Childhood Assets and Cardiometabolic Health in Adolescence

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abstract

BACKGROUND: Research shows that the development of cardiometabolic disease can begin early in life with risk factors accumulating over time, but less is known about protective pathways to positive health. In this study, we use prospective data to test whether childhood assets predict a greater likelihood of being in optimal cardiometabolic health by age 17.

METHODS: Data are from 3074 participants in the Avon Longitudinal Study of Parents and Children (mean age = 17.8). Four childhood assets were prospectively assessed via cognitive tests and parent report when children were between ages 8 and 10: strong executive functioning skills, prosocial behaviors, and low levels of internalizing and externalizing problems. Cardiometabolic health was assessed at ages 9 and 17 by using a composite dysregulation score derived from multiple biological parameters, including cholesterol, blood pressure, C-reactive protein, insulin resistance, and BMI. Associations between assets and optimal health at age 17 (ie, a dysregulation score of $\leq$1) were evaluated with Poisson regression models with robust error variances.

RESULTS: After controlling for covariates (including sociodemographics, correlates of cardiometabolic health, and dysregulation scores at age 9), participants with multiple assets were 1.08 to 1.27 times more likely to be in optimal cardiometabolic health at age 17 compared with those with 0 or 1 asset. Each additional asset conferred a 6% greater likelihood of optimal health over time (relative risk = 1.06 [95% confidence interval: 1.01 to 1.11]).

CONCLUSIONS: Childhood assets predicted cardiometabolic health with seemingly cumulative impacts. Identifying early assets may provide novel targets for prevention and elucidate pathways to positive adult health.

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WHAT'S KNOWN ON THIS SUBJECT: Previous work has found associations between positive psychosocial factors in childhood and cardiovascular health in adulthood, but few studies have examined similar relationships earlier in the life course to assess when potential protective effects may begin to emerge.

WHAT THIS STUDY ADDS: In this study, we found that having more versus fewer childhood assets predicted a greater likelihood of optimal cardiometabolic health by late adolescence. Identifying early protective factors may offer novel insights into future targets for cardiovascular disease prevention.

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Cardiovascular disease (CVD) is the leading cause of mortality worldwide, accounting for >30% of global deaths.[1] Although most children are born with optimal cardiovascular health, less than a quarter of individuals possess it by adulthood.[2] Research on the childhood origins of CVD has revealed that early life factors influence cardiovascular risk over the life course.[3] To date, work in this area has focused primarily on the negative impact of early adversity,[4] while the potential protective effects of childhood assets, such as interpersonal resources (eg, parental warmth) and intrapersonal competencies (eg, effective emotion regulation) have received less attention. Assets reflecting positive cognitive and psychological functioning have been shown to predict academic achievement and thriving in adolescence[5,6] but remain understudied with respect to the establishment of early trajectories of physical health.

Previous research indicates that childhood assets, such as attention regulation and cognitive ability, are associated with favorable cardiovascular health at midlife,[7–10] but it is unclear whether protective effects are evident before adulthood. Research on early life adversity has found that it is associated with poorer cardiometabolic profiles in childhood and adolescence,[4,11,12] suggesting that psychosocial-related biological alterations are observable in the first decades of life. Because risk factors in childhood contribute to early health deteriorative processes, it is plausible that assets may serve a health-promoting function as youth transition to adulthood. Therefore, our goal for this study was to test whether positive childhood assets predict a greater likelihood of being in optimal cardiometabolic health by late adolescence.

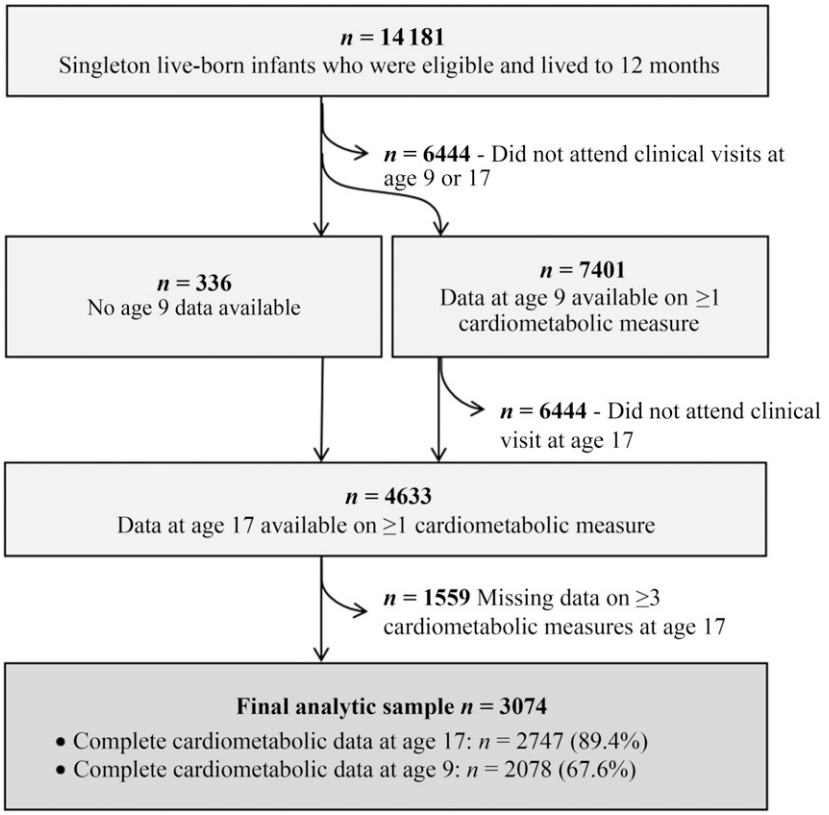

FIGURE 1
Flowchart of study sample composition.

METHODS

Sample

Data are from the Avon Longitudinal Study of Parents and Children (ALSPAC) in England.[13–15] Between April 1991 and December 1992, 14 541 women who were pregnant were enrolled, and the health and development of their children was assessed prospectively through age 17.[16] Additional participants were enrolled in the study when children were 7 years old, resulting in 15 458 total participants. The eligible sample for the current study was a total of 14 181 singleton live-born children who lived past 12 months and did not have an acute infection at ages 9 or 17 (C-reactive protein [CRP] levels of >10 mg/L). The final analytic sample included 3074 participants with available data on at least 4 cardiometabolic measures at age 17. The sample composition over the study period is depicted in the flowchart provided in Fig 1.

Data were collected through questionnaires administered periodically to mothers and children starting during pregnancy and continuing through age 17. Biological measures on child participants were obtained through clinical assessments conducted every 2 years from ages 9 to 17. Detailed information on all data can be accessed on the study Web site.[17] All participants' parents provided written, informed consent for their child to take part in the study, and children also assented to data collection starting at age 9.[16] Research protocols were approved by the ALSPAC Law and Ethics Committee and Local Research Ethics Committee.

Measures

Childhood Assets

Four assets reflecting positive cognitive and psychological functioning were considered: (1)

strong executive functioning (EF) skills, (2) prosocial behaviors, (3) low levels of internalizing problems (eg, being withdrawn or anxious), and (4) low levels of externalizing problems (eg, being aggressive or hyperactive). EF skills were directly measured by using tasks in which children's cognitive functions were assessed, whereas prosocial behavior and internalizing and externalizing problems were assessed through maternal report at age 9 by using the validated Strengths and Difficulties Questionnaire (SDQ).[18,19]

EF Skills

Participants completed computer-based tasks in which 5 discrete EF skills were assessed at 2 points in childhood. At age 8, measures of selective attention, dual attention, and attentional control were obtained by using tasks from the validated Test of Everyday Attention for Children assessment tool.[20] At age 10, participants' working memory was assessed by using the Counting Span Task,[21,22] and inhibitory control was assessed by using the Stop Signal Task.[22–24] More information on EF tasks is provided in the Supplemental Information.

Because EF comprises several related yet distinct skills,[25,26] a composite EF asset measure was constructed by combining data on participants' performance on each task following a procedure similar to that used in previous work on childhood self-regulation.[27] First, to identify participants with a high level of proficiency in each domain, EF measures were dichotomized at the quintile of the sample distribution representing the highest level of task performance. The number of domains in which children performed well were summed to create an EF score, with higher scores indicating skill in multiple EF domains. Children who were top performers on ≥3 domains were considered to have strong EF skills.

Prosocial Behaviors

At age 9, participants' mothers completed the 5-item prosocial subscale of the SDQ,[18,19] endorsing statements about their child's positive behaviors in the previous 6 months on a 3-point scale from 0 (not true) to 2 (certainly true). Items were summed to create a summary score, with higher values reflecting a stronger prosocial tendency (α = .68). Participants scoring within the average range defined in the SDQ manual (≥6) were defined as prosocial.

Low Internalizing or Externalizing Problems

Following previous work, internalizing behaviors were assessed by summing the emotional problems and peer problems subscales on the SDQ (α = .73).[28] Following SDQ manual criteria, we defined low levels of internalizing problems as having scores below the top quintile of the sample distribution (≤4). Externalizing behaviors were measured as the sum of conduct problems and hyperactivity subscale scores (α = .77). We defined low levels of externalizing problems as having scores below the top quintile of the sample distribution (≤6).

Total Childhood Assets

Individual binary assets were summed into a categorical measure of total assets (0–1, 2, 3, or 4 assets). This served as the primary predictor in all analyses.

Cardiometabolic Health at Age 17 Years

The American Academy of Pediatrics recently recommended that clinicians assess youth health profiles on the basis of elevated levels of multiple biological risk markers.[29] Therefore, cardiometabolic health was defined by the absence of multiple dysregulated cardiometabolic parameters. Data for each parameter was collected on-site following standard study protocols[16,30] (see Supplemental Information). A composite measure of cardiometabolic health was created by using continuous data on fasting high-density lipoprotein cholesterol (HDL-C) (millimoles per liter), fasting non–high-density lipoprotein cholesterol (nHDL-C) (total cholesterol − HDL-C; millimoles per liter), systolic blood pressure (SBP) (millimeters of mercury), diastolic blood pressure (DBP) (millimeters of mercury), the homeostatic model assessment of insulin resistance (HOMA-IR) ([fasting glucose × fasting insulin]/22.5),[31] CRP (milligrams per liter), and BMI (kilograms per meter squared). First, continuous measures were dichotomized to indicate dysregulation on the basis of the unhealthiest quintile of the sample distribution for each parameter (eg, ≥80th percentile for nHDL-C, SBP, DBP, HOMA-IR, CRP, and BMI; ≤20th percentile for HDL-C). Scores used to define the unhealthiest quintiles in ALSPAC were largely consistent with thresholds identified in pediatric populations, when available (see Supplemental Table 5). Therefore, for each parameter, dysregulation captured both clinical and subclinical risk. The number of dysregulated parameters was then summed to create an overall dysregulation score (0–7), with higher scores indicating poorer health. Participants were considered to be in optimal cardiometabolic health if they had dysregulated levels on 0 or only 1 parameter (ie, no evidence of clustered dysregulation). More information on the derivation of dysregulation scores is provided in the Supplemental Information.

Cardiometabolic Health at Age 9 Years

During clinical visits at age 9, nonfasting blood samples were collected, precluding the assessment of insulin resistance. Therefore, the parameters used to construct a measure of cardiometabolic health at age 9 included nonfasting nHDL-C

TABLE 1 Distribution of Study Variables Used to Compare Participants Included in the Final Analytic Sample (n = 3074) With Those Who Were Lost to Follow-up (n = 11 107)

	Participants		P[a]
	Final Sample, n (%)	Lost to Follow-up, n (%)	
Total sample	3074 (21.7)	11 107 (78.3)	—
Child characteristics			
Girls	1585 (51.6)	5315 (47.9)	<.001
White race	2891 (95.1)	9993 (94.1)	.006
Birth wt <2500 g	116 (4.0)	468 (4.5)	.2
Childhood chronic condition	298 (11.6)	633 (12.8)	.1
Cardiometabolic dysregulation score of ≥2 at age 9 y	681 (32.8)	1042 (38.5)	<.001
Experienced puberty by 10 y	376 (16.7)	776 (18.5)	.08
Family characteristics			
Parent history of cardiometabolic disease	1247 (43.8)	3595 (39.2)	<.001
Mother with overweight or obesity prepregnancy	501 (19.0)	1785 (21.3)	.009
Maternal education			<.001
Below ordinary level	502 (17.6)	3102 (34.0)	
Ordinary level	919 (32.3)	3223 (35.3)	
Advanced level	849 (29.8)	1860 (20.4)	
University or higher	579 (20.3)	952 (10.4)	
Parental manual labor job	917 (38.5)	3513 (51.8)	<.001
Experienced poverty by clinical visit at 9 y	691 (33.7)	2688 (55.9)	<.001

Sample sizes are based on observed values and may vary because of missing data. —, not applicable.
[a] Calculated by using the χ^2 test.

(millimoles per liter), nonfasting HDL-C (millimoles per liter), SBP (millimeters of mercury), DBP (millimeters of mercury), CRP (milligrams per liter), and BMI (kilograms per meter squared). By using the same procedures described previously, cardiometabolic measures at age 9 were dichotomized to reflect dysregulation on the basis of the unhealthiest quintile of the distribution for each parameter, then summed to create a dysregulation score ranging from 0 to 6.

Covariates

Child- and family-level covariates were assessed by maternal-completed questionnaires. Child confounders included sex, precise chronological age at the age 9 clinical visit, and experiencing puberty by age 10 (Tanner stage 2 or higher, determined by pubic hair growth). Family-level confounders included maternal educational attainment (below ordinary level, ordinary level, advanced level, or university or higher), parental manual labor occupation based on the highest social class reported by either parent (categories III–V of the 1991 British Office of Population and Census Statistics classification), and living in poverty during the participant's childhood (weekly family income of <£200 at age 3, 4, 7, or 8 years). Known correlates of future cardiometabolic health included the child's birth weight (grams), presence of chronic conditions by age 10 (diabetes, asthma, or epilepsy), family history of cardiometabolic disease (ie, diabetes, coronary heart disease, or hypertension), and maternal prepregnancy BMI (kilograms per meter squared). Adolescent health behaviors that may serve as pathway variables included youth-reported, past–30-day cigarette use and weekly alcohol consumption at age 17.

Statistical Analysis

We compared the distribution of study covariates among participants in the final analytic sample with that among participants lost to follow-up by using χ^2 tests. Missing data due to participant attrition was accounted for by using a combination of multiple imputation (MI) and inverse probability weighting (IPW) (see Supplemental Information for more information).[32]

We then examined bivariate associations between total assets and study covariates by using χ^2 tests. Because most participants were healthy at age 17, multivariable associations were assessed by using Poisson regression models with robust error variances to minimize bias in the estimation of risk ratios.[33] All results from Poisson regression analyses were exponentiated for interpretation as relative risk (RR) (ie, likelihood) of being in optimal cardiometabolic health at age 17. Associations between assets and health were tested by using 4 sequentially adjusted regression models, accounting for confounders, correlates of future cardiometabolic health (including cardiometabolic dysregulation scores at age 9), and adolescent health behaviors that may serve as pathway variables. Potential sex differences were evaluated by introducing an interaction term and stratification.

Sensitivity analyses were used to examine whether specific cardiometabolic parameters or specific assets accounted for most of the observed relationships. Separate

TABLE 2 Descriptive Statistics of Study Sample by Total Childhood Assets

	0–1 Asset, *n* (%)	2 Assets, *n* (%)	3 Assets, *n* (%)	4 Assets, *n* (%)	*P*[a]
Total sample	153 (8.4)	359 (19.8)	1144 (63.0)	159 (8.8)	—
Child characteristics					
Sex					.008
Girls	63 (6.7)	184 (19.5)	602 (63.7)	96 (10.2)	
Boys	90 (10.3)	175 (20.1)	542 (62.3)	63 (7.2)	
Birth wt, g					.9
<2500	7 (10.6)	14 (21.2)	40 (60.6)	5 (7.6)	
≥2500	140 (8.4)	327 (19.7)	1045 (62.9)	149 (9.0)	
Childhood chronic condition					.8
Yes	20 (9.3)	40 (18.6)	139 (64.7)	16 (7.4)	
No	125 (8.3)	293 (19.5)	947 (63.1)	137 (9.1)	
Cardiometabolic dysregulation at age 9 y					.7
Dysregulation score of 0 or 1	70 (7.5)	185 (19.8)	598 (64.0)	82 (8.8)	
Dysregulation score of ≥2	39 (9.2)	78 (18.4)	269 (63.4)	38 (9.0)	
Experienced puberty by age 10 y					.4
Yes	29 (11.4)	46 (18.0)	156 (61.2)	24 (9.4)	
No	105 (8.1)	257 (19.9)	817 (63.1)	115 (8.9)	
Family characteristics					
Parent history of cardiometabolic disease					.9
Yes	62 (8.0)	152 (19.6)	491 (63.4)	69 (8.9)	
No	84 (8.8)	190 (19.9)	600 (62.9)	80 (8.4)	
Maternal prepregnancy BMI					.8
Overweight or obesity	112 (8.5)	260 (19.7)	832 (63.1)	114 (8.7)	
<25	21 (6.9)	57 (18.8)	198 (65.1)	28 (9.2)	
Maternal education					.2
Below ordinary level	28 (11.3)	53 (21.5)	150 (60.7)	16 (6.5)	
Ordinary level	49 (9.0)	116 (21.3)	341 (62.5)	40 (7.3)	
Advanced level	41 (7.5)	108 (19.7)	346 (63.0)	54 (9.8)	
University or higher	25 (6.5)	65 (17.0)	251 (65.7)	41 (10.7)	
Parental occupation					.05
Manual labor	48 (9.2)	118 (22.6)	317 (60.7)	39 (7.5)	
Non–manual labor	70 (7.3)	172 (18.0)	621 (65.0)	93 (9.7)	
Ever experienced poverty by age 9 y					<.001
Yes	48 (12.7)	90 (23.9)	213 (56.5)	26 (6.9)	
No	71 (7.4)	174 (18.1)	624 (64.9)	93 (9.7)	
Health behaviors in adolescence					
Smoked in past 30 d at age 17 y					.04
Yes	44 (10.8)	93 (22.8)	238 (58.3)	33 (8.1)	
No	27 (7.5)	60 (16.6)	242 (66.9)	33 (9.1)	
Consumed alcohol weekly at age 17 y					.7
Yes	29 (8.4)	60 (17.3)	224 (64.6)	34 (9.8)	
No	93 (8.2)	227 (19.9)	721 (63.2)	100 (8.8)	

Sample sizes are based on observed values and may vary because of missing data. —, not applicable.
[a] Calculated by using the χ^2 test.

Poisson regression analyses were used to evaluate associations between total assets and having healthy levels of each cardiometabolic parameter, controlling for confounders and cardiometabolic correlates. Separate adjusted analyses were also used to evaluate associations between individual assets and total cardiometabolic health. Finally, to assess the robustness of findings, associations between total assets and dysregulation scores were examined by using linear regression. All analyses were conducted by using Stata MP version 15.0 (Stata Corp, College Station, TX).

RESULTS

Sample Description

The average age of the final analytic sample was 17.8 years. Roughly half of participants were girls, and >95% were white. When considering childhood cardiometabolic-related factors, 11.6% of children had a chronic condition before age 10, and nearly half had a family history of cardiometabolic disease. With respect to attrition, participants who remained in the sample were more likely to be socially advantaged and in optimal cardiometabolic health at age 9 compared with those who were lost to follow-up (see Table 1).

The characteristics of the sample according to participants' total childhood assets are shown in

TABLE 3 Associations Between Total Childhood Assets and Cardiometabolic Health at Age 17 Years (n = 3074)

Total Childhood Assets	Cardiometabolic Health at Clinical Visit at Age 17 y							
	Model 1[a]		Model 2[b]		Model 3[c]		Model 4[d]	
	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*
0–1 asset	Reference	—	Reference	—	Reference	—	Reference	—
2 assets	1.09 (0.93 to 1.28)	.3	1.06 (0.91 to 1.25)	.4	1.08 (0.94 to 1.26)	.3	1.11 (0.88 to 1.40)	.4
3 assets	1.16 (1.01 to 1.33)	.04	1.12 (0.97 to 1.29)	.1	1.11 (0.98 to 1.27)	.1	1.16 (0.95 to 1.43)	.1
4 assets	1.36 (1.16 to 1.60)	<.001	1.29 (1.09 to 1.52)	.002	1.27 (1.09 to 1.48)	.002	1.23 (0.96 to 1.57)	.1
Linear trend	1.08 (1.03 to 1.13)	.001	1.06 (1.01 to 1.11)	.01	1.06 (1.01 to 1.11)	.01	1.06 (0.99 to 1.14)	.08

Exponentiated RRs were estimated by using Poisson regression models with robust error variances, and *P* values were calculated by using F-tests. —, not applicable.

[a] Unadjusted.

[b] Adjusted for confounders, including sex, age at baseline, pubertal status at age 10 y, maternal education, experiencing poverty during childhood, and parental manual labor occupation.

[c] Adjusted for confounders in Model 2 as well as correlates of future cardiometabolic health, including childhood chronic conditions (diabetes, asthma, or epilepsy), birth wt, family history of coronary heart disease or diabetes, maternal prepregnancy BMI, and number of dysregulated cardiometabolic parameters at the clinical visit at age 9 y.

[d] Adjusted for covariates in Models 2 and 3 as well as adolescent health behaviors, including smoking status and weekly alcohol consumption at age 17 y.

Table 2. Assets were common, with 71.8% of participants possessing ≥3. No appreciable differences were observed by correlates of cardiometabolic health; however, assets were socially patterned. Having 1 or no assets was more common among boys (χ^2 = 11.9; P = .005), participants who had parents in a manual labor job (χ^2 = 8.0; P = .05), and those who experienced child poverty (χ^2 = 20.4; $P < .001$). Additionally, participants who reported smoking in the past 30 days had fewer assets (χ^2 = 18.7; $P < .001$).

With respect to cardiometabolic health, 67.2% of participants were in optimal health at age 9, compared with 62.0% at age 17. Half of participants (49.2%) maintained good health from childhood to adolescence, and 15.2% were in poor health at age 9 but optimal health by age 17. Controlling for sex, optimal childhood cardiometabolic health was associated with a 1.67 times greater likelihood of optimal health in adolescence (95% confidence interval [CI]: 1.53 to 1.82; $P < .001$).

Childhood Assets and Cardiometabolic Health at Age 17 Years

A positive association between childhood assets and cardiometabolic health at age 17 was observed across all models, with estimates attenuating slightly with increasing levels of adjustment (see Table 3). As assets accumulated, participants were increasingly more likely to be in optimal health at age 17, even after controlling for all study covariates. Tests for a linear trend revealed that each additional asset conferred a 6% greater likelihood of optimal cardiometabolic health over time (95% CI: 1.01 to 1.11; P = .01). Associations were robust to further adjustment for adolescent health behaviors.

Formal tests for interaction by sex were null. However, sex-stratified analyses appeared to yield larger estimates for boys (see Supplemental Fig 2).

Sensitivity Analyses

Associations between total childhood assets and individual cardiometabolic parameters were in expected directions, with more assets associated with an equal or greater likelihood of being within a healthy range for each cardiometabolic parameter (see Table 4). The largest associations were evident among participants who possessed all 4 assets. Although estimates appeared to be comparable across parameters, the strongest protective associations were observed in relation to SBP and BMI. Mean levels of cardiometabolic parameters by total assets are provided in the Supplemental Information. For all other parameters (except nHDL-C), associations were less pronounced, but having 4 assets was protective. Analyses by individual asset revealed that EF skills (RR = 1.15; 95% CI: 1.05 to 1.26) had the strongest association with cardiometabolic health (see Supplemental Table 7).

Associations between assets and health defined by continuous dysregulation scores supported our initial findings. Participants with more assets had less cardiometabolic dysregulation at age 17 (fully adjusted linear trend β = −.11; P = .05; full results provided in Supplemental Table 5).

DISCUSSION

Our goal for this study was to test whether childhood assets predict positive cardiometabolic health in adolescence. Considering 4 psychological and behavioral assets measured in childhood, children with multiple assets were more likely to have optimal cardiometabolic health at age 17 compared with those with ≤1. Associations persisted after accounting for children's cardiometabolic health at study baseline and controlling for relevant covariates. Our results are consistent with previous research on childhood assets and adult cardiovascular health,[7,8] but extend that work by demonstrating that protective effects may be observed earlier in the life course than previously appreciated.

TABLE 4 Likelihood of Being Below the Unhealthiest Quintile for Individual Cardiometabolic Parameters at Age 17 Years, Adjusted for Confounders and Correlates of Cardiometabolic Health (n = 3074)

Total Childhood Assets	Healthy Range of Cardiometabolic Health Parameters at Clinical Visit at Age 17 y						
	nHDL-C, RR (95% CI)	HDL-C, RR (95% CI)	SBP, RR (95% CI)	DBP, RR (95% CI)	CRP, RR (95% CI)	HOMA-IR, RR (95% CI)	BMI, RR (95% CI)
0–1 asset	Reference	Reference	Reference	Reference	Reference	Reference	Reference
2 assets	0.99 (0.91 to 1.08)	1.03 (0.93 to 1.13)	1.05 (0.96 to 1.15)	1.02 (0.93 to 1.12)	1.03 (0.94 to 1.12)	1.03 (0.93 to 1.14)	1.04 (0.95 to 1.15)
3 assets	1.00 (0.93 to 1.08)	1.03 (0.95 to 1.12)	1.08[a] (1.00 to 1.17)	1.04 (0.96 to 1.14)	1.01 (0.94 to 1.09)	1.05 (0.96 to 1.15)	1.09[a] (1.00 to 1.18)
4 assets	0.99 (0.89 to 1.10)	1.09 (0.98 to 1.21)	1.10[b] (1.00 to 1.21)	1.09 (0.98 to 1.21)	1.08 (0.98 to 1.19)	1.09 (0.97 to 1.21)	1.13[b] (1.02 to 1.26)
Linear trend	1.00 (0.97 to 1.03)	1.02 (0.99 to 1.05)	1.03[a] (1.00 to 1.06)	1.03[b] (1.01 to 1.06)	1.01 (0.98 to 1.04)	1.02 (0.99 to 1.05)	1.04[b] (1.01 to 1.07)

Exponentiated RRs were estimated by using Poisson regression models with robust error variances, and P values were calculated by using F-tests.

[a] $P \leq .10$.

[b] $P \leq .05$.

Also consistent with previous findings,[3] the accumulation of assets appears to drive positive health additively from childhood to adolescence, much like the accumulation of risk factors contributes to health deterioration over time. Sensitivity analyses used to evaluate associations with individual cardiometabolic parameters revealed comparable positive associations, suggesting childhood assets may have protective effects in multiple physiologic systems.

This study has some limitations. Given that this is observational research, causality cannot be determined conclusively. However, we included numerous covariates through a series of nested adjustment models, and all models yielded consistent estimates. Selective attrition is also a potential concern. Study retention was socially patterned because children who were disadvantaged were less likely to remain in the study than youth who were more advantaged. Although previous work suggests that estimates are only slightly affected by attrition from the ALSPAC,[34] we adopted a rigorous approach to account for missing data, combining MI and IPW techniques. Lastly, assets were only assessed between ages 8 and 10. Because repeated measurements were not collected, we were unable to examine the stability of assets or whether acquiring assets during adolescence was associated with better cardiometabolic health over time.

This study also has numerous strengths. Data were from a large community-based sample of parents and children and collected at various time points beginning in fetal life and continuing through adolescence. Cardiometabolic measures were obtained during clinical visits starting in childhood, with data available at the time assets were assessed. Additionally, a rich array of information on health, social factors, and behaviors over the follow-up period was available, making it possible to adjust for many potential confounders and correlates of cardiometabolic health.

If replicated, our results could have important policy implications. Of the assets examined, EF skills appeared to have the strongest impact on participants' future health. Effective interventions targeting this asset have been developed, and previous work has shown that EF skills can be improved in childhood with targeted enrichment.[35] Programs that successfully improve EF skills include computer-based training, martial arts, and the Tools of the Mind and Montessori preschool curricula.[35] These programs are touted for improving school readiness and reducing social disparities in education,[36] but our findings suggest that they may also contribute to reducing cardiometabolic health disparities. Increasing investments in early initiatives to support the development of childhood assets may be a worthwhile future direction for the primordial prevention of CVD.

Our results also have important implications for epidemiological research. The childhood assets included in this study are foundational for developing self-regulation, decision-making skills, and emotional well-being, and are considered critical to lifelong health.[37] Although there is considerable evidence of associations between early psychosocial risk factors and poor cardiometabolic outcomes,[4] no cohort studies have prospectively collected substantive data on childhood assets to identify protective pathways to positive cardiometabolic health. Few studies measure positive functioning, such as children's emotion regulation abilities or positive emotional states. Because of data limitations, we defined 2 assets by the absence of behavior problems, which may not fully capture the unique contributions of

positive psychological functioning to health. Considered in conjunction with emerging research on the health-promoting (and potentially restorative) impact of positive psychological well-being among adults,[38,39] our findings indicate a greater need for large-scale studies to monitor positive factors starting in childhood. A more comprehensive understanding of the distribution of childhood assets in the population and the pathways by which they influence heath and disease over the life course will provide a stronger evidence base with which to inform clinical practice.

CONCLUSIONS

With this study, we contribute to the growing field of positive cardiovascular health[38] and primordial prevention.[40] Although work in this area has historically been focused on adults,[38] we examined pathways earlier in the life course and with respect to a broader array of developmentally informed psychological and behavioral assets. Our findings suggest that early investment in assets may help youth maintain health and combat the accumulation of health-damaging behavioral and biological risk factors over time. Identifying novel targets for early prevention and developing systems to monitor assets in the population are important future directions for pediatric epidemiology.

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ABBREVIATIONS

ALSPAC: Avon Longitudinal Study of Parents and Children
CI: confidence interval
CRP: C-reactive protein
CVD: cardiovascular disease
DBP: diastolic blood pressure
EF: executive functioning
HDL-C: high-density lipoprotein cholesterol
HOMA-IR: homeostatic model assessment of insulin resistance
IPW: inverse probability weighting
MI: multiple imputation
nHDL-C: non–high-density lipoprotein cholesterol
RR: relative risk
SBP: systolic blood pressure
SDQ: Strengths and Difficulties Questionnaire

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