

What predicts depression in cardiac patients: Sociodemographic factors, disease severity or theoretical vulnerabilities?

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What predicts depression in cardiac patients: Sociodemographic factors, disease severity or theoretical vulnerabilities?

Running title: Depressive vulnerabilities in cardiac patients

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Abstract

Depression is associated with increased cardiovascular risk in patients with acute coronary syndrome (ACS), but some argue that elevated depression is actually a marker of cardiovascular disease severity. Therefore, disease indices should be better predictors of depression than established theoretical causes of depression (interpersonal life events, reinforcing events, cognitive distortions, type D personality). However, little theory-based research has been conducted in this area. In a cross-sectional design, hospitalised ACS patients (n=336) completed questionnaires assessing depressive symptoms and vulnerabilities. Nested logistic regression assessed the relative contribution of demographic or vulnerability factors, or disease indices or vulnerabilities to depression. In multivariate analysis, all vulnerabilities were independent significant predictors of depression. Demographic variables accounted for <1% of the variance of depression status, with vulnerabilities accounting for significantly more (pseudo $R^2=.16$, $\chi^2(\text{change})=150.9$, $df=4$, $p<0.001$). Disease indices accounted for 7% of the variance in depressive status (pseudo $R^2=.07$, $\chi^2=137.9$, $p<0.001$). However, adding the vulnerabilities increased the overall variance explained to 22% (pseudo $R^2=.22$, $\chi^2=58.6$, $df=4$, $p<0.001$). Theoretical vulnerabilities predicted depression status better than did either demographic or disease indices. The presence of these proximal causes of depression suggests that depression in ACS patients is not simply a result of cardiovascular disease severity.

Keywords: Depression; acute coronary syndrome; psychological theory; life events; personality; just world beliefs

Introduction

In persons with coronary heart disease (CHD), the prevalence of depression is elevated in comparison to general population samples (Jacobi *et al.*, 2005; Thombs *et al.*, 2006). Furthermore, depression has been associated with poorer prognosis in such patients (Barth, Schumacher, & Herrmann-Lingen, 2004; Nicholson, Kuper, & Hemingway, 2006), but the treatment of such depression does not improve prognosis (The ENRICHD Investigators, 2003; van Melle *et al.*, 2007). Thus, some researchers have postulated that the association between depression and poorer prognosis simply reflects some confounding aspect of somaticised symptoms of disease severity. In this case, the symptoms of depression are assumed to arise from heart disease severity (Lane, Carroll, Ring, Beevers, & Lip, 2000; Lane, Lip, & Carroll, 2004), or patients are assumed to be simply adjusting naturally to a stressful life event (Frasure-Smith & Lesperance, 2003).

When assessing the prognostic value of depression, studies regularly include a wide range of variables (e.g. Carney, Freedland, Miller, & Jaffe, 2002; de Jonge *et al.*, 2006; Frasure-Smith & Lesperance, 2008; Frasure-Smith, Lesperance, & Talajic, 1993; Grace *et al.*, 2005; Lane, Carroll, Ring, Beevers, & Lip, 2002; Nicholson, Kuper, & Hemingway, 2006; The ENRICHD Investigators, 2003). However, there is generally little correlation between coronary disease severity and depression (Carney, Freedland, Miller, & Jaffe, 2002). Furthermore, when statistically controlling for other established risk factors, the association between depression and prognosis remains (Barth, Schumacher, & Herrmann-Lingen, 2004; Nicholson, Kuper, & Hemingway, 2006). However, with such heterogeneous assessment of coronary

disease indices, it is difficult to establish whether all appropriate variables have been assessed, or if any potential mediating variables have been omitted.

One alternative way to address this issue is to ask ‘What causes depression?’, rather than ‘What causes CHD?’. Much of the research into the association between depression and CHD has been atheoretical, and few studies have been conducted on the antecedents of depression in CHD patients. If depression is psychogenic, i.e. has its origin in theoretical proximal causes in CHD patients, then it can be argued that patients are depressed independently of CHD disease severity. Some of the well-established theoretical causes of depression in the general population are stressful life events, reduced positive reinforcement, and distorted cognitions (Beck, 1991; Lewinsohn & Gotlib, 1995; Paykel, 2003). These proximal causes of depression have corresponding theories of causality and evidence-based interventions, respectively: interpersonal theory, behavioural theory and cognitive theory (Beck, 1991; Davidson, Rieckmann, & Lesperance, 2004; Kanter, Callaghan, Landes, Busch, & Brown, 2004; Lewinsohn & Gotlib, 1995). A further vulnerability is personality type (Enns & Cox, 1997; Smith & MacKenzie, 2006).

As a higher prevalence of depression is seen in the CHD population, these vulnerabilities should be elevated in depressed cardiac patients (Davidson, Rieckmann, & Lesperance, 2004). Only one study has demonstrated that several of these theoretical depressive vulnerabilities are present in the depressed cardiac population. Rieckmann *et al.* (2006) assessed 314 acute coronary syndrome (ACS) patients for depression within one week of hospital admission and reported that both mildly (29%) and moderately/severely (18%) depressed patients showed higher levels

of cognitive (dysfunctional attitudes), interpersonal (role transitions) and behavioural (reduced number of pleasant events) vulnerabilities. Also, these vulnerabilities independently contributed to depressive symptoms, i.e. no one vulnerability was mediated by another, although the presence of more than one vulnerability increased the odds of being depressed. These vulnerabilities also predicted depression status independently of sociodemographic factors in a hierarchical logistic regression model. However, results also demonstrated that substantial proportions of mildly depressed (25%) and moderately/severely depressed (14%) did not demonstrate any elevated vulnerability.

The relative independence of the theoretical vulnerabilities highlights the complexity of depression in this population. The finding that 14-25% of ACS patients have no vulnerability is intriguing. However, it is possible that the authors did not consider two key factors – cardiovascular disease severity indices, such as left ventricular ejection fraction (LVEF), were not included in the analyses, and LVEF may be a crucial confounder of the depression-CHD relationship (Barth, Schumacher, & Herrmann-Lingen, 2004; Nicholson, Kuper, & Hemingway, 2006; van Melle et al., 2005); and ‘negative-prone’ personality was not considered as a vulnerability. Type D personality – the tendency to feel negative emotion across situations yet to inhibit these emotions – has been associated with increased depressive symptoms in several studies (Doyle, Conroy, & McGee, 2007; Pedersen & Denollet, 2006; Pedersen et al., 2006). Pedersen et al. (2006) found that type D personality assessed at 6 months post-event predicted the onset of depression in 542 patients 12 months after percutaneous coronary intervention. This was remarkable for two reasons. Firstly, none of the sample had depression at 6 months, and secondly, a new cardiac event occurring 6-12

months from the initial recruitment into the study was not associated with onset of depression. Thus, type D can be considered a vulnerability factor for depressive symptoms, but it is unknown if type D accounts for the other vulnerabilities described above, or the category of individuals who did not show any elevated vulnerability.

The aims of the present study were therefore: 1) to determine whether theoretical vulnerabilities to depression, including type D personality, independently predict depression status in patients with ACS; 2) to determine whether theoretical vulnerabilities independently predict depression status beyond demographic factors and cardiovascular disease severity indices. In other words, the study tests whether depressive symptoms are better accounted for by sociodemographic factors, disease severity indices or by theoretical vulnerabilities.

Method

Study design and participants

The methods have been reported previously (Doyle, Conroy, McGee, & Delaney, in press). This paper presents data from a subgroup of ACS patients who completed questionnaires on theoretical vulnerabilities. Briefly, a multi-centre cross-sectional design was used to determine the association of vulnerabilities with depression. Twelve hospitals participated. Ethical approval was obtained as appropriate. Consecutive patients with confirmed ACS (myocardial infarction or unstable angina) who were literate in English were recruited by coronary care staff to participate in the survey during their hospital stay. Patients were asked for consent to access their charts at 12-months post-hospitalisation, and completed a composite psychological questionnaire.

CAD risk factors assessed as co-variables

The main CAD risk factors assessed as co-variables in the present study included non-modifiable characteristics (e.g. older age, male sex, history of CHD or cardiovascular disease, socioeconomic status [as assessed by the proxy of having private health insurance]), and modifiable characteristics including lifestyle (smoking, elevated cholesterol, hypertension, diabetes). Disease severity indices were also assessed using clinical or procedural indices (ejection fraction, revascularisation procedures during hospital stay) or other proxy variables (length of hospital stay). Major co-morbidities were also recorded, and later recoded as per the Charlson Comorbidity Index (CCI) (Han-Yi Wang, Ghee Chew, Kung, Kun-Jung Chung, & Lee, 2007). As this index contains some of the risk factors already assessed (e.g. MI, diabetes), a modified total score was generated to indicate the CCI score without these variables.

Measures

Depressive symptoms

Hospital Anxiety and Depression Scale – Depression subscale (HADS-D)

The HADS was developed to identify psychological disturbances in general medical and non-psychiatric samples (Zigmond & Snaith, 1983). It aims not to achieve contamination by omitting somatic symptoms which may be prevalent in these samples. The HADS consists of a 7-item depression scale (HADS-D) and a 7-item anxiety scale (HADS-A). The HADS-A was not used here. The optimal cut-off score is >7 for the HADS-D (Bjelland, Dahl, Haug, & Neckelmann, 2002; Roberts, Bonnici, Mackinnon, & Worcester, 2001), and this threshold is adopted in the present study.

Beck Depression Inventory – Fast Screen (BDI-FS)

The BDI-FS is a 7-item screening instrument that minimises the possibility of yielding spuriously high estimates of depression for patients with medical problems by focusing on cognitive (as distinct from somatic) symptoms of DSM-IV criteria for depression (Beck, Guth, Steer, & Ball, 1997). Studies have consistently found that a BDI-FS cut-off score of >3 yields a sensitivity of >0.90 and specificity of >0.85 for detecting major depressive disorders (Beck, Steer, Ball, Ciervo, & Kabat, 1997; Scheinthal, Steer, Giffin, & Beck, 2001). In the present study, the suicidality item of the BDI-FS was omitted, for reasons outlined previously (Doyle, Conroy, McGee, & Delaney, in press; McGee, Doyle, Delaharpe, Shelley, & Conroy, 2006), with the recommended threshold of >3 maintained.

Maastricht Questionnaire – 10 item (MQ-10)

According to ICD-10 (World Health Organisation, 1993), a core diagnostic symptom of depression is fatigue. The above scales deliberately omit this symptom. ‘Vital exhaustion’ – a syndrome of fatigue, irritability and demoralisation – focuses mostly on fatigue (Doyle, Conroy, & McGee, 2007). However, the level of overlap between vital exhaustion and depression questions whether this construct is different from depression. Therefore, the brief 10-item version of the Maastricht Questionnaire (MQ-10; Appels, Hoppener, & Mulder, 1987) was considered a depression scale in the present study. The scale items are scored as binary (yes=1, no=0). The recommended cut-off score of >4 was used, as this has been shown to predict risk in both aetiological and prognostic studies (Appels, 2001).

Assessing depressive vulnerabilities

List of Threatening Experiences Questionnaire (LTE-Q)

The LTE-Q was used to assess the extent of interpersonal and life event difficulties that patients may have experienced in the year prior to their hospital admission. The LTE-Q is a 12-item scale which assesses stressful life events (Brugha, Bebbington, Tennant, & Hurry, 1985; Brugha & Cragg, 1990). Examples of life events on the LTE-Q are experiencing a serious illness or assault, or a relationship break-up. It has been shown to have high test-retest reliability in psychiatric patients, and compared well with an interview technique (sensitivity and specificity for stressful life events were between 0.89-1.0 and 0.74-0.88 respectively). The present study modified the question regarding serious illness (item 1: You yourself suffered a serious illness, injury or assault) to “*illness (not including this current hospitalisation), injury or assault*”.

Pleasant Events Schedule – Alzheimer’s Disease (short version) (PES-AD)

The PES-AD is a 20-item behavioural log that rates a number of pleasant events which people may enjoy doing. This scale was originally developed for persons with Alzheimer’s disease (Logsdon & Teri, 1997), but a variation has recently been used with ACS patients (Rieckmann et al., 2006). Ratings of the frequency of behaviours/events in the past month are understood to correspond to the rate of environmental engagement. Ratings of enjoyment are assumed to reflect positive reinforcement. As in previous research, missing items were coded as 0 if at least half of the 20 items had been answered (Rieckmann et al., 2006). A cross-product produced a total schedule score of positive reinforcement in the past month. As the

PES-AD is a schedule, not a scale, its items are theoretically independent. Thus, reliability statistics are meaningless.

Belief in a Just World – Self scale (BJW-S)

Cognitive distortions, as conceptualised by Beck (1991), were not considered as a theoretical causes of depression in the present study. Such cognitive distortions are only present in a minority of cardiac patients (Martens *et al.*, 2006; Rieckmann *et al.*, 2006), they do not predict cardiovascular prognosis (de Jonge *et al.*, 2006; Doyle, McGee, De La Harpe, Shelley, & Conroy, 2006), and the treatment of such symptoms with cognitive behavioural therapy does not reduce associated morbidity/mortality (The ENRICHD Investigators, 2003). However, a period of adjustment post-event is likely not only for those who have dysfunctional attitudes or distorted cognitions, but for most if not all patients. Therefore, what may be relevant is how all individuals adjust their world-view in regards to themselves and their place in the world post-ACS – rather than the more self-focused depressive vulnerabilities/cognitions that may be prevalent in a minority of persons. Therefore, the concept of belief in a just world (BJW) – the belief that good things happen to good people, and bad things happen to bad people (Furnham, 2003) – was assessed. Previous research has shown that a ‘distorted’ BJW – i.e. believing that the world is not just – was associated with depression (Furnham, 2003; Lipkus, Dalbert, & Siegler, 1996), and that just world beliefs in regard to the self (rather than to others or the population in general) predicted psychological well-being independently of personality (Lipkus, Dalbert, & Siegler, 1996). BJW for self is assessed by the BJW-S, which is an 8-item scale, with a 6-point Likert answer format (Lipkus, Dalbert, & Siegler, 1996). It has not previously been used in a cardiac population. In the validation studies, seven items

loaded moderately to highly on a single factor, but one item was included despite an unsatisfactory item loading during factor analysis (Lipkus, Dalbert, & Siegler, 1996). An overall Cronbach's α was not reported.

For comparability among measures in the present study, and in line with previous research (Rieckmann et al., 2006), the scores of the vulnerability scales above were categorised. The PES-AD and the BJW-S were reverse scored, and then recoded so that the upper quartile indicated a greater theoretical vulnerability to depression (i.e. a *lack* of positive reinforcement, *not* believing in a just world). Scales were divided into quartiles, and then recoded to compare the upper quartile (=1) with the others (=0).

Type D scale – DS14

The DS14 is a 14-item scale which measures whether or not participants have a distressed (type D) personality type (Denollet, 2005). The DS14 consists of two 7-item scales assessing negative affectivity (NA) and social inhibition (SI). A cut-off of 10 on both scales indicates those of the type D personality disposition. The DS14 has been used extensively in cardiac patients, and it has demonstrated good psychometric properties. The NA and SI scales have demonstrated internal consistencies (α) of 0.86 and 0.88, and three-month test re-test reliabilities of 0.72 and 0.82 respectively (Denollet, 2005).

Statistics

Logistic regression was adopted to allow prediction of binary dependent variables, using odds ratios (ORs) as a measure of effect size. Depression was classified as scoring above threshold on any of the scales, as this was most comparable to the

results found by Rieckmann et al. (2006). Univariate and multivariate logistic regression was used to assess differences between depressed and non-depressed samples. Demographic and disease variables which predicted depression with a p-value $<.15$ were included in the multivariate analyses, omitting variables with large numbers of missing values. Nested logistic regression was used to predict the unique contribution of blocks of variables (sociodemographics or disease indices as block 1, vulnerabilities as block 2; the sample size did not allow the inclusion of both at the same time). Nested logistic regression differs from hierarchical regression in that it allows blocks of variables to co-vary with the addition of subsequent blocks, whereas hierarchical regression fixes the variance explained for each block. Goodness-of-fit statistics (χ^2 , pseudo r^2 and F-statistics) were utilised to assess the prediction of depressive symptoms by theoretical vulnerabilities. The pseudo r^2 statistic only approximates the r^2 statistic in linear regression analyses, and cannot be interpreted as such. To account for original clustering of patients within hospitals, analysis was conducted using Huber-White robust variance estimation adjusted standard errors. Missing data was imputed for depression scales, BJW-S and the DS14 scale using Stata SE 9.2's regression-based 'impute' command (see Doyle, Conroy, McGee, & Delaney, in press). Regression-based imputation performs well in comparison to inputting the mean, median or last observation carried forward (Shrive, Stuart, Quan, & Ghali, 2006). For life events and pleasant events, missing data imputation was inappropriate.

Results

The baseline profile of the sample has been reported previously (Doyle, Conroy, McGee, & Delaney, in press). Data from 339 participants with fully completed

vulnerability questionnaires were analysed. Data were compared for those scoring above cut off on any of the HADS-D, BDI-FS or MQ-10 (48%) – Table 1.

Insert Table 1 here

More non-depressed participants had private health insurance, but no other demographic variables differed between subgroups. Depressed participants were more likely to be smokers, to have diabetes, to have lower levels of cholesterol, and have higher prevalence of past CHD and revascularisation. No differences were seen in terms of treatments received for the index hospitalisation. The prevalence of elevated vulnerabilities was significantly higher in depressed patients.

Proportions of vulnerabilities reported

The number of vulnerabilities reported by proportions of the sample, stratified by depression status, is displayed in Fig 1.

Insert Fig 1 here

Of those scoring above thresholds on given depression scales, 22% demonstrated no theoretical vulnerability to depression. Thus, type D did not account for the proportion of patients who did not exhibit any vulnerability as found by Rieckmann et al. (2006), although it was the only vulnerability reported by 13% of those with elevated depressive symptoms. However, the elevated levels of depressive symptoms seen in Rieckmann et al. (2006), and above, suggests that this is an overestimation of

those with elevated depressive symptoms. Using a more realistic estimate of depressive symptoms (the proportion of individuals who scored in the top quintile of depressive symptoms according to at least one scale – 29%) leads to a different answer – <4% of depressed patients did not exhibit any vulnerability (other data not shown).

Predicting depression: multivariate analyses

The first model, predicting depression status using demographic and vulnerability factors (variables with a p -value<.15), is displayed in Table 2 (a). The demographic factors in block 1 predicted <1% of the model (pseudo R^2 =.008, χ^2 =4.0, df =4, p =.134), but the addition of theoretical vulnerabilities in block 2 increased the proportion of the model explained by a significant margin (χ^2 =150.9, df =4, p <0.001), with the model now explaining 16% of the variance (overall model fit: pseudo R^2 =.16, χ^2 =166.5, p <0.001). Thus, the vulnerabilities were far more important than demographic factors for prediction of depression in this sample.

Table 2 here

The analysis was repeated with block 1 consisting of risk factor and disease severity indices (current smoker, prior diabetes, prior CHD, prior or current revascularisation, cardiac arrest), and block 2 consisting of the vulnerabilities. The results are in Table 2 (b). This first block of disease severity variables accounted for 7% of the variance in depressive status (pseudo R^2 =.07, χ^2 =137.9, p <0.001). However, the vulnerabilities added significantly to the model (χ^2 =58.6, df =4, p <0.001), with the overall variance explained increasing to 22% (pseudo R^2 =.22, χ^2 =156.0, p <0.001). Thus,

psychological vulnerabilities accounted for significant variation in depression not explained by disease severity.

Discussion

The aim of this study was to determine if theoretical vulnerabilities to depression, including type D, were independent predictors of depression in ACS patients, and to determine if these vulnerabilities were better predictors of depression than sociodemographics or indices of cardiovascular disease. Results showed that the vulnerabilities were more powerful predictors of depression status than demographic or disease variables, and moreover that these vulnerabilities were each independent predictors of depression.

The vulnerabilities assessed here show good comparability to previous research (Lipkus, Dalbert, & Siegler, 1996; Pedersen & Denollet, 2006; Rieckmann et al., 2006; Schiffer et al., 2006), suggesting that the findings should have a degree of generalisability. The present study argued that the presence of depressive vulnerabilities would provide evidence that patients were depressed independently of the ACS event or severity. In univariate and multivariate nested regression analyses, all vulnerabilities predicted depression. These results support the results of Rieckmann et al. (2006), but extend these findings in two ways. First, vulnerabilities predicted depression status beyond coronary disease markers, including LVEF. Second, the result that type D personality was an independent predictor of depressive symptoms, when controlling for other established vulnerabilities, has not previously been demonstrated in the literature.

Rieckmann et al. (2006) showed that a category of depression may exist that cannot be attributed to particular theoretical vulnerabilities, with between 14-25% of depressed persons (who constituted 47% of their sample) not exhibiting any assessed vulnerability. However, the authors did not assess personality disposition as a possible vulnerability. Although type D did not account for all of this residual category in the current study, this category of depression was smaller when a more conservative (and probably more realistic) threshold criterion was used for depression. Thus, in this instance, it could be argued that type D did account for the majority of the remaining depressed individuals who did not display vulnerabilities in Rieckmann et al. (2006). Indeed, type D was the only vulnerability reported by 13% of those with depression (according to original thresholds). Furthermore, the differences in findings seen in the Rieckmann et al. (2006) study could also be attributable to the differences in categorising of vulnerabilities. For example, Rieckmann et al. (2006) typically used the upper standard deviation (typically about 16% in a normal distribution), whereas the present study used upper quintiles. Thus, the residual 'atheoretical' category may depend on not only how depression is defined as 'elevated', but also how the vulnerabilities are defined as 'elevated'. A further possibility is that there are likely a proportion of patients who are having what would be considered adjustment which will abate naturally over time, rather than actual 'depression' (Frasure-Smith & Lesperance, 2003) – and these could explain the residual category also.

The results of the present study provided support for the notion that vulnerabilities to depression were more important for depression than were demographic or cardiovascular disease variables. The first multivariate model tried to replicate Rieckmann et al. (2006), who had found that although demographic variables were

significant predictors of depressive symptoms, the vulnerabilities were more strongly associated with depression. In the present study, demographic variables accounted for very little of depression status – less than 1% of the model was explained by these factors. The vulnerabilities showed a significant improvement in model fit, and demonstrating that these vulnerabilities were a much more powerful predictor of depression. Furthermore, all vulnerabilities remained significant independent predictors in the nested model, showing that no vulnerability was mediated by another, replicating the previous research.

Although depression was not associated with most cardiovascular disease severity or risk marker indices, these variables did explain a significant, albeit small (7%), proportion of the model in depression in the present study. It is important to highlight that these disease variables did not account for the bulk of the variance in the data, and vulnerabilities accounted for significantly more of the model. This supports other work which has shown that there is little or no association between cardiovascular disease severity indices and depression (Carney, Freedland, Miller, & Jaffe, 2002; Lane, Carroll, Ring, Beevers, & Lip, 2000; Parker *et al.*, 2008; Sorensen *et al.*, 2005). Furthermore, LVEF<40% did not discriminate between depressed and non-depressed patients, and was therefore not included in the multivariate analysis. However, including this variable did not significantly change the results (data not shown). This is perhaps surprising given the previously outlined findings that depression was more prevalent in those with LVEF <30% (van Melle *et al.*, 2005). However, it may be that LVEF needs to be lower than 30% for these hypothesised effects on depression to occur. A further issue is the instrument used in that study – the full-length BDI scale assesses multiple somatic symptoms, which were omitted from the present research.

These symptoms may be simply indicative of CHD symptoms, or even medication side-effects (e.g. insomnia, fatigue). Thus, these methodological differences could account for the findings. Overall, these results suggest that the demographic, disease and treatment indices measured in the present study are unlikely to explain the prevalence of depression – irrespective of whether this prevalence is better accounted for by the theoretical vulnerabilities or not.

If depression in ACS patients is mainly attributed to least one of the vulnerabilities, then theoretically it could be argued that such depression is independent of CHD severity, as these vulnerabilities should be independent of CHD severity. For example, diabetes does not cause (non-health) stressful life events, high cholesterol does not lessen our enjoyment of music or how often we listen to it, and high blood pressure does not cause us to think that the world is an unfair place. This is not to say that prognostic studies should not control for disease severity indices, but this provides an alternative test of whether or not these patients should be considered depressed, rather than simply adjusting to a life event.

The cross-sectional nature of this vulnerability-related data precludes definitive conclusions about directionality or causality of the associations assessed in this study. However, most of the vulnerabilities assessed have predicted depression in longitudinal studies, and some are also pertinent for the treatment of depression. As such, these findings may carry an element of weight with regard to the hypothesised directionality of the relationship. However, previous research has demonstrated a bi-directional relationship between theoretical causes of depression and depressive symptoms, which may have affected present findings. The PES-AD may be

influenced by the negative and pessimistic outlook or response set of depressed individuals (Lewinsohn & Gotlib, 1995). Other studies have demonstrated that although depressed patients report experiencing more life events, depression may actually predispose people to be exposed to further life events, e.g. miscommunication between depressed and non-depressed partners causing further marital conflict (Joiner, 2002; Paykel, 2003; Weissman & Markowitz, 2002). There may also be an element of recall bias, with depressed patients more likely to recall negative events than non-depressed counterparts (Lewinsohn & Gotlib, 1995; Monroe & Hadjiyannakis, 2002). However, reliability studies support the view that severe life events are recalled and reported accurately for up to 10 years (Monroe & Hadjiyannakis, 2002). It could be argued that the BJW-S is not a comprehensive measure of cognitive distortions. However, the present findings showed that the BJW-S was predictive of depression independently of other vulnerabilities, and this supports previous findings that the BJW-S predicted depression independently of personality traits (Lipkus, Dalbert, & Siegler, 1996). Furthermore, there was no measure of BJW prior to the event; therefore it is impossible to know if depressive symptoms associated with lower BJW-S scores were as a result of a 'disrupted' BJW. Importantly, no measure of psychiatric history was available. Sorensen et al. (2005) have shown that previous depression was associated with depression at discharge post-ACS, and that those with a history of depression had more severe depression. Longitudinal studies are required to assess whether ACS patients with such vulnerabilities are at greater risk for developing future depressive episodes.

Conclusions

This study replicates and further extends the previous findings of Rieckmann et al. (2006), by showing that Type D personality can also be considered an independent vulnerability factor for elevated depressive symptoms during hospitalisation for ACS. The results of this study imply that there is very little association between disease severity indices and depression, but that the depressive symptoms were better accounted for by elevated vulnerabilities to depression. This suggests that these depressive symptoms are present independently of the ACS event.

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Table 1: Demographic and risk factor profile of the current sample (n=336 unless otherwise stated), categorised by having elevated depressive symptoms or not

Variable	No depressive symptoms (n=209)	Elevated depressive symptoms (n=199)	Odds-ratio (OR)	95% confidence interval (CI)		p
Demographics						
Age (years)(mean, std dev)	61.7 (11.1)	60.5 (10.4)	0.99	0.96	1.004	0.118
Men	86%	83%	0.8	0.4	1.8	0.648
Has a partner (1=yes)	73%	75%	1.1	0.7	2.0	0.585
Employed (1=yes) (n=252)	23%	15%	0.9	0.5	1.6	0.746
Private health insurance	35%	28%	0.7	0.5	1.0	0.053
Current risk factor profile						
Current smoker	28%	42%	1.9	1.5	2.4	<0.001***
Prior hypertension	45%	44%	1.0	0.7	1.5	0.938
Prior diabetes	4%	14%	3.7	1.9	7.6	<0.001***
Total cholesterol (mmol/l)(mean, SD) (n=252)	4.8 (1.2)	4.4 (1.2)	0.8	0.6	0.9	0.001**
Prior CHD	25%	34%	1.6	1.0	2.4	0.041*
Prior revascularisation	20%	29%	1.7	1.0	2.7	0.031*
Current hospitalisation						
Thrombolysis	28%	20%	0.7	0.3	1.3	0.234
Revascularisation received	75%	66%	0.6	0.4	1.1	0.080
Cardiac arrest confirmed	18%	12%	0.6	0.3	1.1	0.120
Length of hospital stay (median, interquartile range [IQR])	7 (5–11)	7 (4–10)	0.99	0.96	1.02	0.546
Left ventricular function (confirmed as <40%)	16%	12%	0.8	0.4	1.6	0.479
Co-morbidities						
Modified CCI score (median, IQR)	0 (0–0)	0 (0–1)	1.1	0.95	1.4	0.154
Vulnerability scales						
Life events: LTE-Q^ (median, IQR)	0 (0–1)	2 (1–3)	4.7	2.1	10.5	<0.001***
Reinforcing events: PES-AD^ (median, IQR)	27 (23–31)	24 (20–30)	2.2	1.5	3.2	<0.001***
Cognitions: BJW-S^ (median, IQR)	34 (32–36)	31.5 (27–35)	4.0	2.5	6.4	<0.001***
Personality: Type D	17%	44%	3.9	2.3	6.7	<0.001***

*<0.05, **<0.01, ***<0.001

[^]analysed as top quartile (1) versus the rest (0)

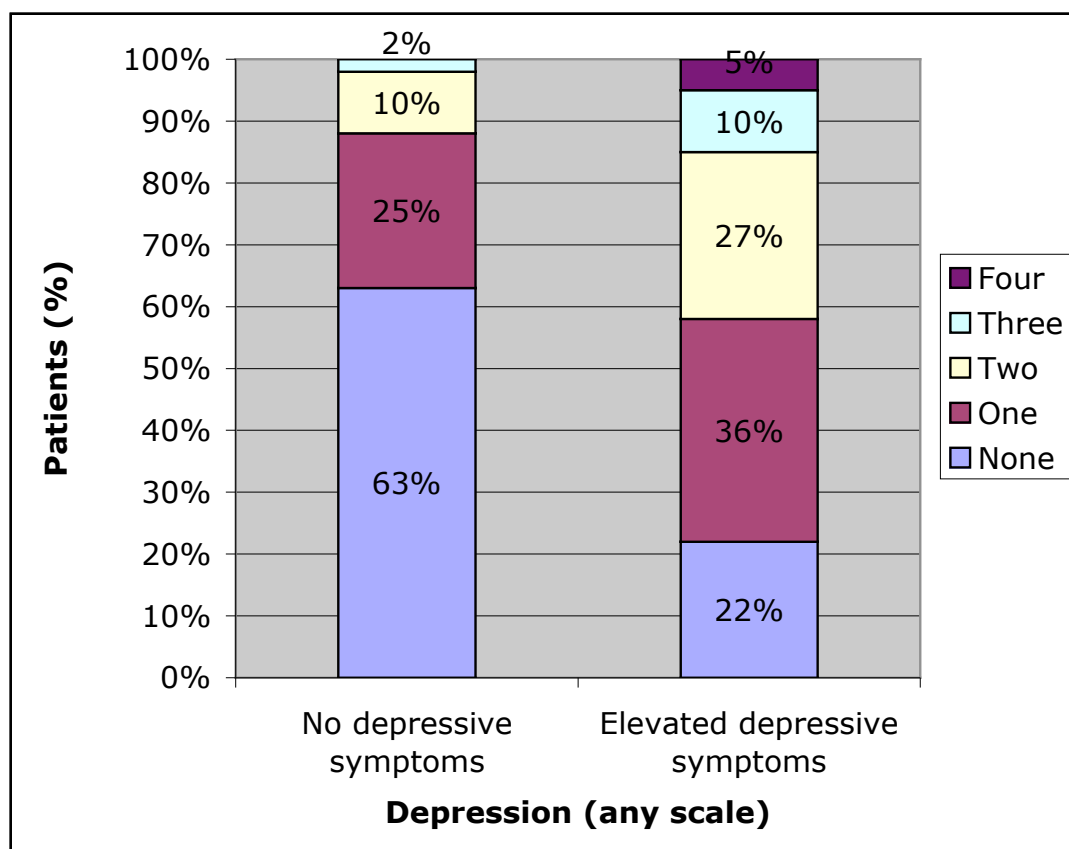


Fig 1: Number of vulnerabilities reported by participants, stratified by depressive symptom status

Table 2: Nested logistic regression model predicting depression with vulnerabilities, demographic (a) and disease severity (b)

a)	OR	95% CI		p
Block 1 - demographics				
Age	1.0	0.98	1.03	0.818
Private health insurance (1=yes)	0.9	0.6	1.3	0.472
Block 2[^]				
LTE-Q (Stressful life events)	3.8	1.8	8.1	0.001**
PES-SV (low reinforcement)	2.1	1.3	3.3	0.002**
BJW (non-belief in a just world)	2.8	1.8	4.3	<0.001***
Type D personality (modified)	3.1	1.9	5.3	<0.001***
b)				
Block 1 – Risk factor and disease severity				
Current smoker	2.5	1.8	3.3	<0.001***
Diabetes	5.5	2.8	10.6	<0.001***
Prior CHD	0.9	0.6	1.5	0.800
Prior revascularisation	1.6	0.9	2.8	0.136
Revascularisation post-event	0.6	0.3	1.0	0.051
Cardiac arrest	0.5	0.3	0.9	0.016*
Block 2 Vulnerabilities[^]				
LTE-Q (Stressful life events)	3.8	1.9	7.7	<0.001***
PES-SV (low reinforcement)	2.0	1.3	3.1	<0.001***
BJW (non-belief in a just world)	2.9	1.7	4.9	<0.001***
Type D personality	3.7	2.1	6.3	<0.001***

Variables which partially discriminated ($p < .15$) between elevated depressive symptoms and no depressive symptoms were included in the models

* <0.05 , ** <0.01 , *** <0.001

[^]analysed as top quartile (1) versus the rest (0)