to a different factor than in the prior study (interpersonal aggression vs. general social integration). Together, these findings build and extend the case that a range of different types of social difficulties, from rule-breaking behavior to lack of social integration to, in the current study, interpersonal aggression well prior to adulthood may be implicated in broad future lifespan health risks. They also help rule out the reverse causal explanation that it is reactions to concurrent health difficulties in adulthood that drive observed links to interpersonal aggression and relationship struggles (Miller et al., 1996).

A second key finding of this study was that the effects of early interpersonal aggression appeared potentially mediated by conflict in relationships with parents and punitive behavior toward peers as reported by those parents and peers. Although data did not permit testing of a fully lagged mediational model including all variables, what we were able to find was that once later markers of punitive behavior and conflict were considered, the predictions from adolescent-era aggression were no longer significant. Thus, although we do not show that adolescent aggression is driving the later relationship behaviors (and indeed it is likely that this aggression was itself a result of prior family, personality, and genetic factors), we do show that its relation to future aging appears to exist only to the extent that it also predicts similar future social difficulties.

This study also appears to be the first to observe links between conflict with parents (fathers in this case) in adolescence and accelerated aging later in adulthood. Notably, only conflict with fathers, not mothers, was found predictive. One explanation for this difference, consistent with findings from research on model organisms, is that father care may have unique effects on the development of the adolescent nervous system (Danoff et al., 2023). Alternatively, from the perspective of social safety theory, males' greater physical strength and propensity for aggression may make conflict with fathers particularly stressful. Prior work has also found that attachment to fathers but not mothers uniquely predicts aggression (Booth-Laforce et al., 2006) suggesting that perhaps father-adolescent conflict portends future interpersonal aggression. Each of these findings is consistent with the notion that fathers, though less often studied than mothers (Lamb & Lewis, 2013), may play a particularly large role in influencing the adolescent's future interactions in the larger social world beyond the family (Phares & Compas, 1992; Shulman et al., 1997).

Peer reports of punitive behavior by the participant from age 21 to 28 were also predictive of accelerated aging, although reported levels of simple peer conflict were not. One explanation for this divergence is that punitive behavior is a better marker of actual interpersonal aggression than simple levels of conflict. These findings are consistent with prior research findings that marital violence is in general significantly more deleterious than simple conflict (Cummings, 1998). It may be that the hostility and aggression embodied in punitive behavior create a relationship context that is inherently unsafe. Alternatively, given that it was the participants' own punitive behavior (not that of their peers) that was predictive, it may also be that these findings are simply an external marker of intense felt hostility and that it was this felt hostility that had deleterious effects. Overall, these findings regarding peer-reported punitive behavior further bolster the connection of interpersonal aggression to accelerated aging, extending prior findings based on marital relations and self-reports to now cover relationships outside of familial and romantic contexts.

This study was also able to assess the prediction of one key physiological variable, adult BMI, which itself has been previously linked to negative health outcomes. After accounting for a rating of body shape in adolescence, this study found evidence that the same mediated process as was identified in predicting accelerated aging was also found to predict adult BMI by age 27–28. One qualification to this finding is that the approach of assessing body shape in adolescence is at only a proxy measure for adolescent adiposity, though one that has demonstrated strong correlations to BMI in prior research on adolescent samples. This is thus one of the first studies to identify *relational* predictors of BMI in adulthood while controlling for a measure of adiposity assessed in adolescence. As with predictions of accelerated aging, both conflict with fathers and punitive behavior toward close friends predicted future BMI, with the latter two factors appearing as potential mediators of the effect of early adolescent interpersonal aggression. Given extensive evidence linking high adult BMI to health difficulties (Nuttall, 2015) and its extensive links to a number of components of biological aging (Levine et al., 2018), these findings have potential importance in identifying a mechanism by which hostile conflict may lead to future health difficulties.

Although not a focus of the study, male gender and lower family income in adolescence also predicted accelerated aging. Notably, in path analyses, the effects of gender and family income were fully accounted for by higher levels of conflict with fathers for males and by higher levels of punitive behavior toward peers for individuals from families with lower incomes. These results add support to the idea that behavioral and relationship factors may at least partly mediate the effects of structural environmental factors on health. Nonetheless, it remains quite possible that other markers of environmental stress might have more direct effects on health than those measured.

One limitation of the current findings is that the measures of biological aging used in this study are relatively new, although they have shown great promise to date (Belsky et al., 2020; Langevin et al., 2022). In addition, the amount of variance predicted over and above

an adolescent body shape measure used as a proxy for adiposity was relatively modest. Confidence in these results is nevertheless bolstered by the near-complete replication of these findings across two different algorithms for compiling the biomarkers in this study.

It should also be made clear that none of the predictions observed was sufficient to establish causal relations among predictors and accelerated aging. It is quite plausible that other unmeasured factors drove both the predictors and aging processes. More specifically, concurrent levels of aggression and hostility were not assessed, and the results in Figure 1 make clear that it is quite likely that any effects of adolescent interpersonal aggression are mediated via subsequent intervening interpersonal factors. Even taking these predictions at face value, they still also leave unclear whether the operative element is actual interpersonal aggression vs. an underlying hostile attitude, or some combination of the two.

Overall, these results extend our understanding of links between interpersonal aggression and health by showing that interpersonally aggressive behavior as early as adolescence is predictive of comprehensive markers of accelerated aging by age 30, and by showing that nonself-report indices of conflict and aggressive behavior beyond adolescence potentially mediate this relationship. They make clear that relationship difficulties beginning in adolescence are, at minimum, markers of long-term risks for potential health difficulties. If further replicated, these results suggest there may be value in early intervention to address these difficulties with potential payoffs not simply for adolescent mental health but also for long-term physical well-being.

Abstracto

Objetivo: Este estudio examinó la agresión interpersonal en la adolescencia temprana, el conflicto posterior con los padres y la agresión hacia compañeros cercanos como predictores del envejecimiento biológico acelerado a los 30 años. ***Métodos:*** Los participantes (N = 123; 46 hombres y 75 mujeres) fueron evaluados repetidamente, junto con sus padres y amigos cercanos, de edades de 13 a 30 años. ***Resultados:*** Se observó que la agresión interpersonal en la adolescencia temprana predecía un envejecimiento acelerado posterior incluso considerando el género, los ingresos familiares, los problemas de salud previos y las clasificaciones corporales en la adolescencia. Los análisis de trayectoria sugirieron que los efectos de la agresión interpersonal temprana estaban posiblemente mediados por los mayores niveles de conflicto entre padres e hijos adolescentes, reportados por los padres en la adolescencia, y por la conducta agresiva hacia compañeros cercanos, reportada por estos últimos en la adultez temprana. Los análisis de seguimiento sugirieron que estos mismos factores también predijeron las puntuaciones del índice de masa corporal en la edad adulta, considerando la forma corporal en la adolescencia. ***Conclusiones:*** Los resultados se interpretan como evidencia de que las dificultades sociales con implicaciones para la salud a lo largo de la vida pueden identificarse a partir de la adolescencia temprana, lo que resalta la importancia potencial de las intervenciones tempranas para abordar estas dificultades.

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Received November 22, 2024
Revision received September 1, 2025
Accepted September 3, 2025 ■