

Posttraumatic stress disorder and risk for coronary heart disease: A meta-analytic review

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Objective The aim of this study was to estimate the association of posttraumatic stress disorder (PTSD) with risk for incident coronary heart disease (CHD).

Design A systematic review and meta-analysis were used as study designs.

Data Sources Articles were identified by searching Ovid MEDLINE, PsycINFO, Scopus, Cochrane Library, PILOTS database, and PubMed Related Articles and through a manual search of reference lists (1948-present).

Study Selection All studies that assessed PTSD in participants initially free of CHD and subsequently assessed CHD/cardiac-specific mortality were included.

Data Extraction Two investigators independently extracted estimates of the association of PTSD with CHD, as well as study characteristics. Odds ratios were converted to hazard ratios (HRs), and a random-effects model was used to pool results. A secondary analysis including only studies that reported estimates adjusted for depression was conducted.

Results Six studies met our inclusion criteria ($N = 402,274$); 5 of these included depression as a covariate. The pooled HR for the magnitude of the relationship between PTSD and CHD was 1.55 (95% CI 1.34-1.79) before adjustment for depression. The pooled HR estimate for the 5 depression-adjusted estimates ($N = 362,950$) was 1.27 (95% CI 1.08-1.49).

Conclusion Posttraumatic stress disorder is independently associated with increased risk for incident CHD, even after adjusting for depression and other covariates. It is common in both military veterans and civilian trauma survivors, and these results suggest that it may be a modifiable risk factor for CHD. Future research should identify the mechanisms of this association and determine whether PTSD treatment offsets CHD risk. (*Am Heart J* 2013;166:806-14.)

Posttraumatic stress disorder (PTSD) is a common, disabling mental health disorder that occurs in individuals who are exposed to traumatic events such as combat, intimate partner violence, or natural disasters.¹⁻³ The estimated lifetime prevalence of PTSD in developed nations is 4.4%,⁴ and ranges from 12.1% to 30.9% in military veterans.⁵⁻⁷ Individuals with PTSD experience a number of disabling symptoms such as intrusive thoughts, nightmares, flashbacks, avoidance of reminders of the traumatic event, and physiological arousal and are at increased risk for suicide, substance abuse, and inability to work.⁸

Although the profound impact of PTSD on mental health has long been recognized, there has more recently been increasing awareness that individuals with PTSD are at increased risk for physical health impairments, most notably with respect to cardiovascular health. Aside from its association with cardiovascular risk behaviors such as smoking,⁹ alcohol abuse,¹⁰ and nonadherence to medications,¹¹ PTSD has been cross sectionally associated with hypertension, dyslipidemia, obesity, and diabetes in young veterans of Operations Iraqi Freedom and Enduring Freedom¹² and with blood-based measures of endothelial dysfunction¹³ and low-grade systemic inflammation in young civilians.¹⁴

Although recent studies are elucidating associations between PTSD and coronary heart disease (CHD) risk factors, it is less clear whether PTSD is independently associated with incident CHD and cardiac-specific mortality risk. Accordingly, we used systematic review and meta-analysis to examine the prospective association of PTSD with incident CHD and cardiac-specific mortality. Because PTSD is often comorbid with depression,¹⁵ a factor shown to increase risk for CHD and mortality,¹⁶ we

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Submitted April 9, 2013; accepted July 16, 2013.

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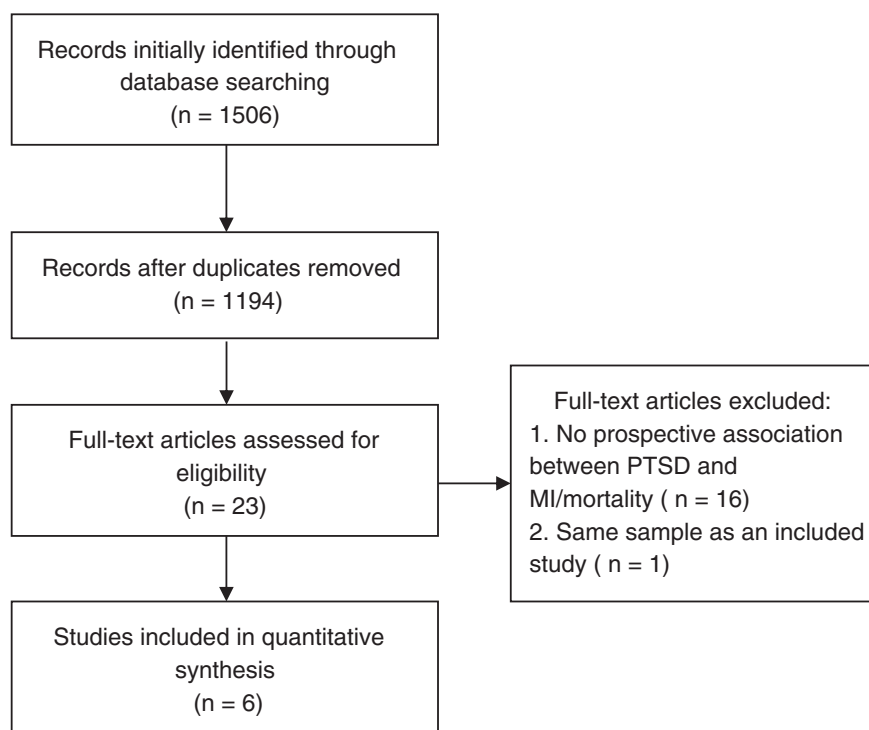
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<http://dx.doi.org/10.1016/j.ahj.2013.07.031>

Figure 1



Search strategy flowchart.

additionally adjusted for depression in determining estimates of the association of PTSD with CHD.

Methods

Data sources and searching

This research was performed in accordance with MOOSE guidelines for meta-analyses of observational studies.¹⁷ Potentially relevant articles were identified by a trained medical librarian (L.F.) who searched publicly available computerized databases. Potentially relevant articles were identified by searching the electronic databases Ovid MEDLINE, PsycINFO, Scopus, PILOTS, and the Cochrane Review. Dates searched were from inception to July 2013. The searches were conducted on August 15, 2012, and July 1, 2013. All relevant subject headings and free-text terms were used to represent PTSD and incident CHD/cardiac-specific mortality, and the sets of terms were combined with AND. Terms for MEDLINE included the following: exp Stress Disorders, Traumatic/OR ptsd.tw. OR (post-traumatic OR (post adj traumatic)).tw OR posttraumatic.tw. OR acute stress disorder\$.tw. OR asd.tw, exp Acute Coronary Syndrome/OR acute coronary.tw. OR acs.tw OR exp Myocardial Infarction/OR myocardial infarct\$.tw. OR (mi OR ami).tw. OR (heart adj attack\$).tw. OR (stemi OR nstemi).tw. OR ((preinfarction OR unstable) adj angina\$).tw. These terms were adapted for the other databases. Additional records were identified by scanning the reference lists of relevant studies and

reviews and by using the Related Articles feature in PubMed and the Cited Reference Search in ISI Web of Science.

Study selection

Eligible study designs included all studies that measured PTSD caused by any traumatic event in initially healthy participants and estimated its association with subsequent CHD or cardiac-specific mortality. Two investigators (D.E., M.M.B.) independently reviewed all citations identified through the literature search, using a predefined protocol to assess study eligibility. Articles that clearly did not meet the inclusion criteria were excluded at the title and abstract level. The remaining articles were selected for full-text review; articles that did not meet the inclusion criteria were excluded. Disagreements regarding the selection of articles were resolved through discussion between the 2 reviewers, and full consensus was achieved at each stage of review.

Data extraction

Two investigators (D.E. and M.M.B.) abstracted information about the dates and cohort sizes of the studies, characteristics of participants enrolled, methods for assessing PTSD, covariates including depression, and the strength of the association between PTSD and incident CHD and cardiac-specific mortality. One study reported estimates for men and women separately,¹⁸ and we thus extracted data separately from this study. Similarly, one study reported estimates for participants with and without depression separately,¹⁹ and we extracted data from this study separately, as well.

Table. Characteristics of studies that estimated an association between ACS-induced PTSD and adverse clinical outcome

Source, y	N	HR not adjusted for depression	HR adjusted for depression; depression assessment	Clinical outcome
Boscarino, 2008 ²⁰	4328	2.25 (1.02-4.95)	2.06 (0.92-4.62); medical record	Heart disease mortality
Jordan et al, 2011 ¹⁸	39,324	1.64 (1.42-1.91)	NA	Self-reported physician-diagnosed heart disease
Kubzansky et al, 2007 ²²	1002	1.42 (1.03-1.95)	1.30 (0.92-1.80); CES-D	Nonfatal MI and CHD mortality
Kubzansky et al, 2009 ²³	1059	3.28 (1.17-8.90)	2.78 (1.10-7.59); DIS	Angina, nonfatal MI, CHD mortality, other cardiac death
Scherrer et al, 2010 ¹⁹	355,999	1.39 (1.33-1.46); age-adjusted only	1.16 (1.00-1.35); ICD-9	Incident MI (ICD-9-CM codes 410-411)
Vaccarino et al, 2013 ²¹	562	2.20 (1.2-4.2)	2.19 (1.1-4.1)	Self-reported hospital admission for heart disease

Abbreviations: ACS, acute coronary syndrome; BMI, body mass index; DSM-III, Diagnostic and Statistical Manual of Mental Disorders, Third Edition; PCL, PTSD Checklist; CES-D, Center for Epidemiological Studies-Depression; DIS, NIMH Diagnostic Interview Schedule.

Data synthesis and analysis

Four studies¹⁸⁻²¹ reported the association of PTSD diagnosis or of a positive screen on a validated PTSD self-report screening questionnaire with incident CHD or cardiac mortality. The other 2 studies^{22,23} reported the risk of incident CHD associated with a 1-SD increase in PTSD symptoms on a self-report PTSD screen questionnaire. For both of those studies, we transformed the findings into a single risk estimate, corresponding to 2 SDs above each study's mean as an approximation of a positive PTSD screen—because the sample scores at that level best approximated published cutoffs for the scales used. Furthermore, although half of the studies reported associations in hazard ratios (HRs), one study²² reported a relative risk and 2^{21,23} reported an odds ratio. For this meta-analysis, we calculated an aggregate point estimate as an HR, using the following equations to convert the relative risk²² and odds ratio²³ to HRs: $RR = \frac{OR}{(1-r)+(r*OR)}$ and $HR = \frac{\ln(1-RR+r)}{\ln(1-r)}$, where RR is the relative risk, OR is the odds ratio, HR is the hazards ratio, and r is the event rate for the reference group (ie, no PTSD).^{24,25}

Comprehensive MetaAnalysis (version 2; BioStat Software, Englewood, NJ) was used for completing all statistical tests and associated graphic results. Estimates of the overall risk of PTSD with incident CHD were pooled using a random-effects model. A secondary pooled analysis was performed by restricting to the 5 studies that adjusted for depression as a covariate for the association of PTSD with incident CHD. Heterogeneity assessments using Cochrane Q statistic and I^2 preceded all meta-analytic tests. There was a statistically significant heterogeneity, justifying the use of a random-effects model to estimate and test effects. Log-transformed HRs and 95% CIs were calculated for each study using the reported effect size and estimates of the SE of each effect drawn from data reported in the article. When articles reported multiple models, we selected the model with the highest level of covariate adjustment. There were too few studies to test for moderator effects; however, we provide descriptive information concerning potential moderators below. To address the issue of

publication bias, we calculated Orwin's fail-safe N and created a funnel plot of SEs by log HRs.

This work was supported by Grants HL-088117, HL-084438, and CA-156709 from the National Institutes of Health (NIH), Bethesda, MD. Its contents are solely the responsibility of the authors and do not necessarily represent the official view of the National Center for Research Resources or the NIH. This work was supported, in part, by Columbia University's Clinical and Translational Science Awards (CTSA) Grant No. UL1RR024156 from National Center for Advancing Translational Sciences-National Center for Research Resources (NCATS-NCRR)/NIH. Dr Kronish is supported by Grant K23-HL098359 from the National Heart, Lung, and Blood Institute. Dr Shaffer is supported by Grant 12CRP8870004 from the American Heart Association and by Grant K23HL112850 from the NIH, Bethesda, MD. No funding bodies had any role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. The authors are solely responsible for the design and conduct of this study, all study analyses, the drafting and editing of the manuscript, and its final contents.

Results

Literature search

A comprehensive search resulted in 1,194 unique articles, of which 23 qualified for full-text review (Figure 1). Six articles met the final inclusion criteria. There was one disagreement²⁶ between the reviewers at the full-text stage, which was resolved by discussion and consensus. A second disagreement²¹ occurred at the data extraction stage because one study published a tightly controlled estimate of the association of PTSD with incident CHD in a sample of twins from a larger registry, which could yield an estimate of the association of PTSD with cardiovascular mortality in a larger sample. We decided to include the

Mean age at baseline	Mean follow-up, y	Sample	PTSD assessment	Covariates included
36.3	15	US Vietnam era veterans	Self-report based on <i>DSM-III</i> criteria	War theater status, age, intelligence, smoking, BMI, depression history
43.9	2.9	World Trade Center survivors	PCL >44	Age, race/ethnicity, education, marital status, smoking, hypertension, diabetes
63	15	US Vietnam era veterans	Mississippi Scale for Combat-Related PTSD (mean + 2 SD)	Age, smoking, systolic and diastolic blood pressure, total cholesterol, BMI, family history of CHD, education, alcohol
44.4	13	Baltimore, Maryland women	Diagnostic Interview Schedule for <i>DSM-III</i>	Age, race/ethnicity, smoking, history of high blood pressure, history of diabetes, alcohol use, education, income
55.7	7	US military veterans	2 outpatient or 1 inpatient PTSD diagnosis codes (<i>ICD-9-CM</i> : 309.81)	Age, sex, race, marital status, insurance, diabetes, hypertension, hyperlipidemia, obesity, alcohol, nicotine
42.6	13	Vietnam era veterans (twin study)	Diagnostic Interview Schedule for <i>DSM-III</i>	Age, education, family income, alcohol, smoking, physical activity, hypertension

more precise estimate from the smaller sample, with the recognition that including the estimate from the larger sample may have increased our overall meta-analytic estimate but would have represented a less rigorous test of our primary study aim.

Study characteristics

Table gives detailed information about study characteristics, which included 402,274 participants cumulatively. Participants were enrolled between 1982²³ and 2000¹⁹ and were followed up for a mean of 2.9¹⁸ to 15 years.²⁰ Most participants were military veterans,^{19,22} but many participants were also drawn from the World Trade Center registry of those exposed to the attacks on 9/11¹⁸ and from a representative sample of women from East Baltimore exposed to various traumas.²³ Accordingly, there were diverse types of traumatic experiences represented in the meta-analysis.

Posttraumatic stress disorder was measured by self-report questionnaire,^{18,20,22} diagnostic interview,^{21,22} or *International Classification of Diseases, Ninth Revision (ICD-9)* diagnostic codes.¹⁹ Articles considered a broad range of covariates, from known cardiac risk factors (eg, systolic blood pressure, hyperlipidemia, and smoking) to intelligence scores. Five of the 6 studies included depression as a covariate.^{19,23} Incident CHD and cardiac-specific mortality were assessed by self-report of physician diagnosis,^{18,21,23} medical records,^{19,20,22} and/or death certificate searches.^{20,23}

Association of PTSD with incident CHD and mortality

There was an indication of heterogeneity in estimates ($Q_5 = 10.17$; $P = .07$; $I^2 = 50.83$), with HRs ranging from

1.39 to 3.28. Figure 2 shows the HRs for incident CHD and cardiac-specific mortality associated with PTSD for each of the 6 studies ($N = 402,274$) with no adjustment for depression. The random-effects model yielded an aggregated HR of 1.55 (95% CI 1.34-1.80).

Although difficult to assess with only 5 studies, there was visual but no statistical evidence of publication bias (Figure 3) because the fail-safe N (number of studies with null findings that would be needed to conclude that the true HR was 1) was 210. Orwin's fail-safe N suggested, however, that 48 studies with null findings would yield a trivial HR of less than 1.04.

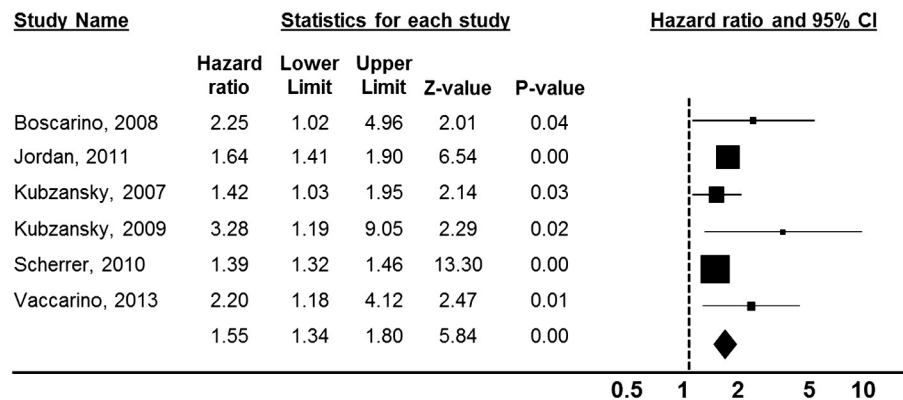
Adjustment for depression

There was also a significant heterogeneity in the depression-adjusted estimates ($Q_5 = 19.09$, $P = .002$; $I^2 = 73.81$), thereby supporting the use of a random-effects model to pool data. The aggregate HR for the 5 depression-adjusted estimates^{19,23} ($N = 362,950$) in a random-effects model was 1.27 (95% CI 1.08-1.49). All studies that adjusted for depression did so after adjustment for clinical and behavioral CHD risk factors. Depression-adjusted estimates reported in each study and the method used for assessing depression in each are given in Table.

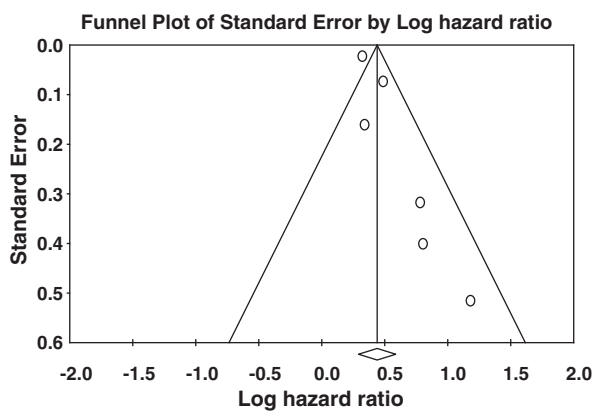
Descriptive information

Possible moderators. Although there were not enough studies to test potential moderators of the association of PTSD with incident CHD, we conducted a number of descriptive analyses for likely moderators.

Sex. There appear to be sex differences in risk for PTSD, and there may be a stronger association of PTSD

Figure 2

Forest plot of the association of PTSD with incident CHD. Note: the area of each square is proportional to the study's weight in the meta-analysis, and each line represents the CI around the estimate. The diamond represents the aggregate estimate, and its lateral points indicate CIs for this estimate.

Figure 3

with incident CHD in women. Without adjustment for depression (only 1 study of women was adjusted for depression), the aggregate HR for the association of PTSD with incident CHD in men was 1.62 (95% CI 1.39-1.89), whereas the HR for women was 1.94 (95% CI 1.13-3.33). Although underpowered to detect a significant difference, a mixed-effects analysis suggested that there may be a sex difference in the association of PTSD with incident CHD ($Q_2 = 4.74$; $P = .09$).

Race/Ethnicity. Some studies suggest that minority race/ethnicity status is associated with increased risk for PTSD, and there are established racial/ethnic differences in CHD risk. We conducted an exploratory meta-regression analysis to determine the association between the racial composition of each study sample and its

estimate of the association between PTSD and CHD by coding each study for the proportion of its participants who identified as non-Hispanic white. Studies ranged from 52% to 100% non-Hispanic white, but racial composition explained less than 1% of the variability in effect size estimates ($P = .89$).

Type of PTSD assessment. Meta-analyses of the rate of PTSD caused by some types of trauma demonstrate that the method for determining a PTSD “case” (ie, positive screen on a self-report questionnaire vs diagnostic interview) influences the estimate of PTSD prevalence.²⁷⁻²⁹ In the included studies, the HR in those that assessed PTSD by clinical interview was 1.81 (95% CI 1.13-2.91), whereas the estimate for those that used screening questionnaires was 1.62 (95% CI 1.42-1.84). Although underpowered to detect a significant difference, a mixed-effects analysis suggested no difference in the association of PTSD with incident CHD by type of PTSD assessment ($Q_1 = 0.21$; $P = .65$).

Date of PTSD assessment. Because definitions of both PTSD and CHD have changed over the last 30 years, we tested whether the date of PTSD assessment was associated with studies' effect size estimates using meta-regression. Date of PTSD assessment accounted for only 1% of the variability in HR estimates ($P = .50$).

Discussion

In this first systematic review and meta-analysis of the prospective association of PTSD with incident CHD and cardiac-specific mortality, we found that PTSD is associated with a 55% increase in risk for incident CHD or cardiac-specific mortality after adjustment for numerous demographic, clinical, and psychosocial factors across 6 studies comprising more than 400,000 participants

followed up for a median of 13 years. After further adjustment for depression, a known contributor to these outcomes, we found that the association between PTSD and CHD was attenuated to 27%, but that risk remained statistically significant.

There are several plausible biological mechanisms linking PTSD with increased CHD risk. Posttraumatic stress disorder is associated with prolonged reactivity and dysregulation of the autonomic nervous system and hypothalamic-pituitary-adrenal axis,² which, in turn, can contribute to the development and progression of atherosclerosis and cardiovascular system damage.³⁰ Neuroendocrine alterations associated with PTSD include increased negative feedback sensitivity of glucocorticoid receptors in the stress-response system and decreased glucocorticoid responsiveness.³¹⁻³³ There is also evidence of autonomic dysfunction in both PTSD and CHD development, as evidenced by decreased heart rate variability, baroreflex dysfunction, and increased QT variability on the electrocardiogram.³³ Reduced heart rate variability has been shown to predict mortality after myocardial infarction (MI),³⁴ reduced baroreflex sensitivity has been linked with carotid atherosclerosis and increased risk of incident CHD,³⁵ and increased QT variability is a predictor of sudden cardiac death.³⁵ Furthermore, von Känel et al^{14,36} found a dose-response relationship between PTSD and inflammatory biomarkers such that as the severity of PTSD symptoms increases, levels of tumor necrosis factor α and interleukin-1 β increase and levels of interleukin-4 decrease. This is an important finding because CHD is understood to be a disease of inflammation³⁷ and the inflammatory markers observed to be dysregulated in PTSD have been linked to CHD events.³⁸ Finally, Ahmadi et al²⁶ recently reported a dose-response relationship between PTSD and coronary artery calcium, a marker of atherosclerosis, and found that the association between PTSD and all-cause mortality was strongest at high levels of coronary artery calcium.

Because depression is an established risk factor for CHD and often comorbid with PTSD, the finding that the association of PTSD with CHD is independent of depression is important. Depression was assessed by self-report,²² interview,^{21,23} and medical record review^{18,20} in the included studies, but only 1 of the studies reported the association of depression (by ICD-9 code) and incident CHD (HR 1.39, 95% CI 1.34-1.45).¹⁸ The same study reported significant associations between ICD-9 code for other anxiety disorders and incident CHD, including panic disorder (HR 1.53, 95% CI 1.36-1.71), generalized anxiety disorder (HR 1.28, 95% CI 1.18-1.38), and anxiety disorder unspecified (HR 1.44, 95% CI 1.37-1.53). These associations are important because PTSD is often comorbid with a host of psychiatric disorders,¹⁵ and to the extent that many are associated with incident CHD (and CHD risk fac-

tors),^{16,27,39} isolating the unique contribution of PTSD from a broader constellation of “distress” is necessary.⁴⁰ The strongest evidence to date that the association of PTSD with incident CHD is independent of general distress is a recent study by Vaccarino et al,²¹ in which it was found that adjustment for depression and other psychiatric diagnoses did not diminish the PTSD-CHD association. Furthermore, in that study, CHD risk was no different for participants with comorbid PTSD and depression than for those with PTSD alone. Future studies that focus on the unique association of PTSD with CHD mechanisms will be important.

Outstanding issues

Numerous behavioral mechanisms may link PTSD with increased risk of CHD and cardiac-specific mortality. Posttraumatic stress disorder has been associated with smoking,⁴¹ drug and alcohol abuse,^{3,10} and obesity.⁴² Most of the included studies adjusted for 1 or more of these behavioral variables when estimating the association of PTSD with incident CHD. None of the included studies, however, directly tested whether these health behavior variables—which may be on the pathway from PTSD to CHD—are mediators of the association. The recent study by Vaccarino et al²¹ showed that, at least in twins who are male Vietnam veterans, the association of PTSD with important CHD health behaviors accounted for almost none of the association of PTSD with incident CHD. That PTSD remained associated with CHD suggests that biological factors may play an important role in the association between PTSD and incident CHD. Of note, we have shown that PTSD is associated with nonadherence to prescribed medications in various patient populations.^{11,43,44} Hence, adherence to cardiovascular medications should also be tested as a potentially important behavioral mediator of the association between PTSD and CHD.

A second outstanding issue is whether sex modifies the association of PTSD with CHD. There are known sex differences in the incidence and presentation of CHD⁴⁵ and PTSD,⁴⁶ so it stands to reason that the link between the 2 may be modified by sex, as well. Given the small number of studies to address the PTSD-CHD link and the myriad differences between the populations sampled that were unrelated to sex, we could only describe the slightly stronger effect size estimate for women in these studies. The one included study that tested the association of PTSD with CHD risk separately for men and women¹⁸ did not show sex differences, but it also failed to control for depression—a CHD risk factor with substantial sex differences. Our descriptive analysis suggested that the risk for CHD associated with PTSD in women may be greater than that in men. Future research should take up this question rigorously.

A third issue is whether minority race and/or ethnicity modifies the association of PTSD with incident CHD. Although our survey of the research to date showed no

evidence of race/ethnicity differences in the PTSD-CHD association, the lack of racial and ethnic diversity both across and within studies highlights the need for future research in this area.

Another issue that is not quite resolved is whether PTSD itself, or the experience of trauma more generally, is responsible for the CHD risk that is reported to be associated with PTSD.⁴⁷ This meta-analysis suggests that PTSD is the primary culprit, as in the 2 included studies to address the issue found that PTSD—and not high combat exposure—was associated with incident CHD.^{20,21} Several studies have measured the associations between trauma exposure and/or PTSD and physical health. Combat exposure has been associated with poor health and mortality in World War II veterans⁴⁸ and incident arterial disease⁴⁹ and self-reported physical health problems⁵⁰ in combat veterans. One large study with 20 years of follow-up, however, found no association between combat exposure and self-reported physical health.⁵¹ In contrast, PTSD has been consistently and independently associated with a near doubling of risk for all-cause and cardiovascular mortality.⁵² Furthermore, although some studies have shown unique effects of both combat exposure and PTSD on health,⁵³ severity of PTSD symptoms often explains the associations between trauma exposure and new onset of disease or health complaints in studies that have considered both simultaneously.^{49,50,54}

Whether a full diagnosis of PTSD—versus a continuous measure of PTSD symptoms assessed by self-report questionnaire—is best for estimating CHD risk is a question currently being considered. The studies included in this meta-analysis suggest that there is a dose-response association of PTSD symptoms reported on self-report screening instruments and incident CHD risk.²¹⁻²³ These findings agree with studies of PTSD after CHD events,^{27,28,55} which have shown a similar dose-response association of PTSD with recurrent CHD.⁵⁶

Limitations

This study should be interpreted in light of its limitations. First, because we did not include unpublished articles or articles from non-peer-reviewed journals, we may have excluded negative findings.⁵⁷ Second, although some articles included in this review were from samples large enough to report useful data on within-sex, race, or ethnicity estimates, there were not enough studies to conduct appropriately powered moderator or meta-regression analyses to determine whether our aggregate estimate was significantly influenced by participant or study characteristics. Third, we only reviewed studies that included measurements of PTSD at 1 time point and its association with a new onset of a CHD diagnosis or mortality, although some of the studies were prospective and some were based on retrospective data review of administrative databases. Other studies that we did not

include in this review have found associations of PTSD with incident CHD using study designs that do not permit conclusions about the temporal relationship between the 2,^{58,59,49,60} or prospective associations of PTSD with other manifestations of cardiovascular disease.⁶¹ Establishing the temporal relationship between PTSD and CHD is important because cardiac events have been shown to cause PTSD in patients with CHD.^{28,55} Finally, all of the included studies were from the United States, so the generalizability of the findings outside the United States cannot be determined.

Implications and conclusions

This systematic review and meta-analysis provides compelling evidence from 6 studies comprising more than 400,000 participants that PTSD is associated with risk for incident CHD and cardiac mortality. Furthermore, it suggests that the association of PTSD with cardiac events and mortality is independent of depression. Many questions concerning the association of PTSD with incident CHD remain open, and more research is needed to determine the precise nature and mechanisms of the association and which populations are at greatest risk. Although questions remain, clinicians should be aware of the cardiovascular implications of PTSD. There is evidence that the likely mechanisms by which PTSD carries cardiovascular risk can be identified early after the PTSD-inducing trauma and that they may accumulate over many years before an acute cardiac event.

Posttraumatic stress disorder represents a major quality of life burden and, as such, should be treated. If PTSD symptoms do not remit, clinicians may still be able to alter the CHD risk trajectory. Unlike many other psychiatric disorders, targeted screening for PTSD is possible because the traumatic events that cause it are identifiable, and screening may allow for early cardiovascular disease risk stratification, particularly once the mechanisms underlying the association of PTSD with cardiac events can be identified. Future research should focus on identifying plausible mechanisms; establishing the feasibility, efficacy, and costs of screening for and tracking CHD risk factors among individuals with PTSD; and determining whether PTSD treatment can offset risk for cardiac events and mortality.

Disclosures

Competing interests: All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf and declare the following: all authors had financial support from the National Institutes of Health for the submitted work, no financial relationships with any organizations that might have an interest in the submitted work in the previous 3 years, and no other relationships or activities that could appear to have influenced the submitted work.

Author contributions: Dr Edmondson conceived the study, led the writing, and is responsible for the statistical analyses. Drs Kronish and Dr Shaffer assisted with analyses and wrote portions of the manuscript. Ms Falzon served as information specialist and created and performed the literature search. Dr Burg served as senior author, abstracted the study data, and was involved with all aspects of manuscript preparation.

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