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Do Children Who Develop Faster Go on to Age Faster in Midlife?

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Abstract

We quantified the association between rate of child development and pace of midlife aging in the Dunedin Study and the Add Health Study. Physical development was quantified in Dunedin participants (N = 1,037; age 45) using a composite comprised of (1) birth weight for gestational age, (2) toddler weight gain, (3) timing of behavioral milestones, (4) adiposity-rebound age, (5) timing of adolescent growth spurt, and (6) tempo of adolescent growth spurt. Social development was quantified using the Vineland Social Maturity Scale. Multi-organ-system (cardiovascular, metabolic, renal, immune, dental, pulmonary) longitudinal biological aging was used as the adult outcome. Fast physical development in childhood was associated with faster midlife aging. This was replicated in Add Health (N = 1,537; age range = 33–44 years), where girls who menstruated earlier had faster midlife aging. By contrast, faster social development in childhood was associated with slower midlife aging. Findings contribute initial empirical evidence characterizing the link between child-development rate and adult aging in humans.

Keywords

child development, aging, biological aging, life course, puberty

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Introduction

Development and aging have historically been conceptualized as distinct and studied separately (Feltes et al., 2015). Development is defined as growth and gain of function during early life, whereas aging is defined as decay and loss of function in later life (Gladyshev, 2021). Accordingly, studies on factors that influence development or aging are usually restricted to populations of younger or older people, respectively.

Yet findings from longitudinal studies have shown that aspects of child development predict aspects of adult aging (Ben-Shlomo, 2013), prompting more exploration linking these two lifespan extremes. One question concerns whether rate of development is related to pace of aging in humans. It is well established that humans exhibit individual differences in the rate of development in early life; humans also exhibit individual differences in trajectories of aging in later life. However, the extent to which these two phenomena are linked remains poorly

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understood. Nonhuman animal models have shown that faster early-life growth precedes faster later-life decline (Yuan et al., 2023). In mice, rapid growth and early reproductive effort is associated with faster aging and shorter lifespans, but this stable pace throughout the lifespan has not been documented in humans. Early-life development is also more complicated in humans compared with other animals, as demonstrated by established literatures in many domains (e.g., physical development, social development).

Because birth-to-late-life studies are rare, it is unclear whether rate of development predicts subsequent pace of aging, and if so, whether this association is positive, negative, or context dependent. This question is relevant for psychological scientists. For psychologists developing behavioral interventions to improve children's health, it is important to quantify potential later-life impacts. For psychologists focused on adult health, it is important to understand antecedents to aging phenomena. And for researchers, this question has theoretical importance: For example, the *developmental origins of health and disease* framework states that biological health is programmed antenatally and shapes both child development and adult aging (Barker, 1990). As another example, *life history theory* in psychology posits that faster development might maximize reproductive success, but the trade-off might be poorer health and faster aging (Nettle & Frankenhuis, 2020). More evidence about the association between development and aging can expand and qualify existing theories.

To our knowledge, there are no studies that relate rate of child development and pace of adult aging in the same individuals. There are several good reasons for this. A study linking rate of development to pace of aging would require three elements. First, it would need repeated, direct measures of development throughout participants' first decades of life. Currently, studies on antecedents of adult health typically index development using one variable, such as birth weight or pubertal timing (Jáni et al., 2022; Wang et al., 2022; Zhang et al., 2023). These variables may be good proxies for development, but they do not capture developmental rate. For example, birth weight could be used as one of many variables in a study measuring developmental rate. But using birth weight on its own may introduce variation that is unduly influenced by factors like maternal height or intrauterine space (Trojner Bregar et al., 2016).

Second, a study linking rate of development to pace of aging would need repeated, direct measurements of aging throughout participants' final decades. Currently, studies that relate child development to adult health use outcomes like disease or mortality, instead of aging itself (Wang et al., 2022). It is challenging to use aging as an outcome because there is little agreement on its definition (Gladyshev et al., 2024). One definition of aging, proposed by geroscientists, is *gradual, progressive, multiorgan system decline* (Kirkwood, 2005). The geroscience definition of aging can be operationalized using longitudinal, repeated measurements of organ functions (D. W. Belsky et al., 2015; Elliott et al., 2021) and can be proxied using DNA methylation in studies that lack longitudinal measurements (D. W. Belsky et al., 2022; Moqri et al., 2023; Rutledge et al., 2022).

Finally, a study linking development and aging would need a prospective, longitudinal design following the same individuals from birth through adulthood. Currently, many studies that relate child development to adult health collect outcome data in adulthood and a retrospective report of the childhood variable (Felitti et al., 1998; Saleh et al., 2017). One limitation of this approach is survivor bias; these studies miss data from individuals who did not survive long enough to be recruited as adults. Longitudinal studies with repeated assessments from an intact birth cohort are needed to mitigate survivor bias.

Here, we aimed to quantify whether an individual's rate of development in childhood can predict their pace of aging in adulthood. We used data from the Dunedin Study, a prospective, population-representative longitudinal cohort following participants from birth to midlife. First, we used six measures to create a composite of physical development. Then we related children's rate of physical development to their pace of adult aging, a biological-aging measure that tracks within-individual, multiorgan-system decline across adulthood in the same Dunedin participants (Elliott et al., 2021). We also benchmarked childhood physical development against social development, to test whether associations between development and aging differed on the basis of domain. Finally, we report a conceptual replication in Dunedin and the National Longitudinal Study of Adolescent to Adult Health (Add Health) of whether fast development, assessed as early menarche, predicts adult aging.

Research Transparency Statement

General disclosures

Conflicts of interest: T. Moffitt and A. Caspi are named as inventors on a license issued by Duke University for DunedinPACE. The algorithm to calculate DunedinPACE is publicly available on Github (https://github.com/danbelsky/DunedinPACE). No other authors have conflicts of interest to report. **Funding:** The Dunedin Study is supported by the U.S. National Institute on Aging (Grant Nos. R01AG032282 and R01AG073207) and by the U.K. Medical Research Council (No. MR/X021149/1). The Dunedin Multidisciplinary Health and Development Research Unit

is supported by the New Zealand Health Research Council (Programme Grant 16-604) and the New Zealand Ministry of Business Innovation and Employment (MBIE). The Add Health Study is funded by Grant R01MD013349 from the National Institutes of Health National Institute on Minority Health and Health Disparities to K. M. Harris and A. Aiello. General support was provided by Grant No. P2CHD050924 from the Carolina Population Center. This research uses data from Add Health, funded by Grant No. P01HD31921 from the National Institute of Child Health and Human Development (NICHD) to K. M. Harris, with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by R. A. Hummer (University of North Carolina at Chapel Hill) and funded by National Institute on Aging cooperative agreements U01AG071448 to R. A. Hummer and U01AG071450 to A. Aiello and R. A. Hummer at the University of North Carolina at Chapel Hill. Add Health was designed by J. R. Udry, P. S. Bearman, and K. M. Harris at the University of North Carolina at Chapel Hill. **Artificial intelligence:** No artificial-intelligence-assisted technologies were used in this research or the creation of this article. **Ethics:** Dunedin Study protocol is approved by the New Zealand Health and Disability Ethics Committee and the Duke University Campus Institutional Review Board. The Add Health study was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill. **Computational reproducibility:** The computational reproducibility of the results in the main article (but not the Supplemental Material) has been independently confirmed by the journal's STAR team. **Open Science Framework (OSF):** To ensure long-term preservation, we registered all OSF files at 10.17605/OSF.IO/EF8G4.

Study 1 disclosures

Preregistration: No aspects of this research were preregistered. **Data:** Data are not publicly available because of confidentiality concerns. Data for this paper are available through managed access (https://sites.duke.edu/moffittcaspiprojects/data-use-guidelines/). Information on how to access Dunedin Study data is provided in the linked data-access table (https://doi.org/10.17605/OSF.IO/FE7VM, under "Materials") or in the Dunedin Study's policy and procedure manual (https://dunedinstudy.otago.ac.nz/for-investigators/policy-statement-code-of-practice). **Materials:** Some study materials are publicly available (https://doi.org/10.17605/OSF.IO/FE7VM). The Vineland Social Maturity Scale items, administration manual, and scoring guide are proprietary to the Educational Test Bureau. To protect test security, these materials are not publicly available. **Analysis scripts:** All analysis scripts are publicly available (https://doi.org/10.17605/OSF.IO/FE7VM). Analyses reported here were checked for reproducibility by an independent data analyst, who used the manuscript to recreate the statistical code and applied it to a fresh copy of the data set.

Study 2 disclosures

Preregistration: No aspects of this research were preregistered. **Data:** Add Health survey data for a subsample of participants are available publicly (https://addhealth.cpc.unc.edu/data/public-use-data-sets/). Survey data for the full sample are available upon request for registered and approved users of restricted data. Add Health epigenetic data are not publicly available because of confidentiality concerns. Add Health genomic data are available through two methods: restricted use, or dbGaP. Information on how to access Add Health epigenetic data through both methods is provided in the linked data-access table (https://doi.org/10.17605/OSF.IO/FE7VM, under "Materials"). **Materials:** All study materials are publicly available (https://doi.org/10.17605/OSF.IO/FE7VM). **Analysis scripts:** All analysis scripts are publicly available (https://doi.org/10.17605/OSF.IO/FE7VM).

Study 1: Modeling the Rate of Child Development and Estimating Its Association With the Pace of Adult Aging

Method

Sample. Participants are members of the Dunedin Multidisciplinary Health and Development Study, a longitudinal birth cohort. The cohort includes all individuals born between April 1972 and March 1973 in Dunedin, New Zealand, who participated in the first follow-up assessment at age 3 ($N = 1{,}037$; 91% of eligible births; 52% male). Assessments were carried out at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, 38, and most recently 45 years, when 94% ($n = 938$) of the 997 study members still alive were assessed.

At each assessment, study members are brought to the Dunedin research unit for a full day of interviews and examinations. The study member's written informed consent is obtained, with study protocol approval by the New Zealand Health and Disability Ethics Committee and Duke University.

The cohort is primarily New Zealand European; 8.6% at age 45 self-identified as having Māori ethnicity. The cohort represents the full range of socioeconomic status in the general population of New Zealand's South Island. As adults, the cohort's members match the New Zealand National Health and Nutrition Survey on key health indicators (e.g., body mass index, smoking, physical activity, physician visits) and the New Zealand census of citizens the same age on educational attainment (Poulton et al., 2022; Richmond-Rakerd et al., 2020).

Child measures of physical development. We included six measurements of physical development in childhood: (1) birth weight for gestational age, (2) weight change from birth to age 3, (3) timing of adiposity rebound, (4) timing of adolescent growth spurt, (5) tempo of adolescent growth spurt, and (6) behavioral milestones (i.e., age of walking, talking, daytime toilet training) reported by parents of study members. We selected these measures because they are all commonly used measures of growth in pediatric settings (Moore et al., 2019; World Health Organization, 1995, 2006). We used these six variables to create a composite measure of developmental rate, and we then also separately investigated each variable's association with our adult pace-of-aging variable, described later.

Height and weight measurements. Birth weight for gestational age was calculated by dividing each study member's birth weight in kilograms (from hospital records) by their gestational age in days. Each study member's weight was measured to the nearest 0.1 kg using calibrated scales at every subsequent time point. Weight change from birth to age 3 was calculated by subtracting study members' birth weight from their age-3 weight measurement.

We also measured each study member's height to the nearest millimeter using a portable stadiometer at each time point (Harpenden; Holtain, Ltd., Dunedin, New Zealand). We calculated each study member's body mass index (BMI) from measures of height and weight. BMI measurements were used in a multilevel longitudinal model to fit individual growth curves of BMI across ages 3 to 13 (Singer & Willett, 2003). Growth curves included linear and quadratic slope curves and were adjusted for sex. From this, we extracted timing of adiposity rebound for each study member, defined as the age at which they reached the nadir of the curve (D. W. Belsky et al., 2012).

We used a nonlinear mixed-effects model to fit individual logistic growth curves to height across ages 3 to 26 years. From this, we extracted each study member's indicators of timing and tempo of adolescent growth spurt. Timing of adolescent growth spurt was defined as the age at which each study member was exactly halfway between the lower and upper asymptotes of their height curve. Tempo of adolescent growth spurt was defined as the rate, with respect to age, at which each study member progressed from the lower to upper asymptote of their height curve.

Behavioral milestones. When Study members were age 3 years, their parents were interviewed about when study members reached three early childhood behavioral milestones: first instance of walking (defined as taking 6 steps), talking (defined as using 6 words), and toilet training (defined as staying dry all day for at least 6 days of the week). We coded the age at which the study member reached each milestone in months. If the child had not reached the milestone by the age-3 study visit, their score was recorded as 44 months (n = 5 children for walking, n = 6 children for talking, and n = 77 children for toilet training). Scores for each milestone were significantly intercorrelated, with coefficients between 0.21 and 0.33. For the current analysis, we summarized the three behavioral milestone variables. We binned each milestone into quintiles and derived the mean of the three binned milestone variables for each Dunedin Study child.

Child measure of social development. We included one measure of social development in childhood. When Dunedin children were aged 3 years, their mothers were asked to complete the Vineland Social Maturity Scale (Doll, 1953). These items examined whether the child was able to complete tasks in the following categories: self-help skills (e.g., dresses self, eats, asks to go to the toilet), communication (e.g., says short sentences, identifies familiar objects), socialization (e.g., plays with other children, "performs" for others), locomotion (e.g., walks around house unattended, uses stairs), and occupation (e.g., marks with pencil/crayon, uses scissors, helps with chores).

Midlife measure of the pace of aging. Our outcome variable was pace of midlife biological aging, measured using a composite score of physiological deterioration across organ systems derived from 19 biomarker measurements from Dunedin Study participants repeated at ages 26, 32, 38, and 45 years. This measure shows evidence of validity in the Dunedin Study, previously published in Elliott et al. (2021). The 19 biomarkers were BMI (kg/m^2), waist-to-hip ratio, hemoglobin A1c, leptin, mean arterial pressure, cardiorespiratory fitness, forced expiratory volume in first second of expiration (FEV1), FEV1 to forced vital capacity ratio, total cholesterol, triglycerides, high-density lipoprotein cholesterol, apolipoprotein B100 to A1 ratio, lipoprotein a, creatinine clearance, urea nitrogen, C-reactive protein, white blood cell count, periodontal disease, and caries-affected tooth surfaces. The linear change in each biomarker for each study member was assessed using linear mixed-effects modeling; the 19 resulting slopes were then summed and scaled so that 1 year of chronological age equated to approximately 1 year of mean change in physiological functioning in the sample. We called this biological aging score the *pace of aging*.

Notably, 955 study members have a pace-of-aging score in this report (more than the 938 who took part in the age-45 assessment), because, in addition to the 938 study members with complete data up to age 45, we were also able to generate a pace-of-aging score for 17

additional study members who were seen up to age 38, but who were not seen at age 45.

Statistical analysis. To operationalize rate of development, we created a composite using the six child-development indicators described above. Because higher values on some indicators correspond to slower developmental rate (e.g., later behavioral milestone attainment, later age at adiposity rebound, later age at growth spurt), these variables were reverse-coded. We then standardized each child-development variable on the full sample. Next, for each study member, we calculated the average of all six standardized indicators, creating a single continuous measure of developmental rate. This composite captures each study member's relative deviation from the cohort-average developmental rate.

We performed linear regression analyses with all variables standardized to a mean of 0 and a standard deviation of 1. We present standardized regression coefficients for the association between the developmental-rate composite and midlife biological aging. Because there are well-documented sex differences in child growth (Hoyt et al., 2020; Kokštejn et al., 2017; Tanner et al., 1976), we also present models adjusting for sex.

To be included in analyses, we required that participants have data on at least one child-development indicator and the age-45 pace-of-aging measure, resulting in an analysis sample of 955. Statistical analyses were performed using R statistical software (version 4.2.2; R Core Team, 2025). Analyses reported here were checked for reproducibility by an independent data analyst, who used the manuscript to recreate the statistical code and applied it to a fresh copy of the data set.

Results

Of the original 1,037 study members, 997 were still alive at the age-45 assessment, and 938 (94.1%) of these study members were assessed. Attrition analyses comparing the full cohort, those alive at age 45, and the study sample showed no statistically significant differences in childhood IQ and childhood socioeconomic status, suggesting no differential drop-out on those variables over the course of the longitudinal study (see Fig. S1 in the Supplemental Material available online).

Correlations between the six child-development variables (for the full sample and stratified by sex) can be found in Table 1. There were no sex differences in correlations between child-development variables.

Faster childhood physical development predicts faster adult aging. Overall, children who had faster physical development went on to age faster in midlife, β = 0.09, 95% confidence interval, or CI =[0.03, 0.16], $p = .005$ (Fig. 1). After we controlled for sex, the association was unchanged, β = 0.11, 95% CI = [0.04, 0.18], $p = .003$. The effect size was significant but modest, indicating that a 1-*SD* faster rate of child physical development predicted a faster pace of aging in midlife by about one tenth of a standard deviation.

Because the rate-of-child-development profiles reflect physical-growth indicators, and BMI is one of 19 biomarkers in the outcome measure of adult pace of aging, we probed whether associations between the rate of child development and pace of aging are primarily the result of associations between physical growth in childhood and adult BMI. We recalculated pace of aging without BMI and regressed this new variable on the developmental-rate composite. The association between faster physical development and faster adult pace of aging, while controlling for sex, remained (β = 0.10, 95% CI = [0.03, 0.17], $p = .003$; see Fig. S2).

In secondary analyses, we tested whether any of the child development measurements were especially important in the prediction of midlife biological aging in linear regression models controlling for sex (Table 2). Variables reflecting physical growth best predicted adult pace of aging. Children who reached adiposity rebound earlier (β = −0.17, 95% CI = [−0.24, −0.11], $p < .001$) and children who grew faster during their adolescent growth spurt (β = 0.09, 95% CI = [0.01, 0.17], $p = .035$) had faster adult pace of aging (Fig. 2). Physical-growth variables that were less strongly associated with adult pace of aging were birth weight for gestational age (β = −0.04, 95% CI = [−0.11, 0.03], $p = .253$), weight change from birth to age 3 (β = 0.06, 95% CI = [−0.01, 0.12], $p = .087$), and timing of adolescent growth spurt (β = −0.06, 95% CI = [−0.14, 0.03], $p = .178$). Parent-reported age at which Dunedin Study children reached behavioral milestones was least related to adult pace of aging (β = −0.01, 95% CI = [−0.08, 0.05], $p = .722$) and was the only child-development variable that did not rely on a measurement of physical growth. There were no sex differences in the association between the child-development variables and midlife pace of aging (Fig. S3).

In Figure S4 and Table S1, we report a complementary analysis that used latent profile analysis to assign Dunedin children to groups on the basis of their rate of development. The results were similar when developmental rate was operationalized using this alternative approach.

Comparing childhood physical development to social development. We examined the association between rate of social development (measured using the Vineland Social Maturity Scale) and midlife pace of aging, controlling for sex: β = −0.13, 95% CI = [−0.20, −0.05], $p = .001$. Dunedin children with faster social development compared with their same-age peers went on to age

Table 1. Correlations Among Child Development Variables in the Full Sample and Stratified by Sex

		Birth weight/ gestational age	Weight gain, birth–age 3	Milestones	Age at adiposity rebound	Age at growth spurt	Tempo of growth spurt
Birth weight for gestational age	Full sample	—					
	Full sample (sex control)	—					
	Male	—					
	Female	—					
Weight gain birth–age 3	Full sample	0.09	—				
	Full sample (sex control)	0.07	—				
	Male	0.06	—				
	Female	0.09	—				
Age at milestone attainment	Full sample	−0.08	−0.01	—			
	Full sample (sex control)	−0.10	−0.03	—			
	Male	−0.09	−0.05	—			
	Female	−0.12	−0.01	—			
Age at adiposity rebound	Full sample	0.00	−0.02	0.05	—		
	Full sample (sex control)	−0.00	−0.02	0.06	—		
	Male	−0.02	0.00	0.06	—		
	Female	0.02	−0.03	0.06	—		
Age at growth spurt	Full sample	−0.01	−0.31	0.15	0.19	—	
	Full sample (sex control)	−0.11	−0.55	0.10	0.29	—	
	Male	−0.09	−0.54	0.10	0.29	—	
	Female	−0.13	−0.56	0.10	0.28	—	
Tempo of growth spurt	Full sample	−0.18	−0.21	−0.11	−0.13	−0.59	—
	Full sample (sex control)	−0.14	−0.13	−0.04	−0.19	−0.33	—
	Male	−0.12	−0.08	−0.05	−0.23	−0.45	—
	Female	−0.17	−0.22	−0.03	−0.15	−0.16	—

Note: This table depicts correlations between child-development variables—for the full sample, for the full sample controlling for sex, for male participants only, and for female participants only. There were no sex differences in correlations among child-development variables. Numbers are Pearson correlation coefficients.

slower in midlife. This can be compared with the association between rate of physical development (measured using the developmental-rate composite) and midlife pace of aging, controlling for sex, which was $\beta = 0.11$, 95% CI = [0.04, 0.18], $p = .003$. Social development and physical development appear to have opposite, but similar-magnitude, associations with adult pace of aging.

Study 2: Conceptual Replication in the Dunedin and Add Health Studies

The novel association between fast development and fast aging in Dunedin was observed in one cohort. Thus, we conducted a replication in a more contemporary cohort in a different country. We used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a longitudinal, multilevel, nationally representative sample of more than 20,000 U.S. adolescents (Harris et al., 2019). The Add Health cohort is slightly younger than the Dunedin cohort. At the last Add Health interview (Wave V in 2016–2018), participants were aged 33 to 44 ($M = 38$). To proxy the rate of development in Dunedin and Add Health, we used menarcheal age. To proxy the longitudinal pace of aging in Add Health, we used the DunedinPACE epigenetic clock.

Method

Samples. We included 449 female Dunedin Study participants and 1,537 female Add Health participants in analyses; inclusion criteria are described in more detail below. Add Health participants were recruited from middle

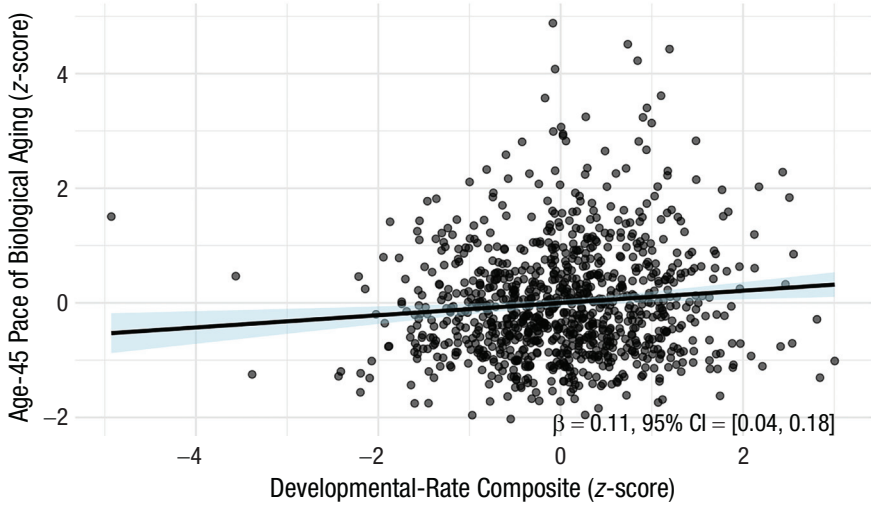

Fig. 1. Children's developmental-rate composite and midlife pace of aging. Linear regression including sex as a covariate showed that children who scored higher on a composite measure of developmental rate had faster pace of aging at age 45, β = 0.11, 95% CI = [0.04, 0.18], $p < .01$. The shaded area is the 95% confidence interval. CI = confidence interval.

and high schools in 1994 to 1995 at ages 11 to 21 years and completed up to five in-person interviews, most recently when participants were aged 33 to 44 years (Harris et al., 2019). Add Health participants represent a race and ethnicity distribution comparable to the U.S. population, but the present analyses included only White participants, to match the demographics of the Dunedin Study. Add Health participants provided written informed consent, and the study was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill.

Measure of menarche. To proxy the rate of child development in Dunedin and Add Health, we used age at menarche. In the Dunedin Study, each girl in the cohort was asked at age 15 to recall the age when she experienced her first menstrual period (*menarche*) in years and months. Nineteen girls had not reached menarche yet at age 15 and were asked again at age 32. Two study members were missing information about menarche. In total, 480 Dunedin girls (96% of the total 502 girls in the cohort) had data for menarche. The distribution of menarcheal age in Dunedin girls is roughly normal: Reported age at menarche ranged from 8.5 to 16.5 years ($M = 13.04$, $SD = 1.11$, $mdn = 13$ years of age).

During the Add Health adolescent interviews at Wave I (1995) and Wave II (1996), girls were asked whether they had ever had a menstrual period, and if yes, how old they were at their very first menstrual period in months and years. About 7% of Add Health girls had not yet reached menarche when the Add Health sample was aged 12 to 20 at Wave II. The distribution of menarcheal age in Add Health girls is normal. Reported age at menarche ranged from 7 to 17 years ($M = 12.22$, $SD = 1.24$, $mdn = 12$ years of age).

Table 2. Earlier Age of Adiposity Rebound and Faster Tempo of Adolescent Growth Spurt Had the Strongest Associations With Faster Pace of Biological Aging in Midlife

	Association with Midlife Biological Aging	
Child-Development Variable	Estimate [95% CI]	*p* value
Birth weight for gestational age	−0.04 [−0.11, 0.03]	.25
Weight gain from birth to age 3	0.06 [−0.01, 0.12]	.09
Age at milestone attainment	−0.01 [−0.08, 0.05]	.72
Age at adiposity rebound	**−0.17 [−0.24, −0.11]**	**< .0001**
Timing of growth spurt	−0.06 [−0.14, 0.03]	.18
Tempo of growth spurt	**0.09 [0.01, 0.17]**	**.04**

Note: Bold denotes statistical significance at the $p = .05$ level. Numbers are standardized regression coefficients (β) and 95% confidence intervals. All child-development variables were residualized for sex.

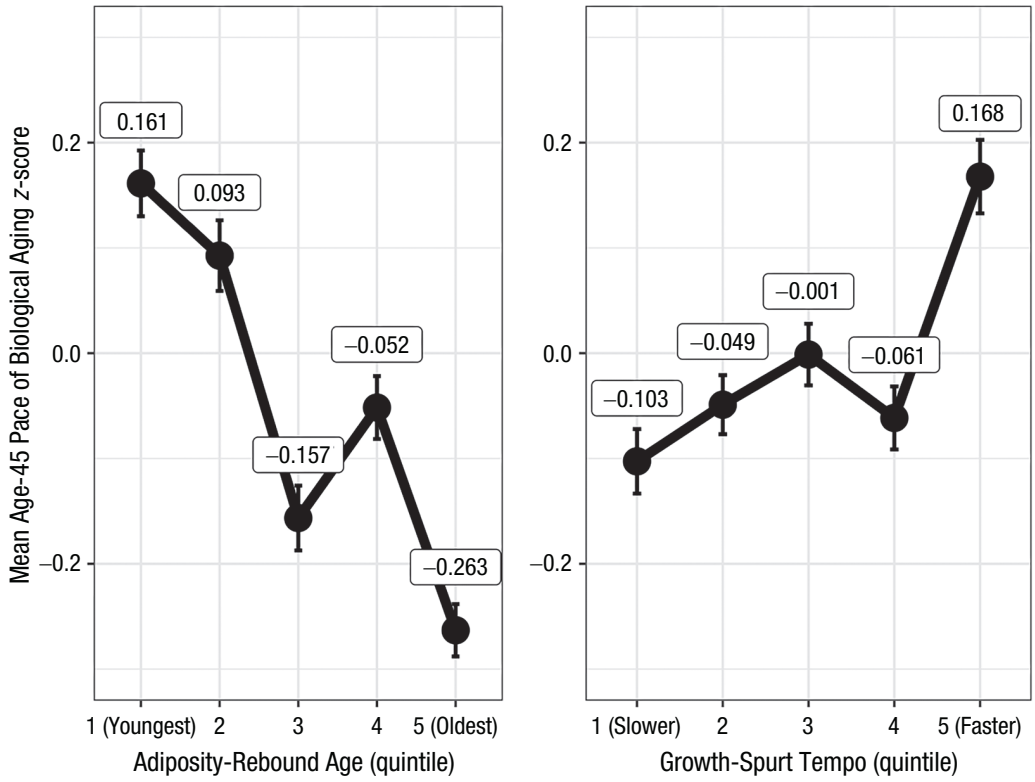

Fig. 2. Children who reached adiposity rebound earlier and grew faster during adolescence spurt had faster midlife pace of aging. Linear regressions including sex as a covariate showed that children who reached adiposity rebound earlier had faster pace of aging at age 45 (β = −0.17, 95% CI = [−0.24, −0.11], $p < .0001$). Children who grew faster during their adolescent growth spurt had faster pace of aging at age 45 (β = 0.09, 95% CI = [0.01, 0.17], $p = .04$). Error bars represent the standard error.

Measure of midlife pace of aging. In the Dunedin Study, midlife pace of aging was measured using our previously published and validated measure of biological aging, termed *pace of aging* and described earlier. In brief, pace of aging tracks longitudinal multi-organ-system decline measured at ages 26, 32, 38, and 45 years.

To quantify pace of aging in Add Health, we used the DunedinPACE algorithm applied to blood DNA methylation. DNA methylation is an epigenetic mechanism that contributes to gene-expression regulation and cellular maintenance. Although DNA methylation is heritable, it can be altered by factors in the environment, including aging processes (Horvath & Raj, 2018; Wu et al., 2023). Machine-learning analysis applied to DNA methylation data from blood cells can produce algorithms that accurately estimate an individual's age and risk for disease and death (Hannum et al., 2013; Horvath, 2013; Levine et al., 2018; Lu et al., 2019). We used these methods to develop a DNA methylation algorithm to measure pace of aging (D. W. Belsky et al., 2020, 2022). We called this measure *Dunedin Pace of Aging Computed from the Epigenome* (DunedinPACE). People with faster DunedinPACE values exhibit increased risk for disease and disability and have shorter lifespans (D. W. Belsky et al., 2022; Bernabeu et al., 2023; Faul et al., 2023). DunedinPACE is also sensitive to exposures that can accelerate aging, such as poverty (Shen et al., 2023), adversity (Felt et al., 2023), and risky lifestyle behaviors (Felt et al., 2023; Harris et al., 2024). Crucially, DunedinPACE can be used in data sets with blood samples, such as Add Health, to estimate individuals' pace of biological aging in the absence of longitudinal physiological profiling (D. W. Belsky et al., 2022).

The Wave V interview in Add Health occurred in 2016 to 2018 when the cohort was aged 33 to 44 years old. Of the 12,300 Wave V participants, 5,381 consented (94% consent rate) to a follow-up in-person home exam, when venous blood was drawn for DNA methylation testing. For details on the methylation sample, data quality, and methods involved in the construction of DunedinPACE in Add Health, see Harris et al. (2024).

Statistical analysis. We tested the association between age at menarche and midlife pace of aging using linear regression models. Because Add Health participants were not all the same age at Wave V, we included a covariate for Wave V chronological age in the Add Health models.

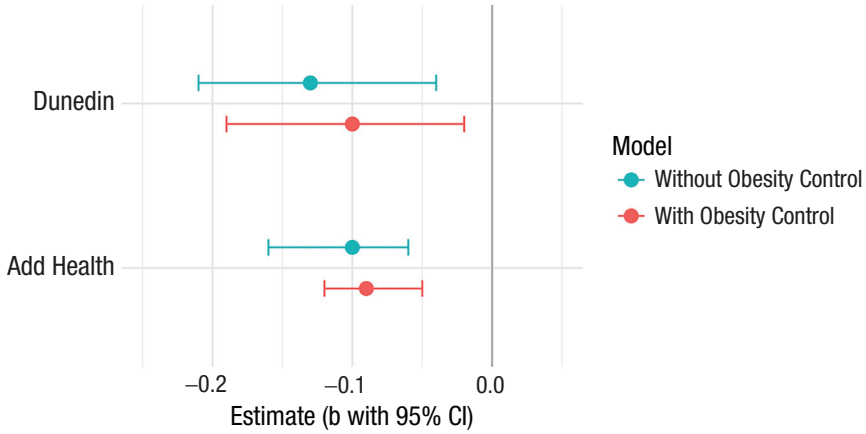

Fig. 3. Girls who experienced menarche earlier had faster adult pace of aging in the Dunedin Study and the Add Health Study. In the Dunedin Study, girls who experienced menarche earlier had faster adult pace of aging (b = −0.13, 95% CI = [−0.21, −0.04], p = .004). This finding was replicated in Add Health using DunedinPACE (b = −0.10, 95% CI = [−0.16, −0.06], p < .001). After controlling for obesity in adolescence, associations were modestly attenuated but remained statistically significant (b = −0.10, 95% CI = [−0.19, −0.02], p = .020 in Dunedin; b = −0.09, 95% CI = [−0.12, −0.05], p < .001 in Add Health).

Because obesity is related to both earlier menarche and faster aging, we also identified girls who were obese in adolescence, and we controlled for this in our models. Obesity was defined at age 13 in the Dunedin Study and at Waves I and II (ages 11–21) in Add Health, using the U.S. Centers for Disease Control and Prevention BMI thresholds. Participants were classified as obese if their BMI was at or above the 95th percentile for their specific age in Add Health and using the cutoff point BMI ≥ 25.46 for girls in Dunedin (Janssen et al., 2005).

In Dunedin, the analysis sample was n = 449 girls who had data on menarche, pace of aging, and BMI. In Add Health, the analysis sample was n = 1,537 girls who had data on menarche, DunedinPACE, and BMI, and who reported their race as White, to match the demographics of Dunedin participants.

Results

In the Dunedin Study, girls who experienced earlier menarche had faster adult pace of aging (b = −0.13, 95% CI = [−0.21, −0.04], p = .004). This finding was replicated in Add Health using DunedinPACE (b = −0.10, 95% CI = [−0.16, −0.06], p < .001). After controlling for obesity in adolescence, associations were modestly attenuated but remained statistically significant (b = −0.10, 95% CI = [−0.19, −0.02], p = .020 in Dunedin; b = −0.09, 95% CI = [−0.12, −0.05], p < .001 in Add Health; Fig. 3). For every 1-year acceleration in menarcheal timing, girls in both cohorts experienced about a 0.1-*SD* acceleration in adult pace of aging, even after controlling for obesity in adolescence. To synthesize findings across Dunedin and Add Health, we conducted a random-effects meta-analysis of the association between menarcheal timing and adult pace of aging (b = −0.11, 95% CI = [−0.15, −0.06], p < .001; Fig. S6).

Discussion

In two longitudinal cohorts, children with faster physical development went on to age faster in midlife. Findings provide initial empirical evidence of the association between fast early-life physical development and fast later-life aging in humans, adding to existing evidence in nonhuman animal models (Yuan et al., 2023). When Dunedin children's developmental rate was summarized using a composite of six physical growth indicators, faster-developing children experienced faster midlife pace of biological aging, compared with their slower-developing peers. The effect size was modest, but striking because it spanned decades of life. An effect size of 0.11 is comparable to those of established early-life risk factors for accelerated aging, such as childhood disadvantage; Aroke et al. (2024) found that children with lower household income had faster DunedinPACE (r = −.17, p = .02), and Raffington et al. (2021) found that children living in more disadvantaged families and neighborhoods had faster DunedinPoAm, a previous iteration of DunedinPACE (r = .18, p = .001). This highlights fast physical development as an additional predictor of aging processes.

Findings further showed that children who were more socially mature went on to age more slowly than their peers, an opposite but similar-magnitude effect compared with what was found for physical development. This adds

to previous research showing that more advanced psychosocial development in midlife is associated with better multidomain functioning in late life (Landes et al., 2014; Malone et al., 2016), although biological aging was not explored in these studies. Nonetheless, the present findings suggest that the protective effects of advanced social development on later aging may take root earlier in the lifespan, in childhood.

When developmental rate was operationalized as menarcheal age in Dunedin and Add Health, every 1-year acceleration in menarcheal timing predicted a 0.1-*SD* acceleration in adult aging. This finding adds to three previous reports linking early menarche to accelerated aging. Two studies used saliva for DNA methylation (DNAm) analyses instead of whole blood. In the Texas Twin Project ($N = 285$, ages 8–18), girls who had experienced menarche had faster DunedinPoAm compared with girls who had not ($r = .10$, $p = .04$; Raffington et al., 2021). In the Healthy Passages study, earlier pubertal timing was associated with faster DunedinPACE when participants were age 19.7 ($\beta = 0.08$, $p < .01$; $n = 1{,}213$) and again at age 27 ($\beta = 0.19$, $p < .001$; $n = 350$; Goering et al., 2024, 2025). Because DunedinPACE was developed using whole blood, the validity of measuring it in saliva is not established. The third study used whole blood and found that women ($n = 183$, ages 25–51) with earlier menarche had higher GrimAge (DunedinPACE not used; $\beta = -0.23$, $p = .005$; Hamlat et al., 2021). Conversely, a study using the National Health and Nutrition Examination Survey ($n = 1{,}033$, ages 50–84) found that retrospectively reported menarcheal age was not associated with any epigenetic clocks, including DunedinPoAm ($\beta = 0.0004$, p value not reported; Daredia et al., 2025).

The present study offers several strengths. First, Dunedin and Add Health are population-representative cohorts with minimal attrition throughout their 45 and 25 respective years of investigation, allowing the opportunity to link development to aging with minimal survivor bias. Second, all variables were direct measurements rather than retrospective self-reports. This is an asset among child-growth studies and preserves measurement accuracy. Third, benchmarking childhood physical development against childhood social development is a test of whether it is *all* fast development that predicts faster adult aging, or whether the association is specific to fast physical development. This is a question that has not been possible to address in nonhuman animal models. Finally, our aging variable directly measures longitudinal biological aging from young adulthood to midlife and captures the aging process itself rather than merely disease incidence or mortality.

This study has limitations. First, Dunedin and Add Health participants represent developed countries and are mainly white. Research in developing countries has shown that as conditions improve, faster development reflects improved health that results in slower aging (De Onis et al., 2012; Nti et al., 2024). Additionally, researchers studying *life-history theory in psychology* argue that the co-occurrence of fast development and aging takes place only with sufficient nutrients to sustain rapid growth (Ellis et al., 2024). Hence, there is a need to qualify the development-aging connection using samples from differently resourced countries. Second, the measures of physical development in the Dunedin Study, used to construct the developmental-rate composite, show relatively modest intercorrelations. This may suggest that the indicators capture somewhat different facets of physical maturation (e.g., skeletal growth, adiposity gain, motor-function gain) and raise questions about the usefulness of a composite. For this reason, we report both the association between the developmental-rate composite and adult pace of aging as well as associations between the composite's constituent indicators and adult pace of aging. Third, the replication in Add Health using menarcheal age and DunedinPACE did not include boys, because there is not a measure of puberty for Dunedin boys that is analogous to the measure of menarcheal age for girls. Finally, participants were in midlife, so it remains to be seen how development relates to aging as samples approach older age.

We do not claim that fast physical development *causes* fast aging, or that fast social development *causes* slow aging. Instead, our findings suggest that despite conventional assumptions that development and aging are distinct, there is a connection between these extremes of the lifespan. Whether this connection reflects common mechanisms or distinct but parallel mechanisms during development and aging warrants future research. We propose several reasons for the connection between development and aging that warrant investigation.

First, there may be genes that influence both child development and adult aging. *Antagonistic pleiotropy theory* states that natural selection favors genes that enhance early-life fitness, even if they cause faster decline (Williams, 1957). Accordingly, studies have identified alleles that enhance early-life survival but increase later-life disease (Lockwood et al., 2024). Nascent research has identified genetic overlap between precocious puberty and shorter lifespan (Hollis et al., 2020). Early-life survival and precocious phenotypes may be proxies for fast physical development, but they do not reflect developmental rate itself. Genetic studies capturing rate of development itself are needed.

Second, physical development and aging may both be accelerated by adversity. Childhood adversity has been associated with faster aging in many cohorts (Cheng et al., 2024; Hamlat et al., 2023; Joshi et al., 2023; Schmitz et al., 2023), including Dunedin (Bourassa et al., 2023).

However, the empirical association between childhood adversity and developmental rate is less clear. On the one hand, adversity has been associated with faster physical development in the form of earlier puberty (Ellis & Essex, 2007; Hamlat et al., 2021). On the other hand, adversity has been linked to slower physical development in the form of stunting (Gunnar & Quevedo, 2007; Walker et al., 2007). It is possible that different types and timing of adversity have different impacts on the rate of development (McLaughlin & Sheridan, 2016).

Third, *life-history theory in psychology* also argues that certain psychosocial traits are associated with both child development and adult aging (Del Giudice, 2014). For example, fast development is observed in children who have a more immediate reward orientation, higher impulsivity, and lower prosociality (Del Giudice, 2014; Han & Chen, 2020). These traits are thought to be adaptive in stressful environments, but at a cost to health, which can take the form of faster aging (J. Belsky, 2024; Kruger, 2021). Outside of life-history-theory framing, these traits have been empirically linked to faster aging (Richmond-Rakerd et al., 2021). By contrast, more advanced social development can enhance cooperation, social support, and self-regulation (Eisenberg et al., 2009). These traits may allow individuals to buffer stress and protect against accelerated aging. Future research may integrate the biological and psychosocial processes involved in the link between child development and adult aging.

This study contributes novel evidence linking fast physical development with fast aging in humans, in two longitudinal cohorts. Findings represent initial evidence that rate of child development is connected to pace of adult aging, conceptualizing these constructs as interconnected. Future research should continue to leverage lifespan longitudinal data to characterize the link between development and aging and explore mediating mechanisms.

Transparency

Action Editor: Julia Stern
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Declaration of Conflicting Interests

T. Moffitt and A. Caspi are named as inventors on a license issued by Duke University for DunedinPACE. The algorithm to calculate DunedinPACE is publicly available on Github (https://github.com/danbelsky/DunedinPACE). No other authors have conflicts of interest to report.

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Artificial Intelligence

No artificial-intelligence-assisted technologies were used in this research or the creation of this article.

Ethics

Dunedin Study protocol is approved by the New Zealand Health and Disability Ethics Committee and the Duke University Campus Institutional Review Board. The Add

Health study was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill.

Open Practices

Open practices for this article are described in the Research Transparency Statement section, which appears at the end of the Introduction section in the main text.

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Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/09567976261483545

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