



Depression, Anxiety, Perceived Stress, and Their Changes Predict Greater Decline in Physical Health Functioning over 12 Months Among Patients with Coronary Heart Disease

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Abstract

Background Although the deleterious impact of psychological distress on patients with coronary heart disease (CHD) is recognized, few studies have examined the influence of change in psychological distress on health outcomes over time. This study investigated whether three common manifestations of distress (depression, anxiety, and perceived stress) and their changes predicted the decline in physical functioning in CHD patients over 12 months. In addition, perceived social support was examined as a buffer of psychological distress or a direct predictor of physical functioning.

Methods Participants were 255 CHD patients with a mean age of 63 (SD = 8.65) years, including 208 men and 47 women. Psychological distress and physical functioning were assessed at baseline, 6 months and 12 months. Hierarchical regression analyses were conducted to examine the influences of psychological factors on physical functioning over 12 months. All models were adjusted for baseline physical functioning, age, gender, marital status, education, BMI, and length of participation at a wellness center.

Results For each psychological distress variable (depression, anxiety, or perceived stress), both the baseline (β s = -0.19 to -0.32, p s = 0.008 to < 0.001) and its respective change over time (β s = -0.17 to -0.38, p s = 0.020 to < 0.001) independently and significantly predicted greater decline in physical functioning at 6 and 12 months, after adjusting for covariates. Perceived social support predicted greater improvement in physical functioning at 12 months (β = 0.13, p = 0.050), but it did not buffer impact of psychological distress.

Conclusions Findings underscore the importance of monitoring various forms of psychological distress continuously over time for CHD patients.

Keywords Depression · Anxiety · Perceived stress · Perceived social support · Physical functioning · Coronary heart disease

Introduction

Coronary heart disease (CHD) is the global leading cause of mortality and disabilities among both men and women, imposing a tremendous burden on the healthcare system worldwide [1]. It is estimated that 110.6 million people in the world are living with CHD [2] and approximately 8 million die from CHD annually, accounting for 16.6% of all deaths globally [1]. In addition to its societal cost, CHD also has a profound impact on patients. Research shows that CHD patients with frequent physical symptoms experience more severe functional impairments, increased use of healthcare services, and substantial reduction in quality of life [3].

Psychological distress, including depression, anxiety, and perceived stress, is not only prevalent among CHD patients, but it also predicts adverse clinical outcomes. Approximately

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40% of CHD patients show noticeable depressive symptoms [4], and about one in five meets the criteria of major depressive disorder [5–9]. Although there is less research on anxiety, a recent review estimated that the prevalence of anxiety disorders among CHD patients was 16% on average and 18% if assessed by trained psychiatric evaluators [10]. In addition, many CHD patients reported high perceived stress as another form of emotional distress, with estimates as high as 50% or more reporting significant levels [11].

Not only is psychological distress common among CHD patients, but it is also a robust predictor of unfavorable clinical outcomes, including relapse, rehospitalization, morbidity, and mortality [12]. For example, depression was found to predict higher mortality within 24 months following myocardial infarction [13], and it was associated with more than a three-fold increase in hospitalization over 18 months [14]. Anxiety is also shown to have detrimental impact on CHD patients [15, 16]. A prospective study reported that anxiety predicted cardiac death, myocardial infarction, cardiac arrest, and revascularization among CHD patients over 2 years [17]. In addition to depression and anxiety, a study of perceived stress reported that marital and relationship stress contributed to a 2.9-fold increase in the risk of recurrent events among women with CHD over 5 years [18].

For cardiac patients, evaluation of health-related quality-of-life (HRQoL) and functional capacities provide valuable health information that facilitates treatment planning and disease management [19]. These measures assess the impact of illness and its treatment on patients, and they also predict subsequent clinical outcomes effectively [20–23]. Psychological distress has been shown to have a deleterious influence on physical functioning and HRQoL in CHD patients. Depression, which has received much research attention, is found to predict subsequent impairment in physical functioning, even after adjusting for baseline functioning and other relevant covariates [23–25]. In assessment of depression in cardiac patients, the somatic items in the measure, such as fatigue, reduced activities, and sleep problems, can be confounded with cardiac symptoms [26, 27, 22]. It has been suggested that measures with affective and cognitive items, but not somatic ones, may better reflect the severity of depressive symptoms among cardiac patients [22, 28].

In addition to depression, fewer studies have examined anxiety and perceived stress, and existing research suggests that they were inversely associated with CHD patients' physical functioning. For example, CHD patients with elevated anxiety showed significantly lower physical functioning at 3-year follow-up [24]. Stressful life events were reported to be associated with lower physical functioning and HRQoL in CHD patients [29]. Furthermore, there has been emerging evidence demonstrating that stress management [30] and complimentary interventions [31, 32] to reduce stress and anxiety

showed beneficial effects in CHD patients, including reduced cardiac risk and improved health functioning.

Despite the findings linking psychological distress and poor health outcomes among CHD patients, the great majority of studies focused only on the initial evaluation of emotional states. Little research has investigated whether the change in distress over time may also impact patients' health outcomes. Considering that individuals' distress levels fluctuate over time, it is conceivable that the change in distress, in addition to the initial status, may provide additional prognostic information. In relevant literature, few studies were found to examine the effect of change in psychological distress on CHD patients' health functioning [33–35]. For example, it was reported that patients with persistent depression or new onset of depressive symptoms were at much higher risk for decreased daily living capability over 30 months [33] and that the changes in depression predicted declines in patients' performance of daily living activities over 5 years [34]. In sum, despite the evidence connecting psychological distress and poor clinical outcomes in CHD patients, there remains a research gap in understanding whether the change in distress may affect health outcomes over time.

As psychological distress is recognized for its detrimental influence, researchers have also explored psychosocial protective factors against emotional disturbances. Social support is commonly construed as a salutary factor in health promotion due to its direct positive effect on health or as a buffer against emotional distress and its health impact [36–38]. A review reported that social support predicted greater HRQoL in patients after CHD diagnosis over a period from a few weeks to over a year [39]. In addition to its direct effect, some studies regarded social support as a distress moderator that buffered the negative influence of emotional disturbances. For example, in a study of patients after myocardial infarction, social support, although not directly associated with cardiac mortality, buffered the impact of depression on mortality over 12 months [36]. However, another study of CHD patients, which examined social integration as a buffer of depression in prediction of prognosis, did not find a significant moderation effect [37]. In sum, social support is generally regarded as a direct protective factor against poor outcomes in CHD patients, but its role as a distress buffer remains uncertain due to a relative lack of research [29, 40].

There are several gaps in existing research. First, most research on psychological distress has focused on depression, leaving other common manifestations of distress, including anxiety and perceived stress, under-investigated. Second, although there is growing longitudinal research, the great majority of these studies only examined psychological distress at initial assessment without considering whether changes in distress might affect health outcomes over time. Third, the measures of depression in research often included somatic items, such as fatigue and sleep disturbances, which could be

confounded with cardiac symptoms and inflate its association with physical dysfunction.

This study is aimed at investigating these issues in hopes of filling the gaps in current research. First, this study investigated whether three major forms of psychological distress, including depression, anxiety, and perceived stress, would predict the decline of physical functioning among CHD patients over a period of 12 months. Second, in addition to the initial distress levels, this study also examined whether its change would independently contribute to the further decline in patients' physical functioning over 12 months. Third, this study investigated whether perceived social support was a protective factor among CHD patients, either as a direct predictor of better physical functioning or as a buffer of harmful impact of psychological distress. In addition, this study tested the influence of depression, either with or without the somatic symptoms potentially confounded with CHD, to determine whether the findings were affected by these items.

Methods

Participants

Participants were 255 patients with CHD recruited consecutively from a community-based Heart Wellness Center (HWC) at the Singapore Heart Foundation. The HWC offered community-based outpatient cardiac rehabilitation for long-term health maintenance. The program consisted of exercise sessions, nutrition counseling, and health classes to support healthy lifestyle and cardiac risk reduction. Eligible participants included those who were at least 21 years of age and diagnosed with CHD verified by a cardiologist. Patients were excluded if they [1] were treated for another major, potentially life-threatening illness (e.g., stroke, cancer and renal failure) that would confound the assessment of physical functioning, [2] showed severe cognitive impairments that hindered their understanding of the interview and questionnaires [3], and exhibited active symptoms of severe psychiatric disorders. Among the 255 participants, 185 and 152 completed follow-up assessments at 6 and 12 months, respectively.

Procedures

Participants were recruited during their outpatient appointments at the Heart Wellness Center. At the baseline evaluation, the participant first met with a research assistant who conducted a brief medical interview in which socio-demographic information and relevant medical history were collected. After the interview, they were administered a packet of measures that assessed their psychosocial characteristics and health functioning. They were instructed to either complete these measures on site or finish at home and return them by mail.

Participants' medical records were reviewed to retrieve relevant clinical history and comorbid conditions. They were reassessed at 6 and 12 months after the baseline. The study was approved by the University Institutional Review Board, with written informed consent obtained from all participants.

Measures

Physical Functioning Physical functioning was measured by the physical health subscale of the MacNew Heart Disease Health-Related Quality of Life Questionnaire (MacNew). The MacNew is a widely used instrument with established reliability, validity, and clinical utility for assessing health functioning of CHD patients [41]. The physical health subscale contains 9 items assessing the physical symptoms and difficulties caused by cardiac diseases, such as chest pain, shortness of breath, aching legs, dizziness, and limitations in exercise or other activities due to heart problems. In this study, the scale demonstrated high internal consistency, with Cronbach's α being 0.82, 0.83 and 0.83 at the baseline, 6 months and 12 months, respectively. Participants rated the severity of each symptom that they experienced in the previous week on a 7-point rating scale. An average score, in the range between 1 and 7, was used in the analysis, with higher values representing better physical functioning.

Depressive Symptoms The depressive symptoms were measured by the Center for Epidemiologic Studies Depression Scale (CES-D). The CES-D is a well-validated measure to assess depressive symptoms with high sensitivity and specificity [42, 43]. It consists of 20 items tapping common depressive symptoms in four domains, including depressive affect, interpersonal dysfunction, well-being, and somatic symptoms [44].

Seven items in the CES-D assess somatic manifestations of depression, such as poor appetite, restless sleep, feeling of excessive effort, talking less, and concentration difficulties. When evaluating CHD patients, there are concerns that these items may be confounded with cardiac symptoms and lead to inaccurate assessment of depression [22]. To rule out the potential confounding, both the full scale and a modified version without somatic items were tested in this study. For the version without somatic items, the sum scores of available items were linearly transformed by dividing them by 13 and then multiplying by 20 to maintain the range being between 0 and 60. The internal consistency reliability of the full scale and modified one was high in this study, with Cronbach's α of the full scale being 0.85, 0.87 and 0.90 at the baseline, 6 months and 12 months, respectively, and the modified scale being 0.81, 0.83, and 0.85 at each time point.

Anxiety Symptom Severity Anxiety was measured by the anxiety subscale of the Hospital Anxiety and Depression Scale (HADS) [45]. The HADS was developed specifically for

medical patients, with demonstrated validity and reliability. It contains seven items tapping common anxiety symptoms, such as worry, fright, inability to relax, and feeling of panic. The participant rated the extent to which one experienced each anxiety symptom on a 4-point scale. The scale showed high internal consistency in this study, with Cronbach's α being 0.81, 0.84, and 0.80 at the baseline and each follow-up. A sum score, ranging from 0 to 21, was used in the analysis, with higher values signifying more severe anxiety.

Perceived Stress Perceived stress was measured by the 10-item Perceived Stress Scale (PSS) [46]. It is a well-validated instrument that assesses the degree to which individuals perceive their life situations as stressful, difficult, overloaded, and beyond their control. Participants rated each item on a 5-point scale. Its internal consistency reliability was high in this study, with Cronbach's α being 0.86, 0.90, and 0.91 at each assessment over 12 months. An average score between 1 and 5 was used in the analysis, with higher values denoting greater perceived stress.

Perceived Social Support Perceived social support was measured by the 19-item Medical Outcome Study Social Support Questionnaire (MOS-SS). It is a valid and reliable instrument tapping the perceived availability of social support in four domains, including emotional, informational, and tangible support, as well as positive social interactions [47]. Participants rated how much they agreed with each statement of support on a 5-point scale. Its internal consistency reliability was excellent, with Cronbach's α being 0.97 in this study. An average score, ranging from 1 to 5, was computed for the analysis, with higher scores reflecting greater perceived social support.

Medical Record Review Participants' medical records were reviewed to retrieve relevant clinical history, treatment received, and comorbid conditions. These included history of cardiovascular diseases (e.g., myocardial infarction and angina), cardiac surgeries, cardiac risk factors (e.g., hypertension, dyslipidemia, diabetes, and smoking), prescribed medications, and length of participation at the Heart Wellness Center.

Statistical Analysis

To compare the differences between participants who remained in the study at follow-ups and those who did not, independent sample *t* tests and Chi-square tests were conducted. Hierarchical multiple regression analyses were conducted to examine whether psychological distress variables (depression, anxiety, and perceived stress) would predict physical functioning at the baseline and each follow-up. Considering that depression, anxiety, and stress were fairly highly

correlated ($r_s = 0.52\text{--}0.62$, $p_s < 0.001$), they were tested in separate models.

All models were adjusted for demographic and medical covariates, including age, gender, marital status, education, BMI, and months at the wellness center. These covariates, which may be confounded with health outcomes, were selected, given that they were commonly included in prior research [39]. In addition, we tested the associations between a number of biomedical and behavioral variables and follow-up physical health functioning, including history of myocardial infarction, angina pectoris, coronary artery bypass grafting, angioplasty, stenting, valve repair or replacement, diabetes mellitus, dyslipidemia, hypertension, cigarette smoking, alcohol consumption, and waist circumference. None of them were significant (all $p_s > .05$), and therefore were not included as covariates.

To examine whether the change in distress, in addition to its baseline, would predict physical functioning over time, change scores of depression, anxiety, and perceived stress over 6 or 12 months were calculated by subtracting the baseline values from those at follow-ups. In the longitudinal models to predict physical functioning at 6 and 12 months, baseline physical functioning was also included as a covariate, thus allowing the examination of predictors of improvement or decline of physical functioning as residualized changes over time. In each regression model, baseline physical functioning and other covariates were entered as a block in the first step, followed by the baseline psychological distress (depression, anxiety, or perceived stress) and its 6- or 12-month change over time in the next step. To examine whether social support was a direct predictor or a moderator (i.e., a buffer) of psychological distress on physical functioning, centered social support variable and its interaction with centered psychological distress variable (depression, anxiety, or perceived stress) were entered in regression model in the last step to predict physical functioning at the follow-ups.

Results

Participant Characteristics

Participants were 255 CHD patients, including 208 men and 47 women, with a mean age of 63.11 (SD = 8.65) and ranging from 36 to 89. The descriptive statistics of their demographic, medical, and psychosocial characteristics are presented in Table 1. The majority of participants were married or partnered (83.9%). Most of them completed high school (42.0%) or technical school (18.0%), and approximately one in three (30.0%) attained a bachelor or graduate degree. Nearly half of them (48.2%) were employed full-time or part-time. The ethnic background of the sample was mostly Chinese (85.1%), followed by Indian (9.0%), Malay (1.6%), or others (4.3%).

Table 1 Descriptive statistics of the participants' demographic and medical characteristics

Participant characteristics	Baseline (<i>N</i> = 255)	6-month follow-up (<i>n</i> = 185)	Dropouts at 6 months (<i>n</i> = 70)	Test of difference at 6 months ^a	12-month follow-up (<i>n</i> = 152)	Dropouts at 12 months (<i>n</i> = 103)	Test of difference at 12 months ^a
Demographic background							
Age	63.11 (8.65)	63.10 (8.58)	63.13 (8.89)	<i>p</i> = 0.980	64.03 (8.57)	61.75 (8.63)	<i>p</i> = 0.039
Married or partnered	214 (83.9%)	154 (83.2%)	60 (85.7%)	<i>p</i> = 0.706	125 (82.2%)	89 (86.4%)	<i>p</i> = 0.392
Ethnicity				<i>p</i> = 0.498			<i>p</i> = 0.066
Chinese	217 (85.1%)	161 (87.0%)	56 (80.0%)		134 (88.2%)	83 (80.6%)	
Malay	4 (1.6%)	2 (1.1%)	2 (2.9%)		1 (0.7%)	3 (2.9%)	
Indian	23 (9.0%)	15 (8.1%)	8 (11.4%)		14 (9.2%)	9 (8.7%)	
Other	11 (4.3%)	7 (3.8%)	4 (5.7%)		3 (2.0%)	8 (7.8%)	
Education				<i>p</i> = 0.347			<i>p</i> = 0.034
< High school	26 (10.2%)	15 (8.1%)	11 (15.7%)		9 (5.9%)	17 (16.5%)	
High school	107 (42.0%)	83 (44.9%)	24 (34.3%)		71 (46.7%)	36 (35.0%)	
Diploma (Polytechnic)	46 (18.0%)	32 (17.3%)	14 (20.0%)		29 (19.1%)	17 (16.5%)	
Bachelor's degree	38 (14.9%)	27 (14.6%)	11 (15.7%)		19 (12.5%)	19 (18.4%)	
Postgraduate degree	36 (14.1%)	26 (14.1%)	10 (14.3%)		22 (14.5%)	14 (13.6%)	
Employed	123 (48.2%)	86 (46.5%)	37 (52.9%)	<i>p</i> = 0.402	66 (43.4%)	57 (55.3%)	<i>p</i> = 0.075
Months at Heart Wellness Center	24.78 (34.00)	25.46 (35.17)	22.99 (30.89)	<i>p</i> = 0.605	27.30 (35.55)	21.07 (31.38)	<i>p</i> = 0.152
Cardiovascular risk factors							
Waist circumference (cm)	89.22 (10.26)	88.32 (10.98)	91.59 (7.59)	<i>p</i> = 0.024	88.23 (11.49)	90.64 (8.01)	<i>p</i> = 0.067
BMI (kg/m ²)	24.43 (3.28)	24.12 (3.18)	25.24 (3.45)	<i>p</i> = 0.015	24.20 (3.18)	24.76 (3.42)	<i>p</i> = 0.186
Type 2 diabetes	46 (18.0%)	29 (15.7%)	17 (24.3%)	<i>p</i> = 0.143	21 (13.8%)	25 (24.3%)	<i>p</i> = 0.046
Hypertension	159 (62.4%)	112 (60.5%)	47 (67.1%)	<i>p</i> = 0.310	96 (63.2%)	63 (61.2%)	<i>p</i> = 0.792
Dyslipidemia	205 (80.4%)	146 (78.9%)	59 (84.3%)	<i>p</i> = 0.477	122 (80.3%)	83 (80.6%)	<i>p</i> = 0.872
Current smoking	6 (2.4%)	4 (2.2%)	2 (2.9%)	<i>p</i> = 1.000	5 (3.3%)	1 (1.0%)	<i>p</i> = 0.405
Medical and health characteristics							
Myocardial infarction	71 (27.8%)	45 (24.3%)	26 (37.1%)	<i>p</i> = 0.042	37 (24.3%)	34 (33.0%)	<i>p</i> = 0.117
Angina pectoris	43 (16.9%)	32 (17.3%)	11 (15.7%)	<i>p</i> = 0.853	29 (19.1%)	14 (13.6%)	<i>p</i> = 0.307
Coronary angioplasty and stents	159 (62.4%)	112 (60.5%)	47 (67.1%)	<i>p</i> = 0.386	90 (59.2%)	69 (67.0%)	<i>p</i> = 0.237
Coronary artery bypass grafting	81 (31.8%)	64 (34.6%)	17 (24.3%)	<i>p</i> = 0.172	55 (36.2%)	26 (25.2%)	<i>p</i> = 0.099
Valve replacement or repair	21 (8.2%)	18 (9.7%)	3 (4.3%)	<i>p</i> = 0.299	15 (9.9%)	6 (5.8%)	<i>p</i> = 0.353
Mental health treatment				<i>p</i> = 1.000			<i>p</i> = 1.000
Depression treatment	3 (1.2%)	2 (1.1%)	1 (1.4%)		2 (1.3%)	1 (1.0%)	

Table 1 (continued)

Participant characteristics	Baseline (<i>N</i> = 255)	6-month follow-up (<i>n</i> = 185)	Dropouts at 6 months (<i>n</i> = 70)	Test of difference at 6 months ^a	12-month follow-up (<i>n</i> = 152)	Dropouts at 12 months (<i>n</i> = 103)	Test of difference at 12 months ^a
Anxiety treatment	4 (1.6%)	3 (1.6%)	1 (1.4%)	<i>p</i> = 1.000	3 (2.0%)	1 (1.0%)	<i>p</i> = 0.650
Physical functioning ^b							
Physical functioning at baseline	5.78 (0.79)	5.85 (0.77)	5.60 (0.84)	<i>p</i> = 0.024	5.80 (0.73)	5.76 (0.88)	<i>p</i> = 0.704
Physical functioning at 6 months		5.87 (0.82)			5.88 (0.79)		
Physical functioning at 12 months					5.82 (0.79)		
Psychological characteristics							
Depression (without somatic items) ^c							
Depression at baseline	9.22 (9.45)	9.16 (8.77)	9.39 (11.10)	<i>p</i> = 0.862	9.19 (8.90)	9.28 (10.24)	<i>p</i> = 0.937
Depression at 6 months		7.95 (8.86)			7.81 (8.81)		
Depression at 12 months					6.70 (8.59)		
Depression (full scale) ^c							
Depression at baseline	9.67 (8.78)	9.65 (7.98)	9.70 (10.68)	<i>p</i> = 0.966	9.56 (7.87)	9.83 (10.01)	<i>p</i> = 0.809
Depression at 6 months		8.90 (8.45)			8.67 (8.57)		
Depression at 12 months					7.98 (8.66)		
Anxiety at baseline ^d	3.04 (2.96)	2.97 (2.69)	3.21 (3.58)	<i>p</i> = 0.553	3.06 (2.70)	3.00 (3.31)	<i>p</i> = 0.876
Anxiety at 6 months ^d		3.02 (3.25)			2.86 (3.01)		
Anxiety at 12 months ^d					2.91 (3.01)		
Perceived stress at baseline ^e	2.35 (0.59)	2.34 (0.60)	2.39 (0.56)	<i>p</i> = 0.534	2.34 (0.59)	2.37 (0.59)	<i>p</i> = 0.701
Perceived stress at 6 months ^e		2.23 (0.64)			2.23 (0.63)		
Perceived stress at 12 months ^e					2.17 (0.64)		
Social support ^f	3.54 (0.94)	3.58 (0.92)	3.43 (0.98)	<i>p</i> = 0.294	3.54 (0.91)	3.53 (0.98)	<i>p</i> = 0.926

^aCategory variables were tested by Chi-square (χ^2) test, and continuous variables were tested by *t* test^bMeasured by the MacNew Heart Disease Health-Related Quality of Life Questionnaire (MacNew)^cMeasured by the Center for Epidemiologic Studies Depression (CES-D)^dMeasured by the anxiety subscale of the Hospital Anxiety and Depression Scale (HADS)^eMeasured by the Perceived Stress Scale (PSS)^fMeasured by the Medical Outcome Study Social Support Questionnaire (MOS-SS)

Among participants, 27.8% reported a history of myocardial infarction, and 16.9% experienced angina pectoris. Almost two-thirds (62.4%) were treated with percutaneous transluminal coronary angioplasty and stent implants, and about one-third (31.8%) underwent coronary artery bypass grafting surgery. For comorbid conditions, 62.4% were diagnosed with hypertension, 80.4% with dyslipidemia, and 18.0% with diabetes. The participants' average body mass index (BMI) was 24.43 (SD = 3.28) kg/m², with a mean waist circumference of 89.22 (SD = 10.26) cm.

Seventy participants did not return for follow-up at 6 months, and 103 did not respond at 12 months. As shown in Table 1, compared with the participants available at 6 months, those who did not return as a group had a larger waist circumference (91.59 cm vs. 88.32 cm; $t = 2.28$, $p = 0.024$), higher BMI (25.24 vs. 24.12 kg/m²; $t = 2.46$, $p = 0.015$), slightly lower physical functioning score (5.60 vs. 5.84; $t = 2.28$, $p = 0.024$), and more likely to have a history of myocardial infarction (37.1% vs. 24.3%; $\chi^2 [1] = 4.35$, $p = 0.042$). At 12 months, those who did not return were more

Table 2 Regression analyses of psychological factors predicting physical functioning at 6 and 12 months

Psychological predictors	Prediction of 6-month health functioning ^a				Prediction of 12-month physical functioning ^a			
Depression								
Model 1 ^b	<i>B</i>	SE	β	<i>p</i>	<i>B</i>	SE	β	<i>p</i>
Baseline depression	-0.02	0.01	-0.19	0.007	-0.03	0.01	-0.32	<0.001
Change in depression	-0.02	0.01	-0.21	0.001	-0.02	0.01	-0.17	0.020
	Model $R^2 = 0.567$				Model $R^2 = 0.499$			
Model 2 ^c	<i>B</i>	SE	β	<i>p</i>	<i>B</i>	SE	β	<i>p</i>
Baseline depression	-0.14	0.07	-0.18	0.031	-0.23	0.07	-0.28	0.002
Change in depression	-0.17	0.05	-0.20	0.001	-0.13	0.06	-0.16	0.023
Baseline social support	0.03	0.05	0.03	0.566	0.05	0.05	0.06	0.393
Social support \times depression interaction	<0.01	0.04	<0.01	0.986	0.02	0.05	0.02	0.765
	Model $R^2 = 0.568$				Model $R^2 = 0.502$			
Anxiety								
Model 1 ^b	<i>B</i>	SE	β	<i>p</i>	<i>B</i>	SE	β	<i>p</i>
Baseline anxiety	-0.07	0.02	-0.24	<0.001	-0.05	0.02	-0.20	0.008
Change in anxiety	-0.10	0.02	-0.29	<0.001	-0.09	0.02	-0.28	<0.001
	Model $R^2 = 0.633$				Model $R^2 = 0.515$			
Model 2 ^c	<i>B</i>	SE	β	<i>p</i>	<i>B</i>	SE	β	<i>p</i>
Baseline anxiety	-0.21	0.05	-0.26	<0.001	-0.15	0.06	-0.19	0.013
Change in anxiety	-0.25	0.04	-0.31	<0.001	-0.22	0.05	-0.28	<0.001
Baseline social support	-0.03	0.04	-0.03	0.531	0.07	0.05	0.08	0.195
Social support \times anxiety interaction	-0.05	0.04	-0.07	0.165	-0.05	0.04	-0.07	0.268
	Model $R^2 = 0.638$				Model $R^2 = 0.525$			
Perceived stress								
Model 1 ^b	<i>B</i>	SE	β	<i>p</i>	<i>B</i>	SE	β	<i>p</i>
Baseline perceived stress	-0.41	0.09	-0.29	<0.001	-0.32	0.12	-0.24	0.006
Change in perceived stress	-0.53	0.08	-0.38	<0.001	-0.50	0.10	-0.35	<0.001
	Model $R^2 = 0.639$				Model $R^2 = 0.530$			
Model 2 ^c	<i>B</i>	SE	β	<i>p</i>	<i>B</i>	SE	β	<i>p</i>
Baseline perceived stress	-0.23	0.06	-0.28	<0.001	-0.16	0.07	-0.21	0.015
Change in perceived stress	-0.31	0.05	-0.38	<0.001	-0.30	0.05	-0.38	<0.001
Baseline social support	0.05	0.04	0.06	0.223	0.15	0.05	0.19	0.003
Social support \times perceived stress interaction	0.01	0.04	0.01	0.770	0.05	0.04	0.07	0.226
	Model $R^2 = 0.642$				Model $R^2 = 0.562$			

^aModels adjusted for age, gender, marital status, education, BMI, months in wellness center, and baseline physical functioning

^bModel 1 predictors include psychological distress (depression, anxiety, or perceived stress), change in psychological distress, and covariates

^cModel 2 further adds social support and its interaction with psychological distress as predictors

likely to be somewhat younger (61.75 vs. 64.03; $t = -2.08$, $p = 0.039$), have lower education levels (16.5% vs. 5.9%; $\chi^2 [4] = 10.45$, $p = 0.034$), and be more likely to have type 2 diabetes mellitus (24.3% vs. 13.8%; $\chi^2 [1] = 4.54$, $p = 0.046$). There were no differences in any psychosocial measures between the two groups. Although no variables showed consistent trends in dropouts at both 6 and 12 months, the overall attrition pattern suggested that the dropouts might have a somewhat lower health status or at higher risk.

Depression and Anxiety Symptom Severity

On the basis of suggested cut-off criteria in prior research of the CES-D [48], 78% of the participants showed no depression (scores < 16), 17% reported mild symptoms (scores between 16 and 26), and 5% endorsed moderate to severe symptoms (scores > 26) at the baseline. At 6 months, 81% reported no depression, 14% mild symptoms, and 5% moderate to severe depression. At 12 months, 86% showed no depression, 10% mild symptoms, and 4% moderate to severe symptoms. For anxiety, based on the suggested guideline of the HADS [45], 92% of the participants showed little or no symptoms (scores < 8), 4% moderate anxiety (scores between 8 and 10), and 4% severe anxiety (scores > 10) at the baseline. At 6 months, 90% reported little or no symptoms, 5% moderate anxiety, and 5% severe anxiety. At 12 months, 91% reported little or no anxiety symptoms, 5% showed moderate anxiety, and 5% severe anxiety.

Cross-Sectional Associations Between Psychological Factors and Physical Functioning

Concurrently at the baseline, higher depression ($\beta = -0.45$, $p < 0.001$), anxiety ($\beta = -0.48$, $p < 0.001$), and perceived stress ($\beta = -0.57$, $p < 0.001$) were all significantly associated with poorer physical functioning in separate regression models, after adjusting for demographic and medical covariates.

Psychological Distress and Its Change in Prediction of Physical Functioning at 6 Months

Table 2 presents the prediction of physical functioning at 6 months by each psychological distress variable and its change. For depression, both the baseline ($\beta = -0.19$, $p = 0.007$) and its increase over 6 months ($\beta = -0.21$, $p = 0.001$) significantly predicted worse decline in physical functioning at the follow-up, while controlling for baseline physical functioning and other covariates. The findings for anxiety and stress demonstrated a similar pattern. Both baseline anxiety ($\beta = -0.24$, $p < 0.001$) and its 6-month increase ($\beta = -0.29$, $p < 0.001$) predicted greater decline in physical functioning at 6 months. Baseline perceived stress ($\beta = -0.29$, $p < 0.001$)

and its increase ($\beta = -0.38$, $p < 0.001$) simultaneously predicted more decline in physical functioning over 6 months. Among covariates, baseline physical functioning ($\beta = 0.57-0.64$, $ps < 0.001$) consistently predicted 6-month physical functioning in all models.

Psychological Distress and Its Change in Prediction of Physical Functioning at 12 Months

The results at 12 months demonstrated similar findings (Table 2). In the model with depression, both the baseline ($\beta = -0.32$, $p < 0.001$) and its increase ($\beta = -0.17$, $p = 0.020$) significantly predicted worse decline in physical functioning at 12 months, after adjusting for baseline physical functioning and covariates. For anxiety, both its baseline ($\beta = -0.20$, $p = 0.008$) and its increase ($\beta = -0.28$, $p < 0.001$) significantly predicted greater decrease in physical functioning at 12 months. Finally, both the baseline perceived stress ($\beta = -0.24$, $p = 0.006$) and its increase over 12 months ($\beta = -0.35$, $p < 0.001$) were significant predictors of greater decline in follow-up physical functioning. Among covariates, baseline physical functioning ($\beta = 0.57-0.65$, $ps < 0.001$) consistently predicted 12-month physical functioning in all models.

Full-Scale Depression in Prediction of Physical Functioning

We tested whether using the full-scale depression measure would affect the results. Both baseline depression ($\beta = -0.27$, $p < 0.001$ at 6 months; $\beta = -0.31$, $p < 0.001$ at 12 months) and its increase over time ($\beta = -0.27$, $p < 0.001$ at 6 months; $\beta = -0.22$, $p = 0.001$, at 12 months) significantly predicted greater decline in physical functioning at each follow-up, while adjusting for covariates. In sum, when full-scale depression measure with somatic items was used, the findings remained similar.

Tests of Social Support as a Moderator of Psychological Distress

Cross-sectionally, social support was significantly associated with patients' physical functioning ($\beta = 0.13$, $p = 0.043$) at the baseline, after adjusting for covariates. Prospectively, baseline social support predicted greater increase in physical functioning at 12 months ($\beta = 0.13$, $p = 0.050$) but not at 6 months ($\beta = 0.07$, $p = 0.224$), after adjusting for baseline physical functioning and other covariates.

To test whether social support buffered the negative influence of psychological distress, its interaction with depression, anxiety, or perceived stress was examined (Table 2). At the baseline, there was no interaction between social support and any psychological distress variable ($Bs = 0.02$ to 0.05 , $ps =$

0.190 to 0.754) in the prediction of concurrent physical functioning. In models to predict physical functioning at 6 or 12 months, no interactions were found between social support and any distress variables ($B_s = -0.05$ to 0.05 , $p_s = 0.165$ to 0.986). In sum, social support predicted improved physical functioning at 12 months, but it did not buffer the impact of psychological distress on physical functioning over time.

Discussion

This study examined the influences of depression, anxiety, and perceived stress, as well as their changes, on physical functioning over 12 months among CHD patients. The results demonstrated that, in addition to baseline distress, the increase in depression, anxiety, or perceived stress also predicted further decline in physical functioning over 12 months. In addition, social support was found to predict improved physical functioning at 12 months, but it did not buffer the negative impact of psychological distress.

With mounting evidence, depression has been recognized for its prevalence and detrimental impact on CHD patients in recovery [12]. The American Heart Association, with the endorsement from American Psychiatric Association, has issued scientific statements declaring depression as a risk factor for poor prognosis of CHD patients [49] and provided practice guidelines for screening and treatment of depression in patients [8]. This study, while affirming past findings and recommended practices, further demonstrated that other common forms of psychological distress, especially anxiety and perceived stress, also warrant clinical attention. Although depression, anxiety, and perceived stress were fairly highly correlated ($r_s = 0.52$ to 0.62), they did not overlap with one another completely. Focusing exclusively on depression could potentially risk overlooking patients who experience aggravating emotional distress expressed in other manners. In contrast with depression, anxiety can manifest as excessive fear, worry, heightened alert, and phobic reactions, and perceived stress is marked by feelings of being overwhelmed and unable to control one's situation [46]. As observed in this study, anxiety and perceived stress, compared with depression, were no less important in the prediction of physical health declines over 12 months. Psychological distress, regardless of its origins or manifestations, appears disruptive and harmful to CHD patients as they strive to regain their health and maintain physical functioning. Finally, although each of the different manifestations of psychological distress, namely depression, anxiety, and perceived stress, predicted greater decline in physical functioning, the findings did not imply that they were distinctive from one another. Different forms of psychological distress may reflect common underlying emotional difficulties when individuals perceive environmental demands as threatening or overwhelming. Future research with more

comprehensive assessments of various forms of distress may illuminate whether specific elements of distress measures, in addition to the common component, would predict physical health decline independently.

In addition to the initial distress levels, exacerbation of psychological disturbances over time, in depression, anxiety, or perceived stress, independently predicted further decline in physical functioning over 12 months. Although the patients' mean scores of physical health functioning and psychological distress, as a group, appeared similar over time, their individuals scores did fluctuate in different magnitudes and directions. As the results showed, the individual differences in patients' baseline psychological distress and its increases predicted greater decline in physical health functioning over time. This deleterious impact of increased distress was above and beyond the influence of baseline distress levels. In other words, patients who experienced worsening of depression, anxiety, or perceived stress over time were vulnerable for even more severe impairments in physical health. The findings underscore the importance of not just assessing CHD patients' distress levels at treatment entry but also tracking their emotional status over time at follow-ups, considering that the increase in distress may contribute to additional health deterioration.

It is noted that the scores of depression and anxiety reported in this study appeared somewhat lower than those in other studies of heart patients [45, 48, 50–52]. It might be due to that the patients were recruited from a community-based heart wellness center where the attendees' illness was likely to be more stable or better managed, whereas patients in prior studies had recent surgeries or more severe pathology [53, 54]. Nevertheless, another plausible explanation is that the CES-D and HADS cutoff scores established in prior research may not apply directly to individuals from a different culture or society. It has been reported that Asian patients appeared more reluctant to acknowledge psychological symptoms and tended to under-report the severity because of perceived stigma of mental illness [55, 56]. Despite the issues regarding cut-off scores, the distress measures in this study were sensitive to individual differences and were robust predictors of health decline over time.

Several psychophysiological and behavioral mechanisms are speculated to explain how psychological distress may lead to health declines in CHD patients. Elevated stress, anxiety, and depression have been associated with disturbances in physiological stress responses orchestrated by the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic-adrenal-medullary (SAM) system [57]. Disturbed stress responses in turn may trigger a cascade of cardiac pathogenic processes, including elevated chronic inflammation, insulin resistance, lipid dysregulation, and endothelial dysfunction, thus leading to the exacerbation of atherosclerosis and CHD progression [58]. In terms of behavioral pathways, elevated

psychological distress is associated with a slew of health-compromising behaviors giving rise to disease exacerbation [59]. For example, depression is marked by loss of interest and diminished motivation, which may lead to non-adherence to medication and other treatment recommendations, such as healthy eating and regular exercise [60, 61]. In addition, excessive anxiety and uncontrollable stress can interfere with the planning and performance of health behaviors required for successful disease management, and they may also weaken patients' resistance to external temptations and relapses back to unhealthy practices [62]. In sum, these potential psychophysiological and behavioral mechanisms may be pathways by which psychological distress can aggravate physical symptoms and hinder health maintenance in CHD patients. Future research with a biopsychosocial approach and longitudinal design is greatly desired to illuminate these pathways between psychological distress, health cognitions, health behaviors, and disease processes in CHD patients.

Both the full-scale CES-D and the version without somatic items showed similar results that both baseline depression and its change predicted worse declines in patients' physical functioning. The results demonstrated that the effect of depression on reduced physical functioning was not driven by the somatic symptoms confounded with cardiac symptoms. The findings suggest that even when the depression measure contains somatic items, it is still a valid tool for identifying patients at risk for more rapid health declines. When applying scoring criteria to identify potential cases for clinical intervention, clinicians are recommended to be aware of the possibility that somatic items in depression measure might inflate the scores. Clinicians may compare the full scale scores and the adjusted ones without somatic items to determine whether these items cause score inflation.

Social support predicted greater improvement of physical functioning at 12 months, although not at 6 months. The results suggested that it might take some time for social support to manifest its cumulative positive influence on health. The findings were largely consistent with prior research demonstrating the salutary influence of social support on clinical outcomes in CHD patients [15, 29, 36, 37, 40]. However, this study did not find that social support buffered the negative influence of psychological distress on physical functioning over time. This study primarily assessed functional social support as perceived availability of support in emotional, instrumental, informational, and social interaction domains. As shown in the literature, social support is a multi-faceted construct with various conceptualizations and measurements [63]. There might be specifically relevant aspects of social support not captured in this study that could be a distress moderator. For example, high marital quality or good family relations could be important supportive resources more effective in alleviating emotional distress. Moreover, it is worth noting that most studies of social support examined it as a direct predictor

of health in CHD patients, rather than a moderator of distress [36]. It may be premature to conclude whether or not social support is an effective distress buffer for health in CHD patients. Further research is needed for a more thorough investigation on various specific types of social support and their roles in health promotion among CHD patients.

The strengths of this study included a relatively sizable clinical sample, a longitudinal design, and comprehensive measures of psychological distress over time. In addition, the participants were of Asian descent, representing a generally under-studied population in clinical research. In contrast, there were limitations of this study that should be heeded, which also suggested directions for future research. The attrition analysis suggested that patients who were younger, had lower education, and were at greater health risk (e.g., higher BMI, a history of myocardial infarction, and comorbid diabetes) appeared more likely to drop out of the study. It is speculated that younger patients and those with lower education might lack financial resources to continue rehabilitation or feel compelled to return to work. It is likely that the patients who participated and remained in the study at follow-ups were healthier and more motivated in managing their health. These attrition biases in clinical study of patients may limit the generalizability of results and should be borne in mind when translating research findings into clinical practice. Moreover, the majority of participants were men, which reflected the patient composition of an outpatient cardiac wellness center. There has been much evidence showing that women with CHD endorsed higher stress and depression symptoms than did male patients [64, 65]. However, it is unclear how this disparity would affect the prediction of physical functioning over time among women. Considering that the sample comprised mostly men, the findings might not be directly generalized to female patients with CHD. Future studies may target diverse patients who are less likely to be included in clinical research. In addition, despite the longitudinal design, given the observational nature of the study, the findings could not determine causal relationships. As discussed earlier, there are a multitude of factors and pathways by which psychological distress may lead to worsening health outcomes in CHD patients. More comprehensive research with integrated perspectives is needed to elucidate how these biopsychosocial pathways interact and affect health maintenance in CHD patients.

As for clinical implications, this study suggests that, instead of viewing psychological distress as a homogenous and static characteristic, clinicians are urged to recognize its diverse manifestations and fluctuating nature. By monitoring CHD patients' psychological status continuously in treatment and during maintenance, health professionals would be more effective at identifying individuals at risk for rapid health declines and delivering timely intervention to prevent relapse and facilitate recovery.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments.

Informed Consent Informed consent was obtained from all individual participants included in the study.

References

- Global Health Estimates 2016. Deaths by cause, age, sex, by country and by region, 2000–2016. In: World Health Organization. Geneva. 2018. http://www.who.int/healthinfo/global_burden_disease/estimates/en/index1.html.
- Roth GA, Johnson C, Abajobir A, Abd-Allah F, Abera SF, Abyu G, et al. Global, regional, and national burden of cardiovascular diseases for 10 causes, 1990 to 2015. *J Am Coll Cardiol*. 2017;70(1):1–25. <https://doi.org/10.1016/j.jacc.2017.04.052>.
- Kohlmann S, Gierk B, Hummelgen M, Blankenberg S, Lowe B. Somatic symptoms in patients with coronary heart disease: prevalence, risk factors, and quality of life. *JAMA Intern Med*. 2013;173(15):1469–71; discussion 71. <https://doi.org/10.1001/jamainternmed.2013.6835>.
- Dickens C. Depression in people with coronary heart disease: prognostic significance and mechanisms. *Curr Cardiol Rep*. 2015;17(10):83. <https://doi.org/10.1007/s11886-015-0640-6>.
- Romanelli J, Fauerbach JA, Bush DE, Ziegelstein RC. The significance of depression in older patients after myocardial infarction. *J Am Geriatr Soc*. 2002;50(5):817–22.
- Dickens C, McGowan L, Percival C, et al. New onset depression following myocardial infarction predicts cardiac mortality. *Psychosom Med*. 2008;70(4):450–5.
- Strik JJ, Lousberg R, Cheriex EC, Honig A. One year cumulative incidence of depression following myocardial infarction and impact on cardiac outcome. *J Psychosom Res*. 2004;56(1):59–66.
- Lichtman JH, Bigger JT Jr, Blumenthal JA, et al. Depression and coronary heart disease: recommendations for screening, referral, and treatment: a science advisory from the American Heart Association Prevention Committee of the Council on Cardiovascular Nursing, Council on Clinical Cardiology, Council on Epidemiology and Prevention, and Interdisciplinary Council on Quality of Care and Outcomes Research: endorsed by the American Psychiatric Association. *Circulation*. 2008;118(17):1768–75. <https://doi.org/10.1161/CIRCULATIONAHA.108.190769>.
- Thombs BD, Bass EB, Ford DE, Stewart KJ, Tsilidis KK, Patel U, et al. Prevalence of depression in survivors of acute myocardial infarction. *J Gen Intern Med*. 2006;21(1):30–8.
- Tully PJ, Cosh SM, Baumeister H. The anxious heart in whose mind? A systematic review and meta-regression of factors associated with anxiety disorder diagnosis, treatment and morbidity risk in coronary heart disease. *J Psychosom Res*. 2014;77(6):439–48. <https://doi.org/10.1016/j.jpsychores.2014.10.001>.
- Otte C, McCaffery J, Ali S, Whooley MA. Association of a serotonin transporter polymorphism (5-HTTLPR) with depression, perceived stress, and norepinephrine in patients with coronary disease: the Heart and Soul Study. *Am J Psychiatry*. 2007;164(9):1379–84. <https://doi.org/10.1176/appi.ajp.2007.06101617>.
- Carney RM, Freedland KE. Depression and coronary heart disease. *Nat Rev Cardiol*. 2017;14(3):145–55. <https://doi.org/10.1038/nrcardio.2016.181>.
- Meijer A, Conradi HJ, Bos EH, Thombs BD, van Melle JP, de Jonge P. Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta-analysis of 25 years of research. *Gen Hosp Psychiatry*. 2011;33(3):203–16. <https://doi.org/10.1016/j.genhosppsy.2011.02.007>.
- Frasure-Smith N, Lesperance F, Talajic M. Depression and 18-month prognosis after myocardial infarction. *Circulation*. 1995;91(4):999–1005.
- Frasure-Smith N, Lesperance F, Talajic M. The impact of negative emotions on prognosis following myocardial infarction: is it more than depression? *Health Psychol*. 1995;14(5):388–98.
- Sirois BC, Burg MM. Negative emotion and coronary heart disease. A review. *Behav Modif*. 2003;27(1):83–102. <https://doi.org/10.1177/0145445502238695>.
- Frasure-Smith N, Lesperance F. Depression and anxiety as predictors of 2-year cardiac events in patients with stable coronary artery disease. *Arch Gen Psychiatry*. 2008;65(1):62–71. <https://doi.org/10.1001/archgenpsychiatry.2007.4>.
- Orth-Gomer K, Wamala SP, Horsten M, Schenck-Gustafsson K, Schneiderman N, Mittleman MA. Marital stress worsens prognosis in women with coronary heart disease: the Stockholm Female Coronary Risk Study. *Jama*. 2000;284(23):3008–14.
- Hofer S, Lim L, Guyatt G, Oldridge N. The MacNew heart disease health-related quality of life instrument: a summary. *Health Qual Life Outcomes*. 2004;2:3. <https://doi.org/10.1186/1477-7525-2-3>.
- Sajobi TT, Wang M, Awosoga O, Santana M, Southern D, Liang Z, et al. Trajectories of health-related quality of life in coronary artery disease. *Circ Cardiovasc Qual Outcomes*. 2018;11(3):e003661. <https://doi.org/10.1161/CIRCOUTCOMES.117.003661>.
- El-Baz N, Ondusova D, Studencan M, et al. Differences between Slovak and Dutch patients scheduled for coronary artery bypass graft surgery regarding clinical and psychosocial predictors of physical and mental health-related quality of life. *Eur J Cardiovasc Nurs*. 2017;1474515117747571.
- Shen BJ, Eisenberg SA, Maeda U, Farrell KA, Schwarz ER, Penedo FJ, et al. Depression and anxiety predict decline in physical health functioning in patients with heart failure. *Ann Behav Med*. 2011;41(3):373–82. <https://doi.org/10.1007/s12160-010-9251-z>.
- Dickens C, Cherrington A, McGowan L. Depression and health-related quality of life in people with coronary heart disease: a systematic review. *Eur J Cardiovasc Nurs*. 2012;11(3):265–75. <https://doi.org/10.1177/1474515111430928>.
- Palacios JE, Khondoker M, Achilla E, Tylee A, Hotopf M. A single, one-off measure of depression and anxiety predicts future symptoms, higher healthcare costs, and lower quality of life in coronary heart disease patients: analysis from a multi-wave, primary care cohort study. *PLoS One*. 2016;11(7):e0158163. <https://doi.org/10.1371/journal.pone.0158163>.
- Kim HS, Kim HK, Kang KO, Kim YS. Determinants of health-related quality of life among outpatients with acute coronary artery disease after percutaneous coronary intervention. *Jpn J Nurs Sci*. 2018.
- Gehi A, Mangano D, Pipkin S, Browner WS, Whooley MA. Depression and heart rate variability in patients with stable coronary heart disease: findings from the Heart and Soul Study. *Arch Gen Psychiatry*. 2005;62(6):661–6.
- Compstell L, Lorenzi S, Russo N, et al. Depressive symptoms, functional measures and long-term outcomes of high-risk ST-

- elevated myocardial infarction patients treated by primary angioplasty. *Intern Emerg Med.* 2017;12(1):31–43. <https://doi.org/10.1007/s11739-016-1504-9>.
28. Shen BJ, Maeda U. Psychosocial predictors of self-reported medical adherence in patients with heart failure over 6 months: an examination of the influences of depression, self-efficacy, social support, and their changes. *Ann Behav Med.* 2018;52(7):613–9. <https://doi.org/10.1093/abm/kay003>.
 29. Staniute M, Brozaitiene J, Bunevicius R. Effects of social support and stressful life events on health-related quality of life in coronary artery disease patients. *J Cardiovasc Nurs.* 2013;28(1):83–9. <https://doi.org/10.1097/JCN.0b013e318233e69d>.
 30. Blumenthal JA, Sherwood A, Babyak MA, Watkins LL, Waugh R, Georgiades A, et al. Effects of exercise and stress management training on markers of cardiovascular risk in patients with ischemic heart disease: a randomized controlled trial. *JAMA.* 2005;293(13):1626–34. <https://doi.org/10.1001/jama.293.13.1626>.
 31. Bradt J, Dileo C. Music for stress and anxiety reduction in coronary heart disease patients. *Cochrane Database Syst Rev* 2009(2):CD006577. doi:<https://doi.org/10.1002/14651858.CD006577.pub2>.
 32. Cho MY, Min ES, Hur MH, Lee MS. Effects of aromatherapy on the anxiety, vital signs, and sleep quality of percutaneous coronary intervention patients in intensive care units. *Evid Based Complement Alternat Med.* 2013;2013:381381. <https://doi.org/10.1155/2013/381381>.
 33. Wilcox ME, Freiheit EA, Farris P, Hogan DB, Patten SB, Anderson T, et al. Depressive symptoms and functional decline following coronary interventions in older patients with coronary artery disease: a prospective cohort study. *BMC Psychiatry.* 2016;16:277. <https://doi.org/10.1186/s12888-016-0986-3>.
 34. Sin NL, Yaffe K, Whooley MA. Depressive symptoms, cardiovascular disease severity, and functional status in older adults with coronary heart disease: the Heart and Soul study. *J Am Geriatr Soc.* 2015;63(1):8–15. <https://doi.org/10.1111/jgs.13188>.
 35. Kendel F, Gelbrich G, Wirtz M, Lehmkühl E, Knoll N, Hetzer R, et al. Predictive relationship between depression and physical functioning after coronary surgery. *Arch Intern Med.* 2010;170(19):1717–21. <https://doi.org/10.1001/archinternmed.2010.368>.
 36. Frasure-Smith N, Lesperance F, Gravel G, et al. Social support, depression, and mortality during the first year after myocardial infarction. *Circulation.* 2000;101(16):1919–24.
 37. Orsten M, Mittelman MA, Wamala SP, Schenck-Gustafsson K, Orth-Gomer K. Depressive symptoms and lack of social integration in relation to prognosis of CHD in middle-aged women. The Stockholm Female Coronary Risk Study. *Eur Heart J.* 2000;21(13):1072–80. <https://doi.org/10.1053/euhj.1999.2012>.
 38. Shen BJ, McCreary CP, Myers HF. Independent and mediated contributions of personality, coping, social support, and depressive symptoms to physical functioning outcome among patients in cardiac rehabilitation. *J Behav Med.* 2004;27(1):39–62.
 39. Pragodpol P, Ryan C. Critical review of factors predicting health-related quality of life in newly diagnosed coronary artery disease patients. *J Cardiovasc Nurs.* 2013;28(3):277–84. <https://doi.org/10.1097/JCN.0b013e31824af56e>.
 40. Lee DTF, Choi KC, Chair SY, Yu DSF, Lau ST. Psychological distress mediates the effects of socio-demographic and clinical characteristics on the physical health component of health-related quality of life in patients with coronary heart disease. *Eur J Prev Cardiol.* 2014;21(1):107–16.
 41. Hiller A, Helvik AS, Kaasa S, Slordahl SA. Psychometric properties of the Norwegian MacNew Heart Disease health-related quality of life inventory. *Eur J Cardiovasc Nurs.* 2010;9(3):146–52. <https://doi.org/10.1016/j.ejcnurse.2010.01.002>.
 42. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas.* 1977;1(3):385–401.
 43. Radloff LS, Locke BZ. The community mental health assessment survey and the CES-D scale. *Community surveys of psychiatric disorders*, vol. 4; 1986. p. 177–88.
 44. Knight RG, Williams S, McGee R, Ollman S. Psychometric properties of the Centre for Epidemiologic Studies Depression Scale (CES-D) in a sample of women in middle life. *Behav Res Ther.* 1997;35(4):373–80.
 45. Zigmond AS, Snaith RP. The hospital anxiety and depression scale. *Acta Psychiatr Scand.* 1983;67(6):361–70.
 46. Cohen S, Williamson G. Perceived stress in a probability sample of the U.S. In: Spacapan S, Oskamp S, editors. *The social psychology of health: Claremont symposium on applied social psychology*. Newbury Park: Sage; 1988. p. 31–67.
 47. Sherbourne CD, Stewart AL. The MOS social support survey. *Soc Sci Med.* 1991;32(6):705–14.
 48. Callahan CM, Wolinsky FD. The effect of gender and race on the measurement properties of the CES-D in older adults. *Med Care.* 1994;32(4):341–56.
 49. Lichtman JH, Froelicher ES, Blumenthal JA, Carney RM, Doering LV, Frasure-Smith N, et al. Depression as a risk factor for poor prognosis among patients with acute coronary syndrome: systematic review and recommendations: a scientific statement from the American Heart Association. *Circulation.* 2014;129(12):1350–69. <https://doi.org/10.1161/CIR.0000000000000019>.
 50. Wiernik E, Meneton P, Empana JP, Siemiatycki J, Hoertel N, Vulser H, et al. Cardiovascular risk goes up as your mood goes down: interaction of depression and socioeconomic status in determination of cardiovascular risk in the CONSTANCES cohort. *Int J Cardiol.* 2018;262:99–105. <https://doi.org/10.1016/j.ijcard.2018.02.033>.
 51. Mommersteeg PM, Pot I, Aarnoudse W, Denollet J, Widdershoven JW. Type D personality and patient-perceived health in nonsignificant coronary artery disease: the Tweesteden mild STenosis (TWIST) study. *Qual Life Res.* 2013;22(8):2041–50. <https://doi.org/10.1007/s1136-012-0340-2>.
 52. Barth J, Martin CR. Factor structure of the Hospital Anxiety and Depression Scale (HADS) in German coronary heart disease patients. *Health Qual Life Outcomes.* 2005;3:15. <https://doi.org/10.1186/1477-7525-3-15>.
 53. Benzer W, Hofer S, Oldridge NB. Health-related quality of life in patients with coronary artery disease after different treatments for angina in routine clinical practice. *Herz.* 2003;28(5):421–8. <https://doi.org/10.1007/s00059-003-2388-9>.
 54. Morys JM, Hofer S, Rynkiewicz A, Oldridge NB. The Polish MacNew heart disease health-related quality of life questionnaire: a validation study. *Cardiol J.* 2015;22(5):541–50. <https://doi.org/10.5603/CJ.a2015.0027>.
 55. Zhuang XY, Wong DFK, Cheng CW, Pan SM. Mental health literacy, stigma and perception of causation of mental illness among Chinese people in Taiwan. *Int J Soc Psychiatry.* 2017;63(6):498–507. <https://doi.org/10.1177/0020764017719303>.
 56. Xu Z, Huang F, Kusters M, Rusch N. Challenging mental health related stigma in China: systematic review and meta-analysis. II. Interventions among people with mental illness. *Psychiatry Res.* 2017;255:457–64. <https://doi.org/10.1016/j.psychres.2017.05.002>.
 57. Steptoe A, Kivimaki M. Stress and cardiovascular disease. *Nat Rev Cardiol.* 2012;9(6):360–70. <https://doi.org/10.1038/nrcardio.2012.45>.
 58. McEwen BS. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiol Rev.* 2007;87(3):873–904. <https://doi.org/10.1152/physrev.00041.2006>.
 59. Ng DM, Jeffery RW. Relationships between perceived stress and health behaviors in a sample of working adults. *Health Psychol.* 2003;22(6):638–42. <https://doi.org/10.1037/0278-6133.22.6.638>.

60. DiMatteo MR, Lepper HS, Croghan TW. Depression is a risk factor for noncompliance with medical treatment: meta-analysis of the effects of anxiety and depression on patient adherence. *Arch Intern Med.* 2000;160(14):2101–7.
61. Gehi A, Haas D, Pipkin S, Whooley MA. Depression and medication adherence in outpatients with coronary heart disease: findings from the Heart and Soul Study. *Arch Intern Med.* 2005;165(21):2508–13. <https://doi.org/10.1001/archinte.165.21.2508>.
62. Snyder HR. Major depressive disorder is associated with broad impairments on neuropsychological measures of executive function: a meta-analysis and review. *Psychol Bull.* 2013;139(1):81–132. <https://doi.org/10.1037/a0028727>.
63. House JS, Umberson D, Landis KR. Structures and processes of social support. *Annu Rev Sociol.* 1988;14(1):293–318.
64. Pilote L, Dasgupta K, Guru V, Humphries KH, McGrath J, Norris C, et al. A comprehensive view of sex-specific issues related to cardiovascular disease. *CMAJ.* 2007;176(6):S1–44. <https://doi.org/10.1503/cmaj.051455>.
65. Mallik S, Spertus JA, Reid KJ, Krumholz HM, Rumsfeld JS, Weintraub WS, et al. Depressive symptoms after acute myocardial infarction: evidence for highest rates in younger women. *Arch Intern Med.* 2006;166(8):876–83. <https://doi.org/10.1001/archinte.166.8.876>.

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