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AUTHOR(S)

Frank Doyle, Hannah Mc Gee, Mary Delaney, Nicola Motterlini, Ronan Conroy

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Depressive vulnerabilities predict depression status and trajectories of depression over one year in persons with acute coronary syndrome

Running title: Vulnerabilities predict post-ACS depression

¹Frank Doyle, PhD, ¹Hannah McGee, PhD, ²Mary Delaney, MA, ³Nicola Motterlini, StatSciD, ⁴Ronán Conroy, DSc

Departments of ¹Psychology and ⁴Epidemiology & Public Health Medicine, ³HRB Centre for Primary Care Research, Division of Population Health Sciences, Royal College of Surgeons in Ireland, 123 St Stephen's Green, Dublin 2, Ireland

²HRB Centre for Health and Diet Research, Department of Food Business and Development, University College Cork, Cork, Ireland

Corresponding author:

Frank Doyle, PhD

Department of Psychology, Division of Population Health Sciences, Royal College of Surgeons in Ireland, 123 St Stephen's Green, Dublin 2, Ireland

Email: fdoyl4@csi.ie, Tel: + 353 1 4022718, Fax: + 353 1 4022764

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Objective

Depression is prevalent in patients hospitalised with acute coronary syndrome (ACS). We determined whether theoretical vulnerabilities for depression (interpersonal life events, reinforcing events, cognitive distortions, Type D personality) predicted depression, or depression trajectories, post-hospitalisation.

Methods

We followed 375 ACS patients who completed depression scales during hospital admission and at least once during three follow-up intervals over one year (949 observations). Questionnaires assessing vulnerabilities were completed at baseline. Logistic regression for panel/longitudinal data predicted depression status during follow-up. Latent class analysis determined depression trajectories. Multinomial logistic regression modelled the relationship between vulnerabilities and trajectories.

Results

Vulnerabilities predicted depression status over time in univariate and multivariate analysis, even when controlling for baseline depression. Proportions in each depression trajectory category was as follows: persistent (15%); subthreshold (37%); never depressed (48%). Vulnerabilities independently

predicted each of these trajectories, with effect sizes significantly highest for the persistent depression group.

Conclusions

Self-reported vulnerabilities – stressful life events, reduced reinforcing events, cognitive distortions, personality – measured during hospitalisation can identify those at risk for depression post-ACS, and especially those with persistent depressive episodes. Interventions should focus on these vulnerabilities.

Keywords: Depression; coronary heart disease; psychological theory; life events; personality; just world beliefs

Depressive vulnerabilities predict depression status and trajectories of depression over one year in persons with acute coronary syndrome

Depression is prevalent in patients with coronary heart disease, with the prevalence estimated at approximately 20% in patients with myocardial infarction [1]. This is significantly higher than that seen in general population samples [2]. The importance of depression is highlighted not only in its prevalence, and its impact on quality of life, but also on the ability of depression to predict cardiovascular prognosis [3-5].

However, while a large literature concerns the prediction of prognosis in depressed cardiac patients, relatively little research is concerned with what happens to depression after the acute hospitalisation phase. Depression is a chronic, episodic condition, and therefore research on what happens to depressive symptoms in the post-acute phase potentially provides vital information for intervention design. While the prevalence of depression is comparatively steady over time, this masks the different trajectories symptoms of depression take [6-8]. Indeed, sophisticated studies have shown different patterns of resolving and persistent depression in patients with heart disease [7, 8]. For example, Martens et al. [7] surveyed 287 patients post-hospitalisation for myocardial infarction at 2 and 12 months. They categorised four groups of patients in relation to depressive symptom status: non-depressed, mildly depressed, moderately depressed and severely depressed. Similarly, Kaptein et

al. [8] followed 475 patients with myocardial infarction every 3 months over one year, and their results showed that five distinct groups regarding depression: no depressive symptoms, mild depressive symptoms, moderate and increasing depressive symptoms, significant but decreasing depressive symptoms and significant and increasing depressive symptoms. Thus, the evolution of depression is complex, and in order to design optimal interventions, more knowledge on the predictors of depressive symptoms and such depressive trajectories is needed [9].

While some research has established predictors of depression in patients with coronary heart disease from easily available variables recorded as part of standard hospital care, the results are often contradictory [7, 8, 10-12]. For example, age, sex, medications and left ventricular function have been shown to predict depression in cardiac patients in some of these findings, but not in others. Furthermore, such findings are atheoretical, and thus provide little clue as to how to intervene in such populations [9, 13]. A paucity of evidence exists assessing the relative importance of theoretical vulnerabilities, and their associated interventions, regarding risk of depression and trajectories of depression after acute coronary syndrome (ACS) [14]. While a small number of studies have assessed theoretical vulnerabilities to depression – for example, stressful life events, personality and cognitions have all been associated with depression in cardiac patients [7, 15, 16] – such studies have not measured these

vulnerabilities simultaneously, or have not assessed their association with trajectories of depression post-ACS.

These vulnerabilities are especially important, given recent findings which suggest that, in patients with ACS, such vulnerabilities predict depression better than do demographic or disease variables [13, 17]. However, both these studies were limited, as they were cross-sectional, and did not allow for the direction of causality to be determined [13, 17]. Also, it was possible that recall bias in depressed patients contributed to a higher self-reported level of such vulnerabilities – thus to inflated correlations between the variables. We therefore report on longitudinal data from our cohort. We aimed to determine a) whether depressive vulnerabilities predicted depression over time, when controlling for baseline depression, and b) whether these vulnerabilities better predicted different types of depression (e.g. persistent depression).

Methods

Study design and participants

The baseline methods have been reported previously [3, 13]. This paper presents data from ACS patients who completed depression questionnaires at baseline (during acute hospital admission), and who responded to at least one of the postal follow-up surveys at 3-, 6- and 12-months (not all participants completed all theoretical vulnerability scales). Briefly, after ethical approval was provided, patients were recruited from 12 hospitals. 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 completed a composite psychological questionnaire while in-hospital, and coronary disease risk factor and treatment data was obtained from medical charts. Major co-morbidities were also recorded as per the Charlson Comorbidity Index [18], and modified by omitting some of the risk factors which are separately assessed in cardiac patients (e.g. MI, diabetes). Patients were then followed up by postal survey, containing measures of depression, at each of the following three phases. Non-respondents were posted a reminder after two weeks, and then telephoned with a further reminder if no response was received after another two weeks.

Measures

Depression scales

Beck Depression Inventory – Fast Screen (BDI-FS)

The BDI-FS is a 7-item scale focusing on cognitive symptoms of depression [19], and has very good sensitivity/specificity ($>0.90/>0.85$) for detecting major depression when using a threshold score of >3 [20, 21]. We omitted the suicidality item, but maintained the threshold of >3 , for reasons outlined previously [3, 6]. Also, the predictive power of the BDI-FS has been shown to be unchanged when removing this item in persons with hepatitis C [22].

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

The HADS is a 14-item measure that was developed to measure anxiety and depression in hospitalised patients, and omits somatic items so scores are not contaminated by symptoms of chronic conditions [23]. We used the 7-item HADS-D only, and adopted the recommended threshold of >7 [24]. The HADS-D focuses mainly on anhedonia.

Scoring above threshold on either scale was considered to indicate depression status at baseline and follow-up.

Depressive vulnerability measures

List of Threatening Experiences Questionnaire (LTE-Q)

Stressful interpersonal and life events (e.g. serious illness or assault, or a relationship break-up) were assessed using the 12-item LTE-Q [25, 26]. This schedule relates to events that have happened in the prior year. The authors showed that the LTE-Q had high test-retest reliability, and compared well with an interview technique (sensitivity/specificity ranges for stressful life events were between 0.89-1.0/0.74-0.88 respectively), in psychiatric patients.

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

Pleasant events were assessed using the PES-AD, a 20-item behavioural log. The scale was originally developed for persons with Alzheimer's disease [27], but has also been used in ACS patients [17]. Environmental engagement is measured by ratings of the frequency of behaviours/events, and enjoyment of

same, in the past month. A cross-product produces a total schedule score of positive reinforcement in the past month. Missing items were coded as zero if at least half of the 20 items had been answered [17].

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

As a period of adjustment post-event is likely for all patients, and not only for those who have distorted cognitions or dysfunctional attitudes or distorted cognitions, we assessed just world beliefs instead of other types of cognitive distortions [13]. BJW refers to the belief that good things happen to good people, and bad things happen to bad people [28], and a ‘distorted’ BJW (i.e. non-belief in a just world) has been associated with depression [13, 28, 29]. BJW for self was assessed by the 8-item BJW-S [29].

Comparing vulnerabilities

For comparability among measures, and in line with previous research [13, 17], the scores of the vulnerability scales above were recoded to indicate a higher risk for depression (i.e. a *lack* of positive reinforcement, *not* believing in a just world, but *higher* numbers of stressful life events). For effect size comparability, scale scores were standardised, with effect sizes representing a one standard deviation increase.

Type D scale – DS14

The distressed (Type D) personality – a combination of both negative affectivity

and social inhibition – was assessed using 14-item DS14 [30]. Scoring above threshold (>10) on both of the subscales indicates those of Type D disposition. The DS14 has been used extensively in cardiac patients, and it has demonstrated good psychometric properties [30].

Statistics

Differences between groups were assessed with χ^2 test or analysis of variance as appropriate. Missing data was imputed for depression and vulnerability scales using Stata's regression-based 'impute' command [3, 13], but imputation was inappropriate for the schedules (LTE-Q, PES-AD). Logistic regression with random effects estimates for panel/longitudinal data was adopted to allow prediction of depression status (person status) throughout the follow-up period, adjusting for baseline depression. Odds-ratios (ORs) were used as a measure of effect size. Latent class analysis of combined HADS-D and BDI-FS score was conducted using the SAS PROC TRAJ command, as in previous research . Adding age, sex, prior CHD and low left ventricular function as co-variables had a negligible effect on the depression trajectories, so the non-adjusted groups were used in subsequent analysis. The lowest Bayesian Information Criterion (BIC) value (-5449.68) lead to 1 category with ~3% of participants, so the next lowest was chosen (-5438.3). Both panel-modelling logistic regression and latent class analysis are designed to account for missing data during follow-up. Multinomial logistic regression, reporting relative risk ratios (RRR) for effect sizes, was then used to model the relationship between vulnerabilities and different categories of

depression during follow-up, using *never depressed* as the reference category. Post-hoc Wald test statistics examined whether the effect sizes were significantly different for each vulnerability when predicting depression categories.

Results

Response rate

During follow-up, 375/430 (87%) patients responded to at least one of the follow-up surveys, and 250/430 (58%) responded to all of the follow-up surveys. This provided, depending on the response to a particular scale at baseline, up to 949 unique observations in the data. Non-respondents to the follow-up phases were less likely to have a partner (OR=0.6, 95% CI 0.4–0.9, $p=0.014$) and less likely to have private health insurance (OR=0.3, 95% CI 0.1–0.7, $p=0.005$), but no other demographic differences were found.

Depression trajectories

The depression trajectories of the combined scales are shown in Figure 1:

Insert Fig 1 here

Numbers/proportions in each depression trajectory category was as follows:
persistent – 57 (15%); subthreshold – 138 (37%); while 180 (48%) did not score

above threshold at any stage (never depressed).

Baseline profile and depression trajectories

The baseline profile of the sample is shown in Table 1, and is subdivided by depression trajectory category.

Insert Table 1 here

There was a significant difference in age among the depression categories – those with persistent depression had the youngest average age. Those in the persistent depression group were also less likely to have private health insurance, while those with subthreshold depression had the lowest prevalence of employment. There were no other major differences among the depression categories.

Predicting depression status longitudinally

The prevalence of depression was as follows during the follow-up waves: 22% (75/335) at 3-months; 25% (75/302) at 6-months; 19% (60/312) at 12-months. The question of whether the vulnerabilities predicted depression status during follow-up is addressed in Table 2.

Insert Table 2 here

Unsurprisingly, depression status at baseline was a very strong predictor of depression status during follow-up (OR=36.7, 95% CI 14.2–94.5, $p<0.001$). As baseline depression was also associated with vulnerabilities [13], we adjusted for baseline depression when assessing the association between individual vulnerabilities and subsequent depression (Table 2, a). Each vulnerability was significantly related to depression during follow-up, with ORs ranging from 1.6–3.7.

Multivariate analysis (Table 2, b), including each of the vulnerabilities and baseline depression in the model, showed that depression status during follow-up was (at least marginally) independently predicted by each of the vulnerabilities. Thus, although depression at baseline was the predictor with the largest effect size of subsequent depression, the effects of the theoretical vulnerabilities were not mediated by initial depression status.

Predicting depression trajectories

Table 3 shows the results of a multinomial logistic regression model predicting depression trajectories, with never depressed as the reference category.

Insert Table 3 here

Each vulnerability was a significant, independent predictor of persistent depression. For example, when compared to those who were never depressed, those with persistent depression were more than twice as likely to have reported being of Type D disposition, or reported having elevated stressful life events or reduced pleasant events in the year prior to the follow-up period, or not to have just world beliefs. Adding age, employment or health insurance status to the model, as these differentiated some the trajectory groups, had little effect on the results (data not shown). For subthreshold depression, only just world beliefs and stressful life events differentiated between this category and the never depressed category.

Visual inspection of the effect sizes for the subthreshold depression category would suggest that these are consistently smaller than those for predicting persistent depression. We tested whether the effect sizes for persistent depression were significantly larger than the effect sizes for the subthreshold category – i.e. whether vulnerabilities had significantly stronger effects for persistent depression overall. Post-hoc Wald statistics confirmed that, with the exception of BJW, the effect sizes for the vulnerabilities when predicting persistent depression were significantly larger than the effects when predicting

the subthreshold category ($p < 0.05$ in each case, data not shown). Thus, elevated levels of stressful life events, reduced pleasant activities, and Type D personality predicted persistent depression to an even greater extent than they did for subthreshold depression. This finding illustrates the power of such vulnerabilities for predicting persistent depression in this population.

Discussion

We longitudinally examined whether theoretical vulnerabilities for depression were independent predictors of depression, and depression trajectories, over one year follow-up in patients with ACS. Results showed not only that the vulnerabilities independently predicted depression status over time, but also predicted the different depression trajectories. Furthermore, vulnerabilities were especially important for persistent depression, being significantly stronger predictors of this category over the subthreshold category.

That depressive vulnerabilities predicted depression status over the follow-up confirms and strengthens the findings of previous cross-sectional reports [13, 17]. Perhaps more importantly, however, was that these vulnerabilities were independently predictive of post-discharge depression when controlling for baseline depression. To our knowledge, this is the first such finding in the literature. That the vulnerabilities are independent predictors probably reflects the heterogeneous nature of the aetiology of depression, and that the vulnerabilities

represent distinct causal theories (i.e. interpersonal, behavioural, cognitive, along with personality [13, 14]).

When predicting trajectories of depression, persistent depression was consistently predicted by the vulnerabilities in comparison to those who were not depressed. Furthermore, with the exception of just world beliefs, these effects were significantly larger than the average effects when predicting the subthreshold depression category, although this is *post-hoc* analysis and needs to be interpreted with caution. Thus, clinicians need to be especially cognisant of patients reporting such theoretical vulnerabilities post-ACS, to determine the probable evolution of depression and the level of intervention needed. Although some of the vulnerabilities were non-significant for predicting subthreshold depression, this may be due to the somewhat lower power and the smaller effect sizes. Future research should address the question of whether these depression trajectories differ in response to intervention, and whether interventions targeting these vulnerabilities can enhance quality of life.

The present results support some previous findings regarding a number of the above vulnerabilities. Cognitions, at least in the form of illness perceptions, have also been associated with new episodes of depression post-myocardial infarction [15]. That personality predicts subsequent depression in cardiac patients has been demonstrated previously [7, 8, 32]. Furthermore, Martens et al. [7] also showed that Type D personality was predictive of persistence of different

categories of depression over time. In contrast to our results, other research showed that stressful life events were not associated with depressive symptoms one-year after myocardial infarction [16]. However, as depression was only measured at two time points, these analyses modelled prevalence of depression, and not depression trajectories as was done here. Also, the authors used a combined depression and anxiety score, rather than just depressive symptoms, which may explain the disparity in findings. Although these studies consolidate the findings of our research, the present findings add to the literature by measuring the vulnerabilities simultaneously.

The trajectories we found closely match those from one study [7], but not others [8, 33]. There may be a number of reasons for the disparities – the scales used or the number of time points during follow-up may explain these differing trajectories.

Our findings differ somewhat from some previous research in that we generally did not show significant associations among demographic factors or coronary disease or treatment indices and depression development/trajectories [7, 8, 10, 11]. However, such results are inconsistent, for example, Spijkerman et al. [10] have shown that women were more likely to be depressed post-MI, whereas others have not [11, 17]. Previous research using similar depression scales as used here has also not shown evidence of sex effects [6, 34]. As regards disease indices, generally it is accepted that coronary disease and depression do not

correlate, although there is some controversy over the status of the relationship between left ventricular function and depression [10, 13, 35, 36]. One reason for this could be the depression scales used in previous research – the full-length BDI has multiple somatic symptoms, and scores on this scale may be more readily contaminated by coronary disease symptoms, which should not be the case in this study. Given the inconsistency in the literature, it is perhaps unsurprising that depression trajectories were not associated with demographic or disease indices in the present study. The present results, along with previous findings [13], demonstrate that these vulnerabilities were more important for depression trajectories than coronary disease indices or demographic factors. Only age, employment and health insurance status were associated with depression trajectories. As such, it is important to stress that although these variables are readily available clinically, they appear to be much less important for predicting depression than vulnerabilities.

Unfortunately, history of depression was unavailable. This may be crucial in determining the persistence or otherwise of the episodes recorded here, as previous research has shown the importance depression history for predicting in-hospital and post-discharge depression [7, 10, 37-39]. The unavailability of history of depression also means that the new onset category analysed here does not directly match those used in other research [40, 41]. Furthermore, it is unclear whether the vulnerabilities measured in this study would continue to predict subsequent depressive symptoms once history of depression was

controlled for. It is probable that the BJW-S is not a comprehensive measure of cognitive distortions, however, it independently predicted depression at baseline, and also in the longitudinal analysis here. It is unclear whether different cognitive distortions would be better predictors of depression or of certain depression trajectories, and future research should address this. We had little power to include disease indices and sociodemographic variables. However, it is unlikely that these variables contributed much as they did not discriminate in univariate analyses [13]. The analysis only contains those who completed at least one follow-up measure, and this limits the generalisability of the findings. Missing data across time points could have led to misclassification of participants, e.g. participants could be considered never depressed if they were not depressed at baseline or at 12 months, but had missing data at the 3, and 6 month follow-up points. Strengths of the present study include the longitudinal design, multiple vulnerability measures, and the ability to adjust for depression at baseline when predicting subsequent depression status. This rules out the possibility that the vulnerabilities predicted depression at baseline simply due to recall bias.

The findings herein are unique, in that for the first time theoretical depressive vulnerabilities have been shown to predict depression post-ACS, and different trajectories of depression also. Furthermore, that these vulnerabilities were particularly important predictors of persistent depression highlights the need for clinicians to be aware of patients with such psychosocial risk factors or characteristics. The recent COPES trial showed that allowing patient preference

for treatment (psychotherapy or antidepressants) in a stepped care model could enhance patient satisfaction with depression treatment [42]. Future studies could address the question of whether the self-reported vulnerabilities as outlined here correlate with patient preference for depression therapy, to determine if such findings have the potential to enhance patient satisfaction or the therapeutic relationship.

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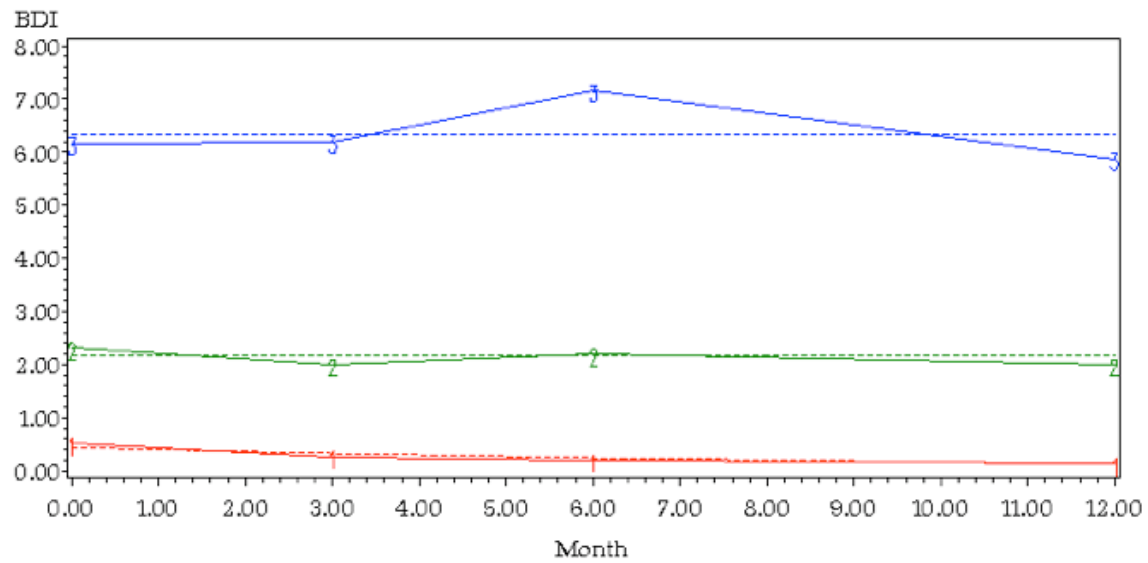
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Figure 1: Depression trajectories, combining both depression scales



1 – Never depressed, 2 – Subthreshold depression, 3 – Persistent depression

Table 1: Sample description (baseline data plus at least one follow-up depression measurement).

	Total (n=375)	Never depressed (n=180)	Subthreshold depression (n=138)	Persistent depression (n=57)	χ^2 (F) statistic	p-value
Demographics						
Age (years)(mean, SD)	61.5 (10.5)	62.8 (9.9)	61.5 (10.8)	57.4 (10.4)	F=5.86	0.003**
Men	79%	82%	76%	79%	1.48	0.477
Has a partner (1=yes)	75%	79%	72%	70%	2.94	0.230
Employed (1=yes)	18%	22%	10%	25%	9.55	0.008**
Private health insurance	33%	38%	30%	21%	5.96	0.051
Risk factor profile						
Current smoker	32%	27%	35%	40%	4.21	0.122
Prior hypertension	48%	52%	47%	40%	2.34	0.311
Prior diabetes	12%	13%	11%	9%	0.86	0.650
Total cholesterol (mmol/l)(mean, SD) (n=284)	4.6 (1.2)	4.7 (1.1)	4.5 (1.2)	4.6 (1.3)	F=1.32	0.269
Prior CHD	29%	28%	27%	37%	2.87	0.238
Prior revascularisation	23%	20%	24%	33%	4.31	0.116
Hospitalisation						
Thrombolysis	24%	28%	20%	23%	3.35	0.188
Revascularisation received	23%	25%	19%	28%	2.56	0.278
Cardiac arrest confirmed	15%	17%	16%	11%	1.29	0.526
Length of hospital stay (mean, SD)	8.6 (6.4)	8.4 (6.5)	8.5 (5.4)	9.0 (8.5)	F=0.16	0.852
Left ventricular function (confirmed as <40%)	13%	12%	15%	14%	0.88	0.644
Co-morbidities						
Modified Charlson Co- morbidity Index score (median, interquartile range)	0 (0–1)	0 (0–0)	0 (0–1)	0 (0–1)	F=0.04	0.956

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

Table 2: Random effects logistic regression models predicting person depression status over time

a) Adjusting for baseline depression	Odds ratio (OR)	95% CI		P-value
LTE-Q (Stressful life events, n=323, observations=829)	1.6	1.04	2.4	0.030*
PES-SV (low reinforcement, n=327, observations=830)	2.6	1.6	4.2	<0.001***
BJW (non-belief in a just world, n=375, observations=949)	1.9	1.3	2.8	0.001**
Type D personality (n=375, observations=949)	3.7	1.7	8.3	0.001**
b) Multivariate (n=295, 756 observations)				
LTE-Q (Stressful life events)	1.5	0.95	2.2	0.084
PES-SV (low reinforcement)	2.1	1.3	3.5	0.002**
BJW (non-belief in a just world)	1.8	1.1	2.7	0.014*
Type D personality	2.2	0.87	5.4	0.097
Baseline depression	14.2	4.9	41.2	<0.001***

Overall multivariate model: $\chi^2=49.5$, df=5, p<0.001

Table 3: Multinomial logistic regression model predicting depression trajectories, with never depressed as reference group (n=295)

	Relative Risk Ratio (RRR)	95% CI		P-value
Never depressed (reference)	-	-	-	-
Subthreshold depression				
LTE-Q (Stressful life events)	1.8	1.2	2.5	0.001**
PES-SV (low reinforcement)	1.3	0.94	1.7	0.124
BJW (non-belief in a just world)	1.9	1.4	2.6	<0.001***
Type D personality	1.6	0.89	2.9	0.140
Persistent depression				
LTE-Q (Stressful life events)	2.6^	1.7	4.1	<0.001***
PES-SV (low reinforcement)	2.5^	1.6	4.0	<0.001***
BJW (non-belief in a just world)	2.3	1.5	3.5	<0.001***
Type D personality	3.6^	1.6	8.5	0.003**

$\chi^2=105.6$, $df=8$, $p<0.001$, pseudo $R^2=0.18$

^ - significant difference in effect size between subthreshold and persistent categories (Wald test, $p<0.05$)