Psychological Well-Being in Childhood and Cardiometabolic Risk in Middle Adulthood: Findings From the 1958 British Birth Cohort

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Psychological Well-Being in Childhood and Cardiometabolic Risk in Middle Adulthood: Findings from the 1958 British Birth Cohort
Abstract

Childhood adversity is linked with poor cardiometabolic outcomes, but less is known about positive childhood factors. Using data from 4,007 members of the 1958 British Birth Cohort, we investigated whether children with greater psychological well-being had lower adulthood cardiometabolic risk. At age 11, participants wrote essays about their future. Two judges rated each essay for nine psychological well-being items (Finn’s $r = .82-.91$), which were combined into a standardized overall score (Cronbach’s $\alpha = .91$). At age 45, nurses assessed participants’ blood pressure, heart rate, lipids, glycosylated hemoglobin, fibrinogen, and C-reactive protein, which were standardized and summed for total cardiometabolic risk. Regressions indicated children with greater psychological well-being had lower cardiometabolic risk ($b = -.14$, 95% confidence interval [CI; -.28, -.06]), specifically healthier total cholesterol ($b = -.04$, 95% CI [-.07, -.03]) and triglycerides ($b = -.06$, 95% CI [-.09, -.02]). Childhood psychological well-being may promote adulthood cardiometabolic health.

Keywords: cardiometabolic risk, cardiovascular disease, childhood, health, life course, primordial prevention, psychological well-being
Statement of Relevance

Childhood adversity is related to poor cardiometabolic outcomes throughout life. However, it is unclear whether positive childhood factors such as psychological well-being (often measured by feelings of life satisfaction, purpose, and optimism) can promote better cardiometabolic health in adulthood. We investigated this question using data in which psychological well-being was assessed from essays written by 11 year old children. This allows direct assessment of the children’s perspectives and avoids bias inherent to retrospective or informant reports. Findings indicated children with greater psychological well-being had lower cardiometabolic risk – for example, healthier lipid levels – more than 30 years later. Associations were small but consistent with related studies. In addition to mitigating adversity, prevention strategies targeting childhood psychological well-being may help foster healthy cardiometabolic outcomes in midlife. Implementing prevention strategies earlier in the life course may help alleviate the burden of cardiovascular disease, which is the leading cause of death worldwide.
Psychological Well-Being in Childhood and Cardiometabolic Risk in Middle Adulthood: Findings from the 1958 British Birth Cohort

Reaching midlife with cardiometabolic risk factors like high levels of cholesterol and blood pressure, poor glucose control, or obesity substantially increases risk for cardiovascular morbidity and mortality (Lloyd-Jones et al., 2006). Furthermore, once cardiometabolic risk factors are in place, removing or mitigating them is difficult (Lloyd-Jones et al., 2006). A more effective strategy for reducing risk may be to implement primordial prevention strategies early in life before health has eroded. Childhood is a sensitive period when psychosocial development, behavioral patterns, and bodily systems set the stage for adult outcomes (Steinberger et al., 2016), yet limited research has identified positive childhood factors that contribute to better cardiometabolic health later in life.

To date, most work has focused on childhood factors that predict poor cardiometabolic outcomes. For example, a systematic review of 43 studies found that experiencing various types of childhood adversity (e.g., poverty, abuse) is associated with greater cardiovascular disease (CVD) risk (Appleton et al., 2017). Furthermore, in two birth cohort studies, childhood emotional distress was associated with greater midlife cardiovascular risk (Appleton, Loucks, et al., 2013; Winning et al., 2015). Thus, adversity and distress in childhood may predispose youth to increased cardiometabolic risk in adulthood.

In contrast, one promising factor that may foster cardiometabolic health is psychological well-being. The multidimensional construct of psychological well-being reflects more than the absence of distress (Ryff et al., 2006) and is characterized by positive thoughts and feelings, such as life satisfaction, positive emotions, purpose, and optimism (Boehm & Kubzansky, 2012). Evidence from adults indicates that psychological well-being independently promotes health
beyond the effects of adversity or distress. For example, one review showed that adulthood psychological well-being was associated with reduced CVD risk and healthier behavioral and biological processes (Boehm & Kubzansky, 2012). Similarly, two meta-analyses found greater life purpose and optimism were related to reduced CVD risk (Cohen et al., 2015; Rozanski et al., 2019). However, most research in this area has been conducted among adults and less is known about whether childhood psychological well-being contributes to healthy cardiometabolic outcomes over the life course.

The few studies that have investigated the long-term cardiometabolic impact of positive childhood factors tend to combine psychological resources with other resources such as socioeconomic status. For example, one U.S. study examined a composite of resources based on adults’ retrospective reports of parental warmth, social support, and socioeconomic status in childhood (Slopen et al., 2017). Adults with more of these resources had healthier scores on a metric encompassing blood pressure, total cholesterol, fasting blood glucose, body mass index (BMI), cigarette smoking, physical activity, and diet (Slopen et al., 2017). Prospective studies using similar composites of childhood psychosocial resources (e.g., children’s self-regulation, parental behaviors, household socioeconomic status) report comparable results with midlife cardiovascular outcomes (Appleton, Buka, et al., 2013; Pulkki-Raback et al., 2015).

Although these findings suggest positive childhood factors are associated with better cardiometabolic health in adulthood, the use of composite measures that include an array of child, parental, and environmental resources in combination with socioeconomic status may inflate associations because CVD and related risk factors are socially patterned (Mensah et al., 2005). Notably, other studies have found similar associations with healthier cardiometabolic outcomes even when socioeconomic factors were not included in composite measures of positive
childhood factors, suggesting psychological well-being may be important in its own right. For example, a composite of positive childhood factors including executive functioning skills and prosocial behaviors assessed prospectively was associated with better cardiometabolic outcomes approximately 8 years later during adolescence, even after adjusting for childhood socioeconomic status (Qureshi et al., 2019).

The current study builds on prior work by investigating whether childhood psychological well-being is associated with cardiometabolic risk more than 30 years later, independent of socioeconomic status. In line with research indicating that the words people use reflect their thoughts, feelings, interests, and priorities (Boyd & Schwartz, 2021), as well as findings that emotion-related word use correlates with emotional experiences and provides insight into psychological functioning (Vine et al., 2020), we used an innovative strategy to assess childhood psychological well-being from essays written by children when they were 11 years old. This approach allowed assessment of children’s psychological functioning without requiring them to have personal insight or relying on retrospective or parent reports. We hypothesized higher levels of overall psychological well-being would be associated with lower cardiometabolic risk in middle-age, independent of sociodemographic and health factors. Because gender norms and cognitive ability may drive essay content (Elliott, 2010), we also examined whether associations were moderated by sex or cognitive ability.

Methods

Participants

Data came from the 1958 British Birth Cohort Study, also known as the National Child Development Study, an ongoing study of 17,638 individuals born in England, Wales, or Scotland during one week in March 1958 (Atherton et al., 2008; Power & Elliott, 2006). The original
A study focused on identifying factors to improve birth outcomes but has since expanded its focus to health, education, and the social environment. In-depth assessments occurred at birth and during nine follow-up waves at ages 7, 11, 16, 23, 33, 42, 46, 50, and 55 years (Power & Elliott, 2006). The present research involves assessments at age 11 (1969; University College London et al., 2020; University of London et al., 2020) and a biomedical assessment at age 45 (University of London et al., 2021). Essays written at age 11 provided information about childhood psychological well-being whereas questionnaire and clinic-based data obtained on a subset of participants at age 45 provided information about adulthood cardiometabolic risk.

A total of 13,732 children wrote essays, which are stored on microfiche in the children’s original handwriting at the Centre for Longitudinal Studies at the University College London (Goodman et al., 2017). In 2016-2017, 10,511 essays were manually transcribed, digitized, and made available to researchers through the UK Data Archive. Trained research assistants coded childhood psychological well-being in essays for all participants who also had relevant health information in adulthood ($n = 5,463$; Supplemental Figure S1). Of these participants, we excluded individuals with essays from which psychological well-being could not be assessed due to essays comprised of only one or two sentences ($n_{missing} = 282$) and those who had insufficient data to derive a cardiometabolic risk score ($n_{missing} = 1,174$). Blood pressure and heart rate had little missing data ($n = 80-81$) but lipids, glycosylated hemoglobin, and inflammatory markers had substantially more missing data ($n = 756-993$) because fewer participants consented to the collection of blood samples (Atherton et al., 2008). Missing data on covariates were imputed so all statistical analyses were based on an analytic sample of 4,007 (Supplemental Figure S1). A priori power analyses suggested this sample size was more than sufficient to detect even very small effect sizes.
Although individuals included in the analytic sample did not differ from those excluded by levels of psychological well-being or sex, they did differ on other variables ($p$-values ≤ .05). Relative to excluded participants with relevant data, participants in the analytic sample tended to be more advantaged and healthier. They were more likely to have fathers in non-manual occupations, had higher childhood cognitive ability scores, wrote more words in their essays, had lower childhood BMI, were less likely to use cardiovascular-related medication as adults, and had lower cardiometabolic risk scores.

Both child participants and their parents provided consent to participate. An Institutional Review Board deemed the present investigation non-human subjects research.

**Assessment of Psychological Well-Being**

Psychological well-being is a multidimensional concept comprised of positive evaluations of one’s life (Boehm & Kubzansky, 2012). Evidence has not determined which specific well-being indicator is most pertinent for health, so we focused on those most commonly described in the literature: positive affect (experiencing positive emotions; Pressman et al., 2019), optimism (expecting favorable outcomes; Carver et al., 2010), purpose in life (having valued goals and activities; McKnight & Kashdan, 2009), life satisfaction (evaluating life overall favorably; Pavot & Diener, 2008), personal growth (ongoing development and improvement of the self; Ryff & Singer, 1998), mastery (controlling one’s actions in the world; Roepke & Grant, 2011), and pleasant experiences (engaging in pleasurable activities; Ryan & Deci, 2001). Such variables can be validly assessed in children and higher levels of childhood psychological well-being tend to be associated with higher levels of adulthood psychological well-being (Richards & Huppert, 2011). Past work also suggests that psychological well-being is generally stable over
time (although it can change, especially in response to distressing life events; Hudson et al., 2019).

In the current study, psychological well-being was assessed from essays written by 11 year old participants who were instructed: “Imagine that you are now 25 years old. Write about the life you are leading, your interests, your home life and your work at the age of 25” (Goodman et al., 2017). They had 30 minutes to complete a written response while in their school classroom. Based on past work suggesting that word use reflects lived experience and writing with more positivity is linked to greater well-being, better health, and longer lives (Vine et al., 2020), trained research assistants coded each essay for nine items assessing psychological well-being; two items each were used to assess optimism and purpose in life whereas all other indicators were assessed with a single item. Coders used face-valid question(s) to rate how strongly each indicator of well-being was manifested in the essay (1 = not at all; 7 = very much; Supplemental Material 1). Research assistants were guided by a codebook that included theoretically-informed operationalizations (Supplemental Material 2) and exemplar essays (i.e., essays for which each aspect of well-being was scored either high or low; Heyman et al., 2014). To avoid bias in coding procedures, coders were blind to all other participant data.

Every essay was rated by two individual coders; a third coder providing ratings in the event of low agreement. We assessed interrater agreement using Finn’s r, which is appropriate for ordinal data and not sensitive to skewness (Heyman et al., 2014). Finn’s r was calculated for each well-being item and ranged from .82-.91 (Supplemental Table S1), indicating acceptable agreement (Heyman et al., 2014). Ratings for each item were averaged across coders (each of the two items for optimism and purpose in life were first averaged across coders and then averaged together). Next, for each participant, an overall composite of psychological well-being was
averaged and standardized ($M = 0, SD = 1$; higher scores indicated greater psychological well-being). This overall composite of psychological well-being served as the predictor variable in all hypothesis-testing analyses. We used this unweighted mean composite to represent psychological well-being because we did not have a priori hypotheses about which indicator of psychological well-being would matter most for cardiometabolic risk, each well-being item was moderately to strongly correlated with other items (Supplemental Table S1), and an exploratory factor analysis yielded a single factor. Consistent with the factor analysis findings, this overall composite of psychological well-being demonstrated high internal consistency reliability (Cronbach’s $\alpha = .91$) and was modestly related to other variables in expected directions. For example, at age 11, psychological well-being was inversely related to teacher-reported internalizing and externalizing symptoms ($r = -.09$ and -.10, respectively) and parent-reported emotional problems ($r = -.05$). Psychological well-being was also positively related to parent-reported social status of father ($r = .07$). To examine differences in psychological well-being across covariates, tertiles of well-being were created based on the analytic sample’s distribution of raw scores (low <5.0 [34.4%], moderate 5.0-5.5 [33.4%], and high >5.5 [32.2%]).

**Assessment of Cardiometabolic Risk**

Following past research in this and other cohorts, we calculated cardiometabolic risk based on nine cardiovascular-related biomarkers (Marino et al., 2014; Winning et al., 2015). Such scores assess risk of CVD and are sensitive to changes in modifiable risk factors (Marino et al., 2014). The individual biomarker components included systolic and diastolic blood pressures, resting heart rate, total cholesterol, triglycerides, high-density lipoprotein cholesterol, glycosylated hemoglobin (HbA1c), fibrinogen, and C-reactive protein (excluding people with values $\geq 10$ mg/L because such levels can indicate acute infection). Biomarkers were assessed
during nurse visits at participants’ homes. Blood pressure and resting heart rate were each assessed three times on participants’ left arm and then averaged. Non-fasting blood samples were collected and then processed according to standard procedures described elsewhere (Fuller et al., 2006). Participants needed data from all biomarker components to be included in analyses. Each of the nine biomarkers was first standardized as a z score ($M = 0, SD = 1$) and then a cardiometabolic risk total score was created by summing across the standardized biomarkers (high-density lipoprotein cholesterol was reverse scored prior to summing; triglycerides and HbA1c were log transformed prior to summing due to skewness and kurtosis). Sensitivity analyses also used conventional clinical cut points to identify whether a participant was high risk (versus not high risk) on each of the nine biomarkers (King et al., 2011; Winning et al., 2015). For each participant, the number of high-risk biomarkers was summed to create a cardiometabolic risk count ranging from 0-9. Additional sensitivity analyses incorporated adulthood cardiovascular medication use into a separate cardiometabolic risk count ranging from 0-10. Higher total scores and counts indicated greater cardiometabolic risk.

Assessment of Covariates

We considered a range of covariates that might confound associations (Appleton, Buka, et al., 2013; Appleton, Loucks, et al., 2013). All but one of the covariates were measured when participants were age 11. Covariates included parent report of their child’s sex (girl or boy) and father’s social class (father absent, father present and employed in non-manual labor, or father present and employed in manual labor; Supplemental Material 3 describes sensitivity analyses that included mother’s social class and why mother’s social class was not considered as a primary covariate). General cognitive ability (continuous) was assessed via verbal and non-verbal tests administered by a teacher familiar with the child. The number of words written in
each essay was determined from the transcribed essays. Trained medical staff assessed height and weight for a calculation of BMI (kg/m\(^2\)) and reported the presence of heart problems (yes or no). Finally, we included use of cardiovascular-related medication in adulthood (yes or no), which was assessed by a computer-assisted interview when participants were ages 44-45. Given that nearly all members of the cohort were white (98.7%) and all were born during a single week in 1958, race and age were not considered as covariates.

Statistical Analyses

Using chi square and one-way ANOVA tests, initial analyses evaluated the distribution of covariates according to tertiles of psychological well-being. To account for missing data on covariates (missing data ranged from \(n = 0\) for sex to \(n = 523\) for BMI at age 11), multiple imputation was used. Twenty imputed datasets (modeled on all relevant indicators of psychological well-being, cardiometabolic risk, covariates, and interactions) were pooled together for hypothesis-testing analyses (Graham, 2009). Linear regression models estimated the relationship between childhood psychological well-being (overall mean score standardized) and the adulthood cardiometabolic risk total score (all relevant assumptions were met). Four models sequentially adjusted for potential confounders. Model 1 was unadjusted whereas Model 2 adjusted for sex and father’s social class in childhood. Model 3 additionally adjusted for other childhood covariates (cognitive ability, essay word count, heart complaints, and BMI) while Model 4 further adjusted for cardiovascular medication use in adulthood. Sensitivity analyses then excluded people using cardiovascular medication from Models 1-3 to see if associations held. Secondary analyses used linear regression models to explore which, if any, of the nine biomarkers might drive the overall association between childhood psychological well-being and adulthood cardiometabolic risk. In these secondary analyses, the overall mean of childhood
psychological well-being (standardized) was the predictor whereas the continuous variable for each of the nine biomarkers served as the outcome in separate regressions.

With clinical relevance in mind, sensitivity analyses used the cardiometabolic risk count as the outcome in a fully adjusted Poisson regression model (first using nine cardiometabolic indicators of high-risk and then separately adding use of cardiovascular medication as the tenth high-risk indicator). The high-risk variable for each of the nine biomarkers was also used as the outcome in fully adjusted logistic regression models to determine whether psychological well-being was associated with reduced likelihood of a high-risk classification. Finally, secondary analyses examined whether sex or cognitive ability moderated the association between psychological well-being and cardiometabolic risk. However, no interaction terms were statistically significant so stratified results are not presented.

Results

Participant Characteristics

Participants were evenly distributed by sex, more than half had a father who worked in a manual job, most were free from heart problems in childhood, and most did not use cardiovascular-related medication in adulthood (Table 1). On average, mean psychological well-being was relatively high considering the possible range from 1 to 7 (raw score median = 5.29; \( M = 5.20; \) \( SD = 0.63; \) actual range = 1.57-7.00). Individuals with higher levels of psychological well-being tended to be girls, have fathers with higher social class, have greater cognitive ability, and wrote more words in their essay (Table 1).

Psychological Well-Being in Childhood and Cardiometabolic Risk in Adulthood

In unadjusted linear regression models, higher levels of psychological well-being were associated with lower levels of cardiometabolic risk (\( r = -.07, \) 95% confidence interval [-.10, -
and associations were maintained after adjusting for a broad range of covariates in childhood and use of medication in adulthood (Table 2). These primary findings were nearly identical when 270 individuals who used cardiovascular medication in adulthood were excluded from analyses (data not shown).

When examining each biomarker of cardiometabolic risk individually in secondary analyses, higher levels of psychological well-being were associated with healthier levels of systolic blood pressure ($r = -0.04, 95\%$ confidence interval $[-0.07, -0.009]$), total cholesterol ($r = -0.05, 95\%$ confidence interval $[-0.08, -0.02]$), triglycerides ($r = -0.07, 95\%$ confidence interval $[-0.10, -0.04]$), and high-density lipoprotein cholesterol ($r = -0.06, 95\%$ confidence interval $[-0.09, -0.03]$) in unadjusted linear regression models (Table 3). In fully adjusted models, only the association between psychological well-being and lower levels of total cholesterol and triglycerides remained robust (Table 3). Psychological well-being was not individually associated with continuous measures of diastolic blood pressure, heart rate, HbA1c, fibrinogen, or C-reactive protein in any linear regression model (Table 3).

Consistent with the primary findings, results from a fully adjusted Poisson model with the cardiometabolic risk count showed that for every standard deviation increase in psychological well-being, risk of a poor cardiometabolic outcome was reduced by 5\% (relative risk $= 0.95, 95\%$ confidence interval $[0.93, 0.98]$). When cardiovascular medication use was also incorporated into the cardiometabolic risk count (i.e., 10 possible high risk components instead of 9), results in a model adjusting for all childhood covariates were comparable (relative risk $= 0.96, 95\%$ confidence interval $[0.94, 0.99]$). In addition, fully adjusted logistic regression models for each component of cardiometabolic risk (defined as high risk or not) showed higher levels of psychological well-being were associated with lower risk for systolic blood pressure,
triglycerides, and high-density lipoprotein cholesterol (Table 4). Although findings were in the expected direction for total cholesterol, psychological well-being was not associated with other high risk components such as diastolic blood pressure, heart rate, HbA1c, fibrinogen, or C-reactive protein (Table 4).

**Discussion**

Using a primordial prevention framework that highlights childhood as a critical period for CVD prevention, this research investigated childhood psychological well-being in relation to midlife cardiometabolic risk. Although researchers increasingly recognize that psychological factors are relevant for CVD, most research has focused on risk rather than protective factors; this is particularly true in studies examining childhood origins of disease (Ladwig et al., 2014). In the current study, we found that 11 year old children whose essays about their future lives reflected greater psychological well-being tended to have lower cardiometabolic risk in adulthood. Associations were evident independent of socioeconomic factors, suggesting that psychological well-being on its own may uniquely contribute to future health. Furthermore, findings were robust to a variety of analytic approaches and persisted after adjusting for sociodemographic, cognitive, and health-related factors. Results in the current study are congruent with past work showing psychological well-being protects against CVD in adulthood (Boehm & Kubzansky, 2012) and wide-ranging positive childhood factors promote healthy cardiovascular outcomes (e.g., Pulkki-Raback et al., 2015).

Such associations may exist because psychological well-being fosters healthy behaviors or buffers the physiological consequences of stress (Boehm & Kubzansky, 2012). For example, findings indicate health behaviors such as physical activity, food consumption, and sleep duration may serve as mechanisms by which psychological well-being is linked to
cardiometabolic risk factors in youth (Thumann et al., 2020). Alternatively, youth with high
levels of psychological well-being may be better equipped to cope with and recover from stress
because they have greater social support and other resources. This, in turn, may attenuate
activation of the body’s stress response and maintain healthy cardiovascular functioning
(Pressman et al., 2019).

In exploratory, secondary analyses examining psychological well-being’s association
with individual cardiometabolic risk factors, consistent relationships were evident with lipids and
systolic blood pressure, although the latter did not always persist after adjusting for covariates.
Psychological well-being was not associated with heart rate, HbA1c, fibrinogen, or C-reactive
protein. Longitudinal research in adults has similarly reported that psychological well-being is
associated with healthier lipids and blood pressure (e.g., Soo et al., 2018), perhaps because they
are tied with the health behaviors that psychological well-being promotes (Kim et al., 2020).
Although prior work has linked adult psychological well-being with lower levels of HbA1c and
inflammatory markers, most evidence is cross-sectional and null findings also exist (Ikeda et al.,
2011; Steptoe et al., 2008; Tsenkova et al., 2016). It is not clear why associations are inconsistent
for these factors, but it is possible that inflammatory biomarkers typically assessed in research
reflect acute rather than chronic inflammation (Rasmussen et al., 2021), even though chronic
dysregulation is likely to be most strongly connected with psychological well-being. Future
studies are needed to confirm associations with individual cardiometabolic risk factors, explore
novel biomarkers (e.g., biomarkers of chronic inflammation), consider potential moderators that
could alter associations (e.g., sex, age), and repeatedly assess biomarkers over time (Steptoe et
al., 2008).
The size of reported associations are not large but are comparable with other findings (e.g., Scheier et al., 2021). Modest associations are not surprising because health is determined by genetic, environmental, and behavioral influences, among others. Any single factor may not have a large impact on health when assessed in isolation, especially when examining associations across more than three decades and adjusting for confounding variables. However, even small associations can have a meaningful impact at the population level (Friedman & Booth-Kewley, 1987), particularly when they accumulate across the lifespan (Funder & Ozer, 2019).

Childhood psychological well-being was assessed via trained judges because self-reported measures were not available. This approach offered numerous advantages. Participants were not required to have self-insight, which may be challenging for children (Conijn et al., 2020). Although children were required to be literate, this approach avoids recall and desirability biases. Additionally, it prevents inconsistencies that occur between parent, teacher, and child reports (Upton et al., 2008). While each assessment method has its own limitations, in the current study, ratings for each indicator of psychological well-being were reliable across judges and correlated with other childhood variables in the expected directions. Human coders may have been better equipped than automated text analysis programs to process the misspellings and grammatical errors in the essays (e.g., “babby” means baby), understand differences in British spelling and words (e.g., flats for apartments), and take context into account (Boyd & Schwartz, 2021; Tausczik & Pennebaker, 2010). On the other hand, a limitation of this approach is it assumes childhood levels of psychological well-being persist into adulthood as other psychological factors do (Roberts et al., 2001). In light of that, we cannot rule out that different indicators of psychological well-being may change over time. It is also possible that assessing
psychological well-being in this way may capture other related psychosocial constructs, including personality and emotional maturity.

The 1958 British Birth Cohort began as a nationally representative sample of individuals born in 1958. Attrition occurred across the multidecade follow-up period; however, rate of loss is comparable to other epidemiologic cohorts and bias appears modest (Atherton et al., 2008). Based on participants who underwent the biomedical assessment at age 45, the cohort reflects the greater British population in sociodemographic characteristics like marital and employment status but is not representative of Britain’s current racial/ethnic composition (Atherton et al., 2008). Moreover, individuals who were included in the analytic sample appeared healthier than those excluded and selection bias may exist because only essays from participants with adulthood health data were included. These issues limit generalizability of the current findings.

This study also has numerous strengths. First, no other epidemiologic cohort has this unique assessment of childhood psychological well-being based on coding children’s text rather than on informant or retrospective assessments. Second, this research had a lengthy follow-up period spanning more than 30 years. Third, the cardiometabolic risk outcome was comprised of clinically assessed biomarkers that eliminate concerns about inflated associations between exposure and outcome due to shared method variance. Fourth, reported associations were statistically adjusted for potential confounders in both childhood and adulthood, including socioeconomic status, cognitive ability, and health. Incomplete data on these covariates was also addressed by multiple imputation. Finally, results were robust when considering adulthood cardiovascular medication use as a covariate, after excluding participants using medication, and when medication use was integrated into the cardiometabolic risk count outcome.
Although CVD and related risk factors are determined by many factors, those that manifest in childhood are understudied. Focusing only on health processes in adulthood ignores substantial life experiences in childhood that shape future health (Matthews & Gallo, 2011). Indeed, both psychological well-being and cardiometabolic health in childhood play a role in adult outcomes (Pool et al., 2021; Richards & Huppert, 2011). Furthermore, childhood may be a key period for establishing healthy psychological functioning because many relevant processes—including cognitive growth, identity formation, and independence—are developing and may be malleable. Indeed, interventions show that psychological well-being can be improved among youth (Carr et al., 2020).

The present study demonstrates that childhood psychological well-being is related to lower cardiometabolic risk in adulthood, even after accounting for childhood socioeconomic status. Although associations may appear small, they are evident across more than three decades from single assessments carried out in childhood and adulthood. However, a question remains as to whether interventions designed to bolster childhood psychological well-being could have long-term impacts on cardiometabolic functioning in adulthood. Preliminary studies among adults are promising (Mohammadi et al., 2020), but no evidence exists for youth. Thus, future work should consider whether interventions targeting childhood psychological well-being may also promote healthy cardiovascular outcomes.
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Table 1. Distribution of covariates overall and according to tertile of psychological well-being.

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Overall N = 4,007</th>
<th>Psychological Well-Being</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Low (n = 1,377)</td>
</tr>
<tr>
<td>Male (%)</td>
<td>1,955 (48.8%)</td>
<td>713 (51.8%)</td>
</tr>
<tr>
<td>Female (%)</td>
<td>2,052 (51.2%)</td>
<td>664 (48.2%)</td>
</tr>
<tr>
<td>Father’s Social Class</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No Male in Household (%)</td>
<td>161 (4.4%)</td>
<td>61 (4.8%)</td>
</tr>
<tr>
<td>Manual Job (%)</td>
<td>2,116 (57.6%)</td>
<td>778 (61.5%)</td>
</tr>
<tr>
<td>Non-Manual Job (%)</td>
<td>1,397 (38%)</td>
<td>426 (33.7%)</td>
</tr>
<tr>
<td>Mean Cognitive Ability (SD)</td>
<td>46.69 (14.29)</td>
<td>43.62 (14.85)</td>
</tr>
<tr>
<td>Mean Word Count (SD)</td>
<td>208.47 (103.64)</td>
<td>183.68 (99.10)</td>
</tr>
<tr>
<td>Heart Complaints</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (%)</td>
<td>3,647 (99.1%)</td>
<td>1,259 (99.7%)</td>
</tr>
<tr>
<td>Yes (%)</td>
<td>32 (.9%)</td>
<td>4 (0.3%)</td>
</tr>
<tr>
<td>Mean Body Mass Index (SD)</td>
<td>17.53 (2.50)</td>
<td>17.55 (2.59)</td>
</tr>
<tr>
<td>Cardiovascular Medication</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (%)</td>
<td>3,723 (93.2%)</td>
<td>1,283 (93.4%)</td>
</tr>
<tr>
<td>Yes (%)</td>
<td>270 (6.8%)</td>
<td>91 (6.6%)</td>
</tr>
</tbody>
</table>

Note: Percentages refer to the column percent of individuals within each tertile of psychological well-being with that characteristic. N = 4,007, although values are presented prior to multiple imputation of missing covariates. As such, some values do not add up to 4,007 due to missing data. All variables in this table were assessed at age 11 except cardiovascular medication, which was assessed at age 45.
Table 2. Association between childhood psychological well-being (standardized) and adulthood cardiometabolic risk total score in linear regression models with missing covariates imputed (N = 4,007).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b (95% CI), p</td>
<td>b (95% CI), p</td>
<td>b (95% CI), p</td>
<td>b (95% CI), p</td>
</tr>
<tr>
<td>Psychological Well-Being</td>
<td>-0.32 (-0.46, -0.18), &lt;0.001</td>
<td>-0.20 (-0.33, -0.06), 0.04</td>
<td>-0.13 (-0.27, 0.01), 0.07</td>
<td>-0.14 (-0.28, -0.006), 0.04</td>
</tr>
<tr>
<td>Sex a</td>
<td>-2.78 (-3.05, -2.51), &lt;0.001</td>
<td>-2.80 (-3.07, -2.53), &lt;0.001</td>
<td>-2.78 (-3.05, -2.51), &lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Father in Manual Labor b</td>
<td>-0.44 (-1.34, -0.45), 0.33</td>
<td>-0.37 (-1.19, -0.46), 0.38</td>
<td>-0.38 (-1.18, -0.43), 0.36</td>
<td></td>
</tr>
<tr>
<td>Father in Non-Manual Labor b</td>
<td>-1.42 (-2.32, -0.52), 0.002</td>
<td>-1.11 (-1.94, -0.27), 0.01</td>
<td>-1.07 (-1.89, -0.25), 0.01</td>
<td></td>
</tr>
<tr>
<td>Cognitive Ability</td>
<td>-0.03 (-0.04, -0.02), &lt;0.001</td>
<td>0 (-0.001, -0.002), 0.68</td>
<td>0 (-0.001, -0.002), 0.65</td>
<td></td>
</tr>
<tr>
<td>Word Count</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart Complaints c</td>
<td>1.56 (-0.82, 3.94), 0.19</td>
<td>1.25 (-0.98, 3.48), 0.27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Mass index</td>
<td>0.19 (0.13, 0.25), &lt;0.001</td>
<td>0.17 (0.11, 0.23), &lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adult Cardiovascular Medication d</td>
<td></td>
<td></td>
<td></td>
<td>2.60 (2.07, 3.13), &lt;0.001</td>
</tr>
</tbody>
</table>

a Males were the reference group
b No male in the household was the reference group
c Parent report; not having heart complaints was the reference group
d No medication was the reference group

Note: All covariates are from age 11 except cardiovascular medication, which is from age 45. Findings are nearly identical when word count of essay is not included in the models.
Table 3. Association between childhood psychological well-being (standardized) and each component of adulthood cardiometabolic risk (continuous) in linear regression models with missing covariates imputed \((N = 4,007)\).

<table>
<thead>
<tr>
<th></th>
<th>Model 1 (b) (95% CI), (p)</th>
<th>Model 2 (b) (95% CI), (p)</th>
<th>Model 3 (b) (95% CI), (p)</th>
<th>Model 4 (b) (95% CI), (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic Blood Pressure</td>
<td>-0.67 (-1.17, -0.17), 0.009</td>
<td>-0.22 (-0.69, 0.25), 0.35</td>
<td>-0.13 (-0.61, 0.36), 0.61</td>
<td>-0.16 (-0.63, 0.32), 0.53</td>
</tr>
<tr>
<td>Diastolic Blood Pressure</td>
<td>-0.26 (-0.60, 0.07), 0.12</td>
<td>-0.01 (-0.33, 0.31), 0.95</td>
<td>0.02 (-0.30, 0.35), 0.89</td>
<td>0.002 (-0.33, 0.33), 0.99</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>-0.22 (-0.54, 0.10), 0.18</td>
<td>-0.24 (-0.56, 0.08), 0.14</td>
<td>-0.15 (-0.48, 0.18), 0.38</td>
<td>-0.15 (-0.48, 0.18), 0.38</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>-0.05 (-0.08, -0.02), 0.003</td>
<td>-0.04 (-0.07, -0.004), 0.03</td>
<td>-0.04 (-0.07, -0.003), 0.03</td>
<td>-0.04 (-0.07, -0.003), 0.03</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>-0.09 (-0.13, -0.05), &lt;0.0001</td>
<td>-0.06 (-0.10, -0.02), 0.001</td>
<td>-0.05 (-0.09, -0.01), 0.008</td>
<td>-0.06 (-0.09, -0.02), 0.005</td>
</tr>
<tr>
<td>High-Density Lipoprotein Cholesterol</td>
<td>0.02 (0.009, 0.03), 0.001</td>
<td>0.01 (-0.001, 0.02), 0.06</td>
<td>0.007 (-0.004, 0.02), 0.21</td>
<td>0.008 (-0.003, 0.02), 0.17</td>
</tr>
<tr>
<td>Glycosylated Hemoglobin</td>
<td>-0.01 (-0.03, -0.006), 0.16</td>
<td>-0.008 (-0.03, -0.01), 0.41</td>
<td>-0.001 (-0.02, -0.02), 0.90</td>
<td>-0.004 (-0.02, -0.02), 0.71</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>-0.01 (-0.03, -0.005), 0.15</td>
<td>-0.01 (-0.03, -0.004), 0.14</td>
<td>-0.004 (-0.02, -0.01), 0.69</td>
<td>-0.004 (-0.02, -0.01), 0.64</td>
</tr>
<tr>
<td>C-Reactive Protein</td>
<td>-0.04 (-0.10, -0.02), 0.16</td>
<td>-0.04 (-0.10, -0.02), 0.16</td>
<td>-0.02 (-0.07, 0.04), 0.59</td>
<td>-0.02 (-0.08, 0.04), 0.53</td>
</tr>
</tbody>
</table>

Note: Model 1 was unadjusted; Model 2 adjusted for child’s sex and father’s social class in childhood; Model 3 adjusted for previous covariates and additional childhood covariates (cognitive ability, essay word count, heart complaints, and body mass index); Model 4 adjusted for previous covariates and adulthood cardiovascular medication use.
Table 4. Association between childhood psychological well-being (standardized) and each high risk (versus not high risk) component of adulthood cardiometabolic risk in logistic regression models with missing covariates imputed (N = 4,007).

<table>
<thead>
<tr>
<th>Component</th>
<th>Fully Adjusted Model OR (95% CI), p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic Blood Pressure</td>
<td>.91 (.83, .99), .03</td>
</tr>
<tr>
<td>Diastolic Blood Pressure</td>
<td>1.00 (.91, 1.10), .95</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>1.00 (.84, 1.17), .96</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>.94 (.88, 1.00), .06</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>.88 (.82, .95), .001</td>
</tr>
<tr>
<td>High-Density Lipoprotein Cholesterol</td>
<td>.88 (.78, .99), .04</td>
</tr>
<tr>
<td>Glycosylated Hemoglobin</td>
<td>1.02 (.83, 1.25), .87</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>1.04 (.88, 1.22), .69</td>
</tr>
<tr>
<td>C-Reactive Protein</td>
<td>.97 (.89, 1.06), .50</td>
</tr>
</tbody>
</table>

Note: Fully adjusted model included child’s sex, childhood covariates (father’s social class, cognitive ability, essay word count, heart complaints, and body mass index), and adulthood cardiovascular medication use. Odds of being high risk on a given component of cardiometabolic risk were modeled. High risk status for each component of cardiometabolic risk was defined according to previous work (King et al., 2011), unless otherwise noted: systolic blood pressure ≥ 140 mm Hg; diastolic blood pressure ≥ 90 mm Hg; heart rate ≥ 90 bpm; total cholesterol ≥ 6.2 mmol/L; triglycerides ≥ 2.25 mmol/L; high-density lipoprotein cholesterol for men < 1.0 mmol/L and for women < 1.3 mmol/L; glycosylated hemoglobin ≥ 6.1% (Bennett et al., 2007); fibrinogen ≥ 4 g/L (Dudek et al., 2010); and C-reactive protein ≥ 3 g/L (Ridker, 2003).