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Psychological Distress and Cardiovascular Disease

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ABSTRACT

- *Objective:* To review the current literature regarding psychological distress in patients with cardiovascular disease (CVD).
- *Methods:* Relevant and current (2005–2015) studies were retrieved by a series of searches conducted in the PubMed and PsychINFO databases using Boolean terms/phrases along with manual extraction from the reference lists of pertinent studies. Narrative and tabular summaries of the findings are reported.
- *Results:* There is a vast literature on psychological distress and CVD. Depression is the most common disorder studied followed by anxiety and post-traumatic stress disorder. Physiologic mechanisms linking psychological distress to CVD are well theorized. Screening for psychological distress in CVD is recommended. Referral and treatment issues need further exploration. Pharmacologic treatment of psychological distress in CVD remains equivocal; however, promising data exists for other therapies such as cognitive behavioral therapy and social support strategies.
- *Conclusion:* Psychological distress has a significant negative impact on patients with CVD and is underrecognized by health care providers. Primary care providers and cardiovascular specialty providers are called upon to improve their recognition of psychological distress in their patients and assure referrals are made to collaborative care teams for proper diagnosis and treatment.

The association between the heart and the mind has been proposed by scientists since the 17th century. However, it was not until the 1970s that the relationship between cardiovascular disease (CVD) and psychological states came into scientific focus. The study of heart-psyche interactions began with investigations of cardiovascular risk and "type A" personality behaviors (aggressiveness, impatience, a sense of time-urgency, intense achievement drive, seeking recognition) [1,2]. Hundreds of studies generated over the last 10 years have yielded an extensive body of literature regarding this complex interaction.

CVD continues to be the leading cause of death globally. Worldwide and in the United States, CVD accounts for 30% of deaths and more than 2000 deaths per day, respectively [3,4]. Psychological distress (specifically depression) has been reported by the World Health Organization (WHO) as the leading cause of disability in the world [4]. Taken together, CVD and depression constitute an immense health burden and result in poor health status, increased care giver burden [5], increased readmission rates to hospitals, increased utilization of primary care services, poor health compliance [6], decreased health related quality of life [7], and a greater than 2 times increase in mortality [8,9].

Despite its devastating consequences, comorbid CVD and psychological distress remains poorly recognized and treated. In this paper, we present a review of the evidence related to key aspects of psychological distress and CVD (for the purposes of this paper, defined as ischemic heart disease and stroke), and provide information to help improve identification among health care providers. Relevant and current (2005-2015) studies for this review were retrieved by a series of searches conducted in the PubMed and PsychINFO databases using Boolean terms/phrases, along with manual extraction from the reference lists of pertinent studies. Due to the breadth and extent of the literature, a comprehensive review of the literature is beyond the scope of this article. However, the reader will be directed to current systematic reviews,

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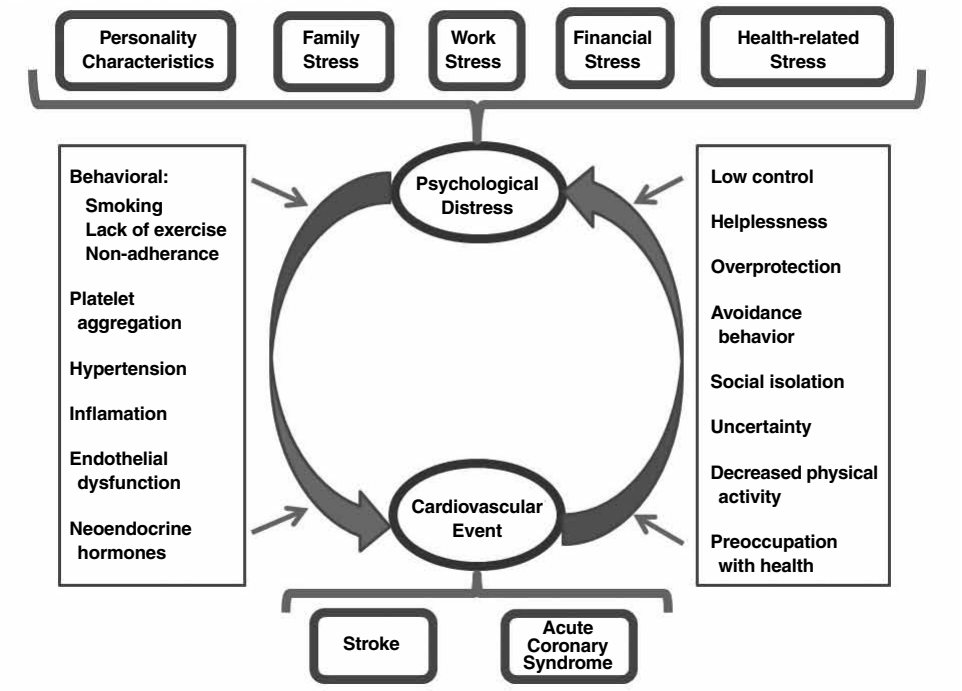

Figure. Mechanisms of psychological distress and cardiovascular disease.

meta-analyses, and recent select research studies sourced for this summary and presented in tabular form.

Mechanisms of Psychological Distress

Psychological distress is created by a number of factors (**Figure**) such as personality characteristics, family stress, work stress, financial stress, and health-related stress [10–12]. These stressors are thought to create an internal physiological environment that is deleterious to the vascular system due to multiple behavioral [13] and physiological mechanisms [14], which ultimately lead to an acute event such as an acute coronary syndrome (ACS) or stroke. In turn, once a CVD event occurs, multiple psychological, sociological, and behavioral factors perpetuate psychological distress, which together with CVD is responsible for numerous poor outcomes.

Multiple behavioral (eg, alcohol intake, physical activity, cigarette smoking) and physiologic (eg, body pain, abdominal adiposity, inflammation, hemodynamic disturbances) mechanisms have been identified as likely mediators between the increase in CVD related to psychological distress [15–17]. Large studies and meta-analyses (**Table 1**) establish that an increased risk and incidence of coronary heart disease (CHD), potentially leading to acute myocardial infarction (AMI), exists in patients with perceived stress [16], stress [17], and psychological distress [15,18]. A meta-analysis equated the increase incidence of coronary heart disease associated with perceived stress to increases in blood pressure, low-density lipoproteins, and smoking [16]. Chronic perceived stress was reported by more than one-third of discharged post-myocardial infarction patients in a secondary analysis of the TRIUMPH (Translational Research Investigating Underlying disparities in acute Myocardial infarction Patients' Health status) study, with half of those describing depressive symptoms [19]. The chronic perceived stress was independently associated with an increase in post-AMI mortality.

Psychological Distress Disorders Related to CVD

Depression, anxiety, and post-traumatic stress disorder (PTSD) are the 3 most common psychological distress disorders related to CVD [20]. Cardiac disease and depression has been most commonly studied. In stroke, the science is not as well evolved due to greater heterogeneity of study samples and outcome measures.

Depression

Dysphoria (feeling blue), anhedonia (inability to experience joy in otherwise enjoyable activities), insomnia or hypersomnia, fatigue or loss of energy, increased guilt or

Table 1. Select Studies of Psychological Distress/Stress and CVD Outcomes

Author	Study Type	Study Group	Predictor Variables	Outcome	Results
Hamer et al (2008) [15]	Prospective study	6576 healthy men and women	Psychological distress (measured using General Health Questionnaire-12), behavioral and pathophysiological risk factors	CVD events	Increased risk of CVD seen in patients with psychological distress (hazard ratio, 1.54). Behavioral factors (eg, alcohol intake, physical activity, and cigarette smoking) explained ~65% of variance using logistic regression). Physiological factors (eg, hypertension and C-reactive protein) accounted for only ~13% and ~5.5%, respectively.
Arnold et al (2012) [19]	Secondary analysis of TRIUMPH study data	4202 AMI patients	Score on Perceived Stress Scale-4 (measures chronic stress) completed during hospitalization	1-yr health status, SF-12, 2-year mortality	AMI patients with moderate/high stress had increased 2-yr mortality compared with those with low levels of stress and had poorer 1-yr health status. Findings persisted after adjusting for confounders (eg, demographics, depression). Increased perceived stress was associated with being unmarried, female, non-Caucasian, less than high school education, and low social support.
Brumby et al (2012) [18]	Cross-sectional descriptive	1792 men and women in farming communities in Australia	Cardiovascular risk factors	Psychological distress (PD)	45.6% reported PD. PD was higher in those < 50 yr compared to those > 50 yr. "Body pain" was significantly associated with PD. There was a positive association between PD and BMI, abdominal adiposity, and metabolic syndrome in the older group.
Richardson et al (2012) [16]	Meta-analysis of 6 studies	118,696 patients without CVD	Self-reported perceived stress	Incident CVD at ≥ 6 mo	High perceived stress was assocated with an increased risk of incident CHD (aggregate risk ratio, 1.27)

worthlessness, decreased concentration, appetite change with significant weight loss or gain, psychomotor retardation or agitation, and suicidal ideation are the symptoms of depression [21]. These symptoms exist on a continuum, ranging from mild symptoms with short duration and limited functional impairment to major depression. Importantly, among otherwise healthy individuals, even minor depressive symptoms have been significantly associated with increased incidence of coronary disease [22].

The breadth of research on depression and CVD is demonstrated by the selected large studies and meta-analyses presented in **Table 2**. In a large study of 43,093 individuals in the National Epidemiologic Survey on Alcohol and Related Conditions, major depressive disorder was shown to increase the odds of any cardiovascular disorder by 50% ($P < 0.001$) [23]. In the longitudinal Netherlands Study of Depression and Anxiety, a 2 to 3 times greater likelihood of developing CVD over a 6-year period was reported in patients with depression compared to those without depression [24]. These recent findings are consistent with the data in the literature presented over the past 2 decades [13]. Though the majority of the literature on psychological distress in CVD represent studies of cardiac patients, a recent meta-analysis found a 29% prevalence of depression in post-stroke patients for up to 10 years [25].

Screening Issues

In recognition of the high prevalence of depression in patients with CHD, an American Heart Association (AHA) science advisory in 2008 recommended routine screening for depression in patients with CHD, with follow-up evaluation for diagnosis and treatment of depression by qualified professionals for positive cases [26]. In 2014, an AHA scientific statement recommended elevating depression to the level of a risk factor in ACS patients [27]. The recommendation for screening was initially met with some concern as being premature [28], when past supporters spoke out against the routine screening of depression in cardiac patients [29]. The dissenting authors claimed that there was

Table 2. Select Studies of Depression and CVD

Author	Study Type	Study Group	Predictor Variables	Outcome	Results
Niranjan et al (2012) [23]	Secondary analysis of data from National Epidemiologic Survey on Alcohol and Related Conditions	Large US sample of noninstitutionalized adults. Survey questions asked about major depressive disorder (MDD) symptoms in addition to alcohol, drugs, etc.	No lifetime depression (n = 34,979) MDD with atypical features (n = 1063) MDD without atypical features (n = 4632)	Physician-confirmed CVD diagnosis	Those with MDD had a 50% increased risk of CVD compared to those without depression (P < 0.001). No greater risk was demonstrated in those with atypical subtype depressive disorder (characterized by increased appetite or weight gain and increased sleep).
Ayerbe et al (2013) [25]	Meta-analysis, 43 studies	20,293 patients with stroke	Stroke	Depression	Depression prevalence was 29% after stroke and remained stable up to 10 years. Cumulative incidence 39%–52% within 5 years of stroke. Stroke severity, depression pre-stroke, disability, cognitive impairment, and anxiety were identified as major predictors of depression.
Seldenrijk et al (2014) [24]	Longitudinal cohort study	2510 CVD-free patients in the Netherlands	DSM-IV diagnostic criteria for depression and anxiety	6-year incidence of CVD	Depressed patients were 2 to 3 times more likely to develop CVD over 6-year study period than those without depression. Increased symptoms resulted in increased odds of developing CVD. Patients with anxiety alone did not have an increased risk of CVD.
Banankhah et al (2015) [48]	Secondary analysis of ENRICHD clinical trial data	770 depressed post-MI patients treated for depression, 130 with treatment-resistant depression (TRD; < 50% decrease in Hamilton Depression score from baseline and > 10 Hamilton score 6 months post treatment)	TRD	Mortality	At mean follow-up of 29 mo, mortality was 13% in TRD patients and 7% in patients without TRD. TRD was a significant predictor of mortality (hazard ratio, 1.995, P = 0.046) after controlling for confounders (eg, age, diabetes, smoking).

a lack of scientific evidence supporting the efficacy of treatment for depression in cardiac patients, and that potential negative effects of routine screening and follow-up treatment were unknown. They argued the following: limited data from randomized controlled trials and/or evidenced-based reviews exist demonstrating improved outcomes in cardiac patients based on screening and referral [30]; antidepressants are not yet recognized to be effective in cardiac populations and there is a lack of evidence related to potential harms [28]; concerns exist about the potential for mass screening to increase health care resource use at the expense of other health care needs [29]; and routine screening may cause unnecessary negative social stigma related to false-positive findings [31].

Although clinical trials of depression treatment in cardiac patients have not demonstrated an increase in survival, treatment has been shown to be effective in reducing depression symptoms, improving patient satisfaction with depression care and improving health related quality of life [32–34]. Further, recent studies described the AHA recommendation as well accepted by cardiac unit staff, not heavily resource intensive, feasible, and accurate [35,36]. Bigger and Glassman [37] published a recent analytical review of the AHA advisory and concluded that the advisory is supported by the literature. A

salient point regarding the depression screening debate is that screening without proper follow-up for further diagnosis and potential treatment may be harmful [28,29,31]. Despite concerns of the potential negative impact of depression screening in cardiac patients raised in the literature, the preponderance of the literature indicates that its benefits are likely to outweigh its risks [32,34,36,38–40].

Outcomes of Depression Treatment

Answers to questions about improvement in cardiovascular and all-cause mortality outcomes with depression treatment remain elusive in the literature. However, data show an improvement in depressive symptoms and quality of life for depressed patients receiving some types of treatment [33,41–45]. The Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) study was a landmark study of MI patients with a 6-month treatment intervention of cognitive behavioral therapy (CBT) plus pharmacologic intervention if indicated for depression [33]. Patients were followed for an average of 29 months post-MI. A significant improvement was seen in depressive symptoms and social isolation in the treatment group; however, there was no improvement in event-free survival [33]. When outcome measures are restricted to mortality alone, subsequent trials of antidepressant medications for treatment of depression in cardiac patients have shown them to be ineffective [46]. However, CBT and other supportive stress management strategies are effective in decreasing depressive symptoms and improving the quality of life in patients suffering with depression and CVD [46].

Promising results are emerging in the literature as researchers refocus their analysis on subgroups of depressed cardiac patients. In one large study of 442 depressed and 325 non-depressed patients, the number of depressive symptoms after an MI irrespective of the pre-MI depression status was associated with worse cardiac outcomes [47]. For every 1 additional depressive symptom reported 1 year post-MI, patients had a 15% increased risk for a new cardiac event in the next 2.5 years [47]. Another study demonstrated an improvement in depressive symptoms by 75.3% in in post-cardiac surgery patients with low ejection fraction (< 40%) after 8 weeks of nurse-guided CBT and worsening in depressive symptoms by 26.8% in usual care patients. More moderate findings were seen in the those with higher ejection fraction receiving the same CBT intervention for depression [45]. A treatment-resistant depression subgroup analyzed in a recent secondary analysis of the ENRICHD trial showed a twofold increase in mortality when compared to those in the non–treatment-resistant depression group [48]. Since treatment does not work for all patients with depression, including depressed post-MI patients, further evaluation with a focus on those who respond to treatment is needed.

Depression in Stroke

Depression has been reported to have a 39% to 53% incidence in stroke patients within 5 years of a stroke [25]. Compared to nondepressed stroke patients, depressed post-stroke patients experience an approximately 5 times increased risk for disability at 3 months post-stroke, along with a fourfold increase in anxiety, a 27% increase in mortality, and lower quality of life [49]. Regarding depression, stroke represents unique screening and treatment challenges. Apathy and depression can occur together or independently after stroke, with apathy having been reported at a higher rate than depression in post-stroke patients [50]. Since apathy is a disturbance of motivation presenting as no obvious emotions, indifference, and contentment with doing nothing, it is easily misinterpreted as depression [51] and occurs in more than half of those with depressed mood. A meta-analysis of 19 studies and 2221 stroke patients reported a 36.6% pooled rate of apathy, which was 3 times higher than the rate of depression [51]. In addition, up to 53% of post-stroke patients report pseudobulbar affect, defined as sudden and socially inappropriate laughing and crying, which adds to the complexity of screening for and diagnosing depression [52]. Given the challenges associated with post-stroke symptoms, screening post-stroke patients has been studied and reported as feasible using the Patient Health Questionnaire (**Appendix**), which is recommended by the AHA for screening in cardiac patients [53].

Issues related to treatment of stroke patients parallel those of depressed patients with cardiac disease, as the effect on mortality and survival is unknown. Depression has been reported to go untreated in up to 67.9% of depressed post-stroke patients [54]. In addition, mismatches between antidepressant prescription and those with depression suggested that some patients without depression were being treated for depression while some patients with depression were not being treated [54].

Table 3. Select Studies of Anxiety and CVD

Author	Study Type	Study Group	Measures	Outcome	Results
Huffman et al (2008) [56]	Prospective study	110 patients 72 hours' post-MI	Anxiety assessed using Beck Anxiety Inventory	In-hospital cardiac complications	Post-MI anxiety significantly associated with serious in-hospital complications ($P = 0.003$). Cardiac illness, demographics, and depression variables were controlled for. Cardiac complication were predicted independently by psychological symptoms of anxiety alone ($P = 0.015$).
Shen et al (2008) [57]	Secondary analysis of Normative Aging Study data	735 older men without CHD or diabetes	Anxiety assessed via 4 scales: 1. Psychasthenia 2. Social Introversion 3. Phobia 4. Manifest Anxiety plus an overall measure derived from the 4	Onset of MI over an average of 12.4 yr	After controlling for demographic and clinical variables, adjusted relative risk of MI associated with overall anxiety was 1.43 (95% CI 1.17 to 1.75). This risk remained significant after additional adjustment for health behaviors (smoking, drinking, caloric intake), medications (hypertension, high cholesterol, diabetes) during follow-up, and further psychologic variables (negative emotions, depression, anger, type A behavior, and hostility).
Roest et al (2012) [59]	Longitudinal, naturalistic cohort study	438 patients with AMI	Generalized anxiety disorder, assessed via Composite Intl Diagnostic Interview	All-cause mortality and CVD-related readmissions	After adjustment for gender and age, there was an increased rate of adverse events (hazard ratio: 1.94; 95% CI 1.14 to 3.30; $P = 0.01$). The twofold increase in adverse events was independent of depression and cardiac disease severity.
D'Aniello et al (2014) [55]	Cross-sectional study	81 chronic post-stroke patients	Assessed for anxiety and depression using Hospital Anxiety and Depression Scale		Greater than 55% prevalence rate for anxiety compared to approximately 20% for depression. Enhanced fear, muscle tension, and nervousness are likely to influence post-stroke cognitive and physical rehabilitation outcomes
Butnoriene et al (2015) [58]	Prospective longitudinal study	1115 middle-aged and elderly primary care patients	Patients evaluated for metabolic syndrome, generalized anxiety disorder (GAD), CVD risk factors	10-year all-cause and CVD mortality	GAD and metabolic syndrome were associated with up to a twofold increase in all-cause mortality and up to a threefold increase in cardiac mortality in middle-aged women but not men. Cardiovascular mortality was approximately doubled (hazard ratio 1.83–1.99; $P < 0.025$) in women with current GAD independent of cardiac risk factors and metabolic syndrome.

Anxiety

Anxiety disorders create behavioral disturbances of fear and avoidance related to an individual's propensity to overestimate dangers [21]. Though a number of anxiety disorders are described in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5), anxiety as a general constellation of symptoms (afraid, inability to relax, worry about everyday problems, feelings of panic) is described in most of the literature related to CVD [55–57]. To a lesser extent, generalized anxiety disorder (GAD), defined as anxiety symptoms on more days than not and lasting more than 6 months [58,59] has also been studied. Because anxiety can be a component of depression, the 2 are often discussed together. Taken together, in the context of CHD, individuals with both anxiety and depression are at significantly greater risk for death (odds ratio 2.35, 95% confidence interval 1.23-4.47, $P = 0.01$), compared with those without symptoms [60].

The negative effect of anxiety in the cardiac and stroke populations is well documented in the literature (**Table 3**). Those with anxiety have a 43% increased risk

of having an AMI and this risk remains high after controlling for traditional risk factors [57]. Post-MI anxiety is independently significantly predictive of cardiac complications with serious in-hospital post-MI complications [56]. One study reported cardiovascular mortality was approximately doubled in women with current GAD independent of cardiac risk factors and metabolic syndrome [58]. Another study reported a similar twofold increase in adverse events in the presence of GAD, independent of depression and cardiac disease severity [59]. In a study of chronic post-stroke patients, the prevalence rate of anxiety was 55%, compared to 20% for depression, and anxiety was correlated negatively with physical rehabilitation and post-stroke cognition outcomes [55]. There is a paucity of data related to formal screening of anxiety in cardiac patients and the effects of treatment for anxiety in both the CHD and stroke populations.

Post-Traumatic Stress Disorder

PTSD presents with a heterogeneous cluster of symptoms that are generally described as avoidance, re-experiencing, arousal, and negative cognitions and mood [21]. Previously classified as a anxiety disorder in DSM-IV, PTSD is in a new category of trauma and stress or related disorders in the revised DSM-V [21]. In addition, the temporal component of the symptoms has been changed to a disturbance lasting more than 1 month, without reference to acuity or chronicity.

Most studies of PTSD include depression, which elevates the effects of PTSD on CHD. A recent large meta-analysis addressed this issue by removing the effect of depression, which reduced the hazard ratio of CHD in PTSD from 1.55 (when depression was included) to 1.27 (when depression was not included) [61]. There is an increased focus on general stress and in particular post-traumatic stress as an independent factor in CHD risk (**Table 4**). In one large study of 637 patients, relative risk of death was 1.48 (95% confidence interval [CI] 1.03 to 2.91, $P = 0.01$) in subjects with PTSD and CAC score > 0 (indicating atherosclerosis) compared to subjects without PTSD and CAC score equal to 0 [62]. In a report from the Heart and Soul Study, patients with CHD and PTSD were 1.5 to 2 times more likely to report poor health behaviors after controlling for income and depression [63]. Having an AMI, on the other hand, may be a mediator of PTSD symptoms in patients with neuroticism and antagonistic personalities, as they likely magnify the cardiac event [64]. In one recent study, over 17% of post-ACS patients met the criteria for significant PTSD, and of those with PTSD over 70% reported they suffered significantly [65]. No large studies were found related specifically to formal screening for PTSD or treating PTSD in patients with CVD.

Implications for Clinical Practice

Despite the extensive body of literature regarding the negative association of psychological distress to health outcomes in CVD patients, there remains a significant practice gap related to screening, referral, and treatment of psychological distress in CVD patients [30,36,66]. Busy clinical practices focused on physical symptoms (which may not be recognized as mental health–related), along with a health system that has historically regarded mental health issues as the sole domain of mental health professionals creates barriers that need to be overcome. Many health care providers are not proactive in screening patients for psychological distress [11]. Additionally, psychological distress is often perceived as a metaphysical, inexact phenomenon, and is not regarded with the same import as physiological indices, such blood pressure and lipid levels [13]. Another significant barrier may be that the importance of psychological distress, especially depression, has been minimized by some investigators and clinicians because of the lack of data that show improvement with treatment of "hard" outcomes, such as mortality. Lastly, no studies have conclusively demonstrated that treating depression in the general population would lower subsequent cardiovascular clinical events, adding to the minimization of the importance of screening, referral, and treatment of psychological distress among clinicians.

The challenges associated with psychological distress and CVD are centered on the perceived role of the health care provider and role of the patient [11,67]. To date, identification of patients with psychological distress in CVD populations has not been considered a part of routine practice, and only a small percent of those identified with psychological distress are treated. In one study, 17.6% of 1181 patients had moderate to severe depression and of those, only 24.5% were recognized as depressed by their health care providers [68]. In a smaller study of 35 patients with depression after an acute MI, only 10% received treatment with antidepressants [66]. Similarly poor treatment rates were seen in a recent study of antidepressant use after stroke and transient ischemic attack (TIA), with 67.9% of stroke patients and 70.0% of TIA patients with persistent depression going untreated [54].

Table 4. Select Studies of Post-Traumatic Stress Disorder (PTSD) and CVD

Author	Study Type	Study Group	Measures	Outcome	Results/Comments
Ahmadi et al (2011) [62]	Prospective descriptive	637 without known CAD	Patients were assessed for PTSD	Atherosclerotic CAD (as assessed via coronary artery calcium score) and mortality	There was a one and a half times greater risk of death in patients with PTSD who had a coronary artery calcium score > 0. Atherosclerosis was greater in those with PTSD and was and predictive of mortality when gender, age, and conventional risk factors were controlled for.
Chung et al (2011) [64]	Prospective descriptive	120 MI patients	Patients completed scales, including the Posttraumatic Stress Diagnostic Scale, GHQ-28	PTSD and general psychological distress	AMI is a likely mediator affecting the degree of PTSD symptoms in those with neurotic traits due to magnification of the cardiac event. Problem-focused coping is influenced by neuroticism such that it affects general psychological distress following an AMI.
Zen et al (2012) [63]	Prospective cohort (Heart and Soul Study)	1022 adults with CVD	Patients were evaluated for PTSD, and physical activity, medication adherance and smoking were assessed		Patients with PTSD and heart disease were 1.5 to 2 times more likely to report physical inactivity, medication nonadherence (skipping medications or forgetting medications) and smoking, even after controlling for depression and income. Poor health behaviors may be involved in the high risk of adverse CVD events in PTSD patients.
Bluvstein et al (2013) [65]	Prospective descriptive	82 MI and CABG survivors admitted to cardiac rehab	Patients were assessed for PTSD symptoms and posttraumatic growth (PTG; a subjective experience of positive psychological change reported by individuals as a result of coping with trauma or highly challenging life crisis)		17.1% suffered significant PTSD symptoms. PTG was moderator between PTSD and mental health outcomes. PTG potentially mitigates the negative effects of PTSD.
Edmondson et al (2013) [61]	Meta-analysis of 6 studies and 402,274 participants	Patients with PTSD and free of CHD		Incident CHD	All but 1 study included the covariate of depression. An independent association exists between PTSD and CHD: hazard ratio (HR) 1.55 before adjustment & HR 1.27 when adjusted for depression.

Stress, depression, anxiety, and PTSD are often not self-contained or experienced in isolation. Rather, some or all of these conditions may present as an interconnected phenomenon [69,70]. There are several reasons for this. First, symptom identification relies on self-report and/or observation of the symptoms, which may confound validity and reliability of diagnosis [70]. Second, the conditions share some symptoms, which may complicate diagnosis of a primary condition. In addition, the overlap of somatic, cognitive, and affective symptoms [71] may deter health care providers from using screening tools that have physical symptoms as part of the screening process. In a recent study of depressed cardiac patients, investigators clustered symptoms and demonstrated that cognitive affective symptoms of depression predict depression in patients with heart disease [72]. Certain events, such as stroke, may make screening especially difficult due to the presence of neurological changes that may complicate the screening process [73]. These issues highlight the need for formalized depression screening tools such as the Patient Health Questionnaire 2-item screening tool (PHQ-2) and/or

Patient Health Questionnaire nine-item screening tool (PHQ-9) (Appendix), as recommended by the AHA. The PHQs have been validated in stroke patients and cardiac patients and have been found to be accurate, easy to use, and feasible [35,36,73].

Patients look to their providers for information and security during vulnerable times in their life. In a 2014 study regarding the perceptions of psychosocial consequences and access to support after MI, patients reported a high sense of security regarding being able to contact their providers [74]. Providers in both the primary and acute care settings should use these opportunities to assess for psychological distress. Many inpatient health care providers assume that it is the primary care providers' responsibility to screen for depression [75]; however, screening should be done in all practice settings caring for patients with CVD. Clinical practice environments should develop policies and procedures to define who, when, and how screening for psychological distress is accomplished using currently available brief screening tools.

A plan for insuring that proper referrals are made following screening to ensure accurate diagnosis, effective treatment, and follow-up should be in place. To date, there is ample evidence that patients benefit from screening in the context of an interdisciplinary treatment approach [34]. Collaborative care, utilizing a team of health professionals (physician, case manager trained in working with patients with psychological distress, and mental health specialist) working with the patient, is an ideal model to improve outcomes [44]. A Cochrane review of 79 RCTs with over 24,000 participants compared utilization of a collaborative team with routine care and found decreased depression and anxiety symptoms in patients receiving team care for up 2 years, along with improved medication adherence, improved quality of life, and improved patient satisfaction with care [39].

Conclusion

Psychological distress remains underrecognized in CVD patients. Brief screening tools such as the PHQ-2 and PHQ-9 are available, easy to use, and reliable for use by clinicians to improve case finding. Primary care providers and cardiovascular specialty providers are called upon to improve the recognition of psychological distress in their patients and assure referrals are made to collaborative care teams for proper diagnosis and treatment of mental health issues. Longitudinal studies focused on the impact of primary/secondary/tertiary psychological distress prevention strategies in the general population, as well as those with CVD, are needed to bring the state of the science forward and provide evidence to enhance the care of those with psychological distress and CVD.

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Appendix. Patient Health Questionnaire (PHQ-9)*

	Not at all	Several days	More than half the days	Nearly every day
1. Over the *last 2 weeks*, how often have you been bothered by any of the following problems?				
a. Little interest or pleasure in doing things	☐	☐	☐	☐
b. Feeling down, depressed, or hopeless	☐	☐	☐	☐
c. Trouble falling/staying asleep, sleeping too much	☐	☐	☐	☐
d. Feeling tired or having little energy	☐	☐	☐	☐
e. Poor appetite or overeating	☐	☐	☐	☐
f. Feeling bad about yourself or that you are a failure or have let yourself or your family down	☐	☐	☐	☐
g. Trouble concentrating on things, such as reading the newspaper or watching television	☐	☐	☐	☐
h. Moving or speaking so slowly that other people could have noticed. Or the opposite: being so fidgety or restless that you have been moving around a lot more than usual.	☐	☐	☐	☐
i. Thoughts that you would be better off dead or of hurting yourself in some way	☐	☐	☐	☐
2. If you checked off any problem on this questionnaire so far, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?	Not difficult at all ☐	Somewhat difficult ☐	Very difficult ☐	Extremely difficult ☐

Scoring:
Count the number (#) of boxes checked in a column. Multiply that number by the value indicated below, then add the subtotals to produce a total score. The possible range is 0–27. Use the table below to interpret the PHQ-9 score.

Not at all (#)_____ x 0 = _____
Several days (#)_____ x 1 = _____
More than half the days (#)_____ x 2 = _____
Nearly every day (#)_____ x 3 = _____
Total score: _____

Score	
0–4	Minimal depression
5–9	Mild depression
10–14	Moderate depression
15–19	Moderately severe depression
20–27	Severe depression

*The PHQ-2 comprises the first 2 items of the PHQ-9 (1a and 1b).