



Psychological resilience to lifetime trauma and risk for cardiometabolic disease and mortality in older adults: A longitudinal cohort study

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ABSTRACT

Objective: Trauma exposure is associated with risk for cardiometabolic disease and mortality, in part through negative psychological sequelae. In contrast, psychological resilience, showing positive psychological health despite experiencing trauma, may offset some of this risk. We examined longitudinal associations of psychological resilience to lifetime trauma and cardiometabolic outcomes and mortality in a large sample of older adults.

Methods: Using data from 6596 US Health and Retirement Study participants who had experienced lifetime trauma (mean age 64), we defined manifested psychological resilience based on trauma burden and psychological health (composite of low distress and high psychological well-being) through 2012. New onset cardiometabolic disease (heart problems, stroke, diabetes) and mortality were assessed across eight years of follow-up (through 2020). Repeated measures regressions determined associations between psychological resilience and outcomes over follow-up, adjusting for sociodemographic factors and prior disease.

Results: Higher levels of manifested psychological resilience were associated with lower risk for developing any cardiometabolic outcomes (relative risk for one SD higher resilience score, RR = 0.91, 95%CI 0.88–0.94) and for all-cause mortality (RR = 0.73, 95%CI 0.63, 0.86), adjusting for sociodemographic confounders. When examining individual diseases, resilience was significantly associated with lower risk for certain heart problems (i.e., congestive heart failure) and diabetes. Associations generally held when additionally adjusting for adult psychosocial and biobehavioral factors that could be potential pathway variables.

Conclusion: Psychological resilience to lifetime trauma may be linked to better cardiometabolic health even later in life. Promoting recovery and psychological resilience to trauma may be a target for more favorable health and longevity.

1. Introduction

While trauma exposure may increase risk for cardiometabolic disease [1,2] and all-cause mortality [3], psychological responses following trauma may be key factors influencing subsequent health outcomes. Moreover, although trauma is a potent risk factor for psychiatric disorders [4], not everyone who experiences trauma develops poor mental health. Many individuals show psychological resilience, broadly defined as “the process and outcome of successfully adapting to difficult or challenging life experiences” [5]. Resilience has been conceptualized in various ways, including as a trait-like capacity (e.g., one's perceived ability to overcome adversity encapsulating intrapersonal features and

behaviors, often measured via self-report scale), a dynamic process over time (e.g., involving interplay of risk and protective resources enabling adaptation to adversity), or an outcome following adversity (e.g., measured by assessing psychological health following trauma or adversity exposure) [6]. In this study, we focus on resilience as an outcome whereby individuals show positive psychological functioning despite prior experiences of trauma, operationalized as the level of psychological health relative to the level of trauma exposure [6–8]. Individuals demonstrating higher psychological resilience to trauma may have lower risk for cardiometabolic outcomes that would otherwise be increased due to trauma exposure.

Growing evidence indicates associations between resilience and

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cardiometabolic disease [9]. Longitudinal analyses of Swedish male military conscripts have identified that stress resilience, defined as psychologist-rated ability to cope with conflict, was associated with lower risk for multiple cardiometabolic diseases across 23–46 years of follow-up [10–13]. In a 20-year longitudinal study of adults (56% women), higher psychological resilience to early adversity using an outcome-based definition was associated with lower risk for incident cardiometabolic diseases [14]. Cross-sectional evidence in an Italian adult cohort (50% women) indicated that higher resilience, measured by self-report trait scale, was associated with lower prevalence of cardiovascular disease (CVD) [15]. Additionally, higher self-reported resilience capacity was associated with lower mortality risk in a large Chinese older adult cohort (59% women, mean age 86) over three years [16,17]. Other analyses failed to identify associations. For example, resilience (measured by self-report trait scale) was not found to be associated with incidence of CVD over 23 years among African American women [18], and resilience was unassociated with diabetes prevalence in the Italian cohort [15]. Thus, there has been some inconsistency in findings across studies to date.

Differing definitions and operationalizations of resilience may have resulted in variability across studies. Manifested resilience can be operationalized in multiple ways, including as a continuous measure of levels psychological health relative to trauma burden [19,20] or as categories based on a priori clinical cutoffs [6,21]. Both operationalizations consider one's trauma exposure and subsequent psychological functioning [7]. Prior studies used self-reported resilience capacity, psychologist-rated resilience capacity, or assessed psychological resilience to early adversity only, rather than manifested resilience to lifetime trauma. Additional studies are needed that define manifested resilience more comprehensively, including applying residual-based resilience measures, and focusing on populations at high risk for cardiometabolic disease. Studies need to include a number of cardiometabolic endpoints and to include older adults who are at higher risk for many chronic conditions, including cardiometabolic diseases [22], such as heart disease, stroke, and diabetes [23]. Furthermore, divergent findings in samples of men versus women indicate there could be gender differences in associations between resilience and cardiometabolic disease, though few studies to our knowledge have examined this explicitly.

In this study, we determined whether psychological resilience to lifetime trauma was associated with cardiometabolic diseases or mortality in the Health and Retirement Study (HRS), a longitudinal cohort of older US adults. We hypothesized that higher levels of residualized psychological resilience to lifetime trauma would be associated with lower risk for cardiometabolic outcomes and mortality over approximately eight years of follow-up.

2. Methods

2.1. Sample

Data came from HRS, a population-based longitudinal study of US adults over age 50 and their spouses, sponsored by the National Institute on Aging (U01AG009740), conducted by the University of Michigan [24], and made publicly available (<https://hrs.isr.umich.edu/data-products>). HRS began in 1992 with US adults born 1931–1941 and has been refreshed with additional birth cohorts [25]. Interviews assessed factors related to health, labor force participation, and aging every two years. Beginning in 2006, psychosocial factors were assessed in supplemental questionnaires administered to half of the sample at alternating years (e.g., half completed the questionnaire in 2006 and again in 2010, the other half completed the questionnaire in 2008 and again in 2012). As psychological resilience was defined using variables from 2006 to 2012 waves, we considered 2012 our study baseline and waves 2014–2020 as follow-up. Among those who participated in at least one wave between 2006 and 2012 ($n = 16,820$), we excluded 7138

individuals who were missing resilience information and 1169 additional individuals who did not complete at least one follow-up wave. Among 8513 remaining individuals, we included 6596 individuals who had experienced at least one lifetime trauma, given our definition of resilience as a manifested response to trauma exposure (Fig. A.1). All participants received an informed consent document, were read a confidentiality statement, and gave oral consent to participate. Ethical approval for HRS was granted from the University of Michigan Institutional Review Board and the study has been conducted according to the principles of the Declaration of Helsinki.

2.2. Measures

2.2.1. Psychological resilience

Psychological resilience was defined based on lifetime trauma burden and psychological health (measure details in Table A.1). Lifetime trauma included 11 lifetime potentially traumatic events [26] assessed in waves 2006–2012 (endorsement during any of four possible waves was considered exposure). Participants reported whether they had experienced any of four events before age 18 (i.e., repeating a year of school, trouble with police [added in 2008], family problems due to parental alcohol or drug use, physical abuse by a parent) and seven events occurring anytime (i.e., death of a child; experienced a major natural disaster; combat exposure; spouse, partner, or child addicted to drugs or alcohol; experienced serious physical attack; experienced life threatening illness or accident; spouse or child experiencing a life threatening illness or accident). Consistent with work on cumulative trauma [27–29], we indexed *trauma burden* by calculating the total number of endorsed event types (potential range 1–11) as a continuous variable. We also created an indicator for any versus no childhood trauma (any of four events before age 18), as early life experiences especially may have potent and lasting impacts on later health [30].

Psychological health was a composite of distress and psychological well-being measures, spanning the mental health spectrum. Distress was measured using past week depressive (8-item sum score from the Center for Epidemiological Studies-Depression scale [31,32]) and anxiety (5-item sum score from the Beck Anxiety Inventory [33]) symptoms, completed in waves 2006–2012 (latest time point was used in the case of multiple assessments). Psychological well-being was captured using multiple related but distinct aspects of well-being spanning hedonic (pleasure or positive affective experience) and eudaimonic (meaning and self-realization) dimensions [34]. Measures included life satisfaction, a hedonic dimension of self-evaluative life quality (5-item Satisfaction with Life Scale [35]); positive affect, a hedonic measure of general positive emotionality (13 items from the Positive and Negative Affect Schedule-Expanded Form [36]); optimism, typically considered eudaimonic and defined as generalized favorable expectancies for one's future (6-item Life Orientation Test-Revised [37]); and purpose in life, a eudaimonic dimension meaning one's sense of goals in the process of living (7 items from the Ryff Measures of Psychological Well-being [38]). As psychological well-being is a complex, multidimensional construct, we included various measures to capture broad psychological functioning that may reflect positive psychological functioning following trauma [39]. Consistent with scoring recommendations, mean scores for each scale were computed. A continuous psychological health composite variable was defined by standardizing each individual distress and psychological well-being score ($M = 0$, $SD = 1$) and creating a weighted sum (equally weighting distress and well-being), with inverted distress symptoms such that higher total scores reflect higher overall psychological health.

Psychological resilience was defined as one's level of psychological health relative to level of trauma exposure, specifically the difference in their true psychological health level from their expected psychological health at a specific trauma exposure level [20,40,41]. Using a regression model, we estimated the linear association of trauma count with level of continuous psychological health. As anticipated, higher trauma

Table 1Distribution of covariates among the analytic sample and associations with psychological resilience scores, $n = 6596$.

Covariate		Full Sample	Resilience Score	p-value ^a
		N (%)	M (SD) or r	
Age, <i>m</i> (SD)		63.8 (11.7)	0.07	0.896
Gender	Women	3882 (58.9)	-0.04 (1.0)	<0.001
	Men	2714 (41.1)	0.06 (0.9)	
Race	White	4810 (72.9)	0.06 (1.0)	<0.001
	Black or African American	1211 (18.4)	-0.15 (1.0)	
	Other	561 (8.5)	-0.19 (1.0)	
Ethnicity	Hispanic	796 (12.1)	-0.18 (1.0)	<0.001
	Not Hispanic	5793 (87.8)	0.03 (1.0)	
Parental Education	<8 years of education	978 (14.8)	-0.24 (1.0)	<0.001
	≥8 years of education	5219 (79.1)	0.07 (1.0)	
Participant Education	Less than high school	924 (14.0)	-0.40 (1.0)	<0.001
	GED	354 (5.4)	-0.41 (1.1)	
	High school graduate	1756 (26.6)	-0.09 (1.0)	
	Some college	1881 (28.5)	0.05 (1.0)	
	College or more	1681 (25.5)	0.33 (0.9)	
Marital Status	Married/partnered	4495 (68.1)	0.11 (0.9)	<0.001
	Divorced/separated	1005 (15.2)	-0.27 (1.1)	
	Widowed	684 (10.4)	-0.11 (1.0)	
	Never married	348 (5.3)	-0.43 (1.1)	
BMI, <i>m</i> (SD)		29.0 (6.4)	-0.11	0.613
Smoking	Current smoker	1106 (16.8)	-0.44 (1.1)	<0.001
	Non-smoker	5310 (80.5)	0.10 (1.0)	
Physical Activity	Never	3314 (50.2)	-0.21 (1.1)	<0.001
	>1 time per month	653 (9.9)	0.17 (0.9)	
	1–3 times per month	700 (10.6)	0.14 (0.9)	
	1–2 times per week	1576 (23.9)	0.31 (0.9)	
	≥3 times per week	155 (2.3)	0.26 (0.8)	
Alcohol Consumption	Never drink alcohol	2415 (36.6)	-0.14 (1.0)	<0.001
	Drink alcohol	4002 (60.7)	0.09 (1.0)	
Any Cardiometabolic Disease History	Ever	2515 (38.1)	-0.16 (1.0)	<0.001
	Never	4081 (61.9)	0.10 (1.0)	

Covariate missingness: race 0.2%, ethnicity 0.1%, parental education 6.0%, marital status 1.0%, smoking 2.7%, physical activity 3.0%, alcohol consumption 2.7%.

^a *p*-values for *t*-tests or ANOVA for resilience scores by categorical covariates, or Pearson correlations (*r*) for resilience scores and continuous covariates.

exposure was associated with lower psychological health. A linear model was best fitting after testing linear, squared, and cubic trauma effects, and we found no violations of linearity, multivariate normality, independence, or homoscedasticity. In linear regression, it is possible that extreme individuals (e.g., very low trauma exposure but low psychological health, or very high trauma exposure but high psychological health) may disproportionately influence model estimates and lead to bias in model fit. To correct for this, we calculated Cook's distance, a measure of how much an individual observation influences overall model fit, for each observation and excluded potential influencers from our final model [20]. From this model excluding influencers, we derived predicted values (what one's estimated psychological health would be, given their trauma exposure level) for the full sample and then extracted residuals (observed psychological health levels minus their predicted values) to index continuous resilience scores. Higher values of these scores indicate relatively more favorable psychological health than would be expected based on trauma burden (i.e., higher resilience).

2.2.2. Cardiometabolic outcomes and mortality

Self-reported physician diagnosis of heart problems (a follow-up question assessed type: heart attack, angina, or congestive heart failure), stroke, and diabetes were assessed at each wave. Mortality (date of death) and cause of death was determined across follow-up from reports of household members and via the National Death Index. Death due to heart problems, stroke, and diabetes for waves 2014, 2016, and 2018 were also included as cardiometabolic cases (2020 cause of death was unavailable at the time of analysis). Indicator variables were created at each wave to define cardiometabolic outcomes (any cardiometabolic outcomes = 1, none by the current wave = 0), for any cardiometabolic outcomes and for each disease individually. We also created an indicator variable for cardiometabolic disease history (any = 1 versus no = 0 cardiometabolic outcomes ever reported), and for each disease

individually (e.g., any = 1 versus no = 0 heart problems ever reported) as of baseline in 2012, to be included in all models to adjust for prior cardiometabolic disease. We also included both all-cause and cardiometabolic-specific mortality by each wave as endpoints.

2.2.3. Covariates

Covariates included sociodemographic confounders, as well as adult psychosocial, health, and behavioral factors that may be mechanisms. Sociodemographic covariates reported at HRS baseline included age, gender, race, ethnicity, parental education attainment, participant education attainment, and marital status. Health and behavioral factors were time-updated at each follow-up wave and included body mass index (BMI, derived as kg/m^2 from self-reported height and weight), smoking, vigorous physical activity, and alcohol consumption. See Table 1 for values of all categorical covariates

2.3. Analyses

We first examined covariate distributions between our analytic sample ($n = 6596$ among 16,820 potential participants) versus those excluded due to missing resilience information, lack of follow-up, or no lifetime trauma exposure. Excluded individuals were older; more likely to be Black or other race, Hispanic, and widowed; had lower education attainment, lower average BMI, and lower physical activity; and were less likely to drink alcohol and more likely to smoke. We additionally examined covariate distributions within our analytic sample, comparing those lost to follow-up ($n = 1442$) versus those retained. Those who dropped out were younger, had lower education attainment, lower average BMI, and were more likely to smoke. Differences indicated potential selection bias with excluded or lost to follow-up individuals having more marginalized identities and lower socioeconomic status. To account for these differences, we used logistic regression to calculate the

Table 2Associations between psychological resilience and cardiometabolic disease and mortality across follow-up, $n = 6569$.

		Predictor: Psychological Resilience Score			
		Model 1	<i>p</i> -value	Model 2	<i>p</i> -value
Outcome: Incident Cases	<i>N</i> (%)	RR (95% CI)	<i>p</i> -value	RR (95% CI)	<i>p</i> -value
Any Cardiometabolic Disease	1167 (17.7)	0.91 (0.88, 0.94)	<0.001	0.92 (0.89, 0.95)	<0.001
Heart Problems	767 (11.6)	0.89 (0.59, 1.36)	0.585	0.90 (0.60, 1.36)	0.607
Heart Attack	98 (1.5)	1.02 (0.32, 3.27)	0.974	1.04 (0.33, 3.31)	0.947
Angina	169 (2.6)	0.90 (0.56, 1.43)	0.630	0.91 (0.57, 1.45)	0.684
Congestive Heart Failure	132 (2.0)	0.60 (0.48, 0.75)	<0.001	0.62 (0.50, 0.77)	<0.001
Stroke	323 (4.9)	0.71 (0.48, 1.06)	0.088	0.74 (0.50, 1.10)	0.126
Diabetes	839 (12.7)	0.91 (0.85, 0.97)	0.004	0.92 (0.87, 0.98)	0.008
Mortality					
All-Cause Mortality	1154 (17.5)	0.73 (0.63, 0.86)	<0.001	0.78 (0.66, 0.92)	0.004
Cardiometabolic Mortality	254 (3.9)	0.81 (0.60, 1.08)	0.149	0.85 (0.63, 1.15)	0.294

CI = confidence intervals; RR = relative risk.

Model 1: adjusted for time, resilience*time, age, gender, race, ethnicity, parental education, and history of cardiometabolic disease; Model 2: additionally adjusted for participant education, marital status, BMI, smoking, physical activity, and alcohol consumption. All models adjusted for inverse probability weighting for selection bias and loss to follow-up, and multiple imputation for missing data. Psychological resilience scores are standardized ($m = 0$, $SD = 1$).

odds of inclusion in the sample (among 16,820 potential participants), and of not being lost to follow-up (among the analytic sample) predicted by baseline covariates. From these models, the inverse probability of each outcome was derived and multiplied to create an overall inverse probability weight, similar to prior work [14]. All analytic models adjusted for these weights, which effectively weight analyses to look more representative of the original sample. Given missing covariate information (see Table 1), we also conducted all analytic models using multiple imputation. Models were run in 25 imputed datasets using the MICE multiple imputation procedure with predictive mean matching in R [42], and parameter estimates and standard errors were obtained by pooling consistent with Rubin's rules [43].

In the analytic sample, we examined associations between covariates and resilience scores. The primary analytic models were repeated measures regressions with generalized estimating equations, which estimate population-level effects while accounting for longitudinal, repeated outcome measures with robust error variance [44,45]. Poisson regressions with log links were used to determine relative risk (RR) of binary outcomes over time. Models were conducted with standardized resilience score predicting any cardiometabolic outcomes and mortality over follow-up. All models were adjusted for time (years since baseline) and included resilience*time interactions to determine whether associations between resilience and outcomes were stable across time. Models first adjusted for sociodemographic confounders that are either stable or likely precede lifetime trauma, including age, gender, race, ethnicity, and parental education, as well as cardiometabolic disease history as of baseline (Model 1). Models then additionally adjusted for time-updated social, health, or behavioral factors, which could be confounders or potential pathway variables including participant education, marital status, BMI, smoking, physical activity, and alcohol consumption (Model 2). All models were run with imputed covariate data and adjusted for inverse probability weights. As secondary analyses, we reran the primary models for resilience with each cardiometabolic disease type (i.e., heart problems [including subtypes: heart attack, angina, and congestive heart failure; there was some missingness of specific subtypes], stroke, and diabetes). These models were adjusted for any prior experience of each individual disease.

We conducted several sensitivity analyses. To determine whether individual resilience components were associated with cardiometabolic outcomes, we reran models with each resilience component as a primary predictor first separately (i.e., trauma count or psychological health as primary predictor) and then co-adjusted for trauma count and psychological health. Additional sensitivity analyses restricted the sample to 4081 individuals who had no cardiometabolic diseases as of 2012, for a cleaner assessment of incident diseases.

Exploratory analyses with interactions were conducted to determine

whether resilience-cardiometabolic disease associations differed by gender and by exposure to childhood trauma.

Analyses were conducted in R, version 4.2.3, all tests were two-sided, and an a priori threshold of $p < .05$ defined statistical significance.

3. Results

3.1. Descriptive statistics

The analytic sample was 63.8 years old on average in 2012, 58.9% were women, 72.9% were White, 18.4% were Black or African American, and 12.1% were Hispanic (Table 1). Among the trauma-exposed sample, 27.2% experienced two, 17.4% experienced three, and 21.0% experienced four or more types of traumatic events in their lifetimes. Measures of psychological well-being were negatively correlated with distress ($r_s = -0.28$ to -0.41), and positively correlated with each other ($r_s = 0.34$ to 0.54 ; Table A.2). As expected, higher trauma count was significantly associated with lower psychological health ($\beta = -0.11$, 95%CI -0.13 , -0.10) in the regression model deriving resilience scores. Resilience scores were associated with most demographic and behavioral covariates, with men, White, and non-Hispanic individuals, those with higher education, and married individuals having higher resilience scores.

As an older sample, unsurprisingly 38.1% of individuals reported at least one cardiometabolic disease by 2012. Over the eight-year follow-up, 17.7% of the sample reported cardiometabolic outcomes. Regarding individual diseases over follow-up, diabetes (12.7%) and heart conditions (11.6%) were most common, followed by stroke (4.9%). A minority (3.2%) had two or three of the queried cardiometabolic outcomes. Across follow-up, 17.5% of the sample died, with 3.9% having cardiometabolic specific causes of death.

3.2. Psychological resilience and cardiometabolic outcomes

Higher psychological resilience was associated with lower risk for any cardiometabolic outcome over time (RR = 0.91, 95%CI 0.88–0.94), adjusting for sociodemographic confounders and history of cardiometabolic diseases (Table 2, Fig. 1). This association was slightly attenuated when adjusting for adult social, health, and behavioral factors. We did not identify a significant resilience*time interaction, indicating that associations between resilience and cardiometabolic outcomes were stable across follow-up. For individual diseases, higher resilience was significantly associated with lower risk for diabetes (RR = 0.91, 95%CI 0.85–0.97), while associations were in the protective direction but not significant for stroke (RR = 0.71, 95%CI 0.48–1.06) and heart problems (RR = 0.89, 95%CI 0.59–1.36). Regarding specific

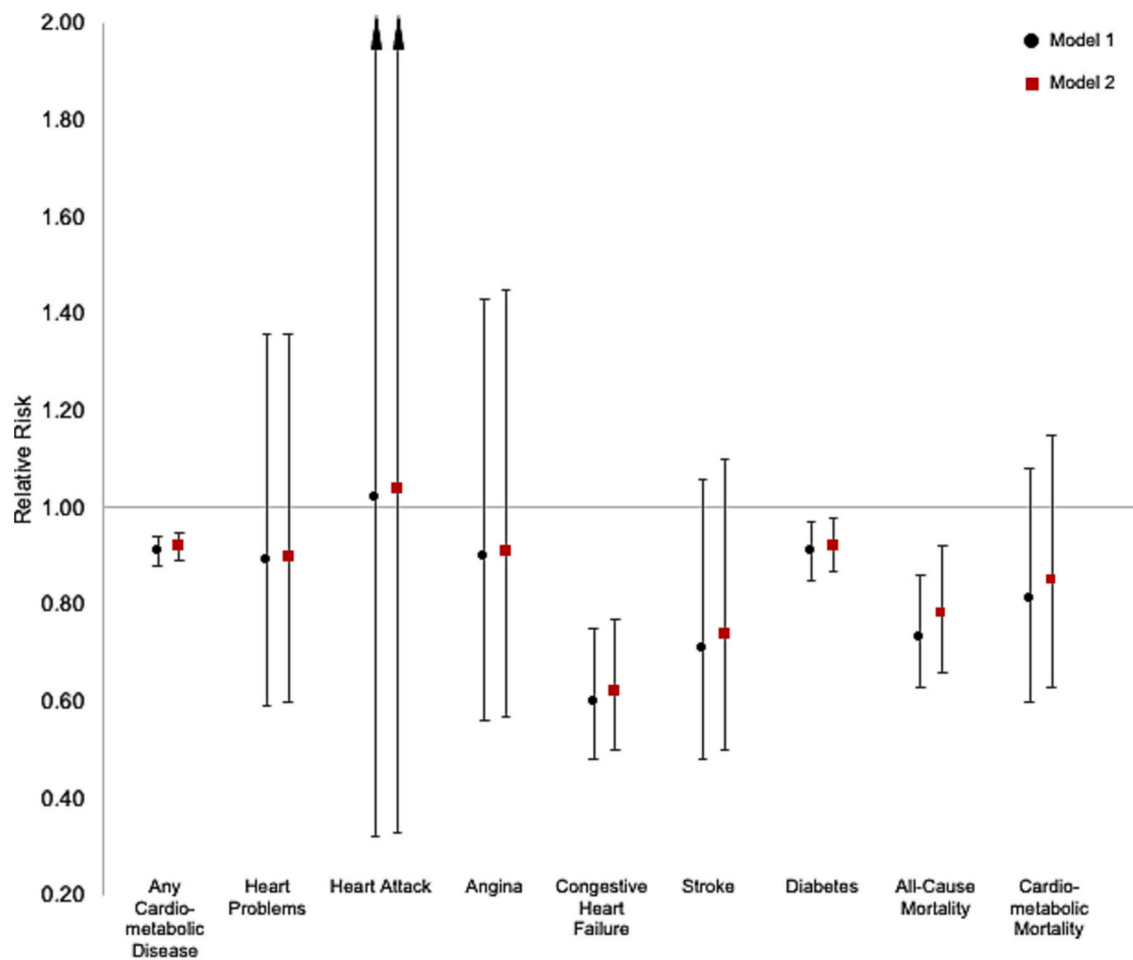


Fig. 1. Relative risk and 95% confidence intervals for associations between psychological resilience and cardiometabolic disease and mortality across follow-up, $n = 6596$.

Relative risks are for one standard deviation increase in resilience score. Model 1: adjusted for time, resilience*time, age, gender, race, ethnicity, parental education, and history of cardiometabolic disease; Model 2: additionally adjusted for participant education, marital status, BMI, smoking, physical activity, and alcohol consumption. All models adjusted for inverse probability weighting for selection bias and loss to follow-up, and multiple imputation for missing data.

heart problems, resilience was significantly associated with lower risk for congestive heart failure (RR = 0.60, 95%CI 0.48–0.75), but not heart attack or angina (Table 2, Fig. 1). Associations between resilience and specific outcomes were only modestly attenuated when adjusting for all covariates.

3.3. Psychological resilience and mortality

One standard deviation higher resilience score was associated with 27% lower risk for all-cause mortality over follow-up, adjusting for sociodemographics and prior cardiometabolic disease (RR = 0.73, 95% CI 0.63–0.86; Table 2, Fig. 1), which remained significant when fully adjusted. Resilience was protective but not significantly associated with lower cardiometabolic mortality.

3.4. Sensitivity analyses

Both trauma count (association for each additional trauma, RR = 1.06, 95%CI 1.04–1.07) and psychological health (association for one SD increase in psychological health, RR = 0.90, 95%CI 0.87–0.93) were significantly associated with cardiometabolic outcomes in individual models (Table A.3). When co-adjusted, both trauma and psychological health attenuated slightly but remained significant independently.

When restricting to individuals who did not report cardiometabolic diseases by 2012, resilience was associated with lower risk for any

cardiometabolic disease (Table A.4). Overall patterns were similar to the primary results, with associations in the same direction and some effect estimates of even stronger magnitudes, while precision was lower (i.e., wider confidence intervals).

We did not identify significant interactions in exploratory models (Table A.5), suggesting associations between resilience and any cardiometabolic disease did not differ by gender or by experience of childhood trauma versus only adulthood/lifetime trauma.

4. Discussion

Manifested psychological resilience to lifetime trauma was protective against cardiometabolic diseases and all-cause mortality in a large community-based sample of older adults. Consistent with our hypotheses, psychological resilience, operationalized as a score of psychological health relative to trauma burden, was associated with lower risk for any cardiometabolic diseases over follow-up. Examining specific outcomes, resilience was associated with lower risk of congestive heart failure and diabetes, but not heart attack, angina, or stroke. These findings, consistent with some prior work [10,11], indicate that individuals who experience psychological health despite trauma could be protected against cardiometabolic disease in later life. Resilience was also protective against all-cause mortality across follow-up, suggesting a broadly protective effect. We add to growing literature linking resilience and cardiometabolic health, extending prior work by using a comprehensive,

empirically derived measure of resilience in relation to lifetime trauma and focusing on older adults at higher risk for poor health outcomes. Moreover, we examined various cardiometabolic disease manifestations, which provides initial evidence of potential mechanisms to explore, and mortality as a comprehensive health endpoint.

Psychological resilience was associated with lower risk of any cardiometabolic outcomes over time, adjusting for confounders. When additionally adjusting for adult social, health, and behavioral factors, which could be contributors to resilience or pathway variables linking resilience with health outcomes, associations were modestly attenuated. Complementing our main findings, sensitivity analyses supported the hypothesis that both trauma exposure and later psychological health matter independently for disease outcomes. Prior findings in this area have been mixed, with several prospective studies indicating significant associations between stress resilience and lower risk for diabetes [12], coronary heart disease [10], and stroke [11]. In contrast, other studies in more diverse populations have produced null associations between psychological resilience and CVD or diabetes [18,46]. As work was generally limited with a focus on specific populations (e.g., Swedish male conscripts) and self-report or psychologist-rated resilience capacity measures, we complement existing evidence by including a population-based study of older adults and an empirically-derived resilience measure.

Our findings indicate that resilience was associated with lower likelihood of certain outcomes, such as congestive heart failure and diabetes, emphasizing the need to examine outcomes individually. Mechanisms underlying the decreased risk for cardiometabolic outcomes could include stress-related physiological and behavioral pathways [1,2,47,48]. Physiologically, chronic stress stemming from trauma exposure and related psychopathology can cause dysregulation of the autonomic nervous system and hypothalamic-pituitary-adrenal axis, chronic low-grade systemic inflammation, dysregulated immune functioning, and accelerated cellular aging via epigenetic effects [1,28,48,49]. These interacting physiological changes can increase risk for cardiometabolic disease via impacts on atherosclerosis, glucose metabolism, and insulin resistance [1,49]. Although not directly studied, showing psychological resilience following trauma may lessen or inhibit these physiological impacts, thereby lowering disease risk. Behaviorally, showing psychological resilience may be associated with more healthy behavior patterns [9,50], as also seen in the current data, with resilience linked to non-smoking and higher physical activity at baseline. While resilience may promote healthier behaviors, which may in turn lower disease risk, accounting for time-updated behaviors in our analyses did not greatly impact associations, suggesting they were not prominent mediating factors. Behaviors in older adulthood may be less mechanistically linked to disease endpoints, compared to behavior earlier in life. Given shared risk factors for heart conditions, stroke, and type 2 diabetes [51], we had hypothesized that psychological resilience would broadly impact disease risk. Interestingly, resilience was not associated with heart attack, angina, or stroke, but protective associations with both congestive heart failure and diabetes suggests that resilience may indeed impact certain shared mechanisms, such as oxidative stress, endothelial dysfunction, atherosclerotic processes, or metabolic dysregulation [48,52] increasing risk for various disease endpoints.

This is among the first studies to link psychological resilience with all-cause mortality. Prior work in a large older Chinese cohort indicated that higher resilience, measured with seven self-report items covering positive and negative affect, was associated with lower mortality risk [16,17]. We found that resilience, considering both lifetime trauma and positive and negative psychological health dimensions, may protect against mortality risk. We did not find significant associations with cardiometabolic mortality, though prevalence was low and misclassification was possible. Although requiring replication, such evidence is promising that psychological resilience may broadly be associated with healthier trajectories across late life.

The timing of our study may have impacted our ability to identify significant associations across cardiometabolic endpoints. Our primary exposure, psychological resilience, was classified relatively late in life and eight years of follow-up began after that assessment. Eight years in older adulthood may not be enough time to identify new outcomes, especially cardiometabolic diseases which can take years to develop. Additionally, our examination of cardiometabolic disease may have been hindered by a healthy survivor effect, with those included in the sample at all and those without cardiometabolic diseases by 2012 reflecting healthier individuals who may be less likely to develop disease in general. Given these factors, our observed results may have underestimated the magnitude of associations. Additional work with longer follow-up and more population-representative samples should be conducted to further examine associations of later life psychological resilience and cardiometabolic disease. Future studies should also examine cardiometabolic disease prognosis. For example, although a large portion of older adults may have developed cardiometabolic disease, those who show higher psychological resilience may have lower disease severity or slower progression.

5. Limitations

As noted, sociodemographic differences were identified between the analytic sample versus those excluded and lost to follow-up. However, we attempted to statistically account for potential biases with inverse probability weighting to adjust for differences. We could not comprehensively assess all potential lifetime traumas and relied on retrospective reports that are subject to recall bias [53,54]. Relatedly, although psychological resilience to trauma is often understood as a dynamic process unfolding over time [7], we assessed a snapshot of psychological resilience, which could have resulted in measurement error. Moreover, there was no assessment of psychological or pharmacological treatments, therefore it is possible that resilience to trauma was due to receiving effective treatment. Our primary outcome was newly reported cardiometabolic disease or mortality over follow-up, and we adjusted for any prior diagnoses, which is not strictly an assessment of incident cases. We chose to adjust for pre-existing disease given the large prevalence (38.1%) of disease as of baseline. However, sensitivity analyses among only those without pre-existing disease, despite limited statistical power, found generally similar results. There was some missingness of specific heart problem types, and potential for misclassification in self-reported disease outcomes in general. Although HRS was developed as a population representative sample of older US adults, given noted issues of selection bias and restrictions to derive our analytic sample, we did not apply HRS survey weights thus cannot generalize more broadly. Relatedly, HRS may reflect a healthier sample than the general population due to selective mortality resulting in healthier and more socio-economically advantaged individuals who can participate in longitudinal cohorts in older adulthood [55].

6. Conclusions

Although we only captured a snapshot of psychological resilience to lifetime trauma in older adulthood, we found associations between higher resilience and lower likelihood of certain forms of cardiometabolic disease and all-cause mortality. Our findings suggest that promoting recovery and supporting mental health following trauma exposure may have additional benefits beyond psychological well-being, which could extend to cardiometabolic health and longevity.

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Declaration of Competing Interest

The authors have no competing interests to report.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jpsychores.2023.111539>.

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