A General Factor Model for the Beck Depression Inventory – II: Validation in a Sample of Patients Hospitalized with Acute Myocardial Infarction

Brett D. Thombs, PhD[1]; Roy C. Ziegelstein, MD[2]; Christine A. Beck, MSc[3]; Louise Pilote, MD, MPH, PhD[3,4]

[1]Department of Psychiatry, Sir Mortimer B. Davis – Jewish General Hospital and McGill University, Montreal, Quebec; [2]Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland; [3]Division of Clinical Epidemiology, the Research Institute of McGill University Health Center, Montreal, Quebec; [4]Division of Internal Medicine, the Research Institute of McGill University Health Center, Montreal, Quebec.

Running Head: BDI-II in Acute Myocardial Infarction

Address for Correspondence:

Brett D. Thombs, Ph.D.

Institute of Community and Family Psychiatry

SMBD-Jewish General Hospital

4333 Cote Ste Catherine Road

Montreal, Quebec H3T 1E4

Tel (514) 340-8222 ext. 5112

Fax (514) 340-8124

E-mail: brett.thombs@mcgill.ca

Acknowledgements

This study was supported by a grant from the Fonds de la Recherche en Santé de Québec (grant no. 961305-104) awarded to Dr. Pilote. Dr. Pilote is a research scholar funded by the Canadian Institutes of Health Research and a William Dawson Professor of Medicine at McGill University. Dr. Ziegelstein is supported by NIH R21NS048593.

Abstract

Objective: Many studies have linked symptoms of depression after an acute myocardial infarction (AMI) to negative health outcomes, including mortality. It has been suggested, however, that this link may be due to biased measurement of depressive symptoms in post-AMI patients related to confounding with somatic symptoms related to AMI. The objective of this study was to validate a factor model for the BDI-II that would allow for modeling of depressive symptoms after explicitly removing bias related to somatic symptom overlap.

Methods: 477 hospitalized post-AMI patients from 10 cardiac care units were administered the BDI-II. Confirmatory factor analysis models for ordinal data were conducted with MPLUS to test the fit of a model with a single General Depression factor (all 21 BDI-II items) and uncorrelated Somatic (5 items) and Cognitive (8 items) factors (G-S-C model) compared to standard correlated 2-factor models.

Results: The G-S-C model fit as well or better than previously published correlated 2-factor models. 73% of variance in BDI-II scores is accounted for by the General Depression factor, whereas 11% and 13% respectively, is accounted for by uncorrelated Somatic and Cognitive factors.

Conclusions: The G-S-C model is a novel approach to understanding the measurement structure of the BDI-II, presents advantageous statistical and interpretive properties compared to standard correlated factor models, and provides a viable mechanism to test links between symptoms of depression, as measured by the General Depression factor, and health outcomes among patients with AMI after explicitly removing variance from somatic symptoms unrelated to the General Depression factor.

Introduction

Major depression is diagnosed in approximately 20% of patients hospitalized with acute myocardial infarction (AMI) [1]. Following AMI, symptoms of depression predict ongoing functional impairment [2], less favorable self-care behaviors [3], substantially higher health-care costs [4], and increased cardiac morbidity and mortality [5]. The high rate of depression among medically ill patients, including patients with AMI, however, has raised questions about the validity of existing methods of symptom assessment because there is substantial overlap in somatic symptoms of depression and symptoms common in medical illness, including fatigue or loss of energy, anhedonia, changes in sleep patterns, and changes in appetite [6].

The Beck Depression Inventory (BDI) [7] and its revised version, the Beck Depression Inventory-II (BDI-II) [8], are the most commonly used assessment tools in research of post-AMI patients [1]. The BDI tends to be used more frequently in research on post-AMI depression [1]. The main difference between the instruments is that items on the BDI-II that reflect agitation, concentration, loss of energy and feelings of worthlessness have replaced items from the BDI related to concerns about appearance, ability to work, change in weight, and worries about health. Specific concerns have been raised, however, about the performance characteristics of the Beck depression scales in patients with medical illness since approximately half of the items on the BDI and BDI-II assess somatic or performance related symptoms [9, 10]. Consistent with this, several authors have argued that studies linking depression to cardiac and all-cause mortality post-AMI have not adequately controlled for potential bias in the measurement of depressive symptoms related to confounding of somatic symptoms of depression and symptoms of the AMI [11-13]. The authors of one systematic review of the association between post-AMI

depression and mortality wrote:

> The BDI is considered a useful screening instrument for depression in the medically ill. However, 7 of the 21 items in the BDI, in fact, assess somatic symptoms. For the particular somatic symptom will 50–75% of the medical patients fulfil it [sic]. Cardiac patients may, therefore, on the sheer basis of somatic symptoms, score close to 10 points which is used as the cutoff score for depression. The group of so-called depressed MI patients may in reality consist of a large number of patients with severe cardiac disease, and the positive relation between so-called depression and mortality may not be caused by the truly depressed patients, but by the patients with severe cardiac disease [12].

Existing studies that have assessed the relationship between depressive symptoms measured during hospitalization for AMI and subsequent outcomes have used linear regression, logistic regression, and survival models, which treat total scores on instruments like the BDI or BDI-II are treated as error-free measures of depressive symptoms. These types of analyses would indeed be susceptible to confounding or bias due to somatic symptom overlap that could artifactually inflate the strength of the relationship between symptoms of depression and health-related outcomes. Total scores on the BDI or BDI-II that are used as predictors in these models potentially include three components: variance related to depressive symptoms; variance systematically related to phenomena, such as somatic symptoms from medical illness, that are unrelated to depression per se; and error variance. Structural equation modeling techniques, on the other hand, present an alternative paradigm that allows for the possibility of explicitly separating variance related to depression per se and variance related to extraneous somatic factors, and can model these types of variance after removing estimates of error variance, thus, creating a “cleaner” depression predictor variable. Indeed, a confirmatory factor analysis (CFA) model of the BDI-II developed by Ward [14] explicitly separates variance from a General Depression factor from variance from unrelated somatic symptoms. Ward demonstrated in five

different samples that a BDI-II model with General Depression, Somatic, and Cognitive factors (G–S–C) that were forced to be orthogonal fit as well or better than previously published correlated two-factor BDI-II models, representing either Somatic-Affective and Cognitive factors (SA–C) [8] or Cognitive-Affective and Somatic factors (CA–S) [15]. In Ward's model, all BDI-II item scores are indicators of the General Depression factor, and some items also load on Somatic or Cognitive factors that are orthogonal to the General factor and each other. The correlated SA-C and CA-S would, similar to Ward's model, offer the advantage of modeling "true" factor scores with estimates of error variance removed. The factors of these models would not, however, be able to be used together to predict outcomes, such as mortality. This is because there is typically a high level of correlation between the factors (.71 to .88) [14] that would result in uncorrectable multicollinearity if one attempted to use them simultaneously as predictors.

Demonstration that Ward's G-S-C model is valid with medically ill post-AMI patients would provide a mechanism for using latent variable models to explicitly model longitudinal outcomes following AMI on a General depression factor independent of variance from an orthogonal Somatic factor and would allow explicit testing of whether depressive symptoms predict negative outcomes when potential bias from somatic symptom overlap is removed. Thus, the objective of this report was to replicate Ward's G-S-C model in a sample of patients hospitalized with AMI.

Methods

Patients and Procedure

Participants in the study included patients who were treated for AMI at any of 10 hospitals in Québec, Canada between December 28, 1996 and November 1, 1998. Patients with

AMI were eligible for enrollment in the study if they survived at least 24 hours after admission and had been admitted through the emergency department rather than as transfers from another hospital. Patients were excluded if they did not read and understand French or English or if they were medically incapable of giving informed consent or responding to a questionnaire. Research nurses approached eligible patients for informed consent and enrollment within 2-3 days after the date of admission. Study participants completed a self-administered questionnaire in the hospital at the time of enrollment that included the BDI-II. The BDI-II [8] is a 21-item self-report inventory of symptoms of depression that has been used with both psychiatric and non-psychiatric samples. Each item consists of four statements, scored 0-3, indicating increasing symptom severity; total scores range from 0-63. A cutoff score of 14 or above is typically used to identify patients with at least *mild* symptoms of depression [8, 16]. Patients in the study were followed through one year post-AMI, and 12-month vital status was obtained from a central death registry for all patients who were lost to follow-up. This study was an ancillary study to a prospective cohort study of quality of life after AMI [17] that received ethical approval from the Montreal General Hospital Ethics Review Board.

Data Analysis

All CFA models were conducted with Mplus (version 3.11) [18], explicitly modeling the BDI-II items as ordinal data. To do this, Mplus initially estimates item thresholds for ordinal outcome variables using maximum likelihood methods. These estimates are then used to estimate a polychoric correlation matrix. Model parameters are subsequently estimated with weighted least squares using the inverse of the asymptotic covariance matrix as the weight matrix [19]. A chi-square goodness-of-fit test and four fit indices were used to assess model fit, including the

Tucker-Lewis Index (TLI) [20], the comparative fit index (CFI) [21], the root mean square error of approximation (RMSEA) [22], and the standardized root-mean-square residual (SRMR) [23]. Since the chi-square test is highly sensitive to sample size and can lead to the rejection of well-fitting models, practical fit indices were emphasized [24]. Guidelines proposed by Hu and Bentler [25] suggest that models with TLI and CFI close to .95 or higher, RMSEA close to .06 or lower and SRMR close to .08 or lower are representative of good fitting models. A CFI of .90 or above [26] and a RMSEA of .08 or less [27], however, are also considered to represent reasonably acceptable model fit.

Three factor models were fit to the BDI-II data following Ward's procedure. The SA-C two-factor model originally reported by Beck et al. [8] was fit with 12 items loading on the Somatic-Affective factor and 9 items on the Cognitive factor. For each model, modification indices were used to identify pairs of items within factors for which model fit would improve if error estimates were freed to correlate, and for which there appeared to be theoretically justifiable shared method effects. For the SA-C model, correlated errors were permitted between two pairs of items: *loss of energy* with *fatigue* and *agitation* with *irritability.* A second correlated two-factor model (S-CA) was also specified with 5 items loaded on a Somatic factor and 16 items on a Cognitive-Affective factor. In this model, four pairs of error terms were freed to correlate: *loss of energy* with *fatigue, agitation* with *irritability, self-dislike* with *self-criticalness*, and *loss of pleasure* with *loss of interest*. Ward's orthogonal G-S-C model was the third model fit to the data. Per Ward's procedure, all 21 items loaded on the General factor. In addition, 5 items loaded on a Somatic factor (*loss of energy, sleep problems, appetite, concentration, fatigue*), 8 items loaded on a Cognitive factor (*pessimism, past failure, guilty feelings,*

punishment feelings, self-dislike, self-criticalness, suicidal thoughts, worthlessness), 2 items loaded on a Self-Criticalness factor (*self-dislike, self-criticalness*), and 2 items loaded on an Anhedonia factor (*loss of pleasure, loss of interest*). The pairs of items that loaded on each of the latter two factors were constrained to equality for model identification purposes. For the G-S-C model, item communalities (h^2s) that represent the percent of variance in each item predicted by the factors were calculated from standardized factor loadings.

Results

Sample Characteristics

The study sample consistent of 477 AMI patients who completed the BDI-II in the hospital. As shown in Table 1, the sample was predominantly male and White, and the majority of patients were married or living as married. The mean age of the sample was 60.1 years (*SD* = 12.2 years, range = 29 to 90). Female patients, non-White patients, and patients who were not married or living as married were significantly more likely to score 14 or above on the BDI-II. Of cardiac disease and health-related variables, only a history of angina was significantly related to scoring 14 or higher on the BDI. A total of 33 patients died by 12 months post-AMI (6.9%), but this was not related to in-hospital BDI scores (P = .544).

Confirmatory Factor Analysis

Model fit statistics for each of the three models tested are shown in Table 2. In addition to chi-square test results and fit indices, factor correlations are shown for the SA-C and CA-S two-factor models. Both the SA-C and CA-S fit reasonably well based on fit indices. All factor loadings for both models were statistically significant with standardized loadings of .46 or higher for all items in the SA-C model and .44 or higher for all items in the CA-S model. The fit of the

G-S-C model (Figure 1) was as good or slightly better than each of the other two models. All factor loadings were significant with the exception of *pessimism* on the Cognitive factor (z = 1.65, p = .10). Removing this factor loading from the model did not meaningfully change the chi-square test ($\chi^2_{76} = 255.3$), any fit indices (CFI = .92, TLI = .96, RMSEA = .07, SRMR = .07), or any model parameters. The Somatic factor of the G-S-C model reflected a sense of fatigue and low energy. The loadings of these two items were .82 and .62, respectively, compared to .19 to .25 for the other three items that loaded on the factor. The Cognitive factor was largely driven by a sense of self-blame, and the loadings from three items related to failure, guilt, and punishment were between .51 and .56, compared to loadings of .29 to .43 for the items *self-dislike, self-criticalness, suicidal thoughts,* and *worthlessness,* and .11 (non-significant) on the item *pessimism.* As in Ward's results, the General factor explained the highest proportion of total covariance (communality = 73%) with the Somatic and Cognitive factors contributing modest amounts (11% and 13%, respectively), and the two minor Self-Criticalness and Anhedonia factors < 1% each. Item endorsement rates, means, and communalities are shown in Table 3.

A simplified version of the G-S-C model that did not include the minor Self-Criticalness and Anhedonia factors was also tested. As shown in Table 2, simplifying the G-S-C to facilitate practical use in model building and to provide a conceptually more coherent model did not meaningfully affect its overall fit to the data. Item-factor loadings and other model data for the three models are available upon request from the corresponding author.

Discussion

The main finding of this study was that, consistent with results reported by Ward [14] in three clinical and two college student samples, the G-S-C model fit as well as or slightly better

than the correlated two-factor SA-C and CA-S models in a sample of patients hospitalized with AMI. The General factor of the G-S-C model explained 73% of total communality, which is within the range of 71% to 82% reported by Ward. The Somatic and Cognitive factors accounted for 11% and 13%, respectively, of total communality, also similar to the ranges of 6% to 11% (Somatic) and 8% to 14% (Cognitive) reported by Ward [14] in non-medical samples. As in Ward's study, all items on the General factor had reasonably strong loadings. The items *loss of interest, indecisiveness,* and *loss of pleasure* had the highest loadings. The three most salient items on the Cognitive factor were *past failure, punishment feelings,* and *guilty feelings,* representing a strong theme related to self-blame. The Somatic factor was largely defined by the items *loss of energy* and *fatigue*, each of which loaded much more robustly than any other items.

As reviewed by Ward [14], the G-S-C model has interpretive advantages over the SA-C and CA-S two-factor models. In each of the two-factor models, the factors are highly correlated (.80 and .72, respectively). Due to multicollinearity problems with correlations this high, the correlated factors could, thus, not be used simultaneously in a model to predict outcomes. In addition, the factors do not tend to be highly stable across samples. Steer et al. [28], for instance, warned that certain items in the SA-C model tend to shift dimensions across samples. In addition, the two-factor model implications can be misleading since many items, including *sadness, irritability, agitation,* and *loss of pleasure*, are not easily described as either purely somatic or cognitive items. The G-S-C model, on the other hand, has been shown to provide a stable fit that is as good or better than the two-factor models across several different samples, including the sample of AMI patients in this study. Furthermore, interpretation of the G-S-C is consistent with the use a single summary score to estimate the severity of depressive symptoms

as described by Beck et al. [8].

Among medically ill patients, including patients hospitalized with AMI, the G-S-C model has additional conceptual and pragmatic advantages. The Somatic factor was dominated by variance from items related to fatigue and loss of energy that was uncorrelated with the General depression factor. Both of these symptoms are commonly experienced during hospitalization for AMI and which may or may not be related to depression. Variance from items on the Cognitive factor is similarly allocated, which takes on special meaning in the context of an AMI given the predominance of self-blame on the Cognitive factor. Cognitive theories of depression [29] associate self-blame with poor adjustment. On the other hand, studies of the consequences of illness attributions among patients with medical illness [30], suggest that self-blame or attribution of consequences to one's own behavior may be related to more positive coping and better subsequent outcomes [31]. These two theoretical models, however, may be addressing two distinct constructs, characterological and behavioral self-blame. Whereas characterological or personality-related self-blame would be expected to be maladaptive, behavioral self-blame may be a useful coping strategy after an acute medical event that provides a sense of controllability of the future and over one's own health [31]. Thus, separating variance from cognitive items that is orthogonal to the General Depression factor may have important implications among medically ill patients. Pragmatically, the G-S-C model also provides a framework for testing models of relationships between symptoms of depression and long-term health outcomes post-AMI. By regressing outcomes on the General Depression factor within a structural equation modeling approach, possible pathways from symptoms of depression to outcomes can be modeled after explicitly removing variance from somatic factors that is unrelated to the General Depression

factor.

One might wonder whether categorical approaches (e.g., DSM-IV diagnosis of depressive disorder) to classifying depression are more appropriate than dimensional approaches to measuring depressive symptoms (e.g., total number of symptoms, points on a rating scale, or continuously defined latent variable), such as the approach that was incorporated in the factor analytic methods of the current study, for research on prognosis. The debate over categorical versus dimensional approaches is long-running and will not be resolved here. A review of methodological issues in using categorical versus dimensional approaches by Kraemer et al. [32], however, is helpful for framing relevant issues. Kraemer et al. demonstrated that categorical and dimensional approaches are fundamentally equivalent, but have properties that make each more useful in different contexts. Categorical approaches are necessary in clinical research, for instance, to make decisions related to inclusion/exclusion or to test treatment effects. On the other hand, the properties of dimensional approaches typically make them much more suitable when the purpose of assessment is related to hypothesis testing because of issues related to reduced power with dichotomized assessments and error variance introduced by misclassification [32].

There are limitations that should be noted in interpreting the results of this study. Depressive disorders were not assessed with a standardized clinical interview, so the prevalence of major or minor depression in the study cohort was not known. An additional limitation in using this sample to develop the G-S-C model for the BDI-II in post-AMI patients is that BDI-II raw total scores were not associated with baseline health status variables or with 12-month all-cause mortality. The G-S-C can potentially add to the literature on post-AMI prognosis by

controlling for potential bias from somatic symptoms in the measurement of depression and testing to see whether significant results continue to be significant after removing the potential confound of bias due to somatic symptom overlap. That, however, could not be demonstrated in this sample since depressive symptoms were not related to mortality even prior to modeling out variance from somatic symptom overlap.

In summary, the G-S-C model provides a reasonably good-fitting explanation of BDI-II data from patients hospitalized with AMI that is as good as or better than model fit from alternative two-factor models. The G-S-C model has important theoretical and practical advantages, including the ability to model the relationship of a General Depression factor with important health outcomes after explicitly removing variance from somatic factors unrelated to the General Depression factor.

References

[1] 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;21:30-8.

[2] Ruo B, Rumsfeld JS, Hlatky MA, Liu H, Browner WS, Whooley MA. Depressive symptoms and health-related quality of life: the Heart and Soul Study. JAMA. 2003;290:215-21.

[3] Ziegelstein RC, Fauerbach JA, Stevens SS, Romanelli J, Richter DP, Bush DE. Patients with depression are less likely to follow recommendations to reduce cardiac risk during recovery from a myocardial infarction. Arch Intern Med. 2000;160:1818-23.

[4] Frasure-Smith N, Lesperance F, Gravel G, Masson A, Juneau M, Talajic M, et al. Depression and health-care costs during the first year following myocardial infarction. J Psychosom Res. 2000;48:471-8.

[5] van Melle JP, de Jonge P, Spijkerman TA, Tijssen JG, Ormel J, van Veldhuisen DJ, et al. Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta-analysis. Psychosom Med. 2004;66:814-22.

[6] Koenig HG, George LK, Peterson BL, Pieper CF. Depression in medically ill hospitalized older adults: prevalence, characteristics, and course of symptoms according to six diagnostic schemes. Am J Psychiatry. 1997;154:1376-83.

[7] Beck AT, Steer RA. Manual for the Revised Beck Depression Inventory. San Antonio, TX: Psychological Corporation; 1987.

[8] Beck AT, Steer RA, Brown GK. Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation; 1996.

[9] Leentjens AF, Verhey FR, Luijckx GJ, Troost J. The validity of the Beck Depression Inventory as a screening and diagnostic instrument for depression in patients with Parkinson's disease. Mov Disord. 2000;15:1221-4.

[10] Moran PJ, Mohr DC. The Validity of Beck Depression Inventory and Hamilton Rating Scale for Depression Items in the Assessment of Depression Among Patients with Multiple Sclerosis. J Behav Med. 2005;28:35-41.

[11] Lane D, Carroll D, Lip GY. Anxiety, depression, and prognosis after myocardial infarction: is there a causal association? J Am Coll Cardiol. 2003;42:1808-10.

[12] Sorensen C, Friis-Hasche E, Haghfelt T, Bech P. Postmyocardial infarction mortality in relation to depression: a systematic critical review. Psychother Psychosom. 2005;74:69-80.

[13] de Jonge P, Ormel J, van den Brink RH, van Melle JP, Spijkerman TA, Kuijper A, et al. Symptom dimensions of depression following myocardial infarction and their relationship with somatic health status and cardiovascular prognosis. Am J Psychiatry. 2006;163:138-44.

[14] Ward LC. Comparison of Factor Structure Models for the Beck Depression Inventory--II. Psychol Assess. 2006;18:81-8.

[15] Whisman MA, Perez JE, Ramel W. Factor structure of the Beck Depression Inventory-Second Edition (BDI-II) in a student sample. J Clin Psychol. 2000;56:545-51.

[16] Frasure-Smith N, Lesperance F. Depression and anxiety as predictors of 2-year cardiac events in patients with stable coronary artery disease. Arch Gen Psych. 2008;65:62-71.

[17] Pilote L, Lauzon C, Huynh T, Dion D, Roux R, Racine N, et al. Quality of life after acute myocardial infarction among patients treated at sites with and without on-site availability of angiography. Arch Intern Med. 2002;162:553-9.

[18] Muthén LK, Muthén BO. Mplus user's guide. Third ed. Los Angeles: Muthén & Muthén; 1998-2004.

[19] Muthén B. A general structural equation model with dichotomous, ordered categorical, and continuous latent variable indicators. Psychometrika. 1984;49:115-32.

[20] Tucker LR, Lewis C. A reliability coefficient for maximum likelihood factor analysis. Psychometrika. 1973;38:1-10.

[21] Bentler PM. Comparative fit indexes in structural models. Psychol Bull. 1990;107:238-46.

[22] Steiger JH. Structural model evaluation and modification: An interval estimation approach. Multivariate Behavioral Research. 1990;25:173-80.

[23] Bentler PM, Wu EJC. EQS structural equations program, Version 6.1. 2003;

[24] Reise SP, Widaman KF, Pugh RH. Confirmatory factor analysis and item response theory: Two Approaches for exploring measurement invariance. Psychol Bull. 1993;114:552-66.

[25] Hu L, Bentler P,M. Cutoff criteria for fit indexes in covariance structure analysis:

Conventional criteria versus new alternatives. Struct Equ Model. 1999;6:1-55.

[26] Kline RB. Principles and practice of structural equation modeling. 2nd ed. New York: Guilford Press; 2005.

[27] Browne MW, Cudeck R. Alternative ways of assessing fit. In: Bollen KA, Long JS, eds. Testing structural equation models Newbury Park, CA: Sage; 1993:136-62.

[28] Steer RA, Ball R, Ranieri WF, Beck AT. Dimensions of the Beck Depression Inventory-II in clinically depressed outpatients. J Clin Psychol. 1999;55:117-28.

[29] Beck AT. Depression: clinical, experimental, and theoretic aspects. New York: Harper and Row; 1976.

[30] Leventhal H, Leventhal EA, Contrada RJ. Self-regulation, health, and behavior: A perceptual-cognitive approach. Psychol Health. 1998;13:717-33.

[31] Hall S, French DP, Marteau TM. Causal attributions following serious unexpected negative events: A systematic review. J Soc Clin Psychol. 2003;22:515-36.

[32] Kraemer HC, Noda A, O'Hara R. Categorical versus dimensional approaches to diagnosis: Methodological challenges. J Psychosom Res. 2004;38:17-25.

Table 1. Demographic and Clinical Characteristics for Total Sample and by BDI Score

	All Patients (N=477)		Patients With BDI < 14 N=370 (77.6%)		Patients With BDI ≥ 14 N=107 (22.4%)		Significance
	n	%	n	%	n	%	P
Male Sex	394	82.6	315	79.9	79	20.1	.007
White	454	95.2	356	96.2	98	91.6	.049
Married or Living as Married	339	71.1	275	74.3	64	59.8	.004
Prior Myocardial Infarction	96	20.1	74	20.0	22	20.6	.899
History of Angina	120	25.2	82	22.2	38	35.5	.005
Q-wave AMI	246	51.6	196	53.0	50	46.7	.255
Killip class > 1	89	18.7	69	18.6	20	18.7	.992
Diabetes mellitus	74	15.5	55	14.9	19	17.8	.467
Hypertension	173	36.3	131	35.4	42	39.3	.466
Hypercholesterolemia	182	38.2	136	36.8	46	43.0	.242
History of smoking	362	75.9	278	75.1	84	78.5	.473
Current smoker	200	41.9	153	41.4	47	43.9	.635
12-month all-cause mortality	33	6.9%	6	5.6%	27	7.3%	.544
	Mean	SD	Mean	SD	Mean	SD	P
Age (years)	60.1	12.2	60.2	12.2	59.6	12.3	.658
BDI- II Total Score	9.2	7.9	5.7	3.7	21.3	6.8	<.001

Table 2. Summary of Results from Confirmatory Factor Analyses

Model	Factor Correlation	χ^2	*df*	p	CFI	TLI	RMSEA	SRMR
SA-C	.80	258.6	77	< .01	.92	.96	.07	.08
CA-S	.72	296.1	76	< .01	.90	.96	.08	.08
G-S-C	-----	**256.1**	**76**	**< .01**	**.92**	**.96**	**.07**	**.07**
G-S-C Simplified	-----	**260.4**	**77**	**< .01**	**.92**	**.96**	**.07**	**.07**

CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; RMSEA = Root Mean Square Error of Approximation; SRMR = Standardized Root Mean Square Residual; SA-C = Somatic/Affective – Cognitive; CA-S = Cognitive/Affective – Somatic; G-S-C = General – Somatic – Cognitive;

Table 3. Standardized Factor Loadings and Communalities from the G-S-C Model.

BDI Item		G	S	C	SC	An	h^{2a}
1	Sadness	.69					.51
2	Pessimism	.74		.11[b]			.60
3	Past Failure	.60		.55			.60
4	Loss of Pleasure	.75				.29	.60
5	Guilty Feelings	.55		.51			.54
6	Punishment Feelings	.53		.56			.57
7	Self-Dislike	.71		.42	.30		.73
8	Self-Criticalness	.61		.39	.30		.55
9	Suicidal Ideation	.59		.43			.51
10	Crying	.71					.49
11	Agitation	.62					.40
12	Loss of Interest	.79				.29	.64
13	Indecisiveness	.77					.56
14	Worthlessness	.71		.29			.61

15	Loss of Energy	.51	.62			.73
16	Sleep Problems	.53	.19			.34
17	Irritability	.57				.38
18	Appetite	.54	.25			.35
19	Concentration	.65	.24			.47
20	Fatigue	.51	.82			.78
21	Sexual Disinterest	.45				.30
	Communality[†]	**.73**	**.11**	**.13**	**.01**	**.01**	

G = General; S = Somatic; C = Cognitive; SC = Self-Criticism; An = Anhedonia; h^2 = item communality. All factor loadings are significant at $p < .05$ except where noted.

[a]Communality (h^2) for each factor is the proportion of total communality that is attributable to the factor.

[b]Factor loading not significant (p = 10).

Figure 1. General-Somatic-Cognitive Confirmatory Factor Model