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Financial assets and cardiometabolic risk in early adulthood: Evidence from add health

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A B S T R A C T

Socioeconomic status (SES), such as education, income, or occupation, is central to our understanding of health disparities. A substantial literature links wealth to health, yet relatively few studies focus specifically on financial assets in early adulthood and on cardiometabolic risk. Using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), this study investigates how household financial assets are associated with cardiometabolic risks, and assesses mediating pathways including health behaviors, stressful life events, and neighborhood disadvantage. Employing Poisson, logistic, and generalized structural equation models, findings reveal significant gender differences: among women, greater financial assets correlate with lower cardiometabolic risk, while no significant associations emerge among men. These results highlight the distinct influence of wealth on health beyond traditional SES indicators and emphasize the need for gender-sensitive analyses to fully capture the socio-economic determinants of health disparities in the context of increasing wealth inequality.

1. Introduction

Health disparities by socioeconomic status (SES) represent one of the most persistent and well-documented patterns in social epidemiology. This literature indicates that individuals with lower SES experience worse health outcomes across nearly all disease categories and life stages, despite substantial advances in healthcare access in recent decades (Brown et al., 2023; Clark et al., 2009; Hayward et al., 2000; Phelan and Link, 2015). These disparities emerge early in the life course and compound over time, with profound implications for population health, healthcare costs, and social inequalities. Understanding the mechanisms through which socio-economic resources shape health outcomes remains a critical priority for demographic research aimed at informing policies to reduce health inequalities.

Past research has established robust associations between socio-economic indicators and health outcomes across the life course. A growing body of research documents links between wealth and health. We build on this literature by focusing on financial assets in early adulthood and by assessing whether these resources are associated with cardiometabolic risk over and above the role of education, income, and occupation. Unlike income, which captures resource flows at specific points in time, wealth represents a stock of accumulated assets that provides long-term economic security and enables households to weather financial shocks (Boen and Yang, 2016). This distinction becomes particularly salient in contemporary America, marked by growing wealth inequality and expanding wealth gaps across demographic groups (Burgard et al., 2013). Evidence suggests that wealth influences health through pathways that may operate independently of those of education and income, including enhanced consumption capacity for health-promoting goods and services, reduced chronic financial stress, and greater temporal flexibility for health-maintaining activities (Charron-Chénier et al., 2017; McEwen and Gianaros, 2010). At the same time, these channels underscore why wealth should not be treated as merely a proxy for income or education. Among individuals with comparable earnings, those holding even modest financial assets can more readily absorb income disruptions, avoid high-cost debt, and finance preventive or specialty care and safer housing, indicating that wealth expands the opportunity set for health-promoting decisions beyond what is captured by traditional SES indicators.

Researchers in this field also reveal important gender differences in the strength and pathways of socio-economic gradients in health. Although women frequently exhibit lower mortality than men at similar socio-economic levels, accumulating evidence suggests that for cardiometabolic outcomes, socio-economic disadvantage may be especially consequential for women (Chichlowska et al., 2008; Loucks et al., 2007; Santos et al., 2008). These patterns motivate our focus on whether wealth-health associations are similarly gendered in early adulthood.

The relationship between wealth and cardiometabolic health warrants particular attention during early adulthood, a critical life stage when health behaviors crystallize, biological risk factors accumulate,

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and socio-economic trajectories begin to diverge. Cardiometabolic disorders—including obesity, diabetes, and cardiovascular disease—represent leading causes of morbidity and mortality, whose origins are often traceable to early adulthood when protective and risk-promoting behaviors become established (Clark et al., 2009; Harris et al., 2006). Despite strong socio-economic gradients in cardiometabolic outcomes, significant gaps remain in our understanding of how wealth, as a distinct dimension of SES, influences biological health during this pivotal developmental period. By concentrating on early adulthood, we offer a conservative test of whether wealth functions as a distinct health-relevant resource: most respondents have had limited time to accumulate assets from their own earnings. If nascent differences in financial assets already predict cardiometabolic risk in early adulthood, this signals the onset of cumulative (dis)advantage that is likely to widen as cohorts age.

This study addresses these gaps by examining the association between household financial assets and cardiometabolic risk during early adulthood using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). We utilize a widely adopted cardiometabolic risk index based on seven biomarkers and analyze its association with financial asset ownership. We employ Poisson regression models to estimate overall associations, logistic regression to examine correlations with individual biomarkers, and generalized structural equation modeling to test potential mediating mechanisms including health behaviors, stressful life events, and neighborhood disadvantage. Recognizing that socio-economic resources may translate into health benefits differently across demographic groups, we conduct sex-stratified analyses throughout our study.

Our findings reveal substantial differences in wealth-health associations between women and men. Among women, higher financial assets are significantly associated with lower cardiometabolic risk, with significant correlations concentrated in cardiovascular, anthropometric, and inflammatory biomarkers. The association appears to operate partially through reduced exposure to neighborhood disadvantage. Among men, however, we observe no significant association between financial assets and cardiometabolic risk. These findings are consistent with established research showing stronger SES gradients in cardiometabolic health among women, but we extend this pattern to the domain of financial assets and suggest that the pathways linking wealth to health may operate through fundamentally different mechanisms for men and women during early adulthood.

This study makes several contributions to demographic scholarship on health disparities. First, it extends existing literature on the socio-economic gradient in health by providing novel evidence on wealth as a distinct dimension of SES, implicated in shaping cardiometabolic outcomes during early adulthood. Second, it reveals substantial gender differences in the wealth-health relationship that underscore the need for a more nuanced understanding of how different economic resources translate differently into health disparities across demographic groups. Finally, situated within the contemporary American context of skyrocketing wealth concentration, this research contributes to our understanding of how growing economic inequality may generate new forms of health disparities with lasting consequences for population health.

The remainder of this paper proceeds as follows. We first review existing literature on the association between socioeconomic status and health, with particular attention to wealth as a distinct dimension and its potential pathways to influence health. We then describe our data, measures, and analytical approach, followed by the presentation of descriptive and multivariable results as well as mediation analyses. We conclude with a discussion of our findings' implications for understanding the social determinants of cardiometabolic health and directions for future research.

2. Background

2.1. Socioeconomic status and health outcomes

A substantial body of evidence highlights a robust socio-economic gradient in health outcomes (Clark et al., 2009; Yang et al., 2020). Despite significant advances in healthcare, individuals with lower socioeconomic status (SES) consistently experience poorer health throughout their lives (Brown et al., 2023; Hayward et al., 2000; Phelan and Link, 2015). Socio-economic dimensions, such as educational attainment, household income, and occupational status, each uniquely contribute to a broad range of health indicators. Individuals at lower socio-economic positions face higher morbidity and mortality rates, elevated risks of cardiovascular diseases, metabolic disorders, and inflammatory conditions (Phelan et al., 2010). These disparities underscore the profound influence that social and economic conditions exert on population health.

However, the relationship between SES and health is nuanced, with each socio-economic dimension influencing health outcomes differently across the life course. Early-life SES exerts consistent protective effects against metabolic disorders into adulthood, whereas the protective impact of education on inflammatory markers like C-reactive protein persists, yet declines with age for metabolic disorders (Yang et al., 2020). This temporal variability indicates that the relative importance of different SES components shifts over time (Willson et al., 2007). While educational attainment offers foundational health benefits established in youth, income gains prominence in middle and older adulthood. Income becomes particularly salient in buffering against physiological dysregulation and cardiometabolic risk in middle and older adulthood (Yang et al., 2020). During these later life stages, economic resources help individuals manage age-related health challenges and health shocks. Additionally, different SES dimensions uniquely correlate with distinct health outcomes. Education robustly predicts general health and overall mortality (Herd et al., 2007), and income facilitates access to health-promoting resources (Boen, 2016). Occupational status impacts health through distinct psychosocial pathways, influencing exposure to workplace hazards, job stability, and associated stressors (Clougherty et al., 2010; Warren et al., 2004). Occupation-related health effects extend beyond income, influencing functional limitations, injuries, and exposure-driven conditions. The psychosocial dimensions of work, such as job control and stability, significantly impact health outcomes beyond purely economic factors (Marmot et al., 1997). Thus, socio-economic dimensions collectively yet distinctly shape health trajectories.

Socio-economic factors shape health through multiple interconnected pathways, prominently via chronic psychosocial stress, social support, health behaviors, and material resources (Adler and Snibbe, 2003; McEwen and Gianaros, 2010). Chronic stress, driven by financial strain, job insecurity, and discrimination, leads to cumulative physiological harm, manifesting as increased cardiometabolic risk (McEwen, 1998). Social integration can mitigate stress effects, yet individuals with lower SES frequently lack adequate social supports (Brown et al., 2023; Yang et al., 2016). Additionally, lower SES is associated with adverse health behaviors, restricted healthcare access, and poorer living environments, all exacerbating health risks (Cutler and Lleras-Muney, 2010; Phelan et al., 2010; Slopen et al., 2012). Understanding these complex, interconnected mechanisms linking SES and health is crucial for addressing persistent health inequalities.

2.2. Wealth as a distinct dimension of socioeconomic status

This study emphasizes wealth as a critical dimension for understanding disparities in cardiometabolic risk. Wealth is increasingly recognized as distinct from other socio-economic dimensions such as education, income, and occupation (Hällsten and Thaning, 2022; Pfeffer and Killewald, 2018), uniquely influencing health through separate pathways. Unlike income, which captures resource flows, wealth

represents accumulated assets offering long-term security against financial shocks (Boen and Yang, 2016; Pollack et al., 2007; Rodems and Pfeffer, 2021). Evidence consistently links wealth ownership to various health indicators, including self-rated health, stress biomarkers, and mortality (Hajat et al., 2010; Pool et al., 2018). Importantly, wealth-health associations persist even after controlling for education and income, underscoring wealth's unique health impact (Boen, 2016; Maskileyson, 2014; Pollack et al., 2007).

Importantly, many of the mechanisms through which wealth operates, such as purchasing health-promoting goods and services, buffering financial strain, or enabling advantageous residential environments, are similar to those emphasized for income. The distinctiveness of wealth lies in the protection and flexibility it provides conditional on current income and education: two adults earning the same income, but with different asset and debt positions, face sharply different constraints when confronting health shocks, job loss, or caregiving demands. Conceptualizing wealth as this stock-based reserve highlights how it modifies exposure to stressors and the feasibility and timing of health-protective choices in ways that are not captured by flow-based SES measures.

Wealth influences health through multiple interconnected mechanisms, notably via consumption capacities. Financial assets enable access to higher-quality healthcare, healthier dietary options, safer housing, and preventive care services (Charron-Chénier et al., 2017). During economic downturns, wealthier households sustain essential health expenditures, while asset-poor households often compromise health-related spending (Wall et al., 2012). These consumption-based mechanisms illustrate how wealth directly supports ongoing investments in health. Consequently, wealth shapes health outcomes significantly through its ability to consistently secure essential health-promoting resources.

Additionally, wealth affects health by altering time use patterns. Households with substantial wealth can outsource household labor and have increased flexibility regarding work hours, thus allocating more time for health-promoting activities such as exercise and preventive medical care (Burgard et al., 2013; Mani et al., 2013). Wealth also alleviates cognitive burdens by reducing the time and mental resources spent managing financial stress, thereby enhancing health management capabilities. This shift in time use not only promotes healthier behaviors but also contributes to broader physical and mental health improvements. Thus, wealth's influence on health extends beyond mere economic security, affecting lifestyle and daily routines.

Wealth significantly impacts health through psychological and stress-related pathways. Economic insecurity associated with limited assets generates chronic stress and anxiety, contributing to physiological dysregulation and increased cardiometabolic risks (McEwen and Gianaros, 2010; Yilmazer et al., 2015). Financial strain arising from low wealth or high debt correlates with elevated inflammation, cardiometabolic risk, and accelerated biological aging (Boen, 2019; Drentea and Reynolds, 2015). Conversely, wealth fosters a sense of security and control, buffering individuals against broader life stressors beyond financial hardships (Boen et al., 2021). Thus, wealth's psychological benefits significantly mitigate health risks associated with chronic stress exposure.

In particular, Boen et al. (2021) argue that wealth is not only a marker of socio-economic advantage but also a resource that conditions exposure to structural constraints, chronic stress, and opportunities for health protection, with consequences that are patterned by race, gender, and cohort. Their findings that wealth retains strong associations with health net of conventional SES measures underscore our focus on financial assets as a distinct buffer against economic insecurity rather than a simple proxy for income or education. Our analyses build on this perspective by asking whether even comparatively modest financial assets in early adulthood already differentiate cardiometabolic risk and whether these wealth returns vary by gender.

Recent research extends beyond total net worth, examining how wealth scarcity, specifically net worth poverty (NWP), impacts health. Defined as having net worth below one-fourth of the federal poverty line, NWP independently predicts adverse health outcomes apart from income poverty (Gibson-Davis et al., 2021). Even minimal asset holdings provide significant health protections, suggesting critical thresholds exist below which health risks substantially escalate (Gibson-Davis et al., 2023). While wealth and debt are closely intertwined, prior research underscores that they represent related but distinct dimensions of economic (in)security (Boen et al., 2021).High-interest and unsecured debts, such as credit card debt or certain student loans, can exacerbate financial strain and psychological distress even for households with some assets, whereas liquid savings and low indebtedness provide protection against shocks and support future-oriented health investments (Boen et al., 2021).

Although wealth-health gradients are often most visible in midlife and older ages, early adulthood is a formative period in which relatively small financial buffers or liabilities can have outsized consequences. Financial assets in the late twenties typically reflect family support, student loan and consumer debt, and early labor-market opportunities rather than decades of accumulated savings; they shape the terms on which young adults enter the labor market, secure housing, and cope with emerging health shocks or caregiving demands. Examining wealth at this stage therefore captures the early imprint of financial (dis) advantage on physiological risk and allows us to interpret any observed associations as a lower bound on the cumulative role of wealth across the life course.

Research on gender differences in SES–health gradients reveals a complex pattern rather than a simple female "immunity" to the harms of economic disadvantage. While some narrative reviews have characterized women as relatively less affected by SES disparities (Phillips and Hamberg, 2015), these conclusions rely in part on vote-counting approaches that are known to be biased in the presence of heterogeneous findings (Hedges and Olkin, 1980). Empirical studies often cited as evidence of attenuation in fact document significant or stronger SES–health associations among women, including for metabolic and cardiovascular outcomes (Chichlowska et al., 2008; Loucks et al., 2007; Santos et al., 2008). Building on this literature, we treat cardiometabolic risk as a domain in which socio-economic disadvantage may be particularly consequential for women and, correspondingly, expect wealth-health gradients to be potentially steeper among women than men (Jenkins and Ofstedal, 2014; Kavanagh et al., 2010). Several explanations have been proposed for these differential patterns, including gender differences in resource utilization, social stigma associated with health outcomes, and distinct life course trajectories that place women with lower educational attainment at greater risk for cardiovascular-related stressors (Finkelstein et al., 2005).

In sum, we expect individuals with more financial assets to report fewer cardiometabolic risks. In addition, we expect this association to be stronger for women than for men.

3. Data & methods

3.1. Sample and data

Data in this study come from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative longitudinal study that followed US adolescents from grades 7-12 in 1994 across five interview waves over 25 years. This analysis utilizes data from Wave I (1994-1995) and Wave IV (2008-2009), along with parent interviews from Wave I. The initial Wave I sample included 20,745 adolescents and 17,670 parent interviews. Wave IV followed up with 15,701 participants aged 24-32 years and incorporated extensive biomarker collection and health assessments.

Our analytic sample consists of respondents who: (1) participated in Waves I and IV; (2) had valid cross-sectional sample weights; (3) had complete biomarker data for constructing the cardiometabolic risk index

in Wave IV; and (4) had valid values for all financial asset, mediator, and health variables. Based on these criteria, our final sample includes 7700 respondents. Appendix Table A1 presents the sequential application of these selection criteria and resulting sample attrition. We present supplementary analyses without excluding observations with missing data on the pathway variables in the online appendix (Table A7 and Fig. A16). Additionally, we note that our results remain unchanged when applying multiple imputation of missing observations on our independent variables (see Table A8 and Figs. A17–A20 in the online appendix).

3.2. Measures

3.2.1. Dependent variable

The primary outcome measure is a cardiometabolic risk (CMR) index constructed from seven biomarkers collected during Wave IV medical examinations. Following (Harris et al. (2017), this index comprises seven physiological indicators representing the biological functioning of the metabolic, inflammatory, and cardiovascular systems: waist circumference, blood pressure, lipid profiles (triglycerides, HDL cholesterol, and LDL cholesterol), glycated hemoglobin (HbA1c), and C-reactive protein (CRP). Each biomarker was dichotomized using predefined disease risk cut points established in prior research (Harris et al., 2017). Waist circumference was considered elevated at ≥102 cm for men and ≥88 cm for women. Blood pressure risk was defined as systolic pressure ≥130 mmHg, diastolic pressure ≥85 mmHg, or current use of antihypertensive medication. For lipids, elevated risk was defined as triglycerides ≥150 mg/dL, HDL cholesterol <40 mg/dL for men and <50 mg/dL for women, or LDL cholesterol ≥130 mg/dL. HbA1c values ≥ 5.7% or current use of diabetes medication indicated elevated risk, and CRP was considered elevated at >3 mg/L. These binary indicators were summed to create a continuous risk score ranging from 0 to 7, with higher scores indicating greater cardiometabolic risk (Harris et al., 2017).

3.2.2. Independent variable

The key independent variable is household financial assets, measured at Wave IV. Respondents were asked to estimate the total value of financial assets held by all household members who contribute to the household budget, excluding home equity but including bank accounts, retirement plans, and stocks. Responses were collected in nine categories: (1) less than $5,000, (2) $5000 to $9,999, (3) $10,000 to $24,999, (4) $25,000 to $49,999, (5) $50,000 to $99,999, (6) $100,000 to $249,999, (7) $250,000 to $499,999, (8) $500,000 to $999,999, and (9) $1,000,000 or more (see Appendix Figure A1 for more information regarding the distribution of financial assets in our sample).

3.2.3. Control variables

Our main analyses include socio-demographic and contextual controls measured at Waves I and IV. Wave I variables include time-invariant characteristics, such as family socio-economic background (standardized composite score from parent interviews incorporating parental education, occupation, household income, and public assistance receipt, as in Belsky et al., 2018), gender (female = 1), and race/ethnicity (non-Hispanic White, non-Hispanic Black, other races). Wave IV variables include age, personal income (log-transformed, annual earnings before taxes), educational attainment (college attendance = 1), occupational prestige (standardized Hauser-Warren socio-economic index, Belsky et al., 2018), marital status (married/partnered = 1), employment status (employed = 1), homeownership (homeowner = 1), and number of living children (see Appendix Figure A2 for a correlation matrix of all variables in our models). Detailed information on the operationalization of all variables is further provided in Appendix B.

3.2.4. Mediators

Stressful Life Events: We measure exposure to stressful life events using an approach based on Adkins et al. (2009). A comprehensive stressful life events (SLE) index is constructed from Wave IV data, encompassing multiple domains of acute and chronic stressors experienced in the 12 months prior to the interview. The SLE variables include death of family members, violence victimization and perpetration, legal troubles, relationship dissolution, pregnancy-related events, health problems, economic hardship, and other major life disruptions. Individual SLE items are coded as binary indicators (1 = occurred, 0 = did not occur) and summed to create a total SLE score.

Health Behavior: We construct a health behavior index following Olson et al. (2017) that captures multiple dimensions of health-related behaviors. These include: (1) fast food consumption, defined as eating at a fast food restaurant 3 or more times in the past 30 days; (2) tobacco use behaviors, combining cigarette smoking in the past 30 days with other tobacco use; (3) binge drinking, defined as consuming 5 or more alcoholic beverages at one time in the last week; (4) substance use behaviors, encompassing prescription drug abuse (ever abused prescription drugs) and illegal drug use (using illegal drugs 5 or more times during their lifetime to date); (5) physical activity behaviors, measured as participation in physical activity in the past 7 days; and (6) healthcare utilization behaviors, combining visits to the doctor or dentist for preventive care in the past year. Each behavioral category is coded as a binary indicator (1 = engaged in healthy behavior, 0 = did not engage), and these binary indicators are summed to create a total health risk behavior score.

Neighborhood Disadvantage: Following the approach of Martin et al. (2019), we construct a neighborhood disadvantage index using county-level contextual data from the 2005-2009 American Community Survey linked to participants' Wave IV residential locations. County-level census indicators are selected to reflect neighborhood socio-economic conditions, including measures of poverty, public assistance, unemployment, educational attainment, income, and household composition. Principal component factor analysis is conducted to create a composite neighborhood disadvantage index. The first principal component, explaining 59.8% of the total variance, is retained as the neighborhood disadvantage measure.

3.2.5. Analytic approach

Our analytical strategy proceeds in three stages. First, we examine the relationship between financial assets and cardiometabolic risk using Poisson regression models. We then unpack the cardiometabolic risk index by estimating the correlation of financial assets separately for each of the seven risks using logistic regression models. Finally, we employ generalized structural equation modeling (GSEM) to examine potential mediating pathways linking financial assets to cardiometabolic risk through health behaviors, stressful life events, and neighborhood disadvantage. We estimate all models among the full sample as well as for sex-specific subsamples. To formally test gender differences in the wealth-health association, we estimate pooled models that include financial assets × gender interaction terms alongside the same set of covariates used in the stratified models.

Our main model takes the following form:

$$log(E[Biorisk_i]) = \beta_0 + \beta_1 W_i + \boldsymbol{X}_i' + \varepsilon_i$$

Where W_i represents the financial asset category of individual i, and $\boldsymbol{X}_i'$ denotes a vector of covariates detailed above. We further estimate logistic regression models for each binary indicator of individual cardiometabolic risk.

To gauge potential pathways through which financial assets affect cardiometabolic risk, we estimate the following generalized structural equation models (GSEM):

$$W_i = \alpha_0 + \boldsymbol{X}_i' + \varepsilon_{1i}$$

$$M_{ki} = \alpha_{1k} + \beta_{1k} W_i + \mathbf{X}'_{ki} + \varepsilon_{2ki}$$

$$log(E[Biorisk_i]) = \alpha_2 + \beta_2 W_i + M_{ki} + \mathbf{X}'_i + \varepsilon_{3i}$$

where W_i denotes our wealth measure and M_{ki} represents the three mediating variables (health behaviors, stressful life events, neighborhood disadvantage) for individual *i*, and *k* indexes the specific mediator. All analyses account for Add Health's complex survey design using sampling weights and clustered standard errors at the school level.

4. Results

Table 1 presents descriptive statistics by financial assets for all variables in our analyses. Individuals with less than $5000 in financial assets score highest, and individuals with more than $1,000,000 score lowest, in terms of their cardiometabolic risk. This pattern provides suggestive evidence regarding the expected negative relationship between asset ownership and cardiometabolic risks. Descriptive statistics of covariates add important insights: Parental socioeconomic status tracks well with financial assets. Individuals with more financial assets on average come from high-SES families while individuals in the lowest asset categories grew up in low-SES families. This observation is in line with previous research underscoring the significance of intergenerational transmission of advantage in wealth attainment (Killewald and Bryan, 2018; Pfeffer and Killewald, 2018, 2019). On average, individuals with more financial assets also report somewhat higher incomes, occupational prestige, and college education, but fewer children. At the same time, there are no substantive differences when it comes to other demographic characteristics, such as age or marital status.

Our three potential pathways, health behaviors, stressful life events, and neighborhood disadvantage, all vary systematically by financial asset ownership. Individuals with fewer financial assets report worse health behavior, more stressful life events, and greater neighborhood disadvantage compared to individuals with more financial assets. These summary statistics suggest that wealthier respondents engage in better health behaviors, and that accumulated financial wealth might provide some protection against adverse life experiences and could facilitate residence in more advantaged communities. Taken together, descriptive patterns highlight the interconnected nature of financial wealth with multiple dimensions of advantage and disadvantage that may independently or jointly influence biological health outcomes. This pattern accords with recent research suggesting that wealth operates as a critical buffer against structural stressors and material hardship even in early adulthood (Boen et al., 2021).

We now turn to our first multivariable regression models. Table 2 shows results for the full sample (left), as well as the male (center) and female subsamples (right). Starting with the bivariate association between financial assets and cardiometabolic risks, the first model indicates a clear, negative relation. This is in line with our expectations and the descriptive statistics presented in Table 1. The relation persists when accounting for all socio-demographic covariates. More specifically, moving from one wealth category to the next higher category is associated with a 1% decrease in the expected cardiometabolic risk.

Next, we expected the relation between financial wealth and cardiometabolic risk to be stronger for females. Consistent with previous research demonstrating stronger SES gradients in cardiometabolic health among women than men, our findings show that the connection between higher financial wealth and lower cardiometabolic risks does not hold for males. In contrast, we find a strong, negative association in the female sample only. The coefficient on financial assets is substantive and highly significant. A formal test using financial assets × gender interaction terms in the full sample model confirms this gender difference is very significant ($p < .01$). Taken together, these subsample analyses suggest that financial wealth matters for the cardiometabolic health of females but not males. This aligns with prior evidence that women's cardiometabolic profiles are especially sensitive to economic resources and stress exposures (e.g., Boen, 2016; Chichlowska et al., 2008; Loucks et al., 2007; Santos et al., 2008), and suggests that financial assets represent a key arena of gendered health returns in early adulthood.

Why is there an association between financial assets and cardiometabolic risk for females but not males? First, to provide a more comprehensive picture of cardiometabolic risk, we replicate our analyses estimating the effect of financial assets separately for each dimension included in the cardiometabolic risk index (waist circumference, blood pressure, triglycerides, HDL cholesterol, LDL cholesterol, glycated hemoglobin, and C-reactive protein). This allows us to get an idea of whether some factors might be driving the sex-specific negative association between financial wealth and cardiometabolic risk (see Appendix Figure A3 for estimates among the full sample).

Table 1
Descriptive statistics by financial assets category.

Variable	Household Assets									Total
	< $5000	$5000-$9999	$10,000-$24,999	$25,000-$49,999	$50,000-$99,999	$100,000-$249,999	$250,000-$499,999	$500,000-$999,999	≥ $1,000,000	
Biological Risk	2.14	2.01	1.94	1.92	1.87	1.82	1.81	1.57	1.70	1.91
Family SES	−0.23	−0.04	0.06	0.24	0.24	0.36	0.49	0.54	0.98	0.12
Income (log)	8.75	9.46	9.71	9.90	10.01	10.00	10.00	9.89	10.52	9.66
Age	28.37	28.34	28.40	28.53	28.56	28.70	28.62	28.51	28.61	28.30
Occupation Prestige	−0.24	−0.09	0.05	0.15	0.23	0.29	0.36	0.29	0.46	0.03
Female (%)	64.29	58.25	54.25	49.29	47.96	49.35	47.79	55.15	44.93	50.03
Race (%)										
NH White	55.30	58.36	60.45	63.50	62.40	65.73	59.80	56.97	65.22	74.10
NH Black	27.93	24.02	21.54	16.83	15.54	13.99	12.25	14.55	10.14	13.28
Other Race	16.77	17.62	18.01	19.67	22.06	20.28	27.94	28.48	24.64	12.62
College Education (%)	59.07	64.65	71.18	70.46	74.10	76.79	80.88	79.39	86.96	68.31
Employment (%)	63.72	66.89	71.83	68.14	67.66	70.61	66.18	60.61	69.57	68.16
Number of Living Children	1.25	1.10	0.87	0.88	0.74	0.75	0.67	0.79	0.42	0.90
Married (%)	30.82	40.52	40.85	49.59	47.25	50.76	51.47	45.45	28.99	43.41
Household Ownership (%)	25.68	32.21	39.99	49.89	53.06	59.33	60.54	56.36	42.03	45.98
Health Behaviors	1.86	1.67	1.53	1.45	1.42	1.37	1.33	1.27	1.33	1.63
Stressful Life Events	2.66	2.03	1.70	1.37	1.32	1.25	1.32	1.24	1.28	1.71
Neighborhood Disadvantage	0.09	0.06	−0.07	−0.09	−0.09	−0.14	−0.16	−0.12	−0.33	−0.09

Table 2
Financial assets and cardiometabolic risk estimated by Poisson regression models.

Variables	Full Sample		Male		Female	
Asset	−0.03**	−0.01*	−0.01	0.00	−0.05**	−0.03**
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Family SES		−0.04**		−0.05**		−0.03*
		(0.01)		(0.02)		(0.01)
Income (log)		−0.00		−0.01		−0.00
		(0.00)		(0.01)		(0.00)
Occupation Prestige		−0.02		−0.01		−0.02
		(0.01)		(0.02)		(0.02)
Age		0.03**		0.03**		0.03**
		(0.01)		(0.01)		(0.01)
Female		0.01				
		(0.02)				
Race (ref. = NH White)						
NH Black		0.16**		0.08†		0.23**
		(0.03)		(0.05)		(0.03)
Others		0.02		−0.03		0.07
		(0.04)		(0.05)		(0.04)
College+		−0.11**		−0.08†		−0.14**
		(0.02)		(0.04)		(0.03)
Living Children		0.02		0.03†		0.00
		(0.01)		(0.01)		(0.01)
Employment Status		0.04		0.04		0.04
		(0.03)		(0.04)		(0.03)
Married		0.06**		0.05		0.08**
		(0.02)		(0.04)		(0.03)
Home Ownership		−0.03		−0.02		−0.04
		(0.02)		(0.03)		(0.03)
Constant	0.76**	0.57**	0.67**	0.51**	0.84**	0.66**
	(0.02)	(0.07)	(0.04)	(0.10)	(0.03)	(0.07)
Observations	7700	7700	3585	3585	4115	4115

School-clustered standard errors in parentheses.
**p < .01, *p < .05, †p < .1.

Fig. 1 presents coefficient estimates from logistic regression models examining the association between financial assets and each of the seven individual biomarkers comprising the cardiometabolic risk index, accounting for the full set of socio-demographic characteristics presented in Table 2. In line with our findings on the cardiometabolic risk index, results show robust, sex-specific differences. Among female respondents, financial assets demonstrate statistically significant associations with blood pressure, waist circumference, and C-reactive protein, indicating that higher wealth is associated with better cardiovascular profiles, reduced central adiposity, and lower inflammation among women. The remaining biomarkers also show negative yet non-significant associations with financial assets among females.

In contrast, male respondents show no statistically significant associations between financial assets and any of the seven biomarkers. What is more, except for glycated hemoglobin, there is not a single positive point estimate for the male sample. To formally assess whether these sex differences are statistically significant, we estimated additional models for the full sample including gender × financial assets interaction terms. The interaction terms were statistically significant for blood pressure ($p < .05$), waist circumference ($p < .001$), and C-reactive protein ($p < .05$). This pattern suggests that women derive greater cardiometabolic benefits from financial wealth than men, with protective effects potentially operating through cardiovascular and anthropometric pathways among females.

4.1. Probing potential pathways

To understand the mechanisms through which financial assets influence cardiometabolic risk, we conducted mediation analyses examining three potential pathways: health behaviors, stressful life events, and neighborhood disadvantage. Figs. 2 and 3 present generalized structural equation modeling results for females and males separately, revealing different mediation patterns by gender (see Appendix Figure A4 for the full sample model).

Among females (Fig. 2), financial assets demonstrate significant associations with all three potential mediators. Higher asset levels are associated with better health behaviors ($p < .01$), fewer stressful life events ($p < .01$), and reduced neighborhood disadvantage ($p < .05$). However, only neighborhood disadvantage emerges as a statistically significant mediator, with exposure to adverse experiences positively predicting cardiometabolic risk ($p < .05$). The direct effect of financial assets on cardiometabolic risk remains substantial and significant ($p < .01$), indicating that the examined pathways explain only a portion of the protective association among women.

The male mediation pattern (Fig. 3) differs substantially from that observed among females. While financial assets maintain significant associations with health behaviors ($p < .05$) and stressful life events ($p < .05$), the association with neighborhood disadvantage is not statistically significant. More importantly, for males, the indirect effects of health behaviors are statistically significant ($p < .05$). However, the direct effect of financial assets on cardiometabolic risk is non-significant, consistent with our earlier findings that wealth-health associations are predominantly observed among women in this sample.

4.2. Supplementary analyses

Our primary analyses utilized Wave IV data to conduct an exploratory analysis on potential pathways linking household financial assets to cardiometabolic risk. However, the cross-sectional nature of this design constrains causal interpretation due to the inability to establish temporal ordering between predictors, mediators, and outcomes. To partially address this limitation, we conducted a supplementary analysis using a time-lagged framework that draws on the longitudinal structure of the Add Health study. Specifically, we measured household financial assets at Wave IV (2008–09, respondents aged 24–32) and examined their associations with mediating variables and cardiometabolic outcomes measured approximately seven to ten years later, in Wave V (2016–18, respondents aged 33–43). This design introduces a temporal

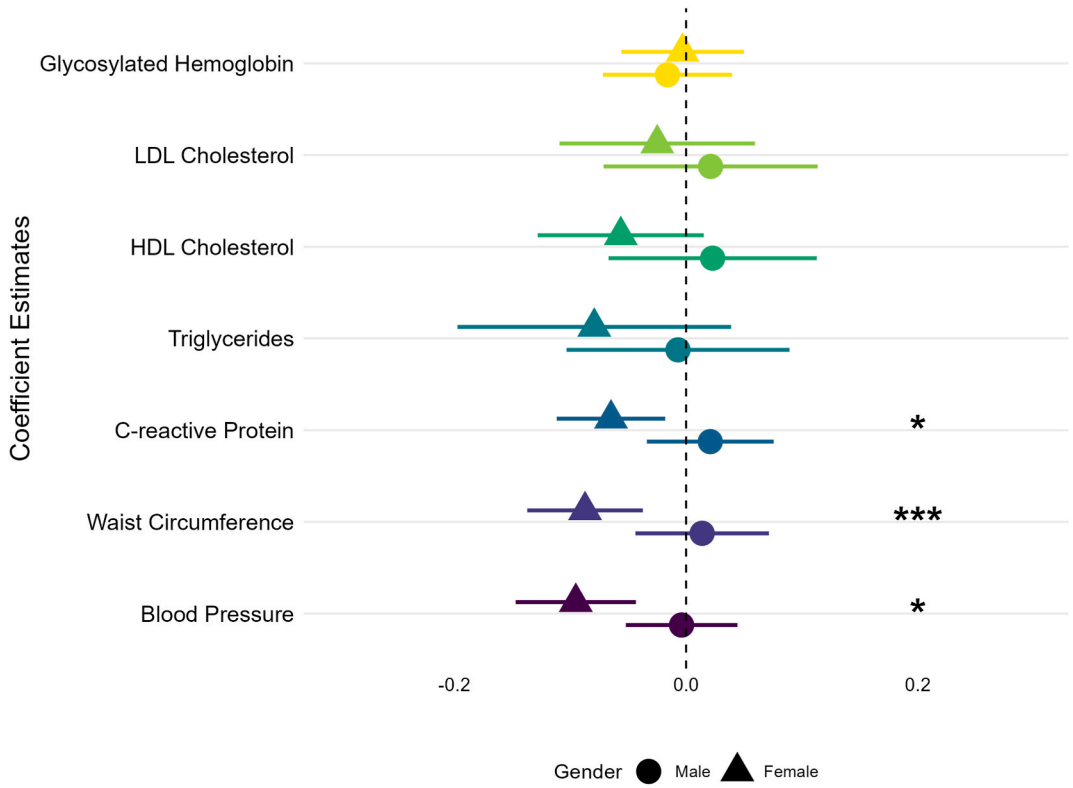

Fig. 1. Estimated coefficient on financial assets (individual biomarker risk by gender)
Notes: Whiskers represent 95% confidence intervals based on robust standard errors clustered at the school level to account for Add Health's complex survey design. Significance levels are denoted as follows: †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$. Significance levels indicate results from formal tests of gender differences based on financial assets × gender interaction terms in full sample models that include the same set of control variables as the stratified models. Estimates are derived from logistic regression models predicting each individual cardiometabolic risk indicator as a function of household financial assets, controlling for parental SES, respondent's age, race/ethnicity, education, personal income, occupational prestige, marital status, employment status, number of children, and homeownership. Models are stratified by sex and incorporate cross-sectional survey weights from Wave IV of Add Health.

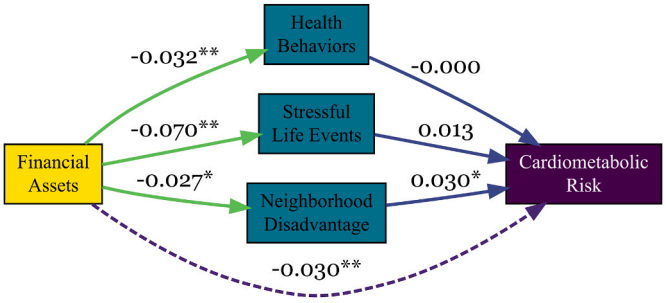

Fig. 2. Mediation pathways from financial assets to cardiometabolic risk (female)
Notes: Path coefficients are derived from generalized structural equation models (GSEM) estimating the direct and indirect effects of household financial assets on cardiometabolic risk. Solid arrows represent indirect pathways through health behaviors, stressful life events, and neighborhood disadvantage. The dashed arrow represents the direct pathway from financial assets to cardiometabolic risk. Coefficient values are displayed along each path, with significance indicated as follows: †$p < .10$, *$p < .05$, **$p < .01$. All models adjust for parental SES, age, race/ethnicity, educational attainment, income, occupational prestige, marital and employment status, number of children, and homeownership. Estimates are based on Wave IV data from Add Health, incorporating survey design weights and school-clustered standard errors.

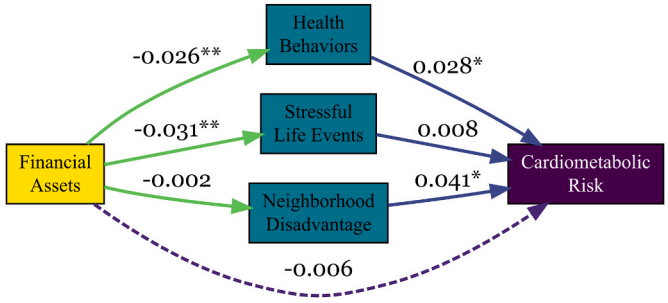

Fig. 3. Mediation pathways from financial assets to cardiometabolic risk (male)
Notes: Path coefficients are derived from generalized structural equation models (GSEM) estimating the direct and indirect effects of household financial assets on cardiometabolic risk. Solid arrows represent indirect pathways through health behaviors, stressful life events, and neighborhood disadvantage. The dashed arrow represents the direct pathway from financial assets to cardiometabolic risk. Coefficient values are displayed along each path, with significance indicated as follows: †$p < .10$, *$p < .05$, **$p < .01$. All models adjust for parental SES, age, race/ethnicity, educational attainment, income, occupational prestige, marital and employment status, number of children, and homeownership. Estimates are based on Wave IV data from Add Health, incorporating survey design weights and school-clustered standard errors.

gap between exposure, mediators, and health outcomes, thereby enhancing the plausibility of directional pathways. These supplementary models yield largely similar results to those presented in the main body of the paper (see Appendix Table A2, as well as Appendix Figures A5, A6, and A7), thus lending further support to the robustness of our findings.

To assess potential early-life health selection, we estimated supplementary models that additionally adjust for Wave I self-rated health, school absenteeism, English grade, binge drinking, and substance use

(Appendix Table A3; Appendix Figures A8-A11). The associations between financial assets and cardiometabolic risk remain substantively unchanged. These results suggest that early-life health and behavioral differences, including any gendered selection processes, do not account for our main findings.

In additional supplementary analyses, we further probe the robustness of the wealth gradients. Because Add Health respondents report financial assets on a nine-category scale, our main models use the categorical measure as provided; we additionally assess whether associations are concentrated at specific levels of assets (Appendix Figure A12). These results show little variation across wealth levels for men and only somewhat stronger associations concentrated in the upper half of the distribution for women, indicating that our conclusions are not driven by a single wealth category. We also re-estimate models using more detailed educational categories and find that, as expected, college education is strongly negatively associated with cardiometabolic risk, while the associations for financial assets remain substantively unchanged (Appendix Table A4; Appendix Figure A13). Finally, models that exclude homeownership yield very similar wealth estimates (Appendix Table A5; Appendix Figure A14), suggesting that our results are not solely capturing real estate holdings.

Next, we assess whether our findings change when adjusting for the level of debt, which includes all types of loans, credit card debt, and medical or legal bills, but excludes mortgage debt. Previous research shows that indebtedness can have adverse consequences for health (Lippert et al., 2022; Sweet et al., 2013), and research on wealth emphasizes that debt leaves an imprint on individuals' lives even when accounting for their assets (Dwyer, 2018). Results presented in Table A6 and Fig. A15 in the Appendix reveal no substantive differences from our main specifications.

5. Discussion

This study aimed to shed light on the distinct role of financial assets in shaping cardiometabolic health disparities during early adulthood, emphasizing the potential for differential associations by gender. Despite extensive research detailing socio-economic and wealth gradients in health, considerably less work has examined financial assets in early adulthood and their relation to cardiometabolic risk profiles. Addressing this gap, we investigated how household financial assets independently associate with biological markers of cardiometabolic risk, explicitly focusing on gender differences and mediating mechanisms. Our motivation stemmed from growing wealth inequalities and their potential implications for population health trajectories, necessitating nuanced explorations of wealth's unique health impacts. Recognizing how financial assets shape health risks during formative life stages might provide critical insights into emerging patterns of inequality. Our focus on financial assets net of education, income, and occupation therefore treats wealth as a distinct reservoir that can differentiate cardiometabolic risk even among individuals with ostensibly similar socio-economic profiles. In doing so, we extend wealth–health research that highlights assets as a buffer against structural constraints and chronic stress, showing that these dynamics are already visible in early adulthood (Boen et al., 2021).

Leveraging data from the Add Health survey, our analyses estimated associations between household financial assets and cardiometabolic risk. Utilizing Poisson regression and generalized structural equation modeling, we found negative associations between financial assets and cardiometabolic risk among women, predominantly driven by cardiovascular, inflammatory, and anthropometric pathways. Specifically, greater financial assets correlated significantly with lower blood pressure, reduced inflammation, and healthier waist circumferences for female respondents. Mediation analyses suggest that reduced exposure to neighborhood disadvantage might partially explain these relationships. Conversely, among men, we observed negligible associations, highlighting the stark gender divergence in how economic resources translate into biological health outcomes. Thus, our findings challenge the assumption that wealth uniformly benefits health in early adulthood, underscoring distinctly gendered pathways. Notably, these patterns emerge among respondents in their late twenties, when individual wealth portfolios are still relatively nascent. The presence of wealth gradients in cardiometabolic risk at this stage suggests that financial resources are already structuring physiological risk in ways that are likely to accumulate and intensify over subsequent decades.

These findings extend previous research demonstrating socio-economic gradients in cardiometabolic health among women to the specific domain of financial assets in early adulthood. While prior studies have established this gendered pattern using traditional SES measures (education, income, occupation) among middle-aged and older adults (Jenkins and Ofstedal, 2014; Phillips and Hamberg, 2015), our results demonstrate that this vulnerability persists for wealth-health associations during the critical developmental period of early adulthood. Rather than implying that women are insulated from the harms of economic disadvantage, our results underscore that cardiometabolic health is a domain in which socio-economic and wealth inequalities may be especially salient for women. Reading our results alongside Boen et al. (2021) suggests that the benefits of financial assets may be unequally distributed across social groups, and that gendered wealth positions in early adulthood represent an important frontier for research on cumulative health inequality.

This study has several limitations. First, our measure of financial assets is based on a nine-category household scale, which restricts our ability to observe meaningful heterogeneity, distinguish specific asset types, or separate asset holdings from particular forms of debt. Second, although we adjust for a broad set of socio-demographic characteristics and early-life health and behavioral indicators, unobserved confounding and selection into both wealth and health likely remain. Moreover, our evidence is based on a single U.S. cohort followed into their late twenties, and patterns may differ for older cohorts or in other institutional contexts, which may limit generalizability.

Our study additionally carries inherent methodological limitations, notably the cross-sectional nature of our primary analyses. Although financial asset data were temporally separated from health outcomes and mediating variables in supplementary analyses, without longitudinal information of wealth trajectories and health developments, the evidence presented here remains correlational. We call for future health surveys to prioritize comprehensive, longitudinal wealth assessments to accurately capture the relevance of financial assets for health outcomes. Such enhanced data infrastructure will profoundly improve understanding of how socio-economic resources perpetuate health disparities.

Despite these limitations, this study advances demographic scholarship on health inequalities by illuminating wealth as an independent determinant of cardiometabolic health during early adulthood. That the association of financial assets among women persists in models adjusting for conventional SES markers underscores that wealth does more than mirror income-based pathways; it captures additional latitude to navigate economic shocks and invest in health over time. Our work enriches our understanding by providing empirical evidence of substantial gender differences in how financial resources mitigate health risks, extending beyond established socio-economic markers such as education and income. Crucially, our results suggest that the health-protective effects of wealth might operate uniquely for women, suggesting gendered sensitivities to economic stressors and resource access. By contextualizing these findings within contemporary patterns of increasing wealth concentration, our research foregrounds the pressing need to integrate wealth more explicitly into health inequality frameworks. Ultimately, a comprehensive understanding of wealth-health intersections not only advances scholarly knowledge but critically informs interventions aimed at reducing entrenched health disparities rooted in economic inequality.

Statement EA

All procedures were performed in compliance with relevant laws and institutional guidelines. No human subjects were involved in this study.

CRediT authorship contribution statement

Haiyi Cheng: Formal analysis, Methodology, Visualization, Writing – original draft, Writing – review & editing. **Manuel Schechtl:** Conceptualization, Funding acquisition, Methodology, Project administration, Supervision, Writing – original draft, Writing – review & editing.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2026.119006.

Data availability

The authors do not have permission to share data.

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