

UCSF

UC San Francisco Previously Published Works

Title

Social support, psychosocial risks, and cardiovascular health: Using harmonized data from the Jackson Heart Study, Mediators of Atherosclerosis in South Asians Living in America Study, and Multi-Ethnic Study of Atherosclerosis

Permalink

<https://escholarship.org/uc/item/2vg5n7gd>

Authors

Park, Jee Won
Howe, Chanelle J
Dionne, Laura A
et al.

Publication Date

2022-12-01

DOI

10.1016/j.ssmph.2022.101284

Peer reviewed



Social support, psychosocial risks, and cardiovascular health: Using harmonized data from the Jackson Heart Study, Mediators of Atherosclerosis in South Asians Living in America Study, and Multi-Ethnic Study of Atherosclerosis

Jee Won Park^{a,b,c}, Chanelle J. Howe^{a,b}, Laura A. Dionne^d, Matthew M. Scarpaci^e, Belinda L. Needham^f, Mario Sims^g, Alka M. Kanaya^h, Namratha R. Kandulaⁱ, Joseph L. Fava^{d,j}, Eric B. Loucks^{b,d}, Charles B. Eaton^{b,k}, Akilah J. Dulin^{a,d,*}

^a Center for Epidemiologic Research, Brown University, Providence, RI, USA

^b Department of Epidemiology, Brown University, Providence, RI, USA

^c Program in Epidemiology, University of Delaware, Newark, DE, USA

^d Center for Health Promotion and Health Equity Research, Department of Behavioral and Social Sciences, Brown University, Providence, RI, USA

^e Hassenfeld Child Health Innovation Institute, Brown University, Providence, RI, USA

^f Department of Epidemiology, University of Michigan, Ann Arbor, MI, USA

^g Department of Medicine, University of Mississippi Medical Center, Jackson, MS, USA

^h Division of General Internal Medicine, University of California, San Francisco, San Francisco, CA, USA

ⁱ Department of Internal Medicine, Northwestern University, Chicago, IL, USA

^j Centers for Behavioral and Preventive Medicine, The Miriam Hospital, Providence, RI, USA

^k Department of Family Medicine, Warren Alpert Medical School of Brown University, Providence, RI, USA

ABSTRACT

Purpose: Social support may have benefits on cardiovascular health (CVH). CVH is evaluated using seven important metrics (Life's Simple 7; LS7) established by the American Heart Association (e.g., smoking, diet). However, evidence from longitudinal studies is limited and inconsistent. The objective of this study is to examine the longitudinal relationship between social support and CVH, and assess whether psychosocial risks (e.g., anger and stress) modify the relationship in a racially/ethnically diverse population.

Methods: Participants from three harmonized cohort studies – Jackson Heart Study, Mediators of Atherosclerosis in South Asians Living in America, and Multi-Ethnic Study of Atherosclerosis – were included. Repeated-measures modified Poisson regression models were used to examine the overall relationship between social support (in tertiles) and CVH (LS7 metric), and to assess for effect modification by psychosocial risk.

Results: Among 7724 participants, those with high (versus low) social support had an adjusted prevalence ratio (aPR) and 95% confidence interval (CI) for ideal or intermediate (versus poor) CVH of 0.99 (0.96–1.03). For medium (versus low) social support, the aPR (95% CI) was 1.01 (0.98–1.05). There was evidence for modification by employment and anger. Those with medium (versus low) social support had an aPR (95% CI) of 1.04 (0.99–1.10) among unemployed or low anger participants. Corresponding results for employed or high anger participants were 0.99 (0.94–1.03) and 0.97 (0.91–1.03), respectively.

Conclusion: Overall, we observed no strong evidence for an association between social support and CVH. However, some psychosocial risks may be modifiers. Prospective studies are needed to assess the social support-CVH relationship by psychosocial risks in racially/ethnically diverse populations.

1. Introduction

Despite the significant declines in cardiovascular disease (CVD) mortality in the United States (U.S.), CVD remains one of the leading causes of death (Virani et al., 2020). Age-adjusted prevalence and mortality rates for CVD differ by race and ethnicity in the U.S., where

African American adults and some Asian adult subgroups (e.g., South Asians) have a higher CVD mortality than White non-Hispanic adults (CDC. Health United States Spotlight, 2019; Jose et al., 2014; Ritchey et al., 2018; Virani et al., 2020). Among Hispanics, the overall CVD prevalence and mortality rates are lower than non-Hispanic adults; however, Hispanic adults are more likely to be hospitalized due to CVD and are younger at the age of death relative to other groups (Balfour

* Corresponding author. Center for Health Promotion and Health Equity Research, Department of Behavioral and Social Sciences, Brown University School of Public Health, Box G-S121-8, Providence, RI, 02912, USA.

E-mail address: akilah.dulin.keita@brown.edu (A.J. Dulin).

<https://doi.org/10.1016/j.ssmph.2022.101284>

Received 9 May 2022; Received in revised form 3 November 2022; Accepted 3 November 2022

Available online 4 November 2022

2352-8273/© 2022 Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

List of acronyms

AHA	American Heart Association
aPR	adjusted prevalence ratio
BMI	body mass index
CI	confidence interval
CVD	cardiovascular disease
CVH	Cardiovascular health
IRB	Institutional Review Board
JHS	Jackson Heart Study
LS7	Life's Simple 7
MASALA	Mediators of Atherosclerosis in South Asians Living in America
MESA	Multi-Ethnic Study of Atherosclerosis
PR	prevalence ratio; U.S. = United States

et al., 2016; Vivo et al., 2009). Thus, reducing CVD incidence/mortality and the related racial/ethnic disparities are a significant public health concern.

Preventing CVD and reducing racial/ethnic disparities are a national priority of the U.S. Department of Health and Human Services through the continued emphasis on increasing overall cardiovascular health (CVH) and promoting healthy behaviors to reduce CVD risk (HP. Washington, 2020HP. Washington). Further, the American Heart Association (AHA) has continued to highlight a national goal to reduce CVD mortality and improve CVH in all Americans by 2030 (HP. Washington, 2020HP. Washington; Lloyd-Jones et al., 2010). The burden of CVD has been attributable to several behavioral and health risk factors that are on mediating pathways to CVD (Global Burden of Cardiovascular Diseases et al., 2018). Using these factors, the AHA proposed Life's Simple 7 (LS7) as a composite measure to evaluate CVH (Lloyd-Jones et al., 2010). The LS7 scores bio-behavioral metrics including smoking, physical activity, diet, body mass index (BMI), blood pressure, total cholesterol, and fasting glucose (Lloyd-Jones et al., 2010), with specific cut-points to indicate poor, intermediate, or ideal CVH.

In addition to behavioral factors, psychosocial factors, such as resilience resources, may potentially improve CVH. Resilience is the ability of individuals to overcome adversities and positively adapt to reduce the negative effects of adversities on physical health (Luthar et al., 2000; Masten & Obradovic, 2006). The Reserve Capacity Model posits that resilience resources, i.e., the reserve capacity an individual may possess, can attenuate the harmful effects of adversities, such as stressors, on adverse CVH outcomes through indirect behavioral pathways (Park et al., 2021). Resilience may have positive effects on physical health through social (e.g., stress buffering), psychological (e.g., appraisals, which influence an individual's subjective evaluation of an event and lead to a reaction), and behavioral (e.g., health-promoting behaviors) mechanisms (Cohen, 1988; Uchino, 2006). Moreover, resilience is a dynamic process where individuals may draw upon resources from multiple levels, such as the individual, interpersonal, or neighborhood-level (Dulin et al., 2018).

Social support is an example of an interpersonal-level resilience resource that may be associated with lower adverse cardiovascular outcomes (Barth et al., 2010; Freak-Poli et al., 2021; Ikeda et al., 2008; Park et al., 2021; Uchino, 2006; Uchino et al., 2018). A potential mechanism through which social support may buffer the harmful effects of stressors on CVH (Cohen et al., 2001; Kamarck et al., 1990; Uchino & Garvey, 1997) and decrease CVD morbidity and mortality (Uchino et al., 2011) is by providing informational, instrumental (i.e., tangible goods/services/aid), or emotional (i.e., caring, empathy, trust) resources that may promote positive adaptive responses to acute or chronic stressors (e.g., psychosocial risks). Thus, social support may be a potential intervention target to improve CVH.

While prior research is informative, there are some limitations. Of the studies on the relationships between social support and LS7, most examine individual metrics of LS7, such as obesity or blood pressure (Johnson et al., 2014; Resnick et al., 2002; Walker et al., 2006; Yang et al., 2015, 2016). Few studies examine the combined LS7 metrics, which is the preferred method of assessing CVH compared to the individual metrics, because the composite LS7 score can substantially increase (or reduce) the risk for CVD and captures the cumulative impact of behavioral and health risk factors on CVD risk comprehensively (Fretz et al., 2018). Additionally, psychosocial risks, defined as psychological or social factors that negatively impact physical health (Martikainen et al., 2002), may be experienced disproportionately in different racial/ethnic groups. It is expected that resilience resources may be most beneficial in reducing or preventing adverse CVH outcomes in the presence of adversities (e.g., psychosocial risks). Thus, the levels of psychosocial risk experienced may modify the relationship between social support and CVH. Past research indicates that psychosocial factors are important to address to reduce or prevent CVD risk or improve CVH (Everson-Rose & Lewis, 2005; Okereke & Manson, 2017). However, most social support literature in the context of CVH does not examine psychosocial risks as potential effect modifiers. Further, there is a lack of longitudinal studies on social support and LS7.

Therefore, this study examines whether social support is associated longitudinally with CVH measured by LS7. We also examine if multilevel psychosocial risks (i.e., individual, interpersonal, and neighborhood-level risks, such as anger, discrimination, and neighborhood deprivation) modify the aforementioned relationship, one at a time, as effect modifiers.

2. Methods

2.1. Study population

We used harmonized data of prospective observational studies of adults from three CVD cohorts in the U.S., including the Jackson Heart Study (JHS), the Mediators of Atherosclerosis in South Asians Living in America (MASALA) study, and the Multi-Ethnic Study of Atherosclerosis (MESA). Briefly, these studies: 1) enrolled adults ages 21–95 years (JHS), 45–84 years (MESA), or 40–79 years (MASALA) at the start of the study, 2) focused on CVD, 3) included information on biological, behavioral, and psychosocial factors, (including measures on risk and resilience), and 4) measured LS7 metrics. JHS enrolled 5306 African American participants residing in the Jackson, Mississippi area, and the first exam was conducted during 2000–2004 with follow-up exams held every 5 years (Taylor et al., 2005). MASALA enrolled 906 South Asian adults without a history of CVD from the San Francisco Bay and Chicago areas in the first exam during 2010–2013, and follow-ups from 2015 to 2018. (Kanaya et al., 2019). MESA enrolled 6814 White, African American, Asian (mainly Chinese American), and Hispanic participants without a history of CVD from 6 different sites (New York (NY), Baltimore (MD), Chicago (IL), Los Angeles (CA), Minneapolis-St. Paul (MN), and Winston-Salem (NC)) and the first exam occurred during 2000–2002. Follow-up exams for the first five exams spanned 10 years (Bild et al., 2002). Data from JHS Exams 1–3, MASALA Exams 1–2, and MESA Exams 1–5 were harmonized. Data harmonization involved examining and applying methods to combine measures/constructs across cohorts via direct (e.g., age and sex/gender) and indirect (e.g., social support using item-to-item matching) harmonization (Pluijm et al., 2005). Harmonized variables were examined using psychometric analyses (e.g., confirmatory factor analysis) (Bentler, 1990) to determine their performance.

Each cohort study protocol was approved by the Institutional Review Board (IRB) at the participating institutions of the three cohorts (JHS, MASALA, and MESA) and all study participants provided written informed consent. The secondary analysis of the data analyzed in this paper was approved by the IRB at the university of the first author.

2.2. Measures

The exposure variable was time-fixed social support measured at Exam 1. Social support was assessed using the Interpersonal Social Support Evaluation List in JHS (Payne et al., 2012) and Social Support Inventory in MESA and MASALA (Mitchell et al., 2003). Since different social support scales were used, a subset of similar items was identified and harmonized indirectly (via item-to-item matching) across the cohorts. Specifically, topics of ‘someone to talk to,’ ‘someone to give advice,’ ‘someone to be there emotionally,’ and ‘someone to help with chores’ were summed, and the means of the sum were standardized onto a 0–1 scale. The median harmonized social support scores were similar across the three cohorts: 0.83 in JHS, 0.81 in MASALA, and 0.75 in MESA. The harmonized social support scale demonstrated acceptable internal consistency (Cronbach’s alpha = 0.79) during our psychometric analysis of the harmonized measures. The harmonized social support variable was classified into tertiles in the analysis.

The outcome variable was an indicator for ideal or intermediate versus poor CVH using the LS7 metrics. We combined the ideal and intermediate CVH categories due to small sample size across the three cohorts (i.e., 6.6% (n = 512)) in the ideal CVH category. The complete set of LS7 metrics from self/proxy-report and/or physical examinations were collected during JHS Exam 1, MASALA Exams 1–2, and MESA Exams 1 and 5. A score of 0, 1, or 2 was assigned to poor, intermediate, or ideal levels of each LS7 metric, respectively, and combined scores between 0 and 7, 8–11, or 12–14 were used to categorize poor, intermediate, or ideal CVH, respectively (Hernandez et al., 2015). A detailed description of the scoring method for each LS7 metric has been noted elsewhere (Brewer et al., 2018; Lloyd-Jones et al., 2010; Ogunmoroti et al., 2017).

Potential confounding variables included age (continuous), sex/gender (female/male), race or ethnicity (African American/White non-Hispanic/Asian/Hispanic), geographical region (West/South/Midwest/Northeast), nativity (U.S.-born/other), marital status (married/never married, separated, divorced, widowed), self-rated health (good/not good), health insurance type (public or private/none), participant history of CVD (yes/no), and family history of CVD (yes/no). All confounding variables were time-fixed, self-reported at Exam 1 and, were considered potential sources of selection bias due to study exclusions and censoring due to dropout.

Potential effect modifiers included the following psychosocial risks: anger, depressive symptoms, chronic stress, education, employment, income, discrimination, neighborhood deprivation, and neighborhood safety. These risks were included because they are established risk factors for CVD, and may modify the relationship between social support and CVH (Boehm et al., 2018; Everson-Rose & Lewis, 2005; Lewis et al., 2014; Rozanski et al., 1999). Anger was harmonized using the Spielberger State-Trait Anger Expression Inventory, specifically the State-Trait for MASALA/MESA, which measured anger as a personality trait, and Anger-Out for JHS, which measured anger as a dynamic expression. Although these are different components of the measure, both anger scales were associated with CVD and were harmonized and categorized into tertiles (Spielberger et al., 1983). Depressive symptoms were measured using the Center for Epidemiologic Studies-Depression (CES-D) scale, and a score of 16 was used to determine a binary depressive symptoms variable (Radloff, 1977). Perceived chronic stress was measured by summing items related to stress from medical problems, job, finances, and relationships from the Global Perceived Stress Scale in JHS and Chronic Burden scale in MESA/MASALA, and categorized as tertiles (Troxel et al., 2003). Variables for educational attainment (less than high school, high school or some college, college degree or higher) and family income (adjusted to the U.S. dollar in the year 2000 to account for inflation during different study periods; \$0–\$19,999, \$20,000–\$49,999, \$50,000 or more) were considered as three-level categorical variables. Employment status was a binary variable comparing employed at least part-time and unemployed. Perceived

discrimination was assessed using the Everyday Discrimination Scale (Williams et al., 1997) and treated as tertiles. Tertiles of neighborhood deprivation was assessed by using the principal component factor analysis scores of neighborhood-level (or census-tract level) SEP measures, such as education, income, and employment; higher scores indicated lower neighborhood deprivation (Diez Roux et al., 2001). Neighborhood safety was a binary variable created from a 1-item neighborhood safety from crime question (safe/not safe). All psychosocial risk measures were self-reported at Exam 1 and were also considered as potential confounding variables and sources of selection bias due to study exclusion and censoring due to dropout; as such, they were included in all outcome models to adjust for confounding and selection bias.

2.3. Statistical analysis

Harmonized data from JHS Exam 1, MASALA Exams 1–2, and MESA Exams 1 and 5 were analyzed. Cohort exams that did not measure the outcome (i.e., complete LS7) by study design were excluded from the analysis. Among JHS, MASALA, and MESA participants (N = 13,284), participants were excluded if they had no social support assessment, no measures for potential confounders, sources of selection bias, or effect modifiers, or no CVH outcome during the relevant follow-up period.

We created a new variable, ‘visit,’ to reflect equal time intervals (6-years) of follow-up time because follow-up times in years were different between exams within and across cohorts. So, we created two visits for our analysis. If a participant had more than one observation (i.e., exams with outcome assessment) within the same visit, we took the furthest observation as our visit; thus, there was a maximum of one observation per visit per participant. However, outcome assessments that were concurrent with the exposure assessment were still included in the first visit. Participants were censored at the second visit due to missing outcome assessment or death during follow-up. We considered death during follow-up as a censoring event due to the small proportion of death (7.3%) and not as an event that was undefined (Chiba & VanderWeele, 2011; Vanderweele, 2011).

Descriptive statistics (chi-square and Wilcoxon Mann-Whitney tests) were used to compare characteristics between included participants and those who were excluded at Exam 1. We used a repeated-measures, modified Poisson regression model fit using generalized estimating equations to estimate prevalence ratios (PRs) for the overall longitudinal relationship between social support assessed at Exam 1 and the binary CVH outcome assessed during the visits. This model allowed for robust variances and accounted for observations clustered within neighborhoods (i.e., census tracts at Exam 1). An independent working correlation structure was chosen in all outcome models (Zou & Donner, 2013). The unadjusted regression model included only the time-fixed social support and visit variable. Our adjusted model controlled for all measured potential sources of confounding and selection bias. The continuous age variable was included in the model using restricted quadratic splines at the 5th, 35th, 65th, and 95th percentiles (Howe et al., 2011). We included social support-visit product terms in both the unadjusted and adjusted outcome models to assess the relationship by visit. We also fit unadjusted and adjusted outcome models without the social support-visit product terms.

To assess for effect measure modification by the level of psychosocial risks, one at a time, we included product terms between social support, visit, and the psychosocial risk as relevant in our adjusted model. P-values from global chi-squared tests indicated whether at least one of the product term coefficients was different from zero.

To determine if inferences changed, for sensitivity analyses, we refit all unadjusted and adjusted models while solely accounting for observations correlated within individuals. We also performed analyses using an exchangeable working correlation structure. Last, we conducted cohort-specific analyses.

In accordance with the recent literature on significance and

hypothesis testing (Amrhein et al., 2019; Greenland et al., 2016; Wasserstein et al., 2019), evidence for an overall association or effect measure modification was interpreted in terms of compatibility with the data (i.e., PRs, 95% confidence intervals (CIs), and p-values), and not whether the 95% CIs excluded the null or p-values were <0.05. Point estimates (i.e., PRs) are the most compatible, and values that lie further away from the point estimate are less compatible with the data. All statistical analyses were performed using SAS 9.4 (SAS Institute, Inc., Cary, North Carolina).

3. Results

We included 7724 participants in our analysis sample (Fig. 1). Out of the 7724 participants included, 564 participants died during follow-up (7.3%) and 4331 were censored administratively (56.1%). Further, 2829 were censored for other reasons (e.g., missing outcome assessment) (36.6%), which included all JHS participants since they did not have CVH outcomes assessed after visit 1. Table 1 compares the characteristics of included participants to those excluded. High social support levels were reported among 32.1% of the included participants compared to 42.3% of the excluded. The included participants' median (25th – 75th percentile) age was 59 years (; ; 51-68) compared to 53 years (; ; 44-63) among the excluded participants. Most included

participants were female (54.7%), African American (40.1%), born in the U.S. (72.2%), married (63.0%), reported good self-rated health (88.0%), and had private or public health insurance (91.0%). Most included participants reported a family history of CVD or stroke (56.3%), and no self-reported history of CVD or stroke (98.6%). Also, most included participants had a high school or higher education, were employed full- or part-time, had an annual income greater than \$50,000, were not depressed, and reported safe neighborhoods.

Table 2 shows the adjusted prevalence ratios (aPR) for ideal or intermediate CVH compared to poor CVH by social support levels among the included participants. Among participants who had high (versus low) social support, the aPR (95% CI) for ideal or intermediate CVH was 0.99 (0.96–1.03). Among those with medium (versus low) social support, the aPR (95% CI) was 1.01 (0.98–1.05). When social support and visit product terms were added to the outcome models, the findings by visit did not change meaningfully compared to findings without the product terms. Therefore, subsequent text refers to the results from outcome models without the social support-visit product terms.

Table 3 shows the assessment for effect measure modifications of the aPRs by levels of psychosocial risks. The 95% CIs overlapped to some extent; however, some psychosocial risks among participants who reported medium (versus low) social support suggested some evidence for effect measure modification. Specifically, for participants who reported

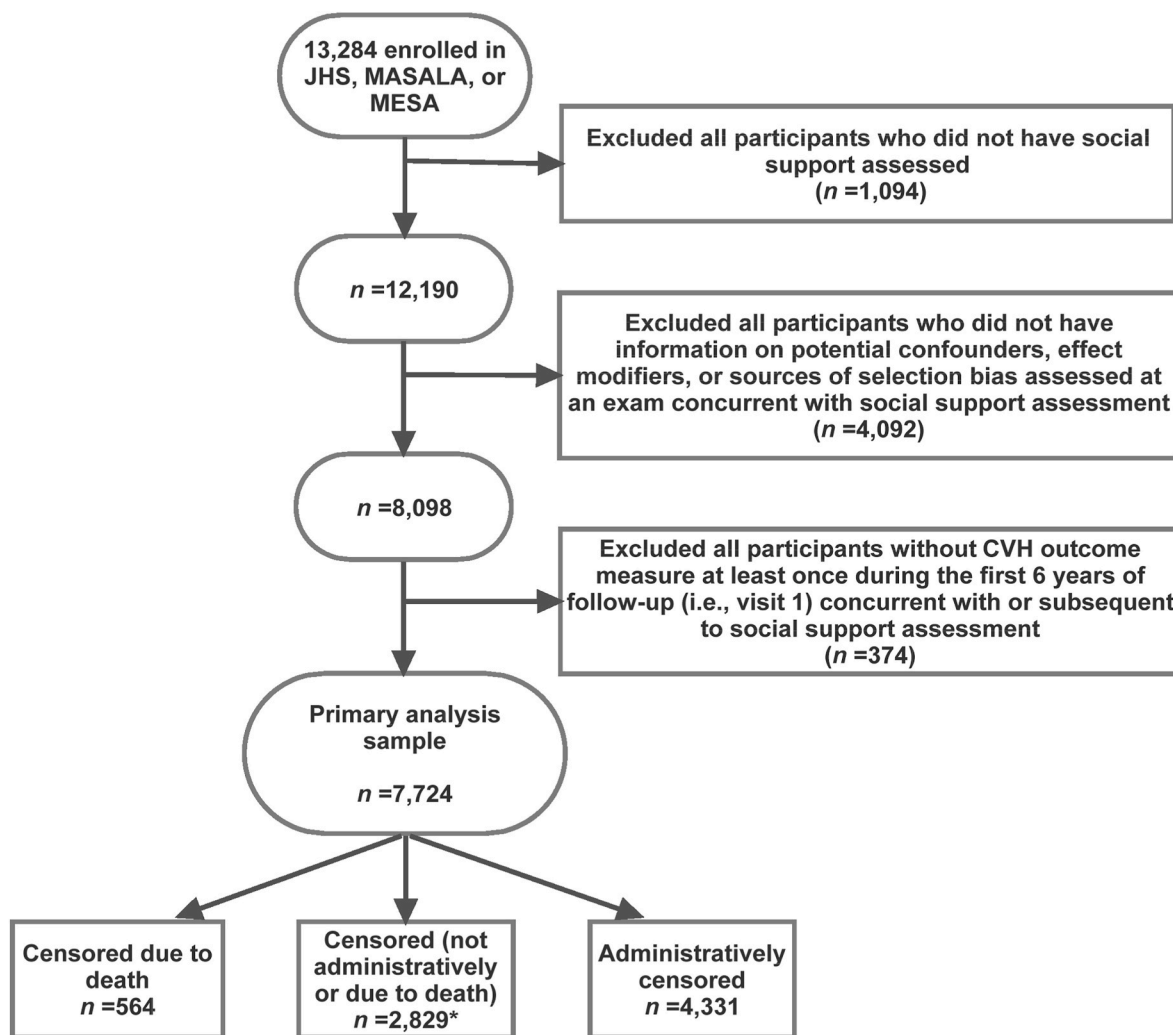


Fig. 1. Flowchart showing the exclusion criteria to identify the 7724 JHS, MASALA, and MESA participants who were included in the primary analysis. Abbreviation: CVH = cardiovascular health, JHS = Jackson Heart Study, MASALA = Mediators of Atherosclerosis Among South Asians Living in America, MESA = Multi-Ethnic Study of Atherosclerosis.

* Includes all JHS participants who were censored because they did not have CVH outcome measure at visit 2.

Table 1
 Characteristics of measures at JHS, MASALA, and MESA Exam 1 comparing the included and excluded participants (i.e., participants with no CVH outcome measured at Exam 1).

Characteristics at Exam 1	Included (n = 7724)		Excluded (n = 374)		P-value ^a
	N	%	N	%	
Social support^b					
Low	2732	35.4	112	30.0	<0.01
Medium	2516	32.6	104	27.8	
High	2476	32.1	158	42.3	
Age^c in years	59 (; ; 51-68)		53 (; ; 44-63)		<0.01
Sex/Gender					
Female	4228	54.7	230	61.5	0.01
Male	3496	45.3	144	38.5	
Race or ethnicity					
White non-Hispanic	2288	29.6	0	0	<0.01
Asian	1081	14.0	15	4.0	
African American	3098	40.1	359	96.0	
Hispanic	1257	16.3	0	0	
Nativity					
Other	2145	28.5	15	2.5	<0.01
U.S.-born	5579	72.2	359	96.0	
Region					
West	1126	14.6	0	0	<0.01
South	2508	32.5	359	96.0	
Midwest	2325	30.1	15	4.0	
Northeast	1765	22.9	0	0	
Marital Status					
Never married, separated/divorced, widowed	2862	37.1	163	43.6	0.01
Married	4862	63.0	211	56.4	
Self-rated health^d					
Not good	925	12.0	120	32.1	<0.01
Good	6799	88.0	254	67.9	
Health Insurance					
None	694	9.0	69	18.5	<0.01
Public or Private	7030	91.0	305	81.6	
Self-history of CVD and Stroke					
No	7616	98.6	343	91.7	<0.01
Yes	108	1.4	31	8.3	
Family history of CVD and Stroke					
No	3372	43.7	165	44.1	0.86
Yes	4352	56.3	209	55.9	
Education					
College degree or more	3173	41.1	146	39.0	0.73
High school or some college	3452	44.7	174	46.5	
Less than high school	1099	14.2	54	14.4	
Employment					
Employed (Part/full-time)	4117	53.3	206	55.1	0.50
Unemployed	3607	46.7	168	44.9	
Income					
\$50,000+	3288	42.6	135	36.1	<0.01
\$20,000-\$49,999	2804	36.3	117	31.3	
\$0-\$19,999	1632	21.1	122	32.6	
Anger^b					
Low	2915	37.7	94	25.1	<0.01
Medium	2507	32.5	70	18.7	
High	2302	29.8	210	56.2	
Depressive symptoms					
No	6660	86.2	273	73.0	<0.01
Yes	1064	13.8	101	27.0	
Chronic stress^b					
Low	3340	43.2	65	17.4	<0.01
Medium	2600	33.7	125	33.4	
High	1784	23.1	184	49.2	
Discrimination^b					
Low	2800	36.3	85	22.7	<0.01
Medium	2580	33.4	101	27.0	
High	2344	30.4	188	50.3	
Neighborhood deprivation^b					
Low	2280	29.5	193	51.6	<0.01
Medium	2676	34.7	106	28.3	
High	2768	35.8	75	20.1	
Neighborhood safety					
Safe	6186	80.1	213	57.0	<0.01
Not safe	1538	19.9	161	43.1	

Abbreviations: CVD, cardiovascular disease; CVH, cardiovascular health; JHS, Jackson Heart Study; MASALA, Mediators of Atherosclerosis in South Asians Living in America; MESA, Multi-Ethnic Study of Atherosclerosis.

^a Pearson's χ^2 -test and Wilcoxon-Mann-Whitney test.

^b Tertiles are not 33% due to ties at boundaries and no participants with the same value being included in different tertiles.

^c Median (25th percentile-75th percentile).

^d Binary variable for self-rated health was used to indicate 'Good' and 'Not good' categories because different self-rated health measures across JHS, MESA, and MASALA studies were harmonized.

medium (versus low) social support, the aPR (95% CI) for ideal or intermediate (versus poor) CVH among those who were unemployed was 1.04 (0.99–1.10) compared to 0.99 (0.94–1.03) for those employed at least part-time. Also, a positive association among those who reported low anger was most compatible with the data (aPR: 1.04, 95% CI: 0.99–1.10), while those with high and medium anger showed aPR (95% CI) of 0.97 (0.91–1.03) and 1.01 (0.96–1.07), respectively. Based on the most compatible estimates, other psychosocial risks did not find meaningful support for effect measure modification. Findings by visit were similar (See [Supplemental Table 1](#) for effect measure modification results by visit).

In sensitivity analyses, our findings did not differ meaningfully from the main analyses when we repeated all of the analyses only accounting for the within-individual correlation or when using the exchangeable working correlation structure.

[Supplemental Table 2](#) shows the results for the cohort-specific analyses. Since only JHS Exam 1 data were included in the study, there were no estimates shown by visit for JHS. The aPRs for MASALA and MESA were similar for both high and medium social support suggesting either no association or a small positive association with ideal or intermediate CVH, while the findings for JHS suggested a small negative association with ideal or intermediate CVH.

4. Discussion

This study examined the longitudinal relationship between social support and CVH measured using LS7 metrics. We also assessed whether this relationship was modified by high, medium, and low levels of psychosocial risks. We did not find evidence to support an association between greater social support and better CVH. The assessment for effect measure modification suggested that some psychosocial risks, such as employment status and perceived anger, may modify the relationship between social support and CVH. Specifically, participants who reported that they were unemployed or had lower anger levels may have better CVH outcomes from having a medium level of social support. Further, by cohort, MASALA and MESA findings suggested a null or weak positive association. For JHS, results indicated a weak negative association; however, the 95% CIs included the null, and both a negative and positive association for social support and ideal or intermediate (versus poor) CVH were compatible with the data.

Although our study findings generally suggested no strong association between social support and CVH, evidence from prior studies has been mixed. In one cross-sectional study using the 2007-08 National Health and Nutrition Examination Survey (NHANES), lack of social support was associated with poor LS7 metrics ([Kieu et al., 2020](#)), while a review study reported that individuals with better social relationships were more likely to achieve or maintain ideal CVH ([Cabeza de Baca et al., 2018](#)). Other studies have also shown that low social support was associated with a greater risk of adverse CVD outcomes ([Angerer et al., 2000](#); [Barth et al., 2010](#); [Berkman et al., 1992](#); [Blazer, 1982](#); [Freak-Poli et al., 2021](#); [Ikeda et al., 2008](#); [Rozanski et al., 1999](#); [Uchino et al., 2018](#)). Conversely, in one study using MESA data, higher social support was not associated with incident CVD, and cross-sectional studies among Black and Hispanic/Latino adults showed that higher levels of social support were not associated with individual LS7 metrics (e.g., BMI,

Table 2
Prevalence ratios (PR) for ideal or intermediate (versus poor) CVH by social support levels among the included JHS, MASALA, and MESA participants (n = 7724).

Outcome	Social support-visit product terms in outcome model		High versus low social support PR (95% CI)		Medium versus low social support PR (95% CI)	
			Unadjusted	Adjusted ^b	Unadjusted	Adjusted ^b
			Ideal or intermediate (versus poor) CVH	No social support-visit product terms	1.01 (0.97–1.05)	0.99 (0.96–1.03)
	Social support-visit product terms are present	Visit 1 ^a	1.00 (0.95–1.04)	0.99 (0.96–1.03)	1.02 (0.98–1.06)	1.00 (0.96–1.04)
		Visit 2 ^a	1.03 (0.98–1.10)	0.99 (0.94–1.05)	1.05 (1.00–1.11)	1.03 (0.98–1.08)

Note: Each repeated-measures modified Poisson regression model accounted for within neighborhood clustering (i.e., census tracts) at Exam 1 (Zou & Donner, 2013). Clustering outcomes within individuals did not change inference.

^a Coefficients for the social support-visit product terms in the unadjusted model: 0.03, 0.04, p = 0.41; adjusted model: 0.03, 0.001, p = 0.60.

^b Adjusted for visit, age, sex/gender, race, nativity, geographic region, marital status, self-rated health, insurance, self-history of CVD and stroke, family history of CVD and stroke, education, income, employment, anger, depressive symptoms, chronic stress, discrimination, neighborhood deprivation, and neighborhood safety.

Table 3
Assessment for the effect measure modification of the adjusted prevalence ratios^a (aPR) for ideal or intermediate (versus poor) CVH by psychosocial risk levels and social support among the included JHS, MASALA, and MESA participants (n = 7724).

Psychosocial risk measures at Exam 1 (Potential effect measure modifiers)	High versus low social support and ideal or intermediate CVH		Medium versus low social support and ideal or intermediate CVH		p ^b
	aPR	95% CI	aPR	95% CI	
Education					
College degree of higher	1.00	(0.96–1.04)	1.00	(0.96–1.05)	0.74
High school or some college	1.00	(0.94–1.06)	1.01	(0.95–1.06)	
Less than high school	0.96	(0.86–1.08)	1.06	(0.95–1.18)	
Employment					
Employed	0.99	(0.95–1.04)	0.99	(0.94–1.03)	0.19
Unemployed	1.00	(0.94–1.05)	1.04	(0.99–1.10)	
Income					
\$50,000+	1.01	(0.96–1.06)	1.01	(0.96–1.06)	0.72
\$20,000–\$49,999	0.98	(0.92–1.05)	1.00	(0.95–1.06)	
\$0–\$19,999	0.97	(0.89–1.07)	1.04	(0.97–1.12)	
Anger					
Low	0.99	(0.94–1.05)	1.04	(0.99–1.10)	0.20
Medium	1.02	(0.97–1.08)	1.01	(0.96–1.07)	
High	0.97	(0.91–1.04)	0.97	(0.91–1.03)	
Depressive symptoms					
No	0.99	(0.95–1.03)	1.01	(0.97–1.05)	0.84
Yes	1.03	(0.90–1.17)	1.00	(0.90–1.11)	
Chronic stress					
Low	1.01	(0.96–1.05)	1.03	(0.98–1.08)	0.79
Medium	0.97	(0.91–1.04)	1.00	(0.94–1.05)	
High	1.00	(0.92–1.09)	0.99	(0.91–1.08)	
Discrimination					
Low	1.00	(0.94–1.05)	1.03	(0.98–1.09)	0.44
Medium	0.99	(0.93–1.05)	1.02	(0.96–1.09)	
High	1.01	(0.94–1.07)	0.97	(0.91–1.03)	
Neighborhood deprivation					
Low	0.95	(0.88–1.04)	1.03	(0.95–1.10)	0.58
Medium	1.01	(0.95–1.08)	1.00	(0.94–1.07)	
High	1.00	(0.96–1.05)	1.01	(0.96–1.06)	
Neighborhood safety					
Safe	0.99	(0.96–1.03)	1.01	(0.97–1.04)	0.85
Not safe	0.99	(0.91–1.09)	1.03	(0.94–1.13)	

Note: Each repeated-measures modified Poisson regression model accounted for within neighborhood clustering (i.e., census tracts) at Exam 1 (Zou & Donner, 2013). Clustering outcomes within individuals did not change inference.

^a Adjusted for visit, age, sex/gender, race, nativity, geographic region, marital status, self-rated health, insurance, self-history of CVD and stroke, family history of CVD and stroke, education, income, employment, anger, depressive symptoms, chronic stress, discrimination, neighborhood deprivation, and neighborhood safety.

^b Global chi-squared p-values.

cholesterol, and blood pressure) (Hernandez et al., 2014, 2018). Evidence from a systematic review of prospective observational studies among participants with CVD suggested a positive association between higher social support and better clinical CVD outcomes (Hemingway & Marmot, 1999); however, conflicting evidence also exists (Lett et al., 2007). Interestingly, studies conducted in Europe have shown, in general, stronger associations between social support and CVD outcomes compared to studies conducted in the U.S (Hemingway & Marmot, 1999). Further, there have been inconsistent findings regarding gender differences in the relationship between social support and CVD outcomes; i.e., some studies suggest stronger associations among men (Hemingway & Marmot, 1999; Hu et al., 2021) while others suggest women may benefit more from social support (Leifheit-Limson et al., 2010; Low et al., 2010). Thus, findings for the relationship between social support and CVH and incident CVD are mixed and may contrast with theories and conceptual frameworks suggesting favorable health benefits of increased social support on CVH outcomes (Cohen & Wills, 1985; Uchino, 2006).

For our effect measure modification assessments, the point estimates and 95% CIs overlapped considerably for most psychosocial risks, suggesting that they did not contribute to meaningful differences in the social support-CVH relationship. However, there was some evidence of effect measure modification by employment status and anger. Specifically, our findings showed that medium social support may be beneficial to achieve ideal or intermediate CVH among individuals who are unemployed or have low levels of anger. The observed differences in the relationship between social support and CVH by certain psychosocial risks may be plausible. Since resilience is a complex and dynamic process operating at multiple levels (Masten, 2014), there may be trade-offs to resilience because of the access to limited resources (Ungar, 2018). For example, our findings showed potential resilience in the presence of some psychosocial risks, but not others, which may suggest that to cope with some adversities, such as individual-level risks, resilience may be facilitated by individuals accessing resources in the form of social support. However, the experience of other adversities, such as neighborhood-level risks, may be too great for resilience to offset negative risks in order to achieve a balanced and optimal CVH outcome (Ungar, 2018). Nevertheless, resilience resources may be protective and potentially more malleable targets than psychosocial risks. The disagreement between prior study findings, including the current study, on the overall relationship between social support and CVH may be due to the different distributions of the psychosocial risks experienced. Thus, additional exploration of resilience resources as interventions to improve CVH in the presence of psychosocial risks among diverse populations are warranted.

4.1. Limitations and strengths

Although informative, our study is not without limitations. First, the

measure for social support was harmonized from different social support measures from three cohorts. We performed psychometric tests to explore how well the harmonized measure performed, and social support showed acceptable internal consistency (Cronbach's alpha = 0.79). However, since our harmonized measure differs from other social support measures, our findings should be interpreted with caution. Also, there is no gold standard measure of social support (Beckers et al., 2020; Hogan et al., 2002); hence, assessing the relationship between social support and CVH outcomes, as well as comparing findings across studies can be challenging. Further, social support was measured at Exam 1 during 2010–2013 in MASALA and during early 2000's in JHS and MESA. Given these different time frames and the study measures available in the cohorts, we could not account for potential variations in regional structural factors that may have affected social support levels and cardiovascular risks among participants. However, the findings for the social support and CVH relationship stratified by cohort did not show meaningfully different results. Second, we did not control for other resilience resources in our analysis, such as optimism and neighborhood social cohesion, which may confound the relationship between social support and CVH because they were assessed after the exposure. Third, there may be residual confounding bias, selection bias, or measurement error due to most measures being self-reported. Also, model misspecification in the outcome models may be an issue despite using restricted quadratic splines (Hernan & Robins, 2020; Howe et al., 2011) for the continuous age variable. Further, our approach does not account for additional correlation in the outcome that may occur because a participant moved to a different neighborhood (i.e., census tract) after Exam 1. However, 86.1% (6652/7724) of the included participants resided in the same census tract after Exam 1 (i.e., at Exam 2). Fourth, the three cohorts were not representative of the U.S. population. Although we used a harmonized data of the three cohorts, our findings may not be generalizable to other populations with varying demographic and psychosocial characteristics. Lastly, we may have had inadequate power to assess for effect measure modification.

Despite these limitations, our study has several strengths. We utilized a harmonized data set of three prospective observational cohort studies with a focus on CVD. Harmonization provided the opportunity for increased sample size and power to explore the relationship between social support and CVH in a racially/ethnically and geographically diverse population. Moreover, we used repeated-measures LS7 metrics for our CVH outcome to assess the longitudinal relationship between social support and CVH. Lastly, using modified Poisson regression models fit with generalized estimating equations to estimate PRs allowed us to obtain robust variances while accounting for clustered data (Zou & Donner, 2013).

5. Conclusion

In summary, our longitudinal study examining the relationship between social support and CVH suggested that there may be some effect measure modification by employment status and anger. Specifically, individuals who are unemployed or have lower anger levels may benefit more, in terms of their CVH outcomes, from having a medium level of social support. However, there was no strong evidence in the overall relationship between social support and CVH. If employment status and anger do in fact serve as effect modifiers, then the observed overall relationship may be different in a population with a different distribution of these effect modifiers. Thus, additional studies are needed. Moreover, future studies should consider changes in social support levels over time by collecting and analyzing data on social support at multiple time points. Additionally, future studies should account for other social constructs, such as social isolation and loneliness. Also, studies should include more frequently measured composite LS7 metrics during longer follow-up periods to prospectively assess ideal, intermediate, or poor CVH outcomes, and account for other resilience measures, such as optimism and neighborhood social cohesion.

Source of funding

Research reported in this publication was supported by the National Heart, Lung, and Blood Institute of the National Institutes of Health under Award Number R01HL135200. One hundred percent of the total project costs (\$1,489,225) are financed with Federal money. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

The Jackson Heart Study (JHS) is supported and conducted in collaboration with Jackson State University (HHSN268201800013I), Tougaloo College (HHSN268201800014I), the Mississippi State Department of Health (HHSN268201800015I) and the University of Mississippi Medical Center (HHSN268201800010I, HHSN268201800011I and HHSN268201800012I) contracts from the National Heart, Lung, and Blood Institute (NHLBI) and the National Institute on Minority Health and Health Disparities (NIMHD).

The Multi-Ethnic Study of Atherosclerosis (MESA) study was supported by contracts 75N92020D00001, HHSN268201500003I, N01-HC-95159, 75N92020D00005, N01-HC-95160, 75N92020D00002, N01-HC-95161, 75N92020D00003, N01-HC-95162, 75N92020D00006, N01-HC-95163, 75N92020D00004, N01-HC-95164, 75N92020D00007, N01-HC-95165, N01-HC-95166, N01-HC-95167, N01-HC-95168 and N01-HC-95169 from the National Heart, Lung, and Blood Institute, and by grants UL1-TR-000040, UL1-TR-001079, and UL1-TR-001420 from the National Center for Advancing Translational Sciences (NCATS). A full list of participating MESA investigators and institutions can be found at <http://www.mesa-nhlbi.org>.

The Mediators of Atherosclerosis in South Asians Living in America (MASALA) was supported by Grant Number R01HL093009 from the National Heart, Lung, and Blood Institute and the National Center for Research Resources and the National Center for Advancing Translational Sciences, National Institutes of Health, through UCSF-CTSI Grant Number UL1RR024131.

The authors also wish to thank the other investigators, the staff, and the participants of the JHS, MESA, and MASALA study for their valuable contributions.

Disclaimer

The views expressed in this manuscript are those of the authors and do not necessarily represent the views of the National Heart, Lung, and Blood Institute; the National Institutes of Health; or the U.S. Department of Health and Human Services.

Data statement

The data that support the findings originate from three separate third party sources. Restrictions apply to the availability of the data, which were used under license for this study. Data from the Jackson Heart Study is available with a request to access at <https://www.jacksonheartstudy.org/Research/Study-Data/Data-Access>. Data from the Multi-Ethnic Study of Atherosclerosis is available with a request to access at <https://www.mesa-nhlbi.org/Publications.aspx>. Data from the Mediators of Atherosclerosis among South Asians Living in America Study is available with permission at <https://www.masalastudy.org/for-researchers>. Code to harmonize and process data collected from sites can be found publicly at <https://doi.org/10.26300/bmr7-sd49>.

Author justification

The data on this project comes from three separate sources, so authors from each site played an integral part in acquiring the data for this project. The results presented in this paper are additionally the culmination of the synthesis of these three separate prospective cohorts into one large dataset for analysis. This process required the input of data harmonization experts in order to ensure the data were combined in a

statistically valid way. Without this process the dataset that these results are derived from would not be possible. Additionally, each of the authors listed were involved in critically revising the document to ensure that all aspects of the manuscript were correct.

Author contributions

Jeon Won Park: Writing – original draft, methodology, formal analysis, **Chanelle J. Howe:** conceptualization, methodology, writing – original draft, supervision, funding acquisition, **Laura A Dionne:** Data curation, writing – review and editing, **Matthew M Scarpaci:** Data curation, formal analysis, writing – review and editing, **Belinda L. Needham:** Supervision, investigation, writing – review and editing, **Mario Sims:** Supervision, investigation, writing – review and editing, **Alka Kanaya:** Investigation, writing – review and editing, **Namratha R. Kandula:** Investigation, writing – review and editing, **Joseph L. Fava:** Supervision, writing – review and editing, **Eric B. Loucks:** Conceptualization, writing – review and editing, **Charles B. Eaton:** Supervision, writing – review and editing, **Akilah J. Dulin:** Conceptualization, methodology, supervision, funding acquisition, project administration, writing – review and editing.

Declaration of competing interest

None.

Data availability

The authors do not have permission to share data.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ssmph.2022.101284>.

References

- Amrhein, V., Greenland, S., & McShane, B. B. (2019). Statistical significance gives bias a free pass. *European Journal of Clinical Investigation*, 49(12), Article e13176.
- Angerer, P., Siebert, U., Kothny, W., et al. (2000). Impact of social support, cynical hostility and anger expression on progression of coronary atherosclerosis. *Journal of the American College of Cardiology*, 36(6), 1781–1788.
- Balfour, P. C., Jr., Ruiz, J. M., Talavera, G. A., et al. (2016). Cardiovascular disease in hispanics/latinos in the United States. *J Lat Psychol*, 4(2), 98–113.
- Barth, J., Schneider, S., & von Kanel, R. (2010). Lack of social support in the etiology and the prognosis of coronary heart disease: A systematic review and meta-analysis. *Psychosomatic Medicine*, 72(3), 229–238.
- Beckers, T., Koekkoek, B., Tiemens, B., et al. (2020). Measuring social support in people with mental illness: A quantitative analysis of the social network map. *Issues in Mental Health Nursing*, 41(10), 916–924.
- Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin*, 107(2), 238–246.
- Berkman, L. F., Leo-Summers, L., & Horwitz, R. I. (1992). Emotional support and survival after myocardial infarction. A prospective, population-based study of the elderly. *Annals of Internal Medicine*, 117(12), 1003–1009.
- Bild, D. E., Bluemke, D. A., Burke, G. L., et al. (2002). Multi-ethnic study of atherosclerosis: Objectives and design. *American Journal of Epidemiology*, 156(9), 871–881.
- Blazer, D. G. (1982). Social support and mortality in an elderly community population. *American Journal of Epidemiology*, 115(5), 684–694.
- Boehm, J. K., Chen, Y., Koga, H., et al. (2018). Is optimism associated with healthier cardiovascular-related behavior? Meta-analyses of 3 health behaviors. *Circulation Research*, 122(8), 1119–1134.
- Brewer, L. C., Redmond, N., Slusser, J. P., et al. (2018). Stress and achievement of cardiovascular health metrics: The American heart association life's simple 7 in blacks of the Jackson heart study. *Journal of American Heart Association*, 7(11).
- Cabeza de Baca, T., Durazo, E. M., & Rodriguez, F. (2018). Achieving optimal cardiovascular health: A social epidemiological approach. *Current Epidemiology Reports*, 5(3), 262–271.
- Chiba, Y., & VanderWeele, T. J. (2011). A simple method for principal strata effects when the outcome has been truncated due to death. *American Journal of Epidemiology*, 173(7), 745–751.
- Cohen, S. (1988). Psychosocial models of the role of social support in the etiology of physical disease. *Health Psychology*, 7(3), 269–297.
- Cohen, S., Gottlieb, B. H., & Underwood, L. G. (2001). Social relationships and health: Challenges for measurement and intervention. *Advances in Mind-Body Medicine*, 17(2), 129–141.
- Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, 98(2), 310–357.
- Diez Roux, A. V., Merkin, S. S., Arnett, D., et al. (2001). Neighborhood of residence and incidence of coronary heart disease. *New England Journal of Medicine*, 345(2), 99–106.
- Dulin, A. J., Dale, S. K., Earnshaw, V. A., et al. (2018). Resilience and HIV: A review of the definition and study of resilience. *AIDS Care*, 30(sup5), S6–S17.
- Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health*, 26, 469–500.
- Freak-Poli, R., Ryan, J., Neumann, J. T., et al. (2021). Social isolation, social support and loneliness as predictors of cardiovascular disease incidence and mortality. *BMC Geriatrics*, 21(1), 711.
- Fretz, A., McEvoy, J. W., Rebholz, C. M., et al. (2018). Relation of lifestyle factors and life's simple 7 score to temporal reduction in troponin levels measured by a high-sensitivity assay (from the atherosclerosis risk in communities study). *The American Journal of Cardiology*, 121(4), 430–436.
- Global Burden of Cardiovascular Diseases, C., Roth, G. A., Johnson, C. O., et al. (2018). The burden of cardiovascular diseases among US States, 1990–2016. *JAMA Cardiol*, 3(5), 375–389.
- Greenland, S., Senn, S. J., Rothman, K. J., et al. (2016). Statistical tests, P values, confidence intervals, and power: A guide to misinterpretations. *European Journal of Epidemiology*, 31(4), 337–350.
- Hemingway, H., & Marmot, M. (1999). Evidence based cardiology: Psychosocial factors in the aetiology and prognosis of coronary heart disease. Systematic review of prospective cohort studies. *BMJ*, 318(7196), 1460–1467.
- Hernandez, R., Carnethon, M., Giachello, A. L., et al. (2018). Structural social support and cardiovascular disease risk factors in hispanic/latino adults with diabetes: Results from the hispanic community health study/study of latinos (HCHS/SOL). *Ethnicity and Health*, 23(7), 737–751.
- Hernandez, R., Kershaw, K. N., Siddique, J., et al. (2015). Optimism and cardiovascular health: Multi-ethnic study of atherosclerosis (MESA). *Health Behav Policy Rev*, 2(1), 62–73.
- Hernandez, D. C., Reitzel, L. R., Wetter, D. W., et al. (2014). Social support and cardiovascular risk factors among black adults. *Ethnicity & Disease*, 24(4), 444–450.
- Hernan, M. A., & Robins, J. M. (2020). *Causal inference: What if*. Boca Raton: Chapman & Hall/CRC.
- Hogan, B. E., Linden, W., & Najarian, B. (2002). Social support interventions: Do they work? *Clinical Psychology Review*, 22(3), 383–442.
- Howe, C. J., Cole, S. R., Westreich, D. J., et al. (2011). Splines for trend analysis and continuous confounder control. *Epidemiology*, 22(6), 874–875.
- [Internet] HP. Washington. DC: U.S. Department of health and human services, office of disease prevention and health promotion <https://health.gov/healthypeople>. (Accessed 23 September 2020).
- Hu, J., Fitzgerald, S. M., Owen, A. J., et al. (2021). Social isolation, social support, loneliness and cardiovascular disease risk factors: A cross-sectional study among older adults. *International Journal of Geriatric Psychiatry*, 36(11), 1795–1809.
- Ikeda, A., Iso, H., Kawachi, I., et al. (2008). Social support and stroke and coronary heart disease: The JPHC study cohorts II. *Stroke*, 39(3), 768–775.
- Johnson, E. R., Carson, T. L., Affuso, O., et al. (2014). Relationship between social support and body mass index among overweight and obese African American women in the rural deep South, 2011–2013. *Preventing Chronic Disease*, 11, E224.
- Jose, P. O., Frank, A. T., Kapphahn, K. I., et al. (2014). Cardiovascular disease mortality in Asian Americans. *Journal of the American College of Cardiology*, 64(23), 2486–2494.
- Kamarck, T. W., Manuck, S. B., & Jennings, J. R. (1990). Social support reduces cardiovascular reactivity to psychological challenge: A laboratory model. *Psychosomatic Medicine*, 52(1), 42–58.
- Kanaya, A. M., Chang, A., Schembri, M., et al. (2019). Recruitment and retention of US South Asians for an epidemiologic cohort: Experience from the MASALA study. *J Clin Transl Sci*, 3(2–3), 97–104.
- Kieu, C., Behforouz, S., & Wong, N. D. (2020). Social isolation and cardiovascular health in US adults. *Scripta Medica*, 51(1), 9–14.
- Leifheit-Limson, E. C., Reid, K. J., Kasl, S. V., et al. (2010). The role of social support in health status and depressive symptoms after acute myocardial infarction: Evidence for a stronger relationship among women. *Circ Cardiovasc Qual Outcomes*, 3(2), 143–150.
- Lett, H. S., Blumenthal, J. A., Babyak, M. A., et al. (2007). Social support and prognosis in patients at increased psychosocial risk recovering from myocardial infarction. *Health Psychology*, 26(4), 418–427.
- Lewis, T. T., Williams, D. R., Tamene, M., et al. (2014). Self-reported experiences of discrimination and cardiovascular disease. *Curr Cardiovasc Risk Rep*, 8(1), 365.
- Lloyd-Jones, D. M., Hong, Y., Labarthe, D., et al. (2010). Defining and setting national goals for cardiovascular health promotion and disease reduction: The American heart association's strategic impact goal through 2020 and beyond. *Circulation*, 121(4), 586–613.
- Low, C. A., Thurston, R. C., & Matthews, K. A. (2010). Psychosocial factors in the development of heart disease in women: Current research and future directions. *Psychosomatic Medicine*, 72(9), 842–854.
- Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*, 71(3), 543–562.
- Martikainen, P., Bartley, M., & Lahelma, E. (2002). Psychosocial determinants of health in social epidemiology. *International Journal of Epidemiology*, 31(6), 1091–1093.

- Masten, A. S. (2014). Global perspectives on resilience in children and youth. *Child Development*, 85(1), 6–20.
- Masten, A. S., & Obradovic, J. (2006). Competence and resilience in development. *Annals of the New York Academy of Sciences*, 1094, 13–27.
- Mitchell, P. H., Powell, L., Blumenthal, J., et al. (2003). A short social support measure for patients recovering from myocardial infarction: The ENRICHD social support inventory. *J Cardiopulm Rehabil*, 23(6), 398–403.
- Ogunmoroti, O., Oni, E., Michos, E. D., et al. (2017). Life's simple 7 and incident heart failure: The multi-ethnic study of atherosclerosis. *Journal of American Heart Association*, 6(6).
- Okereke, O. I., & Manson, J. E. (2017). Psychosocial factors and cardiovascular disease risk: An opportunity in women's health. *Circulation Research*, 120(12), 1855–1856.
- Park, J. W., Mealy, R., Saldanha, I. J., et al. (2021). Multilevel resilience resources and cardiovascular disease in the United States: A systematic review and meta-analysis. *Health Psychology*, 41(4), 278–290.
- Payne, T. J., Andrew, M., Butler, K. R., et al. (2012). Psychometric evaluation of the interpersonal support evaluation list—short form in the ARIC study cohort. *Sage Open*, 2(3), Article 2158244012461923.
- Pluijm, S. M., Bardage, C., Nikula, S., et al. (2005). A harmonized measure of activities of daily living was a reliable and valid instrument for comparing disability in older people across countries. *Journal of Clinical Epidemiology*, 58(10), 1015–1023.
- Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1(3), 385–401.
- Resnick, B., Orwig, D., Magaziner, J., et al. (2002). The effect of social support on exercise behavior in older adults. *Clinical Nursing Research*, 11(1), 52–70.
- Ritchey, M. D., Wall, H. K., Owens, P. L., et al. (2018). Vital signs: State-level variation in nonfatal and fatal cardiovascular events targeted for prevention by million hearts 2022. *MMWR Morb Mortal Wkly Rep*, 67, 974–982.
- Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*, 99(16), 2192–2217.
- Spielberger, C., Gorsuch, R., Lushene, R., et al. (1983). *Manual for the state-trait anxiety inventory*. Palo Alto, CA: Consulting Psychologist Press.
- Taylor, H. A., Jr., Wilson, J. G., Jones, D. W., et al. (2005). Toward resolution of cardiovascular health disparities in african Americans: Design and methods of the Jackson heart study. *Ethnicity & Disease*, 15(4 Suppl 6), S6, 4-17.
- Troxel, W. M., Matthews, K. A., Bromberger, J. T., et al. (2003). Chronic stress burden, discrimination, and subclinical carotid artery disease in African American and Caucasian women. *Health Psychology*, 22(3), 300–309.
- Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine*, 29(4), 377–387.
- Uchino, B. N., Carlisle, M., Birmingham, W., et al. (2011). Social support and the reactivity hypothesis: Conceptual issues in examining the efficacy of received support during acute psychological stress. *Biological Psychology*, 86(2), 137–142.
- Uchino, B. N., & Garvey, T. S. (1997). The availability of social support reduces cardiovascular reactivity to acute psychological stress. *Journal of Behavioral Medicine*, 20(1), 15–27.
- Uchino, B. N., Trettevik, R., Kent de Grey, R. G., et al. (2018). Social support, social integration, and inflammatory cytokines: A meta-analysis. *Health Psychology*, 37(5), 462–471.
- Ungar, M. (2018). Systemic resilience: Principles and processes for a science of change in contexts of adversity. *Ecology and Society*, 23.
- CDC. Health, United States Spotlight. (2019). Racial and ethnic disparities in Heart disease (Accessed) https://www.cdc.gov/nchs/spotlight/HeartDiseaseSpotlight_2019_0404.pdf.**
- Vanderweele, T. J. (2011). Principal stratification—uses and limitations. *International Journal of Biostatistics*, 7(1).
- Virani, S. S., Alonso, A., Benjamin, E. J., et al. (2020). Heart disease and stroke statistics—2020 update: A report from the American heart association. *Circulation*, 141(9), E139–E596.
- Vivo, R. P., Krim, S. R., Cevik, C., et al. (2009). Heart failure in hispanics. *Journal of the American College of Cardiology*, 53(14), 1167–1175.
- Walker, S. N., Pullen, C. H., Hertzog, M., et al. (2006). Determinants of older rural women's activity and eating. *Western Journal of Nursing Research*, 28(4), 449–468. ; discussion 469-474.
- Wasserstein, R. L., Schirm, A. L., & Lazar, N. A. (2019). Moving to a world beyond “p < 0.05”. *The American Statistician*, 73(sup1), 1–19.
- Williams, D. R., Yan, Y., Jackson, J. S., et al. (1997). Racial differences in physical and mental health: Socio-economic status, stress and discrimination. *Journal of Health Psychology*, 2(3), 335–351.
- Yang, Y. C., Boen, C., Gerken, K., et al. (2016). Social relationships and physiological determinants of longevity across the human life span. *P Natl Acad Sci USA*, 113(3), 578–583.
- Yang, Y. C., Boen, C., & Harris, K. M. (2015). Social relationships and hypertension in late life: Evidence from a nationally representative longitudinal study of older adults. *Journal of Aging and Health*, 27(3), 403–431.
- Zou, G. Y., & Donner, A. (2013). Extension of the modified Poisson regression model to prospective studies with correlated binary data. *Statistical Methods in Medical Research*, 22(6), 661–670.