A Bifactor Model of the Beck Depression Inventory and Its Association With Medical Prognosis After Myocardial Infarction

de Miranda Azevedo, Ricardo; Roest, Annelieke M.; Carney, Robert M.; Denollet, Johan; Freedland, Kenneth E.; Grace, Sherry L.; Hosseini, Seyed H.; Lane, Deirdre A.; Parakh, Kapil; Pilote, Louise

Published in:
Health Psychology

DOI:
10.1037/hea0000316

Publication date:
2016

Health Psychology
2016, Vol. 35, No. 6, 614–624

0278-6133/16/$12.00 http://dx.doi.org/10.1037/hea0000316

A Bifactor Model of the Beck Depression Inventory and Its Association With Medical Prognosis After Myocardial Infarction

Ricardo de Miranda Azevedo
and Annelieke M. Roest
University of Groningen

Robert M. Carney
Washington University School of Medicine

Johan Denollet
Tilburg University

Kenneth E. Freedland
Washington University School of Medicine

Sherry L. Grace
York University and University Health Network, Toronto, Ontario, Canada

Seyed H. Hosseini
Mazandaran University of Medical Sciences and Imam Hospital, Sari, Iran

Deirdre A. Lane
Queen Elizabeth Hospital, Birmingham, West Midlands, United Kingdom

Kapil Parakh
John Hopkins Bayview Medical Center

Louise Pilote
McGill University Health Centre

Peter de Jonge
University of Groningen

Objectives: Evidence suggests that depression is associated with adverse outcomes in patients with myocardial infarction (MI). Some of the symptoms of depression may also be symptoms of somatic illness and these may confound the association between depression and prognosis. We investigated whether depression following MI is associated with medical prognosis independent of these somatic symptoms. ***Method:*** The database of an individual patient data meta-analysis was used. Endpoints were all-cause mortality and cardiovascular events. Nine studies were included. Bifactor factor analysis included 13,100 participants and 7,595 participants were included in survival models. Dimensions were generated from the Beck Depression Inventory using factor analyses. The prognostic association was assessed using mixed-effects Cox regression analysis. ***Results:*** A bifactor model, consisting of a general factor and 2 general depression-free subgroup factors (a somatic/affective and a cognitive/affective), provided the best fit. There was a significant association between the general depression factor and all-cause mortality (hazard ratio [*HR*] = 1.25; 95% confidence interval [CI] [1.17, 1.34], $p < .001$) and cardiovascular events (*HR* = 1.18; 95% CI [1.13, 1.23], $p < .001$). After adjustment for demographics, measures of cardiac disease severity, and health-related variables, the association between the general depression factor and all-cause mortality (*HR* = 1.14; 95% CI [1.04, 1.25], $p = .003$) and cardiovascular events (*HR* = 1.16; 95% CI [1.10, 1.23], $p = .014$) attenuated. Additionally, the general depression-free somatic/affective factor was significantly associated with the endpoints, while the general depression-free cognitive/affective was not. ***Conclusions:*** A general depression factor is associated with adverse medical prognosis following MI independent of somatic/affective symptoms that may be partly attributable to somatic illness.

This article was published Online First February 22, 2016.

Ricardo de Miranda Azevedo and Annelieke M. Roest, Interdisciplinary Center Psychopathology and Emotion Regulation (ICPE), Department of Psychiatry, University of Groningen, University Medical Center Groningen; Robert M. Carney, Department of Psychiatry, Washington University School of Medicine; Johan Denollet, CoRPS-Center of Research on Psychology in Somatic Diseases, Department of Medical and Clinical Psychology, Tilburg University; Kenneth E. Freedland, Department of Psychiatry, Washington University School of Medicine; Sherry L. Grace, Faculty of Health, York University, and University Health Network, Toronto, Ontario, Canada; Seyed H. Hosseini, Psychiatry and Behavioral Sciences Research Center, Addiction Institute, Mazandaran University of Medical Sciences, and Psychosomatic Department, Imam Hospital, Sari, Iran; Deirdre A. Lane, Department of Cardiology, Queen Elizabeth Hospital, Birmingham, West Midlands, United Kingdom; Kapil Parakh, School of Medicine and John Hopkins Bloomberg School of Public Health, John Hopkins Bayview Medical Center; Louise Pilote, Division of General Internal Medicine, McGill University Health Centre; Peter de Jonge, Interdisciplinary Center Psychopathology and Emotion Regulation (ICPE), Department of Psychiatry, University of Groningen, University Medical Center Groningen.

Correspondence concerning this article should be addressed to Ricardo de Miranda Azevedo, Interdisciplinary Center Psychopathology and Emotion Regulation (ICPE), Department of Psychiatry, University Medical Center Groningen, Hanzeplein 1, PO Box 30.001, 9700 RB Groningen, The Netherlands. E-mail: r.de.miranda.azevedo@umcg.nl

Keywords: bifactor model, medical prognosis, depression, myocardial infarction

Supplemental materials: http://dx.doi.org/10.1037/hea0000316.supp

Depression following acute myocardial infarction (MI) has been extensively investigated as a risk factor for adverse medical prognosis. A recent meta-analysis including 29 studies reported a significant association between depression and medical prognosis (1.6- to 2.7-fold) in a total of 16,889 MI patients (Meijer et al., 2011). Major depression is a pleomorphic disorder. Although several subtypes have been proposed based on the pattern and severity of symptoms, a recent review did not find compelling evidence to support these subtypes (van Loo, de Jonge, Romeijn, Kessler, & Schoevers, 2012). However, it has been hypothesized that depression in the context of heart disease is not the same as depression in the general population. Ormel and de Jonge suggested that depression in patients with heart disease may consist of a combination of two prototypical subtypes of depression: a cognitive/affective subtype, marked by neuroticism and stress sensitivity, and a somatic/affective subtype, marked by atherosclerosis and sickness behavior (Ormel & De Jonge, 2011).

Several studies have examined depressive symptom dimensions in patients with heart disease (de Miranda Azevedo, Roest, Hoen, & de Jonge, 2014). The same two predominant dimensions have been found: a somatic/affective dimension, which includes insomnia and fatigability, and a cognitive/affective dimension, which includes guilt and self-dislike (Roest et al., 2011). Several of these studies reported differential associations between these symptom dimensions and medical prognosis (de Miranda Azevedo et al., 2014). In a meta-analysis of these studies, only the somatic/affective symptoms of depression were associated with medical prognosis, with a 1-*SD* increase in the somatic/affective symptoms level being associated with a 32% increased risk of adverse cardiac outcomes (de Miranda Azevedo et al., 2014). However, there was significant heterogeneity between the studies, which may be the result of the inclusion of different patient groups, endpoints, depressive symptom measures, and covariates. In addition, the included studies used different techniques to extract symptom dimensions, which is another important limitation of this meta-analysis. Therefore, the question remains whether these factors truly reflect different symptom dimensions of depression and whether these factors are differentially related to medical outcomes.

The limitations of pooling effect estimates from different studies can be avoided by using data of a meta-analysis of individual patient data (IPD). IPD meta-analysis offers several advantages as compared to a regular meta-analysis, including standardization of the analyses (Riley, Lambert, & Abo-Zaid, 2010). Recently, our group compiled data in an IPD meta-analysis to investigate whether depression worsens prognosis in MI patients. This study reported an increased risk of 23% for all-cause mortality and an increased risk of 12% for cardiovascular events per standard deviation in depression score, after adjusting for measures of cardiac disease severity and other health-related variables (Meijer et al., 2013).

Some authors have proposed that symptom dimensions of depression are indicators of a general depression factor, and that these do not reflect distinct constructs (Carney & Freedland, 2012). Bifactor factor analysis techniques address this question by estimating factor scores of a general depression factor that is free of variance of symptoms unrelated to depression (e.g., somatic/affective symptoms reflecting severity of cardiac disease or other somatic comorbidities; Carney & Freedland, 2012). In an earlier study, a similar approach demonstrated a very good fit in a sample of patients with MI, and a general depression factor was associated with mortality after adjusting for the confounding effects of somatic symptoms unrelated to the general depression factor (Thombs, Ziegelstein, Beck, & Pilote, 2008). However, this study was conducted on a single Canadian sample.

The present study used data of multiple studies from multiple countries and the aims are as follows:

- To evaluate whether the Beck Depression Inventory (BDI) fits a bifactor structure consisting of a general depression factor, a general depression-free cognitive/affective factor and general depression-free somatic/affective factor in patients with MI
- To investigate whether the general depression factor is associated with adverse medical prognosis independent of general depression-free cognitive/affective and general depression-free somatic/affective symptoms
- To investigate whether the general depression-free cognitive/affective and the general depression-free somatic/affective factors are associated with adverse medical prognosis.

Method

Study Selection

Data previously collected for an IPD meta-analysis was used. Studies were found through a systematic search of the literature conducted in Medline, EMBASE and PsycINFO from 1975 until January 5, 2011. Any prognostic or intervention study that assessed the association between depressive symptoms and medical prognosis (all-cause mortality and recurrent cardiovascular events) in post-MI patients was eligible for inclusion. Details of this search are available elsewhere (Meijer et al., 2013).

Depressive Symptoms

Only studies that used the BDI to assess depressive symptoms were included. Studies using the BDI-II were not included. The BDI was used because the majority of studies in the database of the IPD meta-analysis used this questionnaire (Meijer et al., 2013). The BDI is a 21-item self-report measure that assesses the presence and severity of symptoms of depression. The items are assessed in a 4-point Likert scale, with sum scores ranging from 0 to 63 (Beck, Steer, & Carbin, 1988).

From the 16 studies used in the previous meta-analysis of IPD, 10 studies used the BDI and nine provided the original data

necessary for the present analyses (Berkman et al., 2003; Denollet, Martens, Smith, & Burg, 2010; Grace et al., 2005; Hosseini, Ghaemian, Mehdizadeh, & Ashraf, 2014; Lane, Carroll, Ring, Beevers, & Lip, 2001; Lauzon et al., 2003; Parakh, Thombs, Fauerbach, Bush, & Ziegelstein, 2008; Spijkerman et al., 2005; van Melle et al., 2007). The sample used for deriving the symptom dimensions was larger than the one used in the survival models, as there were more data available on depressive symptoms than for other variables. For the bifactor factor analysis in the current study, all available BDI data was used. The Enhancing Recovery in Coronary Heart Disease (ENRICHD) study sample was larger in our study than reported elsewhere (ENRICHD Investigators, 2001). The aim of ENRICHD was to evaluate depression treatment efficacy in patients with heart disease. In ENRICHD, patients scoring below 10 on the BDI were excluded from the trial and these data were not used in other reports of the ENRICHD trial (ENRICHD Investigators, 2001). A total of 8,086 participants were initially screened for depression in ENRICHD and were included in the factor analytical models of the present study. A total of 5,238 (65%) participants were further excluded from the ENRICHD trial and 2,848 participants were included in the survival models of the present study.

For some of the participants, some item scores were missing. When fewer than six of the depression items were missing, item scores were imputed with the average of the nonmissing items for that participant, and the imputed values were rounded to the nearest whole number. When a participant had six or more missing items, the participant was excluded from further analyses. This procedure has been chosen because a substantial part of the data was already imputed this way (van Melle et al., 2007).

Prognostic Endpoints

The primary outcome in the present study was all-cause mortality. The secondary outcome was cardiovascular events, represented by both fatal and nonfatal events (cardiac death, recurrent MI, unstable angina, coronary artery bypass graft surgery).

Covariates

Three classes of covariates were used in the present study: demographics (age and sex), measures of cardiac disease severity (left-ventricular ejection fraction [LVEF], Killip class, and previous MI) and health-related risk factors (smoking status, diabetes and body mass index [BMI]). These covariates were selected because they were measured in most of the individual studies and are known to be associated with mortality among patients with MI (Lee et al., 1995; Mueller et al., 1992). LVEF was dichotomized into low ($<40\%$) or normal ($\geq 40\%$) because not every individual dataset contained values continuously measured. Killip class was dichotomized into heart failure (Class II to IV) or no heart failure (Class I), because not every study had the four-category score available (Meijer et al., 2013).

Statistical Analysis

Depressive symptom dimensions. The factor structure of the BDI was derived in a two-step procedure. First, to gain insight on the structure of the data, an exploratory factor analysis (EFA) for ordinal data with a promax rotation was conducted and a Schmid-Leiman transformation was applied. The Schmid-Leiman transformation is a bifactor estimation method that consists of a decomposition of the second-order models, providing a bifactor structure in an EFA framework (Schmid & Leiman, 1957). A minimum residual solution was used as the method to extract factors. Items with loadings $\geq .20$ were assigned to be part of a factor. Two EFA models were built, one restrained to two factors and the other to three factors, as previous studies hypothesized bifactor models with similar structures (Thombs et al., 2008; Brouwer, Meijer, & Zevalkink, 2013). The model with the best fit was chosen. Assessment of the scree plot was also used to determine the optimal number of factors.

The second step consisted of fitting a multiple indicators multiple causes (MIMIC) model (Jöreskog & Goldberger, 1975) including the bifactor structure produced by the EFA. The bifactor model simultaneously assumes a general (G) factor, covering all items of an instrument and other subgroup factors covering only subsets of items (Holzinger & Swineford, 1937; Reise, Moore, & Haviland, 2010; Reise, 2012). The general factor is uncorrelated with the subgroup factors, and the subgroup factors are uncorrelated among each other. The MIMIC model was used because it takes into account the nonindependence of different studies in our sample, by regressing the factors on dummy variables created for each study level. This way, the multilevel structure of the sample could also be taken into account when deriving factor scores.

The goodness of fit was assessed based on comparisons of three fit indices: the Comparative Fit Index (CFI), the Tucker-Lewis Index (TLI) and the Root Mean Square Error of Approximation (RMSEA), and standard cutoffs were used to assess the fit of the models (Browne & Cudeck, 1992; Hu & Bentler, 1999).

To check if the bifactor MIMIC Model A (G-S-C) fitted the present data better than other models, the fit indices were compared with four other models. These models were namely: Model B, a unidimensional model, composed of only one general factor (G); Model C, a correlated-traits model proposed by Beck and Steer, consisting of two dimensions (C-S), in which items 1–14 are labeled as cognitive and items 15–21 are labeled as somatic (Beck et al., 1988); Model D, a bifactor model consisting of a general depression factor composed of all items and a general depression-free somatic/affective factor composed of items 15,16,17,18 and 21, and Model E, a "higher order G-S-C" model, using the same items of Model A but modeled in a higher order format. The main difference between the bifactor and the higher order model is that the general factor in a higher order model is completely mediated by the lower order subgroup factors (i.e., general depression-free somatic/affective and general depression-free cognitive/affective factors). The general factor operates through the lower order factors and only indirectly influences the measured variables (Chen, West, & Sousa, 2006). In the bifactor models (Model A and Model D), the general factor directly influences each measured variable regardless of the influence produced by the subgroup factors.

To indicate the strength of the factors, we computed the explained common variance (ECV). The ECV is the common variance that is explained by a factor divided by the total common variance (Reise, Scheines, Widaman, & Haviland, 2013). An ECV equal or higher than 60% is suggested to indicate that the factor

loadings of the general factor in a bifactor model are close to the factor loadings of the general factor in a unidimensional model.

Mixed-effects Cox proportional hazards model. Mixed-effects Cox proportional hazards models were used to estimate risks for all-cause mortality and cardiovascular events. Unobserved between-study heterogeneity was taken into account by modeling study as a random intercept. A random slope was not included in the model due to low variation between studies.

Seven studies had information on time-to-event data and were included in the survival models (Berkman et al., 2003; Denollet et al., 2010; Lane et al., 2001; Lauzon et al., 2003; Parakh et al., 2008; Spijkerman et al., 2005; van Melle et al., 2007). In these studies, subjects were followed on average for 3.84 years for all-cause mortality, representing a total of 26,035 person-follow-up years. Subjects were followed on average for 2.72 years for cardiovascular events, representing a total of 16,796 person-follow-up years. Two studies did not have time-to-event data, and therefore were not included in these models (Grace et al., 2005; Hosseini et al., 2014).

Two of the included studies assessed if an intervention on decreasing symptoms of depression would improve prognosis (Berkman et al., 2003; van Melle et al., 2007). One study assessed if the effect of cognitive–behavioral therapy, or antidepressant therapy would improve medical prognosis (Berkman et al., 2003). The other assessed if different antidepressant therapies would help improving medical prognosis (van Melle et al., 2007). Depressive symptoms were assessed prior to the treatment in both studies. Only participants with high depression scores were included in these two studies, which could lead to selection bias. To see whether these differences in inclusion criteria would affect the main results, sensitivity analyses were conducted by removing these two intervention studies from the total sample.

All symptom dimensions were included as predictors in the survival models, which allowed us to adjust for the confounding effect of general depression-free cognitive/affective and general depression-free somatic/affective symptoms. To improve interpretability, factor scores were converted to z-scores. A separate model was fit for each of the covariates (previous MI, LVEF, Killip class, BMI, smoking and diabetes). Moreover, two fully adjusted models were fit: one including all heart-disease severity covariates, and another including all covariates. Adjustment for age and sex was performed in all models.

There was some variation with regard to which covariates were measured across individual studies. This led to a variation in the sample size across different models. All studies included demographic variables, history of previous MI, diabetes and smoking, but not every study included LVEF (Berkman et al., 2003; Denollet et al., 2010; Parakh et al., 2008; Spijkerman et al., 2005; van Melle et al., 2007). Therefore, predictive models including this variable had fewer cases than other models.

Mixed-effects logistic regression. All the nine studies had information on all-cause mortality or cardiac events, and therefore a series of mixed-effects logistic regression models were also conducted, including the additional two studies that did not have time-to-event data (Grace et al., 2005; Hosseini et al., 2014). These studies had information available on age, sex, smoking, diabetes and previous MI. Therefore, only models including these covariates were assessed. The same outcomes of the Cox models were used for these analyses.

Exploratory factor analyses were conducted using the *psych* package for R (R Development Core Team, 2008; Revelle, 2014). The MIMIC model was conducted using Mplus 7.0 (Muthén & Muthén, 1998–2012). Multivariable mixed-effects Cox proportional hazards models were conducted using Stata 12.0 (Statacorp, 2011).

Results

Sample Characteristics

The MIMIC model included 13,100 cases with data on depressive symptoms, originating from nine different studies. The studies were conducted in five different countries: the United States of America ($N = 2$), the Netherlands ($N = 3$), Canada ($N = 2$), United Kingdom ($N = 1$), and Iran ($N = 1$). The year of baseline assessment of the included studies ranged from 1995 to 2004. The overall mean sum score of the BDI was 8.4 (± 7.7), with a range from 0 to 59. Mean BDI sum scores of the individual studies ranged from to 5.7 (± 6.1) to 11.9 (± 9.8).

In the sample used to predict prognosis, a total of 7,595 patients were included, ranging from 280 to 2848 within studies. The majority of patients were male (69%) with proportions ranging from 57% to 81% across studies. The mean (*SD*) age of the aggregated sample was 60.9 (± 12.0) years and ranged from 58.2 (± 12.1) to 64.9 (± 12.1) years across studies. Table 1 displays the characteristics of individual studies and the combined sample.

Depressive Symptom Dimensions

The scree plot of the EFA reached a plateau after three points (see Supplemental Materials). These three factors had eigenvalues ≥ 1 (7.4, 1.4, and 1.0). The first factor was a general depression factor, including all the 21 items. In the second factor, namely general depression-free somatic/affective factor, somatic items were predominant: 4 (dissatisfaction), 13 (indecisiveness), 15 (work difficulty), 16 (insomnia), 17 (fatigability), 18 (loss of appetite), 20 (somatic preoccupation), and 21 (loss of libido). The third factor, namely general depression-free cognitive/affective factor, was composed of cognitive/affective items: 1 (sadness), 2 (hopelessness), 3 (sense of failure), 5 (guilt), 6 (punishment), 7 (self-dislike), 8 (self-accusations), 9 (suicidal ideas), and 14 (body-image change).

Table 2 shows the factor loadings of the MIMIC model based on the bifactor EFA (Model A). Table 3 displays the comparison of the goodness of fit between five different MIMIC models. Model A (G-S-C) shows the best fit of the data. Therefore, the factor scores from this solution were used in all survival models.

The ECV for the general depression factor was 79%. For the subgroup factors, an ECV of 9% and 11% was found for the general depression-free somatic/affective and for the general depression-free cognitive/affective factors, respectively.

Mixed-Effects Cox Proportional Hazards Models: General Depression Factor

The assumptions of proportionality of hazards were met for all covariates. Hazard ratios of the associations between symptom dimensions and the outcomes are displayed in Table 4.

Table 1
Characteristics of the Studies Included in the Prediction Models

First author, year	Country and start baseline assessment (year)	*N*	Age (mean, *SD*)	Male (%)	History of MI (%)	LVEF < 40%	Killip class > 1 (%)	Diabetes (%)	Smoking (%)	BMI (mean, *SD*)	Incidence of endpoint (%)	Mean follow-up time (years)
Berkman et al., 2003	United States, 1996	2,848	60.8 (12.3)	58	26	27	21	32	31	28.8 (5.7)	ACM: 12 CVE: 40	ACM: 2.3 CVE: 1.7
Spijkerman et al., 2005	The Netherlands, 1997	499	60.7 (11.7)	81	14	23	14	10	53	26.8 (4.0)	ACM: 22 CVE: 44	ACM: 7.3 CVE: 5.1
Denollet et al., 2010	The Netherlands, 2003	498	59.6 (11.6)	78	14	15	N/A	14	38	27.0 (3.9)	ACM: 8 CVE: 16	ACM: 3.8 CVE: 3.5
Lane et al., 2001	United Kingdom, 1997	288	62.7 (11.5)	75	22	N/A	52	12	43	N/A	ACM: 13	ACM: 2.7
Lauzon et al., 2003	Canada, 1996	552	60.2 (12.2)	79	21	N/A	13	16	40	26.8(4.4)	ACM: 6 CVE: 40	ACM: 1.0 CVE: .6
Parakh et al., 2008	United States, 1995	280	64.9 (12.1)	57	31	30	41	35	29	28.6 (6.1)	ACM: 54	ACM: 6.6
Grace et al., 2005	Canada, 1999	465	60.8 (12.1)	72	22	N/A	20	22	41	N/A	ACM: 6 CVE: 22	ACM: 1.0 CVE: 1.0
Hosseini et al., 2014	Iran, 2004	351	58.2 (12.0)	69	16	N/A	N/A	22	38	N/A	ACM: 14 CVE: 14	ACM: 2.0 CVE: 2.0
Van Melle et al., 2007	The Netherlands, 1999	1,814	61.0 (11.6)	78	13	25	10	12	48	26.5 (3.9)	ACM: 15 CVE: 44	ACM: 6.0 CVE: 4.1
Combined sample	Various	7,595	60.8 (12.0)	69	20	25	19	22	39	27.7 (5.0)	ACM: 14 CVE: 37	ACM: 3.8 CVE: 2.7

Note. ACM = all-cause mortality; BMI = body mass index; CVE = cardiovascular events; LVEF = left-ventricular ejection fraction; MI = myocardial infarction; N/A = not applicable.

In the least-adjusted (baseline) model, an increase of 1 *SD* in the general factor was associated with a 25% increased risk of all-cause mortality. Adjustment for previous MI decreased this association by 14%. Adjustment for LVEF decreased the association by 18% and for Killip class by 22%. Including diabetes in the model reduced the association by 7%, whereas adjustment for smoking and for BMI increased the association by 3%. The models including all cardiac disease severity markers together and the fully adjusted model showed an estimate 41% smaller than in the baseline model.

The risk of having cardiovascular events associated with an increase of 1 *SD* in the general factor was 18%. Adjustment for previous MI decreased the association by 10%. Adjustment for LVEF did not change the association, and for Killip class decreased by 15%. Including diabetes in the model reduced the association by 5%. Adjustment for smoking and BMI did not affect the association. The model with all cardiac disease severity markers showed an estimate 5% smaller than the baseline model. In the fully adjusted model the association has decreased by 10%.

Mixed-Effects Cox Proportional Hazards Models: General Depression-Free Cognitive/Affective and General Depression-Free Somatic/Affective Factors

The general depression-free cognitive/affective factor was not associated with any of the endpoints, with the exception of two subgroups (adjusting for history of MI and fully adjusted) predicting all-cause mortality. The general depression-free somatic/affective factor was associated with both outcomes across all models.

Mixed-Effects Logistic Regression

Four models adjusting for age, sex previous MI, diabetes and smoking were fit. Overall, similar results were found (see Table 5).

Mixed-Effects Cox Proportional Hazards Models: Sensitivity Analyses

Sensitivity analyses were conducted by removing the two intervention studies of the total sample (Berkman et al., 2003; van Melle et al., 2007). In general, the hazard ratios were smaller for the general depression factor in the models predicting all-cause mortality. The association between the general depression factor and all-cause mortality was not statistically significant in three models that included LVEF. For cardiovascular events results were similar to the main analyses. Nonetheless, in two models (adjusting for heart-disease severity markers and fully adjusted), a mixed-effects Cox model could not be computed because these variables were only available for three studies, and two of them were excluded in the sensitivity analyses, leaving data from only one study. Therefore, these models were excluded from the sensitivity analyses. Results are available on Supplemental Table 6.

Discussion

Main Findings

This is the first study using data of an IPD meta-analysis examining whether there is a significant association between a

Table 2
Standardized Factor Loadings of MIMIC Model A

Item	Description	General depression factor	General depression-free somatic/affective factor	General depression-free cognitive/affective factor
BDI 1	Sadness	.768		.062
BDI 2	Hopelessness	.781		.124
BDI 3	Sense of failure	.697		.421
BDI 4	Dissatisfaction	.774	.120	
BDI 5	Guilt	.619		.467
BDI 6	Punishment	.570		.441
BDI 7	Self-dislike	.668		.484
BDI 8	Self-accusations	.574		.479
BDI 9	Suicidal ideas	.643		.231
BDI 10	Crying	.684		
BDI 11	Irritability	.619		
BDI 12	Social withdrawal	.730		
BDI 13	Indecisiveness	.709	.143	
BDI 14	Body-image change	.629		.163
BDI 15	Work difficulty	.565	.542	
BDI 16	Insomnia	.546	.256	
BDI 17	Fatigability	.522	.630	
BDI 18	Loss of appetite	.458	.345	
BDI 19	Weight loss	.271		
BDI 20	Somatic preoccupation	.597	.130	
BDI 21	Loss of libido	.490	.247	

Note. MIMIC = multiple indicators multiple causes; BDI = Beck Depression Inventory.

general depression factor and adverse medical prognosis after taking into account general depression-free somatic/affective and general depression-free cognitive/affective symptoms. A bifactor model of the BDI, consisting of three factors fitted the data better than the competing models.

An increase of 1 *SD* in the general depression factor was associated with a 25% increase in the risk of all-cause mortality and an 18% greater risk of cardiovascular events. The association was attenuated but remained statistically significant after adjustment for cardiac disease severity and health-related markers for both outcomes. Although it has been suggested that smoking may account for poorer prognosis in depressed patients (Ormel & De Jonge, 2011), smoking did not decrease the strength of the association in the present study.

General depression-free somatic/affective symptoms were associated with both outcomes in all models. General depression-free cognitive/affective symptoms were only significantly associated with the outcomes in a model including previous MI and in the fully adjusted model predicting all-cause mortality. Results of the mixed-effects logistic regression indicated the same pattern of associations, where only the general depression factor and the general depression-free somatic/affective symptoms were associated with the outcomes.

Sensitivity analyses indicated that the risk of all-cause mortality is smaller after excluding participants coming from intervention studies. In the models including LVEF, the association between the general depression factor and all-cause mortality was not statistically significant. Statistical power may also play a role, because the sample size of the three models where the general depression factor was not significantly associated with all-cause mortality has decreased by 76%, 83% and 85% after removing these participants, respectively. On the other hand, it might be that LVEF explains the association between the general depression factor and all-cause mortality in participants scoring lower on total depressive symptom score. Nonetheless, general depression-free somatic/affective symptoms were associated with all-cause mortality across all models of the sensitivity analyses. In addition, results were comparable to the main analyses when predicting cardiovascular events. It is unlikely that treatment is responsible for these differences in results because, in both studies, treatment did not have a significant effect on the prognostic outcomes (Berkman et al., 2003; Zuidersma, Conradi, van Melle, Ormel, & de Jonge, 2013).

Table 3
Model Fit Information of the Confirmatory Factor Analyses

Model	CFI	TLI	RMSEA
Model A (G-S-C)	.975	.970	.025
Model B (G)	.835	.821	.052
Model C (S-C)	.877	.864	.045
Model D (G-S)	.957	.952	.032
Model E (higher order G-S-C)	.951	.944	.035

Note. CFI = comparative fit index; G = general unidimensional model; G-S-C = bifactor model; RMSEA = root mean square error of approximation; S-C = somatic/affective and cognitive/affective; TLI = Tucker-Lewis index.

Comparison With Previous Meta-Analyses of Depression After MI

Compared with the previous study using the IPD meta-analysis database, the present associations were weaker (Meijer et al., 2013). For all-cause mortality, all models showed weaker associations between depression and survival. Differences in effect estimates ranged from 18% (after adjustment for LVEF) to 37%

Table 4
Survival Models Assessing the Association Between Symptom Dimensions of Depression and Medical Prognosis

Model covariates	*HR* (95% CI): All-cause mortality	*N* patients (*K* studies)	*HR* (95% CI): Cardiovascular events	*N* patients (*K* studies)
Age, sex	G: 1.25 [1.17, 1.34]** S: 1.25 [1.17, 1.33]** C: 1.06 [.99, 1.13]	6,775 (7)	G: 1.18 [1.13, 1.23]** S: 1.12 [1.08, 1.17]** C: .96 [.92, 1.00]	6,169 (5)
Age, sex, previous MI	G: 1.21 [1.13, 1.30]** S: 1.23 [1.15, 1.32]** C: 1.07 [1.00, 1.15]*	6,691 (7)	G: 1.16 [1.11, 1.21]** S: 1.11 [1.06, 1.16]** C: .96 [.92, 1.00]	6,088 (5)
Age, sex, LVEF	G: 1.20 [1.11, 1.30]** S: 1.22 [1.13, 1.32]** C: 1.08 [.99, 1.17]	4,744 (5)	G: 1.18 [1.12, 1.24]** S: 1.14 [1.08, 1.20]** C: .97 [.92, 1.02]	4,439 (4)
Age, sex, Killip class	G: 1.19 [1.11, 1.28]** S: 1.23 [1.14, 1.31]** C: 1.06 [.99, 1.14]	5,923 (6)	G: 1.15 [1.10, 1.21]** S: 1.11 [1.06, 1.16]** C: .96 [.92, 1.01]	5,326 (4)
Age, sex, diabetes	G: 1.23 [1.15, 1.32]** S: 1.22 [1.14, 1.30]** C: 1.06 [.99, 1.14]	6,739 (7)	G: 1.17 [1.12, 1.22]** S: 1.11 [1.06, 1.16]** C: .96 [.92, 1.01]	6,135 (5)
Age, sex, smoking	G: 1.26 [1.17, 1.35]** S: 1.23 [1.15, 1.32]** C: 1.06 [.99, 1.13]	6,634 (7)	G: 1.18 [1.13, 1.23]** S: 1.12 [1.07, 1.17]** C: .96 [.92, 1.01]	6,030 (5)
Age, sex, BMI	G: 1.26 [1.17, 1.36]** S: 1.27 [1.18, 1.36]** C: 1.07 [.99, 1.15]	6,029 (6)	G: 1.18 [1.13, 1.23]** S: 1.12 [1.07, 1.18]** C: .96 [.92, 1.01]	5,752 (5)
Age, sex, previous MI, LVEF, Killip class	G: 1.14 [1.05, 1.24]* S: 1.23 [1.14, 1.33]** C: 1.08 [.99, 1.17]	4,162 (4)	G: 1.17 [1.11, 1.23]** S: 1.11 [1.05, 1.17]** C: .99 [.94, 1.04]	3,860 (3)
Age, sex, previous MI, LVEF, Killip class, diabetes, smoking, BMI	G: 1.14 [1.04, 1.25]* S: 1.20 [1.10, 1.30]** C: 1.11 [1.02, 1.21]*	3,896 (4)	G: 1.16 [1.10, 1.23]* S: 1.10 [1.04, 1.17]* C: 1.00 [.95, 1.05]	3,632 (3)

Note. *HR* = hazard ratio; CI = confidence interval; MI = myocardial infarction; LVEF = left-ventricular ejection fraction; BMI = body mass index; G = general depression factor; S = general depression-free somatic/affective factor; C = general depression-free cognitive/affective factor. The three factors were simultaneously included in all models.
* $p < .05$. ** $p < .001$.

(after adjustment for cardiac disease severity and health-related markers) for each *SD* increase. For cardiovascular events, the differences were less marked. Our estimates were also weaker in comparison with several other meta-analyses of aggregated data in patients with MI and one in patients with different types of coronary heart disease (Barth, Schumacher, & Herrmann-Lingen, 2004; Meijer et al., 2011; Nicholson, Kuper, & Hemingway, 2006; van Melle et al., 2004). A possible explanation for this discrepancy

Table 5
Mixed Effects Logistic Regression Models for All-Cause Mortality and Cardiovascular Events Adjusting for Age, Sex, Previous MI, Diabetes, and Smoking

Model covariates	*OR* (95% CI): All-cause mortality	*N* patients (*K* studies)	*OR* (95% CI): Cardiovascular events	*N* patients (*K* studies)
Age, sex	G: 1.29 [1.20, 1.40]** S: 1.24 [1.15, 1.35]** C: 1.02 [.95, 1.11]	7,591 (9)	G: 1.24 [1.17, 1.31]** S: 1.14 [1.08, 1.21]** C: .95 [.90, 1.00]	6,987 (7)
Age, sex, Previous MI	G: 1.26 [1.16, 1.36]** S: 1.22 [1.13, 1.33]** C: 1.04 [.96, 1.12]	7,479 (9)	G: 1.21 [1.15, 1.28]** S: 1.12 [1.06, 1.19]** C: .95 [.90, 1.01]	6,878 (7)
Age, sex, Diabetes	G: 1.27 [1.17, 1.37]** S: 1.21 [1.12, 1.31]** C: 1.02 [.94, 1.11]	7,511 (9)	G: 1.22 [1.16, 1.29]** S: 1.13 [1.06, 1.19]** C: .95 [.90, 1.01]	6,909 (7)
Age, sex, Smoking	G: 1.30 [1.20, 1.41]** S: 1.23 [1.13, 1.33]** C: 1.02 [.94, 1.10]	7,427 (9)	G: 1.24 [1.17, 1.31]** S: 1.14 [1.08, 1.21]** C: .95 [.90, 1.01]	6,825 (7)

Note. *OR* = odds ratio; CI = confidence interval; MI = myocardial infarction; G = general depression factor; S = general depression-free somatic/affective factor; C = general depression-free cognitive/affective factor. The three factors were simultaneously included in all models.
** $p < .001$.

in effect sizes is that all previous meta-analyses examined either a sum score of depressive symptoms or a diagnosis of major depression and therefore did not adjust for symptoms unrelated to depression (i.e., a bifactor model was not used in these studies). Sum scores that do not weight items run into the risk of yielding inflated estimates when predicting outcomes (DiStefano, Zhu, & Mindilã, 2009). Thus, somatic symptoms unrelated to depression but measured by depressive symptom questionnaires could have led to inflated estimates of the effect of depression on medical prognosis.

Bifactor Model

The data on the BDI self-report indicated a bifactor structure. To our knowledge, one previous study assessed if a general depression factor of the BDI predicted mortality independent of general depression-free somatic symptoms (Thombs et al., 2008). In this study the bifactor model, as proposed by Ward (Ward, 2006), fitted the data better than a three-factor correlated-traits model. Moreover, this study showed that a general depression factor was associated with 12-month mortality after adjusting for the effect of somatic symptoms unrelated to the general depression factor. Brouwer and colleagues (2013) assessed whether the BDI-II measures a single concept of depression or if other smaller constructs are being measured simultaneously in a sample of psychiatric outpatients without heart disease. They confirmed multidimensionality of this scale, but concluded that a unidimensional model can still represent a unique concept of depression. In the present study, the explained common variance of the general depression factor was also substantially large (79%), suggesting that despite having multiple dimensions, the BDI appears to be a valid measure of depression in patients with MI.

In the present study, Model A (G-S-C) fitted the data better than four different models. The number of factors in Model A was chosen based on a previous study on the factor structure of the BDI (Brouwer et al., 2013) and also on assessment of the scree plot. Although interpreting the scree plot can be rather subjective, we checked whether a two-factor solution would fit the data better than a three-factor solution. Because the three-factor solution fitted the data better than the other competing models, we used this factor solution throughout the manuscript.

It is important to mention that the subgroup factors structure found in the present study is also dependent on the questionnaire used. Because the BDI includes both somatic and cognitive items, we could find such a bifactor structure. This might for instance not be the case for other questionnaires designed to measure depression, such as the Hospital Anxiety and Depression Scale (HADS). The HADS has been created to avoid symptoms that could be secondary to somatic illness (e.g., fatigue and insomnia; Zigmond & Snaith, 1983). In a meta-analysis assessing psychometric properties of the HADS, it was reported that a bifactor model consisting of a general distress factor, and anxiety and depression subgroup factors provided the best fit when compared to other models of the HADS (Norton, Cosco, Doyle, Done, & Sacker, 2013).

The bifactor model presents a unique analytic approach to study the relationship between multidimensional measures and other variables. It gives the possibility to isolate the variance of subgroup factors (i.e., general depression-free cognitive/affective and general depression-free somatic/affective factors) and to yield a more purified measure of the main concept being studied (i.e., depression). Moreover, the bifactor model gives the possibility to investigate how these general depression-free subgroup factors are associated with other variables. Using the factors of a bifactor model allowed us not only to demonstrate that the general depression factor is associated with adverse medical prognosis independent of the general depression-free subgroup factors, but also that the general depression-free somatic/affective symptoms are associated with adverse medical prognosis.

Although the bifactor model used in the present study fitted the data satisfactorily, the names used to entitle the subgroup factors could be somewhat misleading. Most items of the general depression-free somatic/affective factor were clearly somatic, in terms of face-validity, such as fatigue and lack of libido. However, indecisiveness, a cognitive/affective symptom, also loaded on the general depression-free somatic/affective factor. A possible explanation is that, despite being a cognitive/affective symptom, this symptom is more often reported by patients experiencing other somatic/affective symptoms, and therefore clusters with them. These denominations (cognitive/affective and somatic/affective) have been used in previous studies on depressive symptom dimensions. However, it is important to mention that the subgroup factors reported here are different constructs as compared with previous studies on symptom dimensions of depression. Previous studies used correlated-traits models or simply computed sum scores for each of the dimensions, not taking into account that there is a general depression factor. The subgroup factors used in the present study represent variance of cognitive/affective and somatic/affective items that are unrelated to the general depression factor, and therefore we named them general depression-free somatic/affective and general depression-free cognitive/affective factors. Nonetheless, the items loading on the cognitive/affective or somatic/affective factors in previous studies are comparable to the items loading on the general depression-free cognitive/affective factor and on the general depression-free somatic/affective factor of the present study.

We cannot be sure what causes the general depression-free somatic/affective symptoms. It is possible that this factor reflects symptoms of heart disease or its consequences, but these symptoms may also reflect other somatic comorbidities. These general depression-free somatic/affective symptoms are nonspecific, and multiple conditions can simultaneously lead to high levels of these symptoms. Most importantly, however, the symptoms composing the general depression-free somatic/affective factor were consistently associated with adverse medical prognosis, independent of sadness, hopelessness and other common depressive symptoms.

Conceptualizing the general depression-free cognitive/affective factor is a bigger challenge. Ormel and de Jonge hypothesized that personality traits such as neuroticism would be associated with cognitive/affective symptoms of depression (Ormel & de Jonge, 2011). We hypothesize that the general depression-free cognitive/affective factor captures variance related to neuroticism traits that are similar to cognitive/affective symptoms of depression. Items composing the general depression-free cognitive/affective factor have substantial overlap with items used in the neuroticism domain of personality scales, such as the Neuroticism-Extraversion-Openness Personality Inventory 3 (McCrae, Martin, & Costa, 2005). Neuroticism has shown to increase the risk of adverse medical prognosis in initially healthy individuals (Grossardt, Bower, Geda, Colligan, & Rocca, 2009; Shipley, Weiss, Der,

Taylor, & Deary, 2007). However, several other studies have failed to confirm that neuroticism predicts adverse medical prognosis (Almada et al., 1991; Friedman, Kern, & Reynolds, 2010; Iwasa et al., 2008), and some studies have even suggested that neuroticism has a protective effect on adverse medical prognosis (Carinci et al., 1997; Korten et al., 1999; Weiss & Costa, 2005). In the present study, the general depression-free cognitive/affective factor was not predictive of adverse medical prognosis. However, general depression-free cognitive/affective symptoms could substantially affect the quality of life of a patient, and therefore also need to be treated.

The investigation of individual symptoms, rather than groups of symptoms, offers another possibility to study the complex relationship between depression and adverse medical prognosis in patients with MI. Recent evidence suggests that hopelessness is specifically associated with adverse medical prognosis in patients with MI (Denollet, Freedland, Carney, de Jonge, & Roest, 2013). Understanding the prognostic value of individual symptoms of depression in patients with MI could help identifying patients at a higher risk of adverse medical prognosis.

Clinical Implications

Results indicate that participants with high total scores are at increased risk of adverse prognosis, especially when items with higher loadings on the general depression factor (e.g., sadness, hopelessness) are endorsed. Therefore clinicians could routinely assess depressive symptoms to identify patients at higher risk for adverse medical prognosis. Most importantly, however, patients with lower total scores could also be at increased risk, if mostly endorsing somatic/affective symptoms. These patients could easily be overlooked if only total scores are used to identify patients at risk. A study by Wardenaar and colleagues suggests the implementation of person-fit statistics in clinical practice (Wardenaar, Wanders, Roest, Meijer, & De Jonge, 2015). This approach has the goal of assessing if the way a patient responds a questionnaire is atypical within a certain population. Patients with MI appear to mostly endorse somatic/affective symptoms, while an atypical profile may reflect more key-features of depression (Wardenaar et al., 2015). Such procedures could help clinicians to identify patients at risk of clinical depression, but also patients with high somatic/affective symptoms. Future studies could also investigate whether patients with different symptom profiles would benefit from different kinds of interventions.

Strengths and Limitations

The present study has a number of advantages. First, the large sample size provided sufficient statistical power to address our research questions. Second, it included studies from five different countries, which enable us to make wider generalizations and third, it used a bifactor model to assess dimensions of depressive symptoms with the factor solution yielded by the G-S-C Model A fitting the data better than the other models tested.

Only some studies adjusted for LVEF, thereby reducing the sample size of the model predicting all-cause mortality including LVEF from 6,775 to 4,744, which is a limitation of the study. Given that LVEF is an important marker of cardiac disease severity, it is desirable to control for it when predicting medical outcomes in patients with MI.

Although our data is clustered across different studies, assumptions for using multilevel CFA were not met due to low intraclass correlation coefficient for all the 21 items ($\leq$.055), meaning that less than 5.5% of variation in item response is explained by different study levels. The low number of study levels ($N = 9$) was also a concern, because it has been suggested that multilevel CFA performed in datasets with small group-level sample size ($<$100) and low ICC levels ($<$.25) yields inaccurate results (Hox & Maas, 2001). By using the MIMIC model we accounted for the multilevel structure of the sample. A similar procedure has been used elsewhere (Dong et al., 2014).

Another limitation of this study is the form of missing data imputation for depressive symptom data. Mean imputation has been used because a substantial part of the total sample already had missing data imputed this way. To keep it consistent, we used the same strategy to address missing data across the whole sample. Because of this limitation, we could also not quantify the number of cases that were excluded when six or more items were missing.

Despite the sophisticated methodology used to analyze our data, the associations reported here are correlational, and there is no strong evidence to date that treating depression would improve medical prognosis. It could also be that the large sample size used in the present study led to an excess of power. Therefore, care is needed when applying these results to clinical practice.

Conclusion

In this large study using a bifactor model of the BDI in patients with MI, results showed that a general depression factor is associated with adverse medical prognosis, independent of general depression-free somatic/affective symptoms that may in part be caused by somatic illness.

References

Almada, S. J., Zonderman, A. B., Shekelle, R. B., Dyer, A. R., Daviglus, M. L., Costa, P. T., Jr., & Stamler, J. (1991). Neuroticism and cynicism and risk of death in middle-aged men: The Western Electric Study. *Psychosomatic Medicine, 53,* 165–175. http://dx.doi.org/10.1097/00006842-199103000-00006

Barth, J., Schumacher, M., & Herrmann-Lingen, C. (2004). Depression as a risk factor for mortality in patients with coronary heart disease: A meta-analysis. *Psychosomatic Medicine, 66,* 802–813. http://dx.doi.org/10.1097/01.psy.0000146332.53619.b2

Beck, A. T., Steer, R. A., & Carbin, M. G. (1988). Psychometric properties of the beck depression inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8,* 77–100. http://dx.doi.org/10.1016/0272-7358(88)90050-5

Berkman, L. F., Blumenthal, J., Burg, M., Carney, R. M., Catellier, D., Cowan, M. J., . . . the Enhancing Recovery in Coronary Heart Disease Patients Investigators (ENRICHD). (2003). Effects of treating depression and low perceived social support on clinical events after myocardial infarction: The Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) randomized trial. *Journal of the American Medical Association, 289,* 3106–3116. http://dx.doi.org/10.1001/jama.289.23.3106

Brouwer, D., Meijer, R. R., & Zevalkink, J. (2013). On the factor structure of the Beck Depression Inventory-II: G is the key. *Psychological Assessment, 25,* 136–145. http://dx.doi.org/10.1037/a0029228

Browne, M., & Cudeck, R. (1992). Alternative ways of assessing model fit. *Sociological Methods & Research, 21,* 230–258. http://dx.doi.org/10.1177/0049124192021002005

Carinci, F., Nicolucci, A., Ciampi, A., Labbrozzi, D., Bettinardi, O., Zotti, A. M., & Tognoni, G. (1997). Role of interactions between psychological and clinical factors in determining 6-month mortality among patients with acute myocardial infarction. Application of recursive partitioning techniques to the GISSI-2 database. Gruppo Italiano per lo Studio della Sopravvivenza nell' Infarto Miocardico. *European Heart Journal, 18,* 835–845. http://dx.doi.org/10.1093/oxfordjournals.eurheartj.a015349

Carney, R. M., & Freedland, K. E. (2012). Are somatic symptoms of depression better predictors of cardiac events than cognitive symptoms in coronary heart disease? *Psychosomatic Medicine, 74,* 33–38. http://dx.doi.org/10.1097/PSY.0b013e3182405ac4

Chen, F. F., West, S. G., & Sousa, K. H. (2006). A comparison of bifactor and second-order models of quality of life. *Multivariate Behavioral Research, 41,* 189–225. http://dx.doi.org/10.1207/s15327906mbr4102_5

de Miranda Azevedo, R., Roest, A. M., Hoen, P. W., & de Jonge, P. (2014). Cognitive/affective and somatic/affective symptoms of depression in patients with heart disease and their association with cardiovascular prognosis: A meta-analysis. *Psychological Medicine, 44,* 2689–2703. http://dx.doi.org/10.1017/S0033291714000063

Denollet, J., Freedland, K. E., Carney, R. M., de Jonge, P., & Roest, A. M. (2013). Cognitive-affective symptoms of depression after myocardial infarction: Different prognostic importance across age groups. *Psychosomatic Medicine, 75,* 701–708. http://dx.doi.org/10.1097/PSY.0b013e31829dbd36

Denollet, J., Martens, E. J., Smith, O. R., & Burg, M. M. (2010). Efficient assessment of depressive symptoms and their prognostic value in myocardial infarction patients. *Journal of Affective Disorders, 120,* 105–111. http://dx.doi.org/10.1016/j.jad.2009.04.013

DiStefano, C., Zhu, M., & Mindilã, D. (2009). Understanding and using factor scores: Recommendations for the applied researcher. *Practical Assessment, Research & Evaluation, 14,* 1–11.

Dong, G. H., Qian, Z., Fu, Q., Wang, J., Trevathan, E., Ma, W., . . . Simckes, M. (2014). A Multiple Indicators Multiple Cause (MIMIC) model of respiratory health and household factors in Chinese children: The seven Northeastern cities (SNEC) study. *Maternal and Child Health Journal, 18,* 129–137. http://dx.doi.org/10.1007/s10995-013-1245-6

ENRICHD Investigators. (2001). Enhancing Recovery in Coronary Heart Disease (ENRICHD) study intervention: Rationale and design. *Psychosomatic Medicine, 63,* 747–755.

Friedman, H. S., Kern, M. L., & Reynolds, C. A. (2010). Personality and health, subjective well-being, and longevity. *Journal of Personality, 78,* 179–216. http://dx.doi.org/10.1111/j.1467-6494.2009.00613.x

Grace, S. L., Abbey, S. E., Pinto, R., Shnek, Z. M., Irvine, J., & Stewart, D. E. (2005). Longitudinal course of depressive symptomatology after a cardiac event: Effects of gender and cardiac rehabilitation. *Psychosomatic Medicine, 67,* 52–58. http://dx.doi.org/10.1097/01.psy.0000151486.28349.70

Grossardt, B. R., Bower, J. H., Geda, Y. E., Colligan, R. C., & Rocca, W. A. (2009). Pessimistic, anxious, and depressive personality traits predict all-cause mortality: The Mayo Clinic cohort study of personality and aging. *Psychosomatic Medicine, 71,* 491–500. http://dx.doi.org/10.1097/PSY.0b013e31819e67db

Holzinger, K. J., & Swineford, F. (1937). The bi-factor method. *Psychometrika, 2,* 41–54. http://dx.doi.org/10.1007/BF02287965

Hosseini, S. H., Ghaemian, A., Mehdizadeh, E., & Ashraf, H. (2014). Levels of anxiety and depression as predictors of mortality following myocardial infarction: A 5-year follow-up. *Cardiology Journal, 21,* 370–377. http://dx.doi.org/10.5603/CJ.a2014.0023

Hox, J. J., & Maas, C. J. M. (2001). The accuracy of multilevel structural equation modeling with pseudobalanced groups and small samples. *Structural Equation Modeling, 8,* 157–174. http://dx.doi.org/10.1207/S15328007SEM0802_1

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6,* 1–55. http://dx.doi.org/10.1080/10705519909540118

Iwasa, H., Masui, Y., Gondo, Y., Inagaki, H., Kawaai, C., & Suzuki, T. (2008). Personality and all-cause mortality among older adults dwelling in a Japanese community: A five-year population-based prospective cohort study. *The American Journal of Geriatric Psychiatry: Official Journal of the American Association for Geriatric Psychiatry, 16,* 399–405. http://dx.doi.org/10.1097/JGP.0b013e3181662ac9

Jöreskog, K. G., & Goldberger, A. S. (1975). Estimation of a model with multiple indicators and multiple causes of a single latent variable. *Journal of the American Statistical Association, 70,* 631–639. http://dx.doi.org/10.2307/2285946

Korten, A. E., Jorm, A. F., Jiao, Z., Letenneur, L., Jacomb, P. A., Henderson, A. S., . . . Rodgers, B. (1999). Health, cognitive, and psychosocial factors as predictors of mortality in an elderly community sample. *Journal of Epidemiology and Community Health, 53,* 83–88. http://dx.doi.org/10.1136/jech.53.2.83

Lane, D., Carroll, D., Ring, C., Beevers, D. G., & Lip, G. Y. (2001). Mortality and quality of life 12 months after myocardial infarction: Effects of depression and anxiety. *Psychosomatic Medicine, 63,* 221–230. http://dx.doi.org/10.1097/00006842-200103000-00005

Lauzon, C., Beck, C. A., Huynh, T., Dion, D., Racine, N., Carignan, S., . . . Pilote, L. (2003). Depression and prognosis following hospital admission because of acute myocardial infarction. *CMAJ: Canadian Medical Association Journal/Journal De L'Association Medicale Canadienne, 168,* 547–552.

Lee, K. L., Woodlief, L. H., Topol, E. J., Weaver, W. D., Betriu, A., Col, J., . . . the GUSTO-I Investigators. (1995). Predictors of 30-day mortality in the era of reperfusion for acute myocardial infarction. Results from an international trial of 41,021 patients. *Circulation, 91,* 1659–1668. http://dx.doi.org/10.1161/01.CIR.91.6.1659

McCrae, R. R., Martin, T. A., & Costa, P. T., Jr. (2005). Age trends and age norms for the NEO personality inventory-3 in adolescents and adults. *Assessment, 12,* 363–373.

Meijer, A., Conradi, H. J., Bos, E. H., Anselmino, M., Carney, R. M., Denollet, J., . . . de Jonge, P. (2013). Adjusted prognostic association of depression following myocardial infarction with mortality and cardiovascular events: Individual patient data meta-analysis. *The British Journal of Psychiatry, 203,* 90–102. http://dx.doi.org/10.1192/bjp.bp.112.111195

Meijer, A., Conradi, H. J., Bos, E. H., Thombs, B. D., van Melle, J. P., & de Jonge, P. (2011). Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: A meta-analysis of 25 years of research. *General Hospital Psychiatry, 33,* 203–216. http://dx.doi.org/10.1016/j.genhosppsych.2011.02.007

Mueller, H. S., Cohen, L. S., Braunwald, E., Forman, S., Feit, F., Ross, A., . . . Miller, D. (1992). Predictors of early morbidity and mortality after thrombolytic therapy of acute myocardial infarction. Analyses of patient subgroups in the Thrombolysis in Myocardial Infarction (TIMI) trial, Phase II. *Circulation, 85,* 1254–1264. http://dx.doi.org/10.1161/01.CIR.85.4.1254

Muthén, L. K., & Muthén, B. O. (1998–2012). *Mplus user's guide. Seventh Edition*. Los Angeles, CA: Author.

Nicholson, A., Kuper, H., & Hemingway, H. (2006). Depression as an aetiologic and prognostic factor in coronary heart disease: A meta-analysis of 6362 events among 146,538 participants in 54 observational studies. *European Heart Journal, 27,* 2763–2774. http://dx.doi.org/10.1093/eurheartj/ehl338

Norton, S., Cosco, T., Doyle, F., Done, J., & Sacker, A. (2013). The hospital anxiety and depression scale: A meta confirmatory factor analysis. *Journal of Psychosomatic Research, 74,* 74–81. http://dx.doi.org/10.1016/j.jpsychores.2012.10.010

Ormel, J., & de Jonge, P. (2011). Unipolar depression and the progression of coronary artery disease: Toward an integrative model. *Psychotherapy and Psychosomatics, 80,* 264–274. http://dx.doi.org/10.1159/000323165

Parakh, K., Thombs, B. D., Fauerbach, J. A., Bush, D. E., & Ziegelstein, R. C. (2008). Effect of depression on late (8 years) mortality after myocardial infarction. *The American Journal of Cardiology, 101,* 602–606. http://dx.doi.org/10.1016/j.amjcard.2007.10.021

R Development Core Team. (2008). *R: A language and environment for statistical computing. R foundation for statistical computing, Vienna, Austria*. Retrieved from http://www.R-project.org

Reise, S. P. (2012). The rediscovery of bifactor measurement models. *Multivariate Behavioral Research, 47,* 667–696. http://dx.doi.org/10.1080/00273171.2012.715555

Reise, S. P., Moore, T. M., & Haviland, M. G. (2010). Bifactor models and rotations: Exploring the extent to which multidimensional data yield univocal scale scores. *Journal of Personality Assessment, 92,* 544–559. http://dx.doi.org/10.1080/00223891.2010.496477

Reise, S. P., Scheines, R., Widaman, K. F., & Haviland, M. G. (2013). Multidimensionality and structural coefficient bias in structural equation modeling: A bifactor perspective. *Educational and Psychological Measurement, 73,* 5–26. http://dx.doi.org/10.1177/0013164412449831

Revelle, W. (2014). *Psych: Procedures for psychological, psychometric, and personality research* (R package version 1.4.3). Evanston, IL: Northwestern University.

Riley, R. D., Lambert, P. C., & Abo-Zaid, G. (2010). Meta-analysis of individual participant data: Rationale, conduct, and reporting. *British Medical Journal, 340,* c221. http://dx.doi.org/10.1136/bmj.c221

Roest, A. M., Thombs, B. D., Grace, S. L., Stewart, D. E., Abbey, S. E., & de Jonge, P. (2011). Somatic/affective symptoms, but not cognitive/affective symptoms, of depression after acute coronary syndrome are associated with 12-month all-cause mortality. *Journal of Affective Disorders, 131,* 158–163. http://dx.doi.org/10.1016/j.jad.2010.11.018

Schmid, J., & Leiman, J. (1957). The development of hierarchical factor solutions. *Psychometrika, 22,* 53–61. http://dx.doi.org/10.1007/BF02289209

Shipley, B. A., Weiss, A., Der, G., Taylor, M. D., & Deary, I. J. (2007). Neuroticism, extraversion, and mortality in the UK health and lifestyle survey: A 21-year prospective cohort study. *Psychosomatic Medicine, 69,* 923–931. http://dx.doi.org/10.1097/PSY.0b013e31815abf83

Spijkerman, T., de Jonge, P., van den Brink, R. H., Jansen, J. H., May, J. F., Crijns, H. J., & Ormel, J. (2005). Depression following myocardial infarction: First-ever versus ongoing and recurrent episodes. *General Hospital Psychiatry, 27,* 411–417. http://dx.doi.org/10.1016/j.genhosppsych.2005.05.007

StataCorp. (2011). *Stata statistical software: Release 12*. College Station, TX: StataCorp LP.

Thombs, B. D., Ziegelstein, R. C., Beck, C. A., & Pilote, L. (2008). A general factor model for the Beck Depression Inventory-II: Validation in a sample of patients hospitalized with acute myocardial infarction. *Journal of Psychosomatic Research, 65,* 115–121. http://dx.doi.org/10.1016/j.jpsychores.2008.02.027

van Loo, H. M., de Jonge, P., Romeijn, J. W., Kessler, R. C., & Schoevers, R. A. (2012). Data-driven subtypes of major depressive disorder: A systematic review. *BMC Medicine, 10,* 156. http://dx.doi.org/10.1186/1741-7015-10-156

van Melle, J. P., de Jonge, P., Honig, A., Schene, A. H., Kuyper, A. M., Crijns, H. J., . . . the MIND-IT investigators. (2007). Effects of antidepressant treatment following myocardial infarction. *The British Journal of Psychiatry, 190,* 460–466. http://dx.doi.org/10.1192/bjp.bp.106.028647

van Melle, J. P., de Jonge, P., Spijkerman, T. A., Tijssen, J. G., Ormel, J., van Veldhuisen, D. J., . . . van den Berg, M. P. (2004). Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: A meta-analysis. *Psychosomatic Medicine, 66,* 814–822. http://dx.doi.org/10.1097/01.psy.0000146294.82810.9c

Ward, L. C. (2006). Comparison of factor structure models for the Beck Depression Inventory—II. *Psychological Assessment, 18,* 81–88. http://dx.doi.org/10.1037/1040-3590.18.1.81

Wardenaar, K. J., Wanders, R. B., Roest, A. M., Meijer, R. R., & De Jonge, P. (2015). What does the beck depression inventory measure in myocardial infarction patients? A psychometric approach using item response theory and person-fit. *International Journal of Methods in Psychiatric Research, 24,* 130–142. http://dx.doi.org/10.1002/mpr.1467

Weiss, A., & Costa, P. T., Jr. (2005). Domain and facet personality predictors of all-cause mortality among Medicare patients aged 65 to 100. *Psychosomatic Medicine, 67,* 724–733.

Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica, 67,* 361–370. http://dx.doi.org/10.1111/j.1600-0447.1983.tb09716.x

Zuidersma, M., Conradi, H. J., van Melle, J. P., Ormel, J., & de Jonge, P. (2013). Depression treatment after myocardial infarction and long-term risk of subsequent cardiovascular events and mortality: A randomized controlled trial. *Journal of Psychosomatic Research, 74,* 25–30. http://dx.doi.org/10.1016/j.jpsychores.2012.08.015

Received March 3, 2015
Revision received September 2, 2015
Accepted September 28, 2015 ■