Psychological Medicine (2008), **38**, 257–264. © 2007 Cambridge University Press
doi:10.1017/S0033291707001377 Printed in the United Kingdom

ORIGINAL ARTICLE

Cardiac history, prior depression and personality predict course of depressive symptoms after myocardial infarction

E. J. Martens[1]*, O. R. F. Smith[1], J. Winter[2], J. Denollet[1] and S. S. Pedersen[1]

[1] *CoRPS – Center of Research on Psychology in Somatic Diseases, Tilburg University, The Netherlands*
[2] *Department of Cardiology, TweeSteden Hospital Tilburg, The Netherlands*

Background. Although many studies have focused on post-myocardial infarction (MI) depression, there is limited information about the evolution and determinants of depressive symptoms in the first year post-MI. Therefore we examined (1) the course of depressive symptoms during the first year post-MI and (2) the predictors of these symptom trajectories.

Method. To assess depressive symptoms, 287 patients completed the Beck Depression Inventory during hospitalization for MI, and 2, and 12 months post-MI. Personality was assessed with the Type-D scale during hospitalization. We used latent class analysis to examine the evolution of depressive symptoms over a 1-year period and multinomial logit regression analyses to examine predictors of these symptom trajectories.

Results. The course of depressive symptoms was stable during the first year post-MI. Four groups were identified and classified as non-depressed [40%, intercept (IC) 2.52], mildly depressed (42%, IC 6.91), moderately depressed (14%, IC 13.73) or severely depressed (4%, IC 24.54). In multivariate analysis, cardiac history ($\log OR_{severe}$ 2.93, $p=0.02$; $\log OR_{moderate}$ 1.81, $p=0.02$; $\log OR_{mild}$ 1.46, $p=0.01$), history of depression ($\log OR_{severe}$ 4.40, $p<0.001$; $\log OR_{moderate}$ 1.97, $p=0.03$) and Type-D personality ($\log OR_{severe}$ 4.22, $p<0.001$; $\log OR_{moderate}=4.17$, $p<0.001$; $\log OR_{mild}$ 1.66, $p=0.02$) were the most prominent risk factors for persistence of depressive symptoms during the first year post-MI.

Conclusions. Symptoms of depression tend to persist during the first year post-MI. Cardiac history, prior depression and Type-D personality were identified as independent risk factors for persistence of depressive symptoms. The results of this study strongly argue for routine psychological screening during hospitalization for acute MI in order to identify patients who are at risk for chronicity of depressive symptoms and its deleterious effects on prognosis.

Received 2 January 2007; Revised 15 June 2007; Accepted 22 June 2007; First published online 17 September 2007

Key words: Chronicity, depressive symptoms, myocardial infarction, persistence, Type-D personality.

Introduction

Ischaemic heart disease and depression are two of the most prevalent conditions and causes of disability and early death in industrialized countries (Murray & Lopez, 1996, 1997). Furthermore, depression is a common co-morbid disorder with acute myocardial infarction (MI) (Schleifer *et al*. 1989; Frasure-Smith *et al*. 1993). In many well-designed studies, depression has been associated with a two- to fourfold increased risk of adverse clinical outcomes and impaired health status in post-MI patients (Ladwig *et al*. 1994; Frasure-Smith *et al*. 1995; Barefoot *et al*. 1996; Spertus *et al*. 2000; Lespérance *et al*. 2002; Rumsfeld *et al*. 2003; De Jonge *et al*. 2006). Hence, the American College of Cardiology/American Heart Association, and the European Society of Cardiology practice guidelines recommend that the psychosocial status of patients be evaluated, 'including inquiries regarding symptoms of depression', and that 'efforts to relieve stress should be emphasized whenever possible' (De Backer *et al*. 2003; Antman *et al*. 2004).

Despite a growing body of literature stressing the importance of post-MI depression in the pathogenesis of coronary artery disease, there is a paucity of research on the evolution and persistence of depressive symptoms following MI (Schleifer *et al*. 1989). Although evidence suggests that depression is highly prevalent and persistent during the first 12 months post-MI (Davis & Jensen, 1988; Lespérance *et al*. 1996; Lane *et al*. 2002; Lauzon *et al*. 2003), most studies have used relatively short follow-up periods, dichotomous

* Address for correspondence: E. J. Martens, Ph.D., CoRPS, Department of Medical Psychology, Tilburg University, PO Box 90153, 5000 LE Tilburg, The Netherlands.
(Email: e.j.martens@uvt.nl)

This work was presented at the European Conference on Psychosomatic Research, 27 September 2006, Dubrovnik, Croatia.

data, and looked at prevalence rates across time rather than symptom patterns. Accordingly, the question remains if patients exhibit divergent profiles of recovery, as reflected by different symptom levels and course trajectories post-MI.

In a recent report of the National Heart, Lung, and Blood Institute Working Group on Outcomes Research in Cardiovascular Disease, the importance of promoting patient-centred care and its determinants was emphasized, i.e. clinicians need to attend to patients' physical and emotional needs in order to maintain or improve their quality of life (Krumholz *et al*. 2005). Since depression has a major impact on patients' physical and emotional needs (Ladwig *et al*. 1994; Thombs *et al*. 2006) and quality of life (Goyal *et al*. 2005), it is important to determine which factors predict the course of depressive symptoms in post-MI patients. Knowledge of these determinants may point to targets for psychosocial intervention and lead to the design of more successful intervention trials. The two recent randomized controlled trials, ENRICHD (Berkman *et al*. 2003) and SADHART (Glassman *et al*. 2002), showed that a reduction in depressive symptoms did not translate into beneficial effects on survival, although benefits were found in quality of life (Swenson *et al*. 2003). Hence, it remains unclear how to enhance secondary prevention in cardiac patients with depressive co-morbidity. A detailed analysis of the course of post-MI depressive symptoms and its predictors could help in determining the optimal timing for assessment of depression and might guide treatment efforts for depression in post-MI patients.

The aim of the present study was to examine the course of depressive symptoms during the first year post-MI and predictors of these symptom trajectories, using a sound and sophisticated statistical approach and evaluating a wide array of demographic, medical, and psychological variables as possible predictors of those trajectories.

Method

Study design and patient population

Between May 2003 and February 2005, 287 patients hospitalized for acute MI were included from four teaching hospitals (Catharina Hospital, Eindhoven; St Elisabeth Hospital, Tilburg; TweeSteden Hospital, Tilburg; and St Anna Hospital, Geldrop) in The Netherlands. MI was defined according to the following criteria: troponin I levels more than twice the upper limit, with typical ischaemic symptoms (e.g. chest pain) lasting for more than 10 min or ECG evidence of ST segment elevation or new pathological Q-waves.

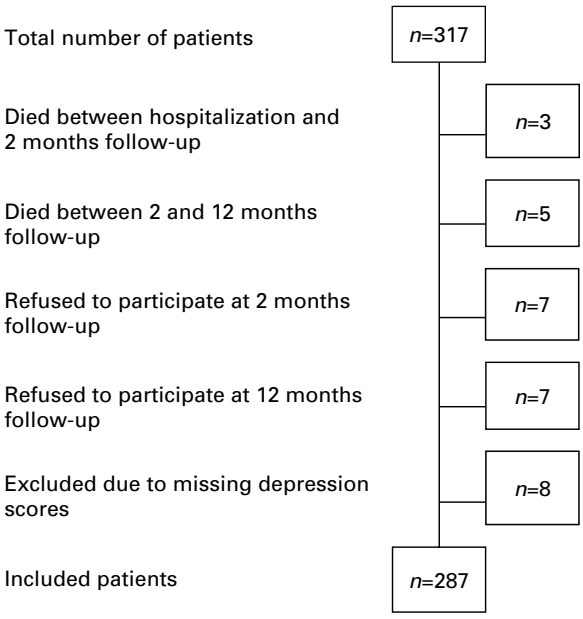

Fig. 1. Flowchart of patient selection.

Patients with significant cognitive impairments (e.g. dementia) and severe co-morbidities (e.g. cancer) were excluded. Patients were assessed during the initial hospitalization for MI, and 2 and 12 months post-MI in the cardiology department of the participating hospitals. The research protocol was approved by the medical ethics committees of the participating hospitals, and the study was conducted in accordance with the Helsinki Declaration. After a complete description of the study to the patients, written informed consent was obtained.

Patients completed self-report measures of depressive symptoms during hospitalization, and at 2 and 12 months follow-up. In addition, they were assessed on Type-D personality and lifetime diagnosis of major depressive disorder (MDD). Demographic and clinical variables were obtained from the medical records. Of the original 317 patients, 287 patients were included in the final analyses (Fig. 1).

Measures

Demographic and clinical characteristics

Demographic variables included age, sex, marital status, educational level, and occupational status. Clinical variables included co-morbidity (defined as arthritis, renal insufficiency or chronic obstructive pulmonary disease), cardiac history [defined as MI, angina, percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) surgery prior to the MI], multi-vessel disease, diabetes mellitus, PCI *versus* conservative treatment, anterior MI location,

https://doi.org/10.1017/S0033291707001377 Published online by Cambridge University Press

obesity (BMI $\geqslant$30), smoking status (self-report), participation in cardiac rehabilitation, cardiac medications [β-blockers, angiotensin-converting enzyme (ACE) inhibitors, anticoagulants, statins, diuretics, and aspirin] and psychotropics.

Depressive symptoms

The Beck Depression Inventory (BDI) is a 21-item self-report measure developed to assess the presence and severity of depressive symptoms (Beck & Steer, 1993). Each item is rated on a 0–3 scale. A total score is obtained by adding up all item scores. The BDI is a reliable and well-validated measure of depressive symptomatology with a Cronbach's α of 0.81 in non-psychiatric samples (Beck *et al*. 1988; Welch *et al*. 1990), and is a widely used self-report measure of depression. A BDI score $\geqslant$10 is indicative of at least mild to moderate symptoms of depression and has been associated with poor prognosis in MI patients (Frasure-Smith *et al*. 1995, 1999; Lespérance *et al*. 2002).

Clinical diagnoses of depression

The Composite International Diagnostic Interview (CIDI; WHO, 1990) was used to assess a lifetime diagnosis of MDD based on DSM-IV diagnostic criteria (APA, 1994).

Type-D personality

The 14-item Type-D scale (DS14) was included to assess Type-D personality (Denollet, 2005). Items are answered on a 5-point Likert scale from 0 to 4. The scale consists of two 7-item subscales, negative affectivity (e.g. 'I often feel unhappy') and social inhibition (e.g. 'I am a closed person'). Patients were categorized as Type-D using a standardized cut-off score $\geqslant$10 on the negative affectivity and social inhibition subscales (Denollet, 2005). The DS14 is a valid and reliable scale with a Cronbach's α of 0.88/0.86 and a test–retest reliability over a 3-month period with $r = 0.72/0.82$ for the two subscales, respectively (Denollet, 2005). Type-D has been associated with a four- to eightfold increased risk of adverse clinical outcome (Denollet *et al*. 1996, 2000; Pedersen *et al*. 2004; Denollet, 2005) and comprises a risk factor for the onset of depressive symptoms 12 months post-PCI (Pedersen *et al*. 2006).

Statistical analysis

Latent class analysis (SAS procedure TRAJ) was used to examine trajectories of depressive symptoms in post-MI patients over a 1-year period (Jones *et al*. 2001). TRAJ fits a finite mixture model to identify groups of individuals following similar patterns of behaviour over time. The time-course of each trajectory is modelled using a polynomial with a maximum order of 3. Subsequently, associations between group membership and covariates can be examined. To determine the optimal number of trajectories, the Bayesian Information Criterion (BIC) was used, with a higher BIC indicating a better fit. BIC is often used for comparing models, as it trades off model fit and model complexity. The optimal polynomial order of each trajectory was examined. Due to the number of measurement points, only constant, linear and quadratic parameters were used. The significance of all parameters (intercept, linear, quadratic) was investigated. If the quadratic parameter did not reach significance, it was dropped from the analysis and was tested for a linear trajectory. If the linear parameter did not reach significance, it was tested for a constant trajectory. Again, models were compared using the BIC. Multinomial logit regression was used to evaluate demographic, medical, and psychological variables as possible predictors of trajectories of depressive symptoms. Variables that were significant at $p < 0.05$ in univariate analysis were evaluated in a final analysis by entering them simultaneously into a multivariate model.

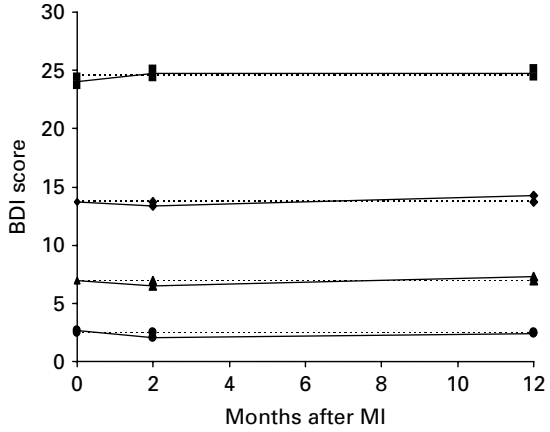

Fig. 2. Trajectories of depressive symptoms (- - -, predicted; ——, observed) in post-myocardial infarction (MI) patients. Severely depressed (■); moderately depressed (◆); mildly depressed (▲); non-depressed (●).

Results

Prevalence and course of depressive symptoms

Based on the BIC criteria, a four-group model was found to be the best fitting model. This model was defined by intercept only. Quadratic and linear parameters were not significant. The level of depressive symptoms over time was stable in this sample of post-MI patients (Fig. 2) The first group (40%) was classified as the non-depressed group with a predicted

https://doi.org/10.1017/S0033291707001377 Published online by Cambridge University Press

Table 1. *Univariate predictors of depressive symptom persistence*[a]

	Mildly depressed			Moderately depressed			Severely depressed		
Covariate	Log OR	S.E.	*p*	Log OR	S.E.	*p*	Log OR	S.E.	*p*
Cardiac history[b]	**1.44**	**0.51**	**0.005**	**1.78**	**0.56**	**0.002**	**2.70**	**0.80**	**0.001**
Depression history	N.S.	N.S.	N.S.	**1.52**	**0.55**	**0.006**	**3.71**	**0.90**	**<0.001**
Type-D personality	**1.52**	**0.65**	**0.02**	**3.20**	**0.66**	**<0.001**	**3.10**	**0.84**	**<0.001**
Female sex	N.S.	N.S.	N.S.	**1.52**	**0.48**	**0.002**	**1.51**	**0.70**	**0.03**
Age >60 yr	N.S.	N.S.	N.S.	**0.97**	**0.43**	**0.03**	N.S.	N.S.	N.S.
Single	N.S.	N.S.	N.S.	**1.04**	**0.47**	**0.03**	**1.52**	**0.69**	**0.03**
High educational level	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Working	N.S.	N.S.	N.S.	**−1.35**	**0.51**	**0.009**	N.S.	N.S.	N.S.
Smoking[c]	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Obesity[d]	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Cardiac rehabilitation	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Anterior MI location	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Multi-vessel disease	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
PCI[e]	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Diabetes	**1.19**	**0.53**	**0.03**	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Co-morbidity[f]	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	**1.96**	**0.68**	**0.004**
β-blockers	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
ACE inhibitors	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Anticoagulants	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Statins	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Diuretics	N.S.	N.S.	N.S.	**1.92**	**0.49**	**<0.001**	N.S.	N.S.	N.S.
Aspirin	**−1.53**	**0.67**	**0.02**	**−1.64**	**0.67**	**0.01**	N.S.	N.S.	N.S.
Psychotropics	N.S.	N.S.	N.S.	**1.57**	**0.54**	**0.004**	**2.31**	**0.65**	**<0.001**

MI, Myocardial infarction; PCI, percutaneous coronary intervention; ACE, angiotensin-enzyme converting; OR, odds ratio; S.E., standard error; N.S., not significant.

[a] Non-depressed group was used as reference category.

[b] MI, angina, PCI or coronary artery bypass graft prior to the MI.

[c] Self-report.

[d] BMI ⩾30.

[e] Reference group: conservatively treated.

[f] Arthritis, renal insufficiency, chronic obstructive pulmonary disease.

mean BDI score of 2.52 (95% CI 1.69–3.35). The level of depressive symptoms in the second group (42%) was slightly higher than in the first group with a mean BDI score of 6.91 (95% CI 5.72–8.11); hence, this group was classified as mildly depressed. According to the standardized BDI cut-off score of ⩾10, groups 3 and 4 displayed significant depressive symptomatology. Group 3 (14%) had a predicted mean BDI score of 13.73 (95% CI 12.46–14.99) and was therefore classified as moderately depressed. The fourth group (4%) was described as severely depressed, with a mean BDI score of 24.54 (95% CI 22.88–26.20). Accordingly, based on the standardized BDI cut-off score of ⩾10, the prevalence rate of depressive symptoms during the first year post-MI was 18%.

The derived finite mixture model suggests a stable course of depressive symptoms in post-MI patients. In order to validate this result, change in depression scores was examined at 2 and 12 months using the baseline depression score as a reference. A clinically significant decrease at follow-up was defined as having a BDI score <10 *and* a 50% decrease in BDI score. A clinically significant increase at follow-up was defined as having a BDI score ⩾10 *and* a 50% increase in BDI score. Only a minority of patients improved (severe group, $n=0$; moderate group, $n=6$) or deteriorated (mild group, $n=29$; non-depressed group, $n=1$) respectively. This suggests that even though there were clinically significant changes at the individual level, at group level the course of depressive symptoms was indeed stable.

Predictors of depressive symptom persistence

Univariate predictors of depressive symptom persistence are presented in Table 1. The estimates reported

https://doi.org/10.1017/S0033291707001377 Published online by Cambridge University Press

Table 2. *Multivariate predictors of depressive symptom persistence*[a]

	Mildly depressed			Moderately depressed			Severely depressed		
Covariate	Log OR	S.E.	*p*	Log OR	S.E.	*p*	Log OR	S.E.	*p*
Cardiac history[b]	**1.46**	**0.58**	**0.01**	**1.81**	**0.77**	**0.02**	**2.93**	**1.21**	**0.02**
Depression history	N.S.	N.S.	N.S.	**1.97**	**0.89**	**0.03**	**4.40**	**1.26**	**<0.001**
Type-D personality	**1.66**	**0.68**	**0.02**	**4.17**	**0.85**	**<0.001**	**4.22**	**1.13**	**<0.001**
Female sex	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Age >60 yr	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	−2.12	1.09	0.05
Single	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Working	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Diabetes	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Co-morbidity[c]	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	**2.78**	**1.21**	**0.02**
Diuretics	N.S.	N.S.	N.S.	**1.88**	**0.80**	**0.02**	N.S.	N.S.	N.S.
Aspirin	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Psychotropics	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	2.02	1.01	0.05

OR, Odds ratio; S.E., standard error; N.S., not significant.

[a] The non-depressed group was used as the reference category.

[b] Myocardial infarction (MI), angina, percutaneous coronary intervention or coronary artery bypass graft prior to the MI.

[c] Arthritis, renal insufficiency, chronic obstructive pulmonary disease.

in Table 1 are log odds ratios (OR), using the non-depressed group as the reference category. Cardiac history was the most prominent clinical risk factor for depressive symptom persistence over the course of 1-year post-MI, with log odds varying from 1.44 to 2.70. In addition, history of MDD and Type-D personality had the highest associated risk, with log odds varying from 1.52 to 3.71. Other predictors varying according to the level of depressive symptoms comprised gender, age, marital status, occupational status, diabetes, co-morbidity, diuretics, aspirin and psychotropics.

Variables that were significantly associated with one of the symptom trajectories were entered into a multivariate model. As displayed in Table 2, patients with a cardiac history and Type-D personality were more likely to be in the mildly, moderately and severely depressed groups compared to the non-depressed group. Furthermore, patients with a history of MDD and patients taking diuretics were more likely to be in the moderately depressed group, whereas patients with a history of MDD and co-morbidities were more likely to be in the severely depressed group.

In *post-hoc* analyses, the risk of being in the mildly, moderately or severely depressed group was stratified by the most prominent risk factors cardiac history, history of MDD and Type-D personality. There was a continuous relationship between the severity of depression group and number of risk factors, with the largest risk incurred in patients with all three risk factors.

Discussion

The aim of the current study was to examine the course of depressive symptoms and its predictors during the first year post-MI. Depressive symptoms were highly prevalent and the course of depressive symptoms stable. Four trajectories were observed, namely non-depressed, mildly depressed, moderately depressed and severely depressed. Cardiac history, history of MDD and Type-D personality comprised the most prominent risk factors for experiencing depressive symptoms across the trajectories, with patients with all three risk factors being at highest risk for the moderately and severely depressive symptom trajectories.

The present results indicate that symptoms of depression following MI are not a transient phenomenon, with levels of depressive symptomatology persisting during the course of the first year post-MI. Although a minority of patients showed a significant change in the level of depressive symptoms at group level the course of depressive symptoms was stable. These results are in line with a recent review on the prevalence of depression in post-MI patients, showing that the majority of MI patients with depression during initial hospitalization remain depressed 1–12 months later (Thombs *et al*. 2006). In addition, in a prospective study of 288 MI patients, depressive symptoms were found to be prevalent and persistent, with only 6.7% of patients depressed in hospital experiencing no further symptoms of depression during the first year post-MI (Lane *et al*. 2002). Although our

https://doi.org/10.1017/S0033291707001377 Published online by Cambridge University Press

results are consistent with previous studies, the majority of these studies had a relatively short follow-up period, used dichotomous data and did not assess symptom patterns but rather looked at prevalence rates across time.

One recent study, however, used the same methodology as applied in the current study to examine the course of depressive symptoms during the first year post-MI (Kaptein *et al*. 2006). They identified five distinct courses in 475 post-MI patients, with 18% of patients experiencing elevated depressive symptom levels during the first year post-MI. These findings are fairly consistent with our results, even though they did find unstable trajectories. It should, however, be borne in mind that they had a considerably larger sample size making it more likely to find significant linear and quadratic parameters, and that the timing of the follow-up assessments was different (i.e. 3, 6, and 12 months in the Kaptein study *versus* 2, and 12 months in the current paper). The percentage of patients in their unstable groups was quite small (<10%), confirming our findings that the majority of post-MI patients display stable patterns of depressive symptoms over time. In *post-hoc* analysis, we made an effort to validate our model by examining changes using the standardized BDI cut-off. Those results indicated that only a minority of patients improved or deteriorated respectively. Moreover, Kaptein *et al*. (2006) showed that history of MDD and several psychological measures (e.g. neuroticism) played an important role in predicting a significantly greater risk for severe/persistent symptom trajectories. Patients with persistent symptoms, which were of at least moderate intensity during all follow-up assessments, had the highest rate of new cardiovascular events, indicating the importance of examining the course of depressive symptoms.

Additionally, our findings are compatible with the study by Spijkerman and colleagues reporting that personality was strongly related with ongoing and recurrent depression in 468 post-MI patients (Spijkerman *et al*. 2005*a*). Cardiac history, however, was not found to be a significant predictor of depression following MI, although some trends were observed (Spijkerman *et al*. 2005*a*; Van Melle *et al*. 2006). In another recent study, prior MI was associated with depressive symptoms post-MI, irrespective of whether they persisted, subsided, or newly developed in the first month after hospitalization (Parashar *et al*. 2006). In addition, history of MDD has been found to be a strong independent predictor of the development of post-MI depressive symptoms (Spijkerman *et al*. 2005*b*; Parashar *et al*. 2006).

Insight into the course of depressive symptoms after MI and its predictors is important for clinical practice. First, the chronicity of post-MI depressive symptoms found in the present study strongly argues for routine psychological screening during hospitalization for acute MI. Hospitalization is not only the most convenient moment to screen patients for depression, but early identification and treatment of depression may also prevent symptoms from becoming chronic and improve patients' quality of life. Since depressed cardiac patients have a higher risk of morbidity, mortality and impaired health status after MI (Frasure-Smith *et al*. 1993, 1995; Ladwig *et al*. 1994; Barefoot *et al*. 1996; Lespérance *et al*. 1996; Spertus *et al*. 2000; Grace *et al*. 2005; Parashar *et al*. 2006), it is important to identify patients with elevated levels of depressive symptoms as early as possible. Those patients should be considered for treatment, be it pharmacological or psychological or a combination thereof. Furthermore, knowledge about the predictors of depressive symptom trajectories may point to targets for psychosocial intervention and lead to the design of more successful intervention trials. The management of depression in MI patients is both a clinical and interdisciplinary challenge, with the development of collaborative and integrative approaches combining the medical and psychological expertise being imperative. However, an important first step in the management of post-MI depression comprises the unravelling of the determinants of depression, which probably consist of a combination of clinical, psychosocial, and biological factors. Studying the biological determinants in future studies may be of particular value, as treatment options for post-MI depression should also have a positive effect on cardiovascular biology (Von Känel & Begré, 2006).

The results of the current study should be interpreted with some caution. First, we had no information on left ventricular ejection fraction. However, we included multi-vessel disease to adjust for disease severity. Second, only 4% of patients were in the severely depressed group, which may have led to unreliable results due to the relatively small number of patients within this particular group. Nevertheless, the most prominent risk factors were consistent across trajectories. Third, prior to setting up a multivariate model to predict depressive symptom trajectories, we evaluated a large number of predictors in univariate analysis. Although most of the variables have been associated with depressive symptoms in previous studies, this procedure may have let to overfitting of our regression model. Finally, cardiac history was defined as MI, angina, PCI, or CABG surgery prior to the MI. Since negative affectivity has been associated with thoracic pain unrelated to cardiac iscahemia, it is possible that a higher level of negative affectivity has contributed to misdiagnosed angina in this study. Yet,

all clinical variables were obtained from the patients' medical records.

Depressive symptoms following MI are not a transient phenomenon, with levels of depressive symptoms persisting throughout the first year post-MI. Cardiac history, history of MDD and Type-D personality were the most prominent risk factors for depressive symptom trajectories. These findings support the importance of including both clinical and psychological variables in cardiovascular research. Since this is one of the first studies to look at trajectories of depressive symptoms in the year following MI and its determinants, further research is warranted to replicate these findings using this new methodology to describe courses of post-MI depressive symptoms. However, the results of this study strongly argue for routine psychological screening during hospitalization for acute MI in order to identify patients who are at risk for chronicity of depressive symptoms and its deleterious effects on prognosis.

Declaration of Interest

None.

References

APA (1994). *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn. American Psychiatric Association: Washington, DC.

Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, Hochman JS, Krumholz HM, Kushner FG, Lamas GA, Mullany CJ, Ornato JP, Pearle DL, Sloan MA, Smith Jr. SC, Alpert JS, Anderson JL, Faxon DP, Fuster V, Gibbons RJ, Gregoratos G, Halperin JL, Hiratzka LF, Hunt SA, Jacobs AK, Ornato JP (2004). ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of patients with acute myocardial infarction). *Journal of the American College of Cardiology* **44**, E1–E211.

Barefoot JC, Helms MJ, Mark DB (1996). Depression and long-term mortality risk in patients with coronary artery disease. *American Journal of Cardiology* **78**, 613–617.

Beck AT, Steer RA (1993). *Manual for the revised Beck Depression Inventory*. Psychological Corporation: San Antonio, USA.

Beck AT, Steer RA, Garbin MC (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review* **8**, 77–100.

Berkman LF, Blumenthal J, Burg MM, Carney RM, Catellier D, Cowan MJ, Czajkowski SM, De Busk R, Hosking J, Jaffe A, Kaufmann PG, Mitchell P, Norman J, Powell LH, Raczynski JM, Schneidermann N, Writing Committee for the ENRICHD Investigators (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.

Davis T, Jensen L (1988). Identifying depression in medical patients. *Journal of Nursing Scholarship* **20** 191–195.

De Backer G, Ambrosioni E, Borch-Johnsen K, Brotons C, Cifkova R, Dallongeville J, Embrahim S, Faergeman O, Graham I, Mancia G, Cats VM, Orth-Gomer K, Perk J, Pyorala K, Rodicio JL, Sans S, Sansoy V, Sechtem U, Silber S, Thomsen T, Wood D (2003). European guidelines on cardiovascular disease prevention in clinical practice: Third Joint Task Force of European and other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of eight societies and by invited experts). *European Heart Journal* **24**, 1601–1610.

De Jonge P, Spijkerman T, Avan den Brink RH, Ormel J (2006). Depression after myocardial infarction is a risk factor for declining health related quality of life and increased disability and cardiac complaints at 12 months. *Heart* **92**, 32–39.

Denollet J (2005). DS14. Standard assessment of negative affectivity, social inhibition, and Type D personality. *Psychosomatic Medicine* **67**, 89–97.

Denollet J, Sys SU, Stroobant N, Rombouts H, Gillebert TC, Brutsaert DL (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *Lancet* **347**, 417–421.

Denollet J, Vaes J, Brutsaert DL (2000). Inadequate response to treatment in coronary heart disease: adverse effects of type D personality and younger age on 5-year prognosis and quality of life. *Circulation* **102**, 630–635.

Frasure-Smith N, Lespérance F, Juneau M, Talajic M, Bourassa MG (1999). Gender, depression, and one-year prognosis after myocardial infarction. *Psychosomatic Medicine* **61**, 26–37.

Frasure-Smith N, Lespérance F, Talajic M (1993). Depression following myocardial infarction: impact on 6-month survival. *Journal of the American Medical Association* **270**, 1819–1825.

Frasure-Smith N, Lespérance F, Talajic M (1995). Depression and 18-month prognosis after myocardial infarction. *Circulation* **91**, 999–1005.

Glassman AH, O'Connor CM, Califf RM, Swedberg K, Schwartz P, Bigger Jr. JT, Krishnan KR, van Zyl LT, Swenson JR, Finkel MS, Landau C, Shapiro PA, Pepine CJ, Mardekian J, Harrison WM, Barton D, Mclvor M, for the Sertraline Antidepressant Heart Attack Randomized Trial (SADHART) Group (2002). Sertraline treatment of major depression in patients with acute MI or unstable angina. *Journal of the American Medical Association* **288**, 701–709.

Goyal TM, Idler EL, Krause TJ, Contrada RJ (2005). Quality of life following cardiac surgery: Impact of the severity and course of depressive symptoms. *Psychosomatic Medicine* **67**, 759–765.

Grace SL, Abbey SE, Kapral MK, Fang J, Nolan RP, Stewart DE (2005). Effect of depression on five-year

mortality after an acute coronary syndrome. *American Journal of Cardiology* **96**, 1179–1185.

Jones BL, Nagin DS, Roeder K (2001). A SAS procedure based on mixture models for estimating developmental trajectories. *Sociological Methods & Research* **29**, 374–393.

Kaptein KI, de Jonge P, van den Brink RH, Korf J (2006). Course of depressive symptoms after myocardial infarction and cardiac prognosis: a latent class analysis. *Psychosomatic Medicine* **68**, 662–668.

Krumholz HM, Peterson ED, Ayanian JZ, Chin MH, De Busk RF, Goldman L, Kiefe CI, Powe NR, Rumsfeld JS, Spertus JA, Weintraub WS (2005). National Heart, Lung, and Blood Institute working group. Report of the National Heart, Lung, and Blood Institute working group on outcomes research in cardiovascular disease. *Circulation* **111**, 3158–3166.

Ladwig KH, Roll G, Breithardt G (1994). Post-infarction depression and incomplete recovery 6 months after acute myocardial infarction. *Lancet* **343**, 20–23.

Lane D, Carroll D, Ring C, Beevers DG, Lip GY (2002). The prevalence and persistence of depression and anxiety following myocardial infarction. *British Journal of Health Psychology* **7**, 11–21.

Lauzon C, Beck CA, Huynh T, Dion D, Racine N, Carignan S, Diodati JG, Charbonneau F, Dupuis R, Pilote L (2003). Depression and prognosis following hospital admission because of acute myocardial infarction. *Canadian Medical Association Journal* **168**, 547–552.

Lespérance F, Frasure-Smith N, Talajic M (1996). Major depression before and after myocardial infarction: its nature and consequences. *Psychosomatic Medicine* **58**, 99–110.

Lespérance F, Frasure-Smith N, Talajic M, Bourassa MG (2002). Five-year risk of cardiac mortality in relation to initial severity and one-year changes in depression symptoms after myocardial infarction. *Circulation* **105**, 1049–1053.

Murray CJ, Lopez AD (1996). *The Global Burden of Disease*. Harvard University Press: Cambridge, MA.

Murray CJ, Lopez AD (1997). Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. *Lancet* **349**, 1436–1442.

Parashar S, Rumsfeld JS, Spertus JA, Reid KJ, Wenger NK, Krumholz HM, Amin A, Weintraub WS, Lichtman J, Dawood N, Vaccarino V (2006). Time course of depression and outcome of myocardial infarction. *Archives of Internal Medicine* **166**, 2035–2043.

Pedersen SS, Lemos PA, van Vooren PR, Liu TK, Daemen J, Erdman RA, Smits PC, Serruys PW, van Domburg RT (2004). Type D personality predicts death or myocardial infarction after bare metal stent or sirolimus-eluting stent implantation: a rapamycin-eluting stent evaluated at rotterdam cardiology hospital (RESEARCH) registry substudy. *Journal of the American College of Cardiology* **44**, 997–1001.

Pedersen SS, Ong AT, Sonnenschein K, Serruys PW, Erdman RA, van Domburg RT (2006). Type D personality and diabetes predict the onset of depressive symptoms in patients after percutaneous coronary intervention. *American Heart Journal* **151**, 367E1–E6.

Rumsfeld JS, Havranek E, Masoudi FA, Peterson ED, Jones P, Tooley JF, Krumholz HM, Spertus JA, Cardiovascular Outcomes Research Consortium (2003). Depressive symptoms are the strongest predictors of short-term declines in health status in patients with heart failure. *Journal of the American College of Cardiology* **42**, 1811–1817.

Schleifer SJ, Macari-Hinson MM, Coyle DA, Slater WR, Kahn M, Gorlin R, Zucker HD (1989). The nature and course of depression following myocardial infarction. *Archives of Internal Medicine* **149**, 1785–1789.

Spertus JA, McDonell M, Woodman CL, Fihn SD (2000). Associations between depression and worse disease-specific functional status in outpatients with coronary artery disease. *American Heart Journal* **140**, 105–110.

Spijkerman T, de Jonge P, van den Brink RHS, Jansen JH, May JF, Crijns HJ, Ormel J (2005*a*). Depression following myocardial infarction: first-ever versus ongoing and recurrent episodes. *General Hospital Psychiatry* **27**, 411–417.

Spijkerman T, van den Brink RHS, Jansen JHC, Crijns HJ, Ormel J (2005*b*). Who is at risk of post-MI depressive symptoms? *Journal of Psychosomatic Research* **58**, 425–432.

Swenson J, O'Connor S, Barton D, van Zyl LT, Swedberg K, Forman LM, Gaffney M, Glassman AH (2003). Influence of depression and effect of treatment with sertraline on quality of life after hospitalization for acute coronary syndrome. *American Journal of Cardiology* **92**, 1271–1276.

Thombs BD, Bass EB, Ford DE, Stewart KJ, Tsilidis KK, Patel U, Fauerbach JA, Bush DE, Ziegelstein RC (2006). Prevalence of depression in survivors of acute myocardial infarction. Review of the evidence. *Journal of General and Internal Medicine* **21**, 30–38.

Van Melle JP, de Jonge P, Kuyper AMG, Honig A, Schene AH, Crijns HJ, van den Berg MP, van Veldhuisen DJ, Ormel J, MIND-IT Investigators (2006). Prediction of depressive disorder following myocardial infarction. Data from the Myocardial INfarction and Depression-Intervention Trial (MIND-IT). *International Journal of Cardiology* **109**, 88–94.

Von Känel R, Begré S (2006). Depression after myocardial infarction: Unraveling the mystery of poor cardiovascular prognosis and role of beta-blocker therapy. *Journal of the American College of Cardiology* **48**, 2215–2217.

Welch G, Hall A, Walkey F (1990). The replicable dimensions of the Beck Depression Inventory. *Journal of Clinical Psychology* **46**, 817–827.

WHO (1990). *Composite International Diagnostic Interview* (*CIDI*). World Health Organisation: Geneva, Switzerland.