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ORIGINAL RESEARCH

Adolescent Psychological Assets and Cardiometabolic Health Maintenance in Adulthood: Implications for Health Equity

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BACKGROUND: Positive cardiometabolic health (CMH) is defined as meeting recommended levels of multiple cardiometabolic risk factors in the absence of manifest disease. Prior work finds that few individuals—particularly members of minoritized racial and ethnic groups—meet these criteria. This study investigated whether psychological assets help adolescents sustain CMH in adulthood and explored interactions by race and ethnicity.

METHODS AND RESULTS: Participants were 3478 individuals in the National Longitudinal Study of Adolescent Health (49% female; 67% White, 15% Black, 11% Latinx, 6% other [Native American, Asian, or not specified]). In Wave 1 (1994–1995; mean age=16 years), data on 5 psychological assets (optimism, happiness, self-esteem, belongingness, and feeling loved) were used to create a composite asset index (range=0–5). In Waves 4 (2008; mean age=28 years) and 5 (2016–2018; mean age=38 years), CMH was defined using 7 clinically assessed biomarkers. Participants with healthy levels of ≥6 biomarkers at Waves 4 and 5 were classified as maintaining CMH over time. The prevalence of CMH maintenance was 12%. Having more psychological assets was associated with better health in adulthood (odds ratio $[OR]_{linear\ trend}$, 1.12 [95% CI, 1.01–1.25]). Subgroup analyses found substantive associations only among Black participants (OR, 1.35 [95% CI, 1.00–1.82]). Additionally, there was some evidence that racial and ethnic disparities in CMH maintenance may be less pronounced among participants with more assets.

CONCLUSIONS: Youth with more psychological assets were more likely to experience favorable CMH patterns 2 decades later. The strongest associations were observed among Black individuals. Fostering psychological assets in adolescence may help prevent cardiovascular disease and play an underappreciated role in shaping health inequities.

Key Words: cardiometabolic health ■ health assets ■ health equity ■ life course ■ psychological well-being ■ social epidemiology

Cardiovascular disease is the leading cause of death in the United States,[1] and much of this burden is attributable to elevated blood pressure, blood sugar, adiposity, and cholesterol levels in the population.[2] Although the clustering of risk factors is a commonly studied early marker of disease development,[3,4] these cardiometabolic risk indicators are also useful for characterizing levels of healthy functioning among young people with few diagnosable conditions.[5,6] In cardiovascular epidemiology, *positive cardiovascular health* (CVH) is defined as meeting clinical recommendations for multiple cardiovascular disease-related risk factors in the absence of manifest disease (eg, body mass index [BMI] levels between 18.5 and 25 kg/m^2).[7,8] Different terminology is often used to distinguish positive CVH measures on the basis of which risk factors are considered and how thresholds for healthy levels are defined. For example, *ideal/favorable CVH* describes meeting clinical recommendations for ideal levels of 8 biological and behavioral risk factors

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CLINICAL PERSPECTIVE

What Is New?

- This prospective study examined associations between 5 psychological assets (optimism, happiness, self-esteem, belongingness, and feeling loved and wanted) in adolescence and cardiometabolic health assessed using 7 biomarkers measured twice in adulthood.
- Youth with more assets were more likely to reach adulthood in good health and to maintain it over time, and associations were stronger for Black youth.

What Are the Clinical Implications?

- Racial disparities in cardiometabolic health were less pronounced among individuals with more assets, suggesting that efforts to bolser psychological assets early in life may be a useful complement to broader work addressing the structural determinants of health inequities.

Nonstandard Abbreviations and Acronyms

CMH	cardiometabolic health
CVH	cardiovascular health
CV	coefficient of variation

(total cholesterol, blood pressure, blood glucose, BMI, nonsmoking, physical activity, sleep, and diet),[7,9,10] whereas *cardiometabolic health* (CMH; also referred to as clinical CVH[11]) refers only to biological risk factors and may be defined more liberally as the absence of high-risk levels.[5,12,13] Despite these differences, studies consistently find that positive CVH is remarkably rare,[14] with prevalence estimates in the United States ranging from 0.5% to 12% among adults[12,14] and <50% among youth.[5,6] Because adolescence marks an important juncture in the life course when the foundations of adult health are established,[15] it is critical to identify and enhance assets that can support positive CVH trajectories through the transition to adulthood.

Observational and intervention research suggests that facets of psychological well-being (eg, optimism, happiness) may be important modifiable assets[16,17] that promote healthy cardiovascular functioning over time.[8,18,19] However, most of this work was conducted among older adults, despite evidence suggesting that health promoting processes triggered by psychological assets may manifest before adulthood.[20,21] To date, we are aware of only 2 studies that explicitly examined the impact of psychological assets on positive CVH starting earlier in life. In a prospective study of 415 participants in the Collaborative Perinatal Project, 7-year-old children with high levels of multiple assets (including attention regulation and cognitive ability) were over 4 times more likely to have favorable CVH 35 years later.[9] More recent work among three thousand 9-year-olds in the Avon Longitudinal Study of Parents and Children also found that youth with multiple assets (including strong prosocial skills, strong executive functioning skills, and low levels of internalizing and externalizing behaviors) were 8% to 27% more likely to have optimal CMH at age 17.[13] Although these prospective studies found early psychological assets were related to better health later in life, we know of no longitudinal work that examined repeated measures of positive CVH or the likelihood of maintaining positive CVH over time. If similar protective relationships are noted with health maintenance, it may suggest that assets play an active role in preserving positive health states across the life course.

Another gap in the existing literature relates to studies conducted among racially and ethnically diverse samples.[20–24] It is well established that minoritized racial and ethnic groups in the United States have lower levels of positive CVH compared with White populations.[5,25] More specifically, Black and Latinx youth are the least likely to have optimal CMH[5] and sustain it over time[11] compared with youth of other races and ethnicities, highlighting the important role psychological assets may play for these groups in particular. To our knowledge, no studies have examined group differences in the relationship between assets and positive CVH early in life. Work conducted among diverse samples of older adults indicate that assets may confer similar health benefits across racial and ethnic groups,[20–23] but competing theories have also been presented. For instance, cumulative advantage theories posit that assets may disproportionately benefit youth from privileged groups who face fewer structural barriers to translate resources into positive health, whereas resource substitution theories postulate that assets may help socially disadvantaged youth overcome adversity by compensating for the absence of other important health-enhancing factors.[26,27] Furthermore, although past research has evaluated potential effect modification by race and ethnicity, joint interactions[28]—which additionally encompass the extent to which racial and ethnic differences in health may be attributable to differences in the population distribution of psychological assets—remain largely unexamined. These relationships have important implications for primordial prevention, as they may provide greater insight into the role psychological assets can play in mitigating racial and ethnic health inequities.

With that in mind, the goal of this study was 2-fold: (1) to investigate prospective associations between psychological assets in adolescence and

CMH maintenance measured twice over a roughly 10-year period in young adulthood, and (2) to examine potential interactions by race and ethnicity. More specifically, we explored whether associations between assets and CMH maintenance varied by race and ethnicity and also whether racial and ethnic disparities in CMH differed depending on the number of assets individuals possessed in adolescence. Using data from the Add Health (National Longitudinal Study of Adolescent Health), we examined 5 psychological assets—optimism, happiness, self-esteem, belongingness, and feeling loved and wanted—which were selected based on previous cardiovascular-related research[8,20–23] and also on work in developmental psychology linking these assets with better behavioral outcomes for youth.[29,30] With respect to positive CVH, we chose to investigate CMH owing to a lack of adequate information on participants' health behaviors. Extending prior work in Add Health that defined positive CVH using measures of blood pressure, glucose, and cholesterol,[31] we constructed a more comprehensive CMH measure[5,12,13] that also included information on inflammation and adiposity given their strong links with disease development over the life course.[32,33]

Following prior work,[13,24,34,35] we hypothesized youth with more psychological assets would be more likely to maintain CMH across adulthood. When considering interactions by race and ethnicity, we expected to observe widespread protective associations, in line with earlier research conducted among older adults.[20–23] However, given the various plausible hypotheses that have been presented relating to effect modification,[26,27] analyses examining differences in the magnitude of associations were exploratory. With respect to racial and ethnic differences in CMH maintenance, we anticipated sizable health inequities would be observed at all levels of psychological assets, because such inequities have deeply rooted, structural origins.[36] All analyses adjusted for a robust set of confounders (eg, sociodemographic characteristics and negative affect[37]) as well as correlates of CMH (eg, family health history and health status in adolescence).

METHODS

Participants

Data came from Add Health, a school-based survey of adolescents in the United States that followed participants into adulthood.[17] Because of the sensitive nature of the data collected for this study, requests to access the data set from qualified researchers trained in human subject confidentiality protocols may be sent to cpc_dataportal@unc.edu. In the 1994 to 1995 school year, a nationally representative sample of 20 745 youth in grades 7 to 12 were enrolled in the study from 145 schools across the country. At baseline (Wave 1; mean age=16 years, range=11–21 years), students and their parents completed in-home interviews covering a range of topics related to health and development. In follow-up assessments in 2008 (Wave 4 [mid-young adulthood]; mean age=28 years; range=24–33 years) and 2016 to 2018 (Wave 5 [late young adulthood]; mean age=38 years; range=33–44 years), trained study staff collected direct measurements of cardiometabolic biomarkers from participants. Add Health was approved by the institutional review board of the University of North Carolina, Chapel Hill, and all participants provided written informed consent to take part in data collection activities.

The present analysis was limited to individuals who consented to the most recent biomarker assessment in Wave 5 and had complete cardiometabolic data available at both Waves 4 and 5 (n=3597). From this sample, participants were excluded if they were missing sampling information (n=83), if they reported a history of chronic health conditions in adolescence (ie, heart conditions, asthma, or diabetes; n=23), or if they were missing self-reported race and ethnicity data (n=13). This resulted in a final analytic sample of 3478 participants. Roughly 40% of our analytic sample had complete data; therefore, missing information on psychological assets (0.4%) and study covariates (0%–24%) was multiply imputed in 60 data sets using chained equations[38] (for information on missingness by individual study variables, see Table S1). To produce estimates that are nationally representative of the population of in-school youth at the study's baseline and also protect against selection bias, all analyses used weights derived by Add Health that account for design effects as well as differential attrition from baseline to Wave 5.

Measures

Adolescent Psychological Assets

Five psychological assets measured in Wave 1 were identified based on past research[8,20–23,29,30] and available data. *Optimism* and *happiness* were each assessed using responses to individual questions drawn from a modified version of the Center for Epidemiologic Studies Depression Scale,[39] which asked how often participants felt hopeful about the future (1 item) and happy (2 items) in the past 7 days. Responses ranged from 0 ("never or rarely") to 3 ("most or all of the time"). *Self-esteem* was measured using positively framed items adapted from the Rosenberg Self-Esteem Scale,[40] which asked youth to rate their agreement with 3 statements ("you have a lot of good qualities," "you have a lot to be proud of," and "you like yourself just the way you are"). Responses ranged from 1 ("strongly agree") to 5 ("strongly disagree"). The same 5-point

scale was used to rate *belongingness* (1 item; "you feel socially accepted") and whether participants felt *loved and wanted* (1 item) using survey items designed specifically for Add Health. Items for self-esteem, belongingness, and feeling loved and wanted were reverse coded for all analyses so that higher scores indicated higher levels of each asset.

Because research suggests that assets confer health benefits that may be unique from the absence of risk factors,[41] we created 5 separate binary variables that distinguished youth who possessed distinctly high levels of each asset (defined as the highest level of endorsement for individual survey items) from those with lower levels. For assets that were measured using multiple items, participants had to report the highest level of endorsement for the majority of items. A composite psychological asset index was then created by summing across each binary measure (range=0–5). Index scores were also grouped into a categorical measure (0–1, 2–3, 4–5 assets). Correlations between individual binary assets ranged from Φ=0.17 to 0.58 (see Table S2).

CMH Maintenance in Young Adulthood

Following prior work,[13–18] CMH was defined using 7 cardiometabolic biomarkers collected in Waves 4 and 5 by trained study staff during in-home clinical visits: (1) *high-density lipoprotein cholesterol* (HDL-C), (2) *non-HDL-C* (calculated as total cholesterol [TC] – HDL-C), (3) *systolic blood pressure*, (4) *diastolic blood pressure*, (5) *hemoglobin A1c*, (6) *C-reactive protein* (CRP), and (7) *BMI*. We did not examine low-density lipoprotein cholesterol because most participants did not fast before their blood draw, and low-density lipoprotein cholesterol was ascertained using the Friedewald equation, which may provide invalid estimates in nonfasting samples.[42,43]

Dried blood spots were collected via capillary finger prick in Wave 4, and venous blood samples were collected via phlebotomy in Wave 5. Lipids were measured using colorimetric and fluorometric assays in Wave 4 (CV [coefficient of variation]$_{TC}$=10.9%, CV$_{HDL\text{-}C}$=10.9%; intraclass coefficient [ICC]$_{TC}$=0.40, ICC$_{HDL\text{-}C}$=0.39), and Wave 5 assays were run using an Ortho VITROS 5600 Integrated System (CV$_{TC}$=3.0%–3.9%, CV$_{HDL\text{-}C}$=3.2–3.8; ICC$_{TC}$=0.88, ICC$_{HDL\text{-}C}$=0.95). Because Wave 4 lipid data were provided in ordered decile ranks to reduce measurement bias related to the use of dried whole blood, only decile measures of lipids were used in longitudinal analyses. In both Waves 4 and 5, 3 blood pressure readings were obtained in 30-second intervals from a seated position after a 5-minute rest period using an oscillometric blood pressure monitor, and final values were calculated as the average of the last 2 measurements. For hemoglobin A1c, Wave 4 assays were conducted using a Roche COBAS INTEGRA 700 Analyzer (CV=2.2%–2.4%; ICC=0.97), and Wave 5 assays were conducted using an Ortho VITROS 5600 Integrated System (CV=6.0–6.2%; ICC=0.99). CRP measurements were obtained using an enzyme-linked immunoabsorbent assay in Wave 4 (CV=8.1%; ICC=0.70) and a particle-enhanced immunonephelometric assay in Wave 5 (CV=6.3%–7.9%; ICC=0.82). Finally, BMI was calculated using height (obtained to the nearest 0.5 cm in a standing position) and weight measurements obtained by study staff following the same standardized protocols in both assessments.

Continuous values of each cardiometabolic metric were used to construct composite CMH scores (range=0–7) at Waves 4 and 5 that reflected participants' total number of biomarkers that did not fall within high-risk levels, as determined by established clinical guidelines. At each assessment, classifications of healthy levels were defined using either decile ranks or clinical thresholds. Healthy levels of HDL-C and non-HDL-C were defined as ≥20th and ≤80th percentile of the sample distribution because these values were found to closely approximate the established clinical thresholds of 40 mg/dL for HDL-C and 160 mg/dL for non-HDL-C in Wave 5.[13,44,45] Healthy values for systolic blood pressure and diastolic blood pressure were defined as <120 and <80 mm Hg,[32] hemoglobin A1c as <6.5,[33] CRP as <3.0 mg/dL,[21] and BMI as values between 18.5 and 25 kg/m^2.[13] Composite measures of CMH were then created using information on participants' history of cardiometabolic-related diagnoses, when available. Because this information was only collected in Wave 4, slightly different approaches were used to define CMH at Waves 4 and 5. In Wave 4, participants reported their age of diagnosis for 4 cardiometabolic conditions (hypercholesterolemia, high blood pressure, diabetes, or heart disease), allowing for optimal health to be defined as both receiving no cardiometabolic diagnoses since the study's baseline and meeting healthy criteria for ≥6 biomarkers. At Wave 5, data were not available on participants' new diagnoses; therefore, optimal CMH was characterized using biomarker values only. *CMH maintenance* (yes/no) was defined as achieving and sustaining CMH over time, meaning having CMH in both Waves 4 and 5. Two alternative outcome measures were also created to mitigate potential sources of bias. First, we constructed a measure that defined CMH as having healthy levels of ≥4 of 5 biomarkers excluding lipid values because of concerns regarding the impact of different blood sample collection methods on lipid measurements. Second, we constructed a measure that did not use any information on cardiometabolic-related diagnoses to ensure the same health information was used at both time points. For secondary analyses using our primary outcome measure, we also considered patterns over

time using a 4-category measure that differentiated participants who maintained CMH from those whose CMH improved, declined, or remained at suboptimal levels throughout the follow-up period.

Covariates

Information collected in adolescence via self-report, interviewer assessment, or parent report was included as a covariate if it was identified in prior literature as a potential confounder or related to cardiometabolic outcomes later in life.[13,24,35] Youth self-classified their race and ethnicity as Hispanic, Black non-Hispanic, Asian non-Hispanic, Native American non-Hispanic, other non-Hispanic, and White non-Hispanic. Because of small samples among certain subgroups, race and ethnicity were reclassified into 4 mutually exclusive categories: Black, Latinx, other (which included Native American and Asian youth, as well as those ethnicities not mentioned), and White. Other demographic characteristics included age (years), sex (male; female), parent-reported annual family income (<$24 999; $25 000–49 999; $50 000–$74 999; ≥$75 000), parental education (less than college; college or higher), and parent marital status (married; not married [ie, single, widowed, divorced, or separated]). To address potential confounding by baseline health, health-related covariates included parent history of obesity or diabetes (yes; no) and youth BMI (kg/m^2; measured by study staff during in-home interviews conducted in Wave 2). For descriptive analyses, a 3-category measure of BMI was created to differentiate youth who were underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), and overweight or obese (≥25 kg/m^2). Lastly, negative affect was also included as a covariate to account for associations between negative psychological states and psychological assets as well as cardiometabolic functioning.[37] A composite measure of negative affect was constructed using 4 items from the Center for Epidemiologic Studies Depression Scale assessing whether participants felt depressed, lonely, sad, or had "the blues" in the past week.[46] Responses ranging from 0 ("never or rarely") to 3 ("most of the time or all of the time") were summed to create an overall score (range=0–12). For descriptive analyses, a binary measure of high levels of negative affect (low-moderate vs high levels) was also constructed based on tertiles of the sample distribution.

Statistical Analysis

Nationally representative sample characteristics were calculated among all participants and within racial and ethnic subgroups using population-weighted means and proportions. We then summarized the distribution of study covariates by level of psychological assets using a categorical measure that differentiated youth by the number of assets they possessed. Differences in the prevalence of both psychological assets and CMH maintenance were also summarized by race and ethnicity.

Our first set of primary analyses were conducted in the overall sample and evaluated associations between psychological assets and CMH maintenance using a series of logistic regression models that sequentially adjusted for study covariates. Model 1 was unadjusted. Model 2 adjusted for age, sex, race and ethnicity, family annual income, parental education, and parent marital status. Model 3 additionally adjusted for family health history and youth BMI. Finally, Model 4 further accounted for negative affect. Associations were evaluated with assets modeled both as a categorical measure and as a continuous measure, allowing for the examination of linear trends. We also conducted a series of sensitivity analyses. First, we excluded participants classified as underweight in adolescence (ie, BMI <18.5 kg/m^2; n=364), as underweight status may serve as a confounder. Next, we excluded participants with CRP values ≥3 SD above the sample mean at either assessment in adulthood (n=103) to account for outlier values that may indicate an acute infection rather than systemic inflammation related to cardiometabolic risk.[47] We also evaluated associations using our 2 alternative outcome measures: one that excluded information on lipid measures to address potential measurement bias and another in which CMH was defined using only biomarkers to ensure that our findings were not influenced by the lack of cardiometabolic-related diagnosis data at Wave 5. Finally, other patterns in CMH were also considered in secondary analyses using multinomial regression models to evaluate associations between psychological assets and (1) a greater likelihood of CMH improvement and (2) a lower likelihood of CMH decline.

Our second set of primary analyses examined interactions by race and ethnicity.[28] Associations between total psychological assets and CMH maintenance were evaluated stratified by racial and ethnic subgroup using the same adjustment procedures described previously. Comparisons between White participants and those from minoritized racial and ethnic groups were formally tested by introducing interaction terms to the fully adjusted model. To gain additional insight into the potential role psychological assets play in shaping disparities in positive CVH, we also evaluated racial and ethnic differences in CMH maintenance separately among participants at different asset levels. More specifically, we used logistic regression models to examine disparities in CMH maintenance between individuals from minoritized racial and ethnic groups and White individuals by considering race and ethnicity as our primary exposure. To illustrate the magnitude of these disparities, we also graphically depicted subgroup

differences in the prevalence of CMH maintenance by asset level. All analyses were conducted using Stata MP 17.0.

RESULTS

Sample Description

The mean age of participants was 15.5 years at baseline, 28.3 years at Wave 4, and 37.9 years at Wave 5. A description of the sample is provided in Table 1, both overall and stratified by race and ethnicity. Roughly half of participants were female (52%), and two-thirds were White (67%; 15% Black, 11% Latinx, 6% other). The prevalence of psychological assets by study covariates are listed in Table S3. Overall, 55% of youth had only 0 to 1 asset, 29% had 2 to 3 assets, and 16% had 4 to 5 assets. Youth with only 0 to 1 asset tended to be older at baseline, female, and to have a parent with less than a college-level education. On the other hand, youth with 4 to 5 assets tended to be younger and to come from higher income families with more educated parents. Figure 1A illustrates differences in the prevalence of assets by race and ethnicity. On average, Black youth had the most assets, followed by White and Latinx youth; those from other racial or ethnic groups had the fewest.Figure 1B illustrates patterns in CMH over the follow-up period, both overall and by racial and ethnic group. Although 21% of the sample reached mid-young adulthood in optimal health, only 12% maintained it into late young adulthood. By contrast, two-thirds of participants (66%) were in persistently poor CMH at both follow-up assessments. There was some fluctuation in health over time, as 13% of participants experienced CMH improvements and 9% experienced CMH declines. Focusing on our primary outcome of CMH maintenance, it was less prevalent among Black (8%) and Latinx participants (9%), and more prevalent among White participants (13%) and those of other ethnic groups (13%).

Psychological Assets and CMH Maintenance

Results from analyses examining associations between psychological assets and CMH maintenance are provided in Table 2. The grouped measure of psychological

Table 1. Sample Description*

	Total sample (N=3478)	White (n=2183)	Black (n=597)	Latinx (n=430)	Other† (n=268)
	n (%)	n (%)	n (%)	n (%)	n (%)
Age at baseline					
11–15 y	1666 (49.2)	1088 (51.4)	289 (44.4)	175 (47.2)	114 (40.8)
16–21 y	1810 (50.8)	1095 (48.6)	306 (55.6)	255 (52.8)	154 (59.2)
Female sex	2103 (51.5)	1306 (53.0)	396 (50.7)	253 (47.0)	148 (45.4)
Annual family income					
≤$24 999	641 (26.8)	311 (21.5)	163 (40.9)	127 (41.0)	40 (24.7)
$25 000–$49 999	956 (31.8)	616 (31.2)	155 (33.5)	112 (31.0)	73 (35.9)
$50 000–$74 999	689 (22.8)	537 (27.1)	67 (12.6)	43 (13.3)	42 (17.5)
≥$75 000	459 (18.6)	345 (20.2)	46 (13.0)	33 (14.7)	35 (21.9)
Parent education					
Less than college	2159 (73.7)	1371 (71.6)	333 (75.0)	322 (86.6)	133 (69.4)
College or higher	895 (26.3)	600 (28.4)	164 (25.0)	49 (13.4)	82 (30.6)
Parent marital status					
Single	803 (26.2)	393 (21.8)	253 (46.8)	111 (29.0)	46 (20.0)
Married	2264 (73.8)	1585 (78.2)	245 (53.2)	264 (71.0)	170 (80.0)
Parent obesity or diabetes	842 (28.2)	552 (28.4)	139 (27.8)	99 (24.9)	52 (32.9)
Body mass index					
Underweight	364 (14.3)	250 (15.9)	35 (7.0)	39 (12.8)	40 (16.4)
Healthy	1705 (58.1)	1096 (58.1)	287 (59.3)	205 (59.0)	117 (54.5)
Overweight or obese	585 (27.6)	348 (26.0)	118 (33.7)	84 (28.2)	35 (29.1)
Negative affect					
Low or moderate	1961 (58.1)	1298 (60.0)	324 (59.3)	204 (52.0)	135 (46.5)
High	1514 (41.9)	885 (40.0)	271 (40.7)	225 (48.0)	133 (53.5)

*n's may vary because of missing values. Column percentages are population-weighted estimates using imputed data.
†The "other" category includes Native American and Asian individuals, as well as other ethnicities not previously listed.

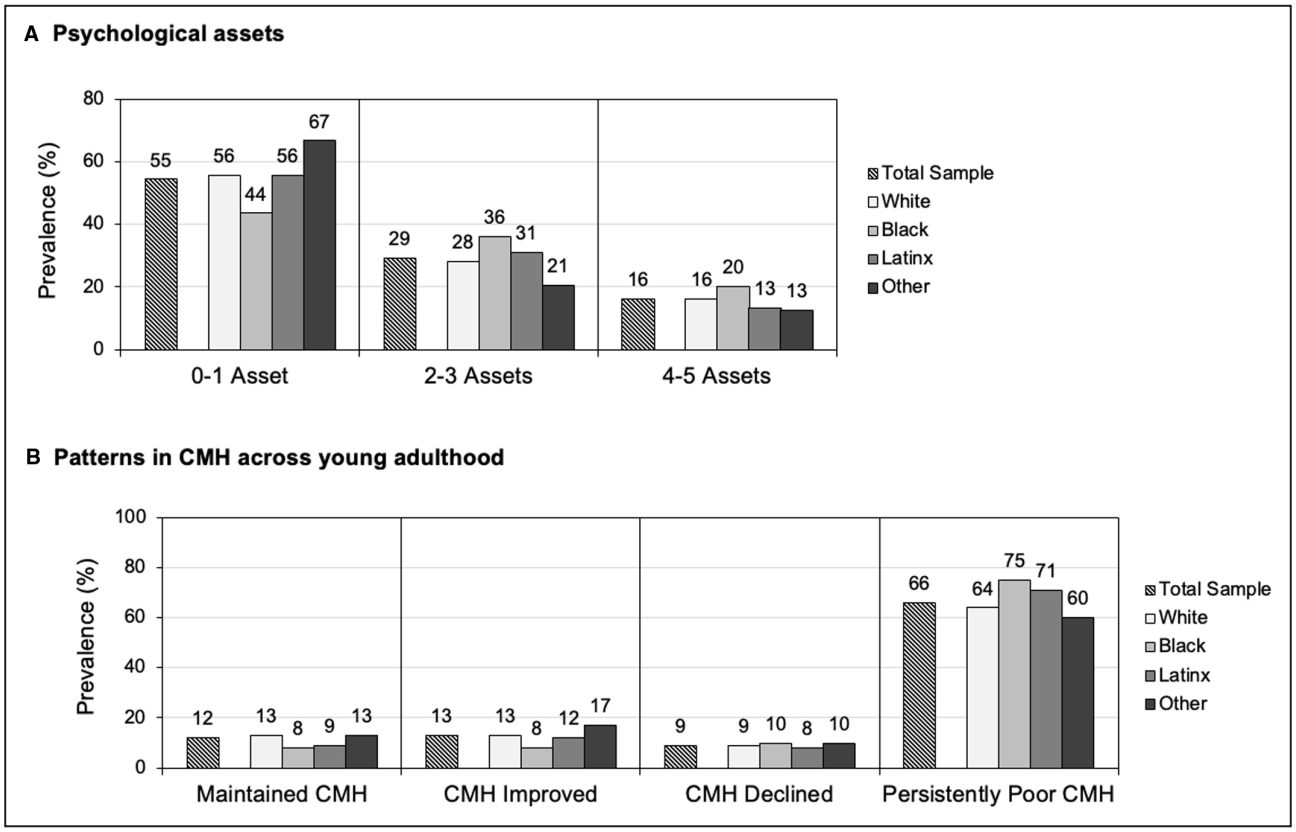

Figure 1. Prevalence of (A) adolescent psychological assets and (B) patterns of CMH across young adulthood in the total sample and stratified by race and ethnicity.
CMH indicates cardiometabolic health.

asset categories was used in all analyses to increase our power to detect associations, but results using a complete 6-category measure (0 assets through 5 assets) mirrored the main results and can be found in Table S4. Unadjusted analyses indicated that compared with youth with only 0 to 1 asset, those with 2 to 3 assets experienced a 18% greater likelihood of CMH maintenance (OR, 1.18 [95% CI, 0.82–1.71]), whereas those with 4 to 5 assets experienced a 69% greater likelihood (OR, 1.69 [95% CI=1.14–2.51]). Associations were consistent after adjusting for all study covariates (2–3 assets: $OR_{Model\ 4}$=1.24, 95% CI=0.81–1.90; 4–5 assets: $OR_{Model\ 4}$, 1.69 [95% CI, 1.07–2.65]). Tests for linear trend found that each additional asset was related to a 12% greater likelihood of CMH maintenance (eg, $OR_{Model\ 4}$, 1.12 [95% CI, 1.01–1.25]). Sensitivity analyses

Table 2. Associations Between Psychological Assets in Adolescence and the Likelihood of Maintaining Cardiometabolic Health in Young Adulthood (N=3478)*

	CMH maintenance				
	n_{Cases}†	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)
Psychological assets					
0–1 assets	246	Reference	Reference	Reference	Reference
2–3 assets	137	1.18 (0.82–1.71)	1.28 (0.86–1.89)	1.23 (0.82–1.84)	1.24 (0.81–1.90)
4–5 assets	104	1.69 (1.14–2.51)**	1.75 (1.16–2.64)**	1.68 (1.09–2.58)*	1.69 (1.07–2.65)*
Linear trend		1.11 (1.01–1.22)*	1.13 (1.03–1.24)**	1.12 (1.01–1.24)*	1.12 (1.01–1.25)*

*$P \leq 0.05$; **$P \leq 0.01$; ***$P \leq 0.001$.
*Model 1 is unadjusted. Model 2 adjusts for age, sex, race and ethnicity (in total sample), family annual income, parental education, and parent marital status. Model 3 further adjusts for parental obesity or diabetes, and participants' body mass index in adolescence. Model 4 additionally adjusts for negative affect. CMH indicates cardiometabolic health; and OR, odds ratio.
†Case counts refer to the number of study participants that maintained CMH within each asset category.

excluding those who were underweight in adolescence or those who had high CRP levels in adulthood yielded similar results (Table S5). Results from analyses using alternative outcome measures were largely consistent with our main findings (Table S6). Secondary analyses examining the relationship between assets and patterns in CMH over time did not find appreciable associations with a greater likelihood of health improvement or a lower likelihood of health declines (Table S7).

Interactions by Race and Ethnicity

Results from stratified models examining total assets in relation to CMH maintenance by racial and ethnic group are presented in Table 3. In fully adjusted models, each additional asset was associated with a 35% greater likelihood of CMH maintenance among Black individuals (OR, 1.35 [95% CI, 1.00–1.82]); a smaller association was apparent among Latinx individuals (OR, 1.24[95% CI, 0.88–1.76]) while no substantive associations were apparent among White individuals (OR, 1.09 [95% CI, 0.97–1.22]) or those of other ethnicities (OR, 0.99 [95% CI, 0.63–1.56]). Relatively few cases of CMH maintenance were observed among minoritized racial and ethnic subgroups, resulting in somewhat imprecise point estimates with wide CIs; as such, tests for interactions by race and ethnicity did not indicate substantial differences between groups (all $P>0.05$).

When examining the prevalence of CMH maintenance by race and ethnicity, somewhat sizable disparities were observed at the low end of the asset distribution, particularly for Black and Latinx participants (Figure 2). Among those with only 0 or 1 asset, the prevalence of CMH maintenance was 6% for Black and Latinx individuals compared with 12% for White individuals. By contrast, disparities were slightly smaller at the high end of the asset distribution, as 13% of both Black and Latinx youth maintained CMH among those with 4 to 5 assets compared with 17% of White youth. Prevalence estimates among participants of other ethnicities were less reliable owing to small sample sizes.

Results from regression models quantifying these disparities are provided in Table 4. In unadjusted models, Black youth with 3 or fewer assets were less likely to maintain CMH in adulthood than White youth ($OR_{1–2\ assets}$, 0.45 [95% CI, 0.19–1.07]; $OR_{2–3\ assets}$, 0.46 [95% CI, 0.22–0.93]). Among youth with 4 to 5 assets, these disparities were smaller, and after adjusting for all study covariates, no longer apparent (OR, 0.99 [95% CI, 0.39–2.50]). Similar trends were noted among Latinx participants, but disparities were less pronounced. Estimates of disparities among participants of other ethnicities were less clear and characterized by very wide CIs that included the null value; therefore, these findings should be interpreted with caution.[48]

DISCUSSION

In this prospective study, we evaluated whether psychological assets may help youth preserve positive CVH states in ways that may delay or prevent the onset of cardiovascular disease. Previous work examining psychological assets early in life found protective associations in prospective studies examining different composite measures of positive CVH assessed at a single time point later in life.[9,13] Few studies considered associations with repeated measures of positive CVH, which may provide unique insight into processes underlying health preservation across the lifespan. Using repeated measures of CMH assessed at approximately ages 28 and 38 years, we found that participants who had more psychological assets as adolescents were more likely to have CMH when they reached mid-young adulthood and to maintain it into late young adulthood.

Although the mechanisms linking psychological assets with healthier functioning are not fully understood, researchers posit that assets may serve a

Table 3. Associations Between Total Psychological Assets in Adolescence and the Likelihood of Maintaining Cardiometabolic Health in Young Adulthood, Stratified by Race and Ethnicity*,†

CMH maintenance	White (n=2183)		Black (n=597)		Latinx (n=430)		Other§ (n=268)	
	n_{Cases}‡	OR (95% CI)	n_{Cases}‡	OR (95% CI)	n_{Cases}‡	OR (95% CI)	n_{Cases}‡	OR (95% CI)
Psychological assets	377		48		53		52	
Model 1		1.10 (0.99–1.22)		1.33 (1.02–1.74)*		1.14 (0.87–1.48)		1.12 (0.72–1.74)
Model 2		1.10 (1.00–1.22)		1.32 (1.01–1.74)*		1.17 (0.90–1.53)		1.09 (0.68–1.74)
Model 3		1.09 (0.98–1.21)		1.34 (0.99–1.80)		1.16 (0.86–1.56)		1.08 (0.68–1.72)
Model 4		1.09 (0.97–1.22)		1.35 (1.00–1.82)*		1.24 (0.88–1.76)		0.99 (0.63–1.56)

CMH indicates cardiometabolic health. *$P\leq0.05$, **$P\leq0.01$, ***$P\leq0.001$.

*Model 1 is unadjusted. Model 2 adjusts for age, sex, family annual income, parental education, and parent marital status. Model 3 further adjusts for parental history of obesity and diabetes, and participants' body mass index in adolescence. Model 4 additionally adjusts for negative affect.

†Tests for interactions did not find evidence of differences between White participants and members of minoritized racial and ethnic groups ($P_{Black\ vs.\ White}=0.19$; $P_{Latinx\ vs.\ White}=0.85$; $P_{Other\ vs.\ White}=0.96$).

‡Case counts refer to the number of study participants that maintained CMH within each racial and ethnic group category.

§The "other" category includes Native American and Asian individuals, as well as other ethnicities not previously listed.

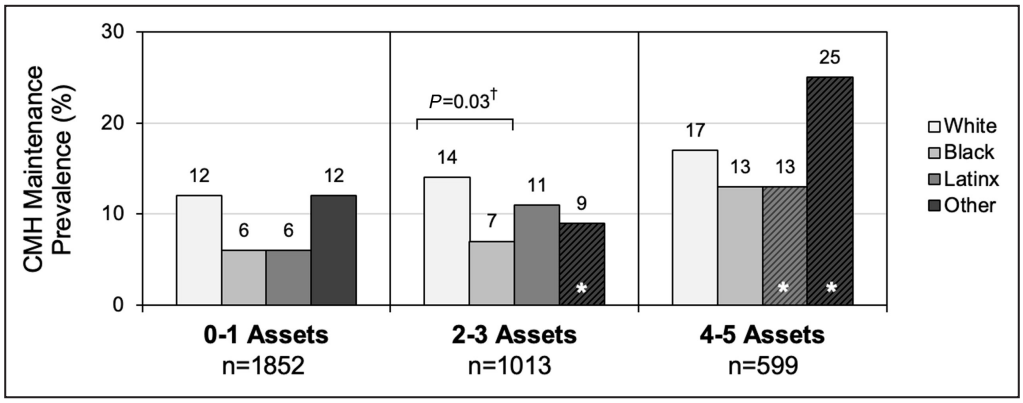

Figure 2. Racial and ethnic differences in the prevalence of maintaining CMH across young adulthood by levels of psychological assets.
*Indicates unstable estimates owing to low sample sizes (ie, n<100). †*P* values indicate unadjusted differences in comparison to White individuals. CMH indicates cardiometabolic health.

protective function by hindering health deteriorative processes and supporting restorative ones.[49] For example, studies show that psychological assets in adolescence buffer youth against the deleterious effects of social stress.[50,51] Assets like optimism and other facets of psychological well-being also predict greater health-enhancing behaviors across multiple domains, including physical activity, diet, and tobacco use.[34,52] In addition, it is possible that psychological assets have direct effects on biological processes because associations have been noted independent of health behaviors.[20,21,24,35]

In analyses examining racial and ethnic differences, we found that patterns in CMH maintenance were similar to those reported in prior work,[5,12] in that Black and Latinx participants had the lowest prevalence of CMH maintenance compared with White participants. Strikingly, Black individuals also had more psychological assets in adolescence, and these assets appeared to provide them with greater health benefits compared with other youth. At first glance, these findings may appear counterintuitive. If psychological assets play a role in explaining the lower levels of CMH maintenance observed among minoritized racial and ethnic groups, one might expect Black youth to possess fewer of these assets or for the health benefits they confer to be less robust. Yet, neither of these trends were apparent in our analyses. When disparities in CMH maintenance were examined by levels of psychological assets, we further found that racial and ethnic differences were pronounced among participants who had fewer assets in adolescence, and smaller (or altogether absent) among those with more assets. Taken together, this suggests that the absence of assets may be particularly health damaging for youth from minoritized racial and ethnic groups and may play an underappreciated role in shaping population differences in CMH maintenance.

Table 4. Racial and Ethnic Disparities in Maintaining Optimal Cardiometabolic Health Across Young Adulthood by Levels of Psychological Assets*†

	CMH maintenance					
	0–1 assets (n=1852)		2–3 assets (n=1013)		4–5 assets (n=599)	
	Model 1	Model 4	Model 1	Model 4	Model 1	Model 4
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
White	Reference	Reference	Reference	Reference	Reference	Reference
Black	0.45 (0.19–1.07)	0.76 (0.30–1.94)	0.46 (0.22–0.93)*	0.45 (0.19–1.05)	0.73 (0.30–1.77)	0.99 (0.39–2.50)
Latinx	0.51 (0.26–0.99)*	0.67 (0.33–1.36)	0.76 (0.36–1.59)	0.78 (0.33–1.85)	0.71 (0.21–2.40)	0.82 (0.29–2.35)
Other†	1.03 (0.50–2.11)	1.26 (0.61–2.61)	0.58 (0.11–2.99)	0.59 (0.10–3.64)	1.58 (0.37–6.77)	1.86 (0.37–9.35)

*Results describe the coefficient for race and ethnicity from regression models, which indicate racial disparities in CMH maintenance on the multiplicative scale. Analyses were conducted only among participants with complete psychological asset data.
†The "other" category includes Native American and Asian individuals, as well as other ethnicities not previously listed.
CMH indicates cardiometabolic health. *$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$.
†Model 1 is unadjusted. Model 4 adjusts for age, sex, family annual income, parental education, parent marital status, parental history of obesity or diabetes, participants' body mass index in adolescence, and negative affect.

To understand the implications of these findings, it is useful to consider the role structural racism plays in shaping health inequities. Racial and ethnic differences in cardiovascular disease-related outcomes are not attributable to innate biological differences but rather reflect generations of inequitable policies, institutions, and cultural norms that define a racial hierarchy in the United States that disproportionately benefits White individuals and harms people of color, especially Black individuals.[36,53] As described in a recent call to action from the American Heart Association,[36] structural racism is a fundamental cause[54] of poor health outcomes among minoritized racial and ethnic groups because of its pervasive and interrelated impacts on social institutions (eg, schools), economic conditions (eg, employment), and the health care system (eg, access to care), yet it remains largely unexplored in cardiovascular research. Although our study was not designed to explore this issue directly, the smaller racial and ethnic disparities in CMH maintenance we identified at the high end of the psychological asset distribution provide suggestive evidence that these assets may play a role in buffering Black and Latinx youth against the health deteriorative effects of structural racism, perhaps by helping compensate for other health-enhancing factors that may be lacking or absent.[27,55] Furthermore, the disparities observed at the low end of the asset distribution may indicate that population-based interventions to bolster assets (eg, in schools[56]) may be particularly beneficial. Prior work has shown that early psychological assets may serve as resilience factors[50,51,57]; therefore, future research should consider whether efforts to enhance these assets may be a useful complement to broader systems-based approaches to redressing the damaging effects of structural racism on population health.

Our findings should be considered in light of some limitations. Given the observational nature of this study, causal associations cannot be definitively determined. It is possible that our results were influenced by unmeasured confounding, potentially by baseline health behaviors (eg, diet, physical activity), which we did not account for given a lack of adequate baseline measures. Adequate baseline data were also not available on participants' timing of pubertal maturation, which may also serve as a potential confounder. Although female participants self-reported their age of menarche, male participants reported only their perceived bodily changes and not when these changes occurred, preventing our ability to construct an objective and accurate measure of maturational timing. In post hoc analyses, we considered associations adjusting for a somewhat crude measure of pubertal timing constructed following procedures used in prior research[58] and our findings were nearly identical (see Table S8 for more information). Nevertheless, we were able to comprehensively adjust for a wide array of other relevant confounders based on prior literature, including negative affect. There were some limitations related to our outcome measure. Although prior cardiometabolic research often uses composite scores derived as the sum of multiple *Z* scored biomarkers,[59] we chose not to employ this approach in the present study given our focus on positive CVH as opposed to cardiometabolic dysfunction. According to the American Heart Association, positive CVH is a "more positive construct than just the absence of clinically evident disease."[7] As such, we derived a measure of CMH that specifically captured whether individuals had distinctly healthy levels of 7 cardiometabolic biomarkers in the absence of manifest disease. Although this approach had power limitations because it required dichotomizing continuous biomarker values, it allowed us to examine associations with positive CVH maintenance during a critical period in the life course when individuals typically experience notable changes in health. Another limitation regarding our outcome measure relates to the manner in which we incorporated information on health conditions. We defined CMH in part using self-reported data on cardiometabolic-related diagnoses, but we did not incorporate data on medication use. When examining information about medication use, we found that nearly a quarter of the sample had medication use histories that were congruent with their CMH classification (ie, those who reported no medication use were already classified as maintaining CMH, whereas those with a history of medication use were classified as not maintaining CMH). On the other hand, over three-quarters of the sample reported no history of medication use but were classified as not maintaining CMH. Therefore, medication use proved to be a less informative measure of CMH than the directly assessed biomarker levels we used to construct our CMH outcome.

With respect to our sample, we were also limited by the relatively small number of individuals from minoritized racial and ethnic groups included in Wave 5 of Add Health, which likely explains the imprecision of our subgroup estimates. Although the survey was designed to be nationally representative, the demographics of the United States have changed considerably since the study's inception, warranting further evaluation of these relationships among underrepresented groups, particularly Latinx youth, as well as Asian and Native American youth, who were aggregated into a single category in our analyses. Another limitation relates to measurement. Changes in biospecimen collection methods used in Wave 4 (dried blood spots) and Wave 5 (phlebotomy) is a possible area of concern. Measurements obtained through dried blood spots have been shown to produce valid estimates of the biomarkers evaluated in this study, with the exception of lipid measures. To address potential concerns

about lipid measurement, we used their decile rank values rather than raw values in all analyses. Because there is no evidence that biomarker levels ascertained through dried blood spots are systematically higher or lower than their true values,[60] we believe differences in study protocols likely introduced nondifferential measurement error to our analyses, which typically biases results toward the null.[61] Therefore, we believe our findings may be somewhat conservative estimates of the true association between psychological assets and CMH maintenance. Regardless, we also conducted sensitivity analyses using an alternative outcome measure that excluded information on lipids, and our results were nearly identical. With respect to our measure of psychological assets, the survey items we used were either taken from existing instruments (eg, the Center for Epidemiologic Studies Depression, the Rosenberg Self-Esteem Scale) or were single items designed for Add Health and not previously validated. However, when we examined the relationship between psychological assets and negative affect, we found expected associations, which provides preliminary evidence of construct validity. New and existing cohort studies should include more substantive, validated measures of psychological assets.

This study also has several strengths. Although prior studies on psychological assets and CMH-related outcomes evaluated whether associations were modified by race and ethnicity,[20,21] we considered potential interactive effects by also evaluating the extent to which racial and ethnic disparities in CMH varied at different levels of psychological assets. Furthermore, we conducted a prospective analysis in which assets were evaluated before CMH assessments, which minimized the possibility of baseline health driving observed associations. We further mitigated concerns regarding reverse causality by excluding participants who had a history of chronic health conditions at baseline and by adjusting for both BMI in adolescence and parental health. Moreover, to investigate health maintenance in adulthood we evaluated CMH using objective clinical assessments of 7 cardiometabolic biomarkers that were measured at 2 time points roughly 10 years apart using data from a large, nationally representative study of racially and ethnically diverse adolescents.

CONCLUSIONS

In this longitudinal study spanning over 2 decades, we examined whether adolescent psychological assets contribute to CMH maintenance later in life and whether interactions were apparent by race and ethnicity. We found that possessing more assets in adolescence was associated with a greater likelihood of achieving and maintaining CMH in young adulthood. Furthermore, we also found that the health benefits conferred by these assets appear to be the strongest among Black youth, and that although substantive racial and ethnic disparities in CMH maintenance were apparent among those who had few or no assets in adolescence, they were less pronounced among those who had most or all of the assets we assessed. Because racial and ethnic disparities in health are a manifestation of inequitable social structures,[36,53,54] these findings provide preliminary evidence that psychological assets may be an underappreciated source of resilience for youth from minoritized racial and ethnic groups. Future work should evaluate whether population-based efforts to build psychological assets early in life may bolster the impact of broader structural interventions aimed at eliminating racial and ethnic health inequities.

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Disclosures

None.

Supplemental Material

Table S1–S8

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SUPPLEMENTAL MATERIAL

Table S1. Percentage of participants missing data on study covariates (n = 3,478).

	n	%
Variable		
Age at baseline	2	0.1
Sex	0	0.0
Race and ethnicity	0	0.0
Annual family income	733	21.1
Parent education	424	12.2
Parent marital status	411	11.8
Parent obesity	451	13.0
Parent diabetes	474	13.6
Body mass index	824	23.7
Negative affect	3	0.1
Psychological assets	14	0.4

Table S2. Φ coefficients for individual psychological assets.

	1	2	3	4	5
1. Happiness	--				
2. Optimism	0.37	--			
3. Self-Esteem	0.27	0.25	--		
4. Belongingness	0.23	0.17	0.54	--	
5. Feeling Loved	0.27	0.21	0.57	0.58	--

Table S3. Prevalence of psychological asset levels by study covariates.*

		0-1 Assets	2-3 Assets	4-5 Assets
	n	n (%)	n (%)	n (%)
Total Sample	3,478	1,852 (54.5)	1,013 (29.2)	599 (16.3)
Age at baseline				
11-15 Years	1,666	841 (51.1)	510 (32.0)	310 (16.9)
16-21 Years	1,810	1,011 (57.9)	502 (26.5)	288 (15.6)
Sex				
Female	2,103	1,156 (56.2)	591 (28.1)	347 (15.8)
Male	1,375	696 (52.8)	422 (30.4)	252 (16.8)
Race and ethnicity				
White	2,183	1,182 (55.6)	604 (28.1)	391 (16.3)
Black	597	248 (43.5)	224 (36.1)	123 (20.3)
Latinx	430	253 (55.6)	120 (31.1)	53 (13.3)
Other	268	169 (66.8)	65 (20.6)	32 (12.5)
Annual family income				
≤$24,999	641	332 (50.5)	212 (34.9)	93 (14.6)
$25,000-$49,999	956	539 (59.9)	256 (24.7)	160 (15.5)
$50,000-$74,999	689	371 (55.2)	184 (26.5)	132 (18.4)
≥$75,000	459	224 (50.2)	144 (31.0)	90 (18.8)
Parent education				
Less than college	2,159	1,191 (56.1)	611 (28.3)	349 (15.5)
College or higher	895	431 (50.0)	273 (29.6)	189 (20.4)
Parent marital status				
Single	803	444 (54.1)	242 (31.7)	116 (14.2)
Married	2,264	1,186 (54.7)	647 (27.7)	423 (17.6)
Parent obesity or diabetes				
Yes	842	460 (55.7)	237 (28.2)	142 (16.0)
No	2,636	1,392 (54.0)	776 (29.6)	457 (16.4)
Body mass index				
Underweight	364	185 (48.4)	105 (31.1)	73 (20.5)
Healthy	1,705	885 (54.0)	518 (29.2)	296 (16.7)
Overweight or obese	585	330 (58.8)	159 (26.0)	91 (15.3)
Negative affect				
Low or moderate	1,961	851 (44.5)	643 (33.6)	461 (21.9)
High	1,514	1,001 (68.5)	370 (23.1)	137 (8.4)

* n's may not sum to total sample size due to missing values. Prevalence estimates are population-weighted using imputed data.

Table S4. Associations between psychological assets in adolescence and the likelihood of maintaining cardiometabolic health (CMH) in young adulthood (N=3,478).[*]

		CMH Maintenance			
		Model 1	Model 2	Model 3	Model 4
	n_{Cases}[†]	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Psychological Assets					
0 Assets	163	Reference	Reference	Reference	Reference
1 Asset	83	0.92 (0.62, 1.36)	0.97 (0.66, 1.43)	0.97 (0.65, 1.46)	0.97 (0.65, 1.46)
2 Assets	67	1.19 (0.75, 1.90)	1.26 (0.76, 2.07)	1.19 (0.71, 1.98)	1.19 (0.70, 2.04)
3 Assets	70	1.10 (0.69, 1.75)	1.27 (0.78, 2.06)	1.25 (0.76, 2.07)	1.26 (0.75, 2.13)
4 Assets	58	1.56 (0.93, 2.62)	1.70 (0.97, 2.98)	1.67 (0.93, 3.00)	1.68 (0.93, 3.04)
5 Assets	46	1.77 (1.01, 3.09)	1.77 (1.02, 3.09)	1.64 (0.92, 2.95)	1.66 (0.90, 3.05)
Linear Trend		1.11 (1.01, 1.22)[*]	1.13 (1.03, 1.24)[**]	1.12 (1.01, 1.24)[*]	1.12 (1.01, 1.25)[*]

[*] Model 1 is unadjusted. Model 2 adjusts for age, sex, race and ethnicity (in total sample), family annual income, parental education, and parent marital status. Model 3 further adjusts for parental obesity or diabetes, and participants' BMI in adolescence. Model 4 additionally adjusts for negative affect.

[†] Case counts refer to the number of study participants that maintained CMH within each asset category.

[*] $p \leq 0.05$, [**] $p \leq 0.01$, [***] $p \leq 0.001$

Table S5. Fully adjusted associations between total psychological assets in adolescence and the likelihood of maintaining cardiometabolic health (CMH) in young adulthood in the total sample compared to that (i) among participants with adolescent body mass index (BMI) ≥18.5 kg/m^2), and (ii) among participants with C-reactive protein (CRP) levels less than 3-SD above the sample mean at either follow-up assessment.[*]

	Total Sample N=3,478		Adolescent BMI ≥18.5 kg/m^2 N=3,114		Adult CRP ≤3-SD Above the Mean N=3,352	
	n_{Cases}[†]	OR (95% CI)	n_{Cases}[†]	OR (95% CI)	n_{Cases}[†]	OR (95% CI)
Psychological Assets						
0-1 Assets	246	Reference	193	Reference	244	Reference
2-3 Assets	137	1.18 (0.82, 1.71)	109	1.24 (0.77, 1.99)	137	1.25 (0.81, 1.92)
4-5 Assets	104	1.69 (1.14, 2.51)[**]	87	2.12 (1.30, 3.48)[**]	104	1.70 (1.08, 2.69)[*]
Linear Trend		1.11 (1.01, 1.22)[*]		1.17 (1.04, 1.32)[**]		1.12 (1.01, 1.25)[*]

[*] Model 1 is unadjusted. Model 2 adjusts for age, sex, race and ethnicity, family annual income, parental education, and parent marital status. Model 3 further adjusts for parental obesity and diabetes, and participants' BMI in adolescence. Model 4 additionally adjusts for negative affect.

[†] Case counts refer to the number of study participants that maintained CMH within each racial and ethnic group category.

[*] $p \leq 0.05$, [**] $p \leq 0.01$, [***] $p \leq 0.001$

Table S6. Results from fully adjusted models examining associations between total psychological assets in adolescence and the likelihood of maintaining cardiometabolic health (CMH) in young adulthood defined using (i) information on biomarkers assessed at Waves 4 and 5 and cardiometabolic-related diagnoses at Wave 4, (ii) information on biomarkers assessed at Waves 4 and 5 only, and (iii) information on biomarkers (excluding lipid levels) and cardiometabolic-related diagnoses at Wave 4.[*]

	CMH Defined as ≥6 of 7 Biomarkers and No Diagnoses		CMH Defined as ≥6 of 7 Biomarkers Only		CMH Defined as ≥4 of 5 Biomarkers (Excluding Lipids) and No Diagnoses	
	n_{Cases}[†]	OR (95% CI)	n_{Cases}[†]	OR (95% CI)	n_{Cases}[†]	OR (95% CI)
Psychological Assets						
0-1 Assets	246	Reference	262	Reference	334	Reference
2-3 Assets	137	1.18 (0.82, 1.71)	142	1.22 (0.80, 1.84)	184	1.11 (0.79, 1.57)
4-5 Assets	104	1.69 (1.14, 2.51)[**]	110	1.69 (1.07, 2.67)[*]	124	1.46 (1.00, 2.13)[*]
Linear Trend		1.11 (1.01, 1.22)[*]		1.12 (1.01, 1.25)[*]		1.07 (0.98, 1.16)

[*] Model 1 is unadjusted. Model 2 adjusts for age, sex, race and ethnicity, family annual income, parental education, and parent marital status. Model 3 further adjusts for parental obesity and diabetes, and participants' BMI in adolescence. Model 4 additionally adjusts for negative affect.

[†] Case counts refer to the number of study participants that maintained CMH within each racial and ethnic group category.

[*] $p \leq 0.05$, [**] $p \leq 0.01$, [***] $p \leq 0.001$

Table S7. Associations between total psychological assets in adolescence and patterns in cardiometabolic health across young adulthood (n=3,478).[a,b]

		Model 1	Model 2	Model 3	Model 4
	n	RRR (95% CI)	RRR (95% CI)	RRR (95% CI)	RRR (95% CI)
Maintained CMH	490	1.14 (1.01, 1.29)*	1.16 (1.03, 1.31)*	1.14 (1.01, 1.29)*	1.14 (1.00, 1.30)*
CMH Improved	471	1.01 (0.93, 1.10)	1.01 (0.93, 1.10)	1.01 (0.93, 1.10)	1.01 (0.86, 1.10)
CMH Declined	350	0.97 (0.90, 1.05)	0.97 (0.89, 1.05)	0.96 (0.89, 1.04)	0.96 (0.88, 1.04)
Persistently Poor CMH	2,167	Reference	Reference	Reference	Reference

[a] Associations describe linear trends noted with each additional asset.

[b] Model 1 is unadjusted. Model 2 adjusts for age, sex, race and ethnicity, family annual income, parental education, and parent marital status. Model 3 further adjusts for parental obesity and diabetes, and participants' BMI in adolescence. Model 4 additionally adjusts for negative affect.

* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$

Table S8. Associations between psychological assets in adolescence and the likelihood of maintaining cardiometabolic health (CMH) in young adulthood additionally adjusting for pubertal timing (N=3,478).[*]

	CMH Maintenance	
	Model 4 Original Analyses	Model 4 + Pubertal Timing
	OR (95% CI)	
Psychological Assets		
0-1 Assets	Reference	Reference
2-3 Assets	1.24 (0.81, 1.90)	1.23 (0.80, 1.89)
4-5 Assets	1.69 (1.07, 2.65)[*]	1.69 (1.07, 2.64)[*]
Linear Trend	1.12 (1.01, 1.25)[*]	1.12 (1.01, 1.25)[*]

[*] Model 4 adjusts for age, sex, race and ethnicity, family annual income, parental education, parent marital status, parental obesity or diabetes, participants' BMI in adolescence, negative affect.

[*] $p \leq 0.05$, [**] $p \leq 0.01$, [***] $p \leq 0.001$