On becoming depressed or anxious in late life: similar vulnerability factors but different effects of stressful life events

EDWIN DE BEURS, AARTJAN BEEKMAN, SANDRA GEERLINGS, DORLY DEEG, RICHARD VAN DYCK and WILLEM VAN TILBURG

Background Little research has been done on the uniqueness of risk profiles for depression and anxiety in late life.

Aims Delineating risk factors for the decline of mental health in older persons, comparing risk profiles for developing symptoms of pure depression, pure anxiety and both anxiety and depression in a prospective design.

Method Self-report data on depression and anxiety were collected from community-dwelling older respondents (≥55 years) on two occasions, 3 years apart. Data from emotionally healthy respondents (n=1810) were used to investigate the effects of long-standing vulnerability factors and stressful life events.

Results After 3 years 9% of the subjects had scored beyond the thresholds for symptoms. Vulnerability for depression and anxiety was quite similar, but life events differed: onset of depression was predicted by death of a partner or other relatives; onset of anxiety was best predicted by having a partner who developed a major illness. No support for moderator effects between vulnerability factors and stress was found; the effects were purely additive.

Conclusions Depression and anxiety have many risk factors in common, but specific risk factors also were found, especially in subjects developing both depression and anxiety.

Declaration of interest Funding detailed in Acknowledgements.

Research shows that symptoms of anxiety and depression are highly concordant (Clark & Watson, 1991). Also, there is a high comorbidity of anxiety and depression at the caseness level (Barbee, 1998), especially in late life (Kirby et al, 1997). Among older persons about 50% of the clinically depressed suffer from comorbid anxiety disorders and 25% of patients with anxiety suffer from major depression (Beekman et al, 2000). Current knowledge on the phenomenology of psychopathology in late life is limited, but the scarce data available do suggest that distinctive features of depression and anxiety become even less pronounced with rising age (MacKinnon et al, 1994). However, an important aspect on which depression and anxiety in late life may be distinguished is the risk profiles for these conditions. Delineating risk profiles is crucial to the development of focused preventive measures aimed at emotional problems in late life. The present study sets out to compare risk profiles for anxiety and depression. Risk factors were organised according to the stress–vulnerability model (Brown & Harris, 1978; Goldberg & Huxley, 1992), which states that destabilisation (getting symptoms) is the result of long-lasting vulnerability factors acting in concert with exposure to environmental stressors, usually one or more highly stressful events.

METHOD

Sample and procedure

To investigate risk profiles for developing depression and anxiety in older persons we used data from the Longitudinal Aging Study Amsterdam (LASA; Deeg et al, 1993). The LASA is a longitudinal study in a large representative sample aged 55–85 years. Data were collected by interviewing respondents in their homes by trained and intensively supervised interviewers. All interviews were audiotaped to allow random quality checks. The first cycle of LASA data collection (T_1) took place between the autumn of 1992 and the autumn of 1993; the second cycle (T_2) took place 3 years later (1995–1996). A random sample of older men and women, stratified for age and gender, was drawn from the population registers of 11 municipalities in three regions of The Netherlands. Older men initially were oversampled to ensure sufficient respondents in these strata for a later phase of the study. In the first cycle of LASA, 3107 respondents were interviewed. This sample has been described extensively in previous publications of LASA (Beekman et al, 1995; de Beurs et al, 1999). Three years later the respondents were contacted again and 2302 (74%) were willing and still able to be interviewed again. Of the 805 non-participants, 417 (13.4% of the T_1 sample) had died, 253 (8.1%) were interviewed by telephone, which did not include the depression and anxiety assessment, 38 (1.2%) were too ill or cognitively impaired to be interviewed, 90 (2.9%) indicated that they were no longer interested in partaking in the study and 17 (0.5%) could not be contacted. Owing to item non-response on measures for depression and anxiety at T_1 or T_2, a further 139 respondents were lost, leaving 2163 subjects for whom depression and anxiety scores were available at both time points (69.6% of the T_1 respondents). The 944 non-participants were compared with the 2163 participants on key variables, to check for selective attrition. No significant difference was found between participants and non-participants regarding anxiety symptoms, but higher depression did make attrition more likely. For other variables we also found an association with attrition: non-participants were more likely to be male, unmarried, older, to be of lower socio-economic status and to have a chronic disease, more functional limitations and a lower score on the Mini-Mental State Examination (MMSE; Folstein et al, 1975). Thus, the subjects who were lost from T_1 to T_2 comprised the less healthy and worse functioning part of the initial participants at T_1.

At T_1 120 respondents (5.5%) had depression symptoms, 84 (4.0%) had anxiety symptoms and 147 (6.8%) had both depression and anxiety symptoms. Because we aimed to investigate *becoming* depressed or anxious, respondents with symptoms were removed from the sample, leaving 1810 respondents. Descriptive statistics and data on health and functioning of the final study sample are presented in Table 1.

Measures

Depression and anxiety

Depression was measured with the Center for Epidemiological Studies Depression scale (CES–D; Radloff, 1977). Depressive symptoms were considered present if the CES–D score was $\geqslant 16$. Anxiety was measured with the Anxiety sub-scale of the Hospital Anxiety and Depression Scale (HADS–A; Zigmond & Snaith, 1983). On the HADS–A the recommended cut-off of $\geqslant 7$ was applied to decide whether a respondent developed symptoms of anxiety or not. However, merely crossing the cut-off on the CES–D or HADS–A at T_2 was deemed insufficient to consider a respondent changed or destabilised, because it can result from random fluctuations due to measurement error on the symptom scale. Therefore, the additional requirement of statistically reliable change was set. To be considered reliably changed, a respondent has to change beyond the standard error of the difference score from T_1 to T_2 (Speer, 1992). For our respondents this implied a change of at least seven scale points on the CES–D and four scale points on the HADS–A. Subjects who crossed the cut-off but failed to meet the requirement of statistically reliable change were considered unchanged and categorised as below threshold.

Vulnerability

The following four sets of variables, all measured at T_1, were selected as vulnerability factors for depression and anxiety: demographics, variables regarding health and functioning, personality characteristics and social functioning.

Demographic vulnerability factors included female gender, higher age, not/no longer married, living in Amsterdam and lower socio-economic status. We used a weighted score composed of level of education, occupation and income (range 0–100) as a measure of socio-economic status (van Tilburg et al, 1995).

Variables regarding health and functioning were included as a separate set of vulnerability factors. In the stress–vulnerability model, somatic illness usually is considered a stressor and is included as such among the stressful life events. However, in view of the fact that health declines during old age often are gradual, ill health and functional limitations also may act as age-specific vulnerability factors. Physical health status was determined in the interview and cross-checked with information from general practitioners (Kriegsman et al, 1996). Physical health includes the number of chronic diseases reported by the respondent, a self-rated health assessment using a single item (Centraal Bureau voor de Statistiek, 1989) and functional limitations, assessed with an adaptation of the Organisation of Economic Collaboration and Development (OECD) Questionnaire (van Sonsbeek, 1988; Kriegsman et al, 1997). Problems with perception (eyesight and hearing) were assessed in the interview by asking the respondent whether they could see/hear "well enough" on a four-point scale ("Yes, no difficulty" to "No, I cannot see/hear"; Centraal Bureau voor de Statistiek, 1989). Responses were recoded into one variable with two levels (no or minor problems v. much difficulty or inability with either eyesight or hearing). Cognitive functioning was assessed with the MMSE.

Three personality characteristics of the respondents were assessed: mastery, neuroticism and self-efficacy. Mastery was assessed in the interview with the abbreviated five-item locus of control scale (Pearlin & Scooler, 1978). A higher score means a more external locus of control or less mastery. Neuroticism (15 items) was measured through the abbreviated sub-scale of the Dutch Personality Inventory (Luteijn et al, 1985). This self-report scale was completed after the interview and mailed in by the respondent. Not all respondents complied: 540 of the 2163 respondents (25%) failed to return fully completed questionnaires. Non-response on the self-report data was not related to the gender of the respondent but was related to higher age (more non-response in older respondents). Finally, coping was measured in the interview with a 12-item version of the General Self-efficacy Scale (Sherer et al, 1982; Bosscher & Smit, 1998).

Social resources were assessed by estimating the size of the social network. Respondents were asked to name people they regularly socialised with and whom they deemed important in various domains of life (relatives, neighbours, work, church, etc). The validity of the network size index was supported in a previous study (van Tilburg, 1994). Also, respondents were questioned about the exchange of emotional support with key members of their social network. Because "emotional support received" could be an important protective factor for developing psychopathology, in particular depression, this variable was included in the analyses as well.

Life events

In the T_2 interview it was assessed retrospectively whether stressful life events had occurred in the time interval between T_1 and T_2. Regarding the question of why some people develop depression whereas others develop anxiety, Finley-Jones & Brown (1981) have suggested that the type of event may be a decisive factor: stressful life events involving loss (e.g. death of a loved one, retirement) are more likely to lead to depression, whereas stressful events involving threat (e.g. being a victim of crime) lead to anxiety. The following stressful events were assessed: illness of one's partner, death of one's partner, illness of a relative, death of a relative, a major conflict with others, income loss (of at least £30 a month), being a victim of crime and relocation. Life events were analysed individually and we also obtained a single composite score for stress by differentially weighting life events. Weights for various life events were derived from Tennant & Andrews (1976).

Table I Descriptive data at T_1 of the sample (n=1810)

	n	%
Gender		
Male	904	49.9
Female	906	50.1
Age (years)		
55–69	1022	56.5
70–85	788	45.5
Marital status		
Not/no longer married	572	31.6
Married	1238	68.4
Urbanisation		
Amsterdam	444	24.5
Elsewhere	1366	75.5
Chronic diseases		
None	850	47.0
One	628	34.7
Two or more	332	18.3
Functional limitations		
None	1233	68.1
One or more	580	31.9
Hearing/eyesight		
No difficulty	1657	91.5
Difficulties	153	8.5
Cognitive functioning		
MMSE $\geqslant 24$	1707	94.7
MMSE < 24	96	5.3

MMSE, Mini-Mental State Examination.

All assessment instruments used in the study had been validated previously in The Netherlands or their psychometric properties had been evaluated in LASA pilot studies (Deeg *et al*, 1993).

Statistical analyses

A series of multivariate logistic regression analyses were undertaken, each time comparing one of the symptom groups with the non-symptomatic controls. First, vulnerability factors were analysed in four sets of conceptually related variables: demographics, health and functioning, personality and social functioning as measured at T_1. Thus, variables could be found that were associated significantly with destabilisation, while controlling for the effect of other variables within the same set. This approach limits the number of predictor variables and decreases the risk of collinearity among predictors. Next, the association of each life event and destabilisation for each symptom group was analysed with multiple logistic regression analyses. Finally, in order to attain the most parsimonious set of predictors, we used only the variables that appeared to have predictive value in previous analyses and performed a series of stepwise logistic regression analyses using a forward inclusion criterion of $P < 0.20$ (Menard, 1995).

To test hypotheses regarding interaction of vulnerability and stress, the interaction term was added to the main effects for each of the three models (Baron & Kenny, 1986; Hosmer & Lemeshow, 1989). In these analyses, we used the composite score for stressful life events.

RESULTS

Emergence of depression and anxiety symptoms

The 1810 respondents of the sample below threshold at T_1 were divided into four groups: 1642 (90.7%) respondents remained below threshold, 73 (4.0%) became depressed, 43 (2.4%) became anxious and 52 (2.9%) scored beyond threshold for both depression and anxiety. Inspection of the mean level of anxiety and depression in the three groups with symptoms revealed that the group with both anxiety and depression had the most serious condition because they suffered from more depression and anxiety compared with the groups with pure depression or pure anxiety. The T_2 mean score on the CES–D was $M=25.0$ (s.d.=8.5) for both depressed and anxious

subjects; $M=20.6$ (s.d.=4.9) for depressed subjects and $M=11.5$ (s.d.=4.9) for anxious subjects. Corresponding mean scores on the HADS–A were $M=10.7$ (s.d.=3.1), $M=3.7$ (s.d.=2.0) and $M=8.7$ (s.d.=2.7). All means were significantly different according to pairwise comparisons with t-tests.

Vulnerability to becoming depressed

Demographics, health and functioning personality and social functioning

Table 2 presents the results of multiple logistic regression analyses of variables representing vulnerability. The results indicate that for all three symptom groups the initial symptom level (averaged score on the CES-D and HADS-A at T_1) was predictive of destabilisation. For demographic variables the findings show that gender, age and socio-economic status were associated with scoring beyond threshold: females were overrepresented among the purely depressed and the purely anxious; the older old were more likely to be purely depressed; and low socio-economic status increased the chance for depression with anxiety. Results for social functioning were mixed: a smaller social network was predictive of becoming depressed, but not anxious, whereas less emotional support was predictive of anxiety but not depression. Social functioning was not a predictive factor for getting symptoms of both depression and anxiety. Regarding health status and functioning, we found that worse self-rated health predicted pure depression and pure anxiety, but not both anxiety and depression. Furthermore, functional limitations predicted the onset of depression and hearing/eyesight problems predicted the onset of both depression and anxiety. Of the personality variables, neuroticism was predictive of symptoms, especially for the group scoring beyond threshold on both depression and anxiety. Self-efficacy was predictive in all groups that scored beyond threshold at T_2.

Stepwise multivariate analyses

Next we evaluated all variables that significantly predicted scoring beyond threshold in three stepwise logistic regression analyses. For pure depression, the number of significantly predictive variables was reduced from eight to five (female gender, higher age, worse self-rated health, more functional limitations, and neuroticism). The likelihood ratio of the final model was χ^2

(6)=75.0, pseudo $R^2=0.16$. Four variables remained to predict anxiety (higher initial symptom level, female gender, less received emotional support and lower self-efficacy). The likelihood ratio of this model was χ^2 (4)=34.2, pseudo $R^2=0.11$. Getting symptoms of both anxiety and depression was predicted by four variables (higher initial symptom level, lower socio-economic status, having hearing or eyesight problems and neuroticism); the likelihood ratio was χ^2 (5)=40.2, pseudo $R^2=0.12$. All of the stepwise logistic regression analyses were repeated while omitting neuroticism (the variable was 25% missing values owing to non-response). The pattern of associations of the remaining variables on this larger sample did not differ meaningfully from the results described above. By and large, the findings of the stepwise analyses concur with the findings when vulnerability factors were analysed in sets. Apparently, most of the significant predictors of Table 2 have independent predictive value for destabilisation.

Life events associated with depression and anxiety

Stressful life events

Next, the predictive value of stressful life events that occurred in the time interval between both assessments was evaluated. Odds ratios were calculated representing the risk of crossing the threshold when an event had occurred. Table 3 presents the frequency of occurrence of events in the full sample and their odds ratios (with 95% CIs) for each of the three symptom groups. Statistically significant odds ratios are printed in bold typeface.

To investigate which events had an association with destabilisation while controlling for the effects of other events, we also performed a multiple logistic regression with stepwise selection of events. For pure depression, death of the partner, death of another family member and a major conflict were associated with destabilisation. The main events associated with an increase in anxiety symptoms were illness of the partner or a family member and having had a major conflict. Finally, events associated with crossing the threshold for both depression and anxiety were the death of a family member and having been victimised by crime.

Interaction of vulnerability factors and stressful life events

Finally, interactions between significant vulnerability factors (neuroticism, mastery,

social network size and coping style) and a selected set of stressful life events were studied. Low mastery was expected to amplify the effect of stress due to loss, and high neuroticism was expected to amplify the adverse effects of stress related to threat on mental health. Regarding social functioning, a protective effect of a large social network was hypothesised, moderating the adverse influence of stressful life events in general. Likewise, higher self-efficacy was hypothesised to protect for the effect of general stress. Hypotheses regarding effect modification were investigated by testing the main effect for each pair of predictors, as well as their interaction. Results showed that in analyses involving neuroticism and mastery none of the interaction terms were significant. Thus, no augmenting effect of neuroticism or low mastery on negative life events for developing depression and/or anxiety symptoms was found. This finding indicates that vulnerability factors and stressful life events, rather than interacting, add to each other in increasing the odds for destabilisation. Regarding the buffering effect of social functioning, only the interaction between received emotional support and stress for developing pure anxiety was marginally significant: respondents who reported more social support were less likely to develop anxiety when faced with stressful events (standardised $\beta=1.21$, OR$=3.3$ (0.9–12.6), $P=0.07$). No significant interaction of self-efficacy and general stress was found.

Table 2 Association of vulnerability factors[1] measured at T_1 with exceeding thresholds[2] for symptoms of depression, anxiety and both depression and anxiety according to multiple logistic regression analyses

	Became depressed ($n=73/1642$)	Became anxious ($n=43/1642$)	Both depressed and anxious ($n=52/1642$)
	OR (95% CI)	OR (95% CI)	OR (95% CI)
Symptom level at T_1			
Depression/anxiety	**1.3** (1.2–1.5)	**1.4** (1.2–1.6)	**1.4** (1.2–1.6)
Demographics			
Gender	**3.1** (1.7–5.4)	**5.1** (2.3–11.2)	1.1 (0.6–2.0)
Age	**2.0** (1.2–3.3)	1.2 (0.6–2.2)	1.1 (0.6–2.0)
Marital status	1.2 (0.7–2.0)	0.9 (0.5–1.8)	1.2 (0.6–2.2)
Socio-economic status	1.4 (0.9–2.3)	0.7 (0.4–1.5)	**1.8** (1.0–3.3)
Urbanisation	1.2 (0.7–2.1)	1.4 (0.7–2.7)	1.2 (0.7–2.3)
Social functioning			
Social network size[3]	**1.7** (1.1–2.9)	0.8 (0.4–1.6)	1.4 (0.8–2.6)
Received emotional support[3]	0.8 (0.5–1.3)	**2.1** (1.0–4.1)	1.3 (0.7–2.4)
Health status and functioning			
No. of chronic diseases	1.0 (0.6–1.8)	1.0 (0.5–2.0)	0.8 (0.4–1.5)
Self-rated health	**2.6** (1.5–4.3)	**2.1** (1.1–4.1)	2.4 (0.8–4.5)
Functional limitations	**1.7** (1.3–3.6)	1.3 (0.7–2.7)	1.6 (0.4–3.0)
Hearing/eyesight problems	1.4 (0.7–2.8)	0.7 (0.2–2.5)	**2.4** (1.2–4.9)
Cognitive functioning[4]	1.8 (0.8–3.8)	0.5 (0.2–1.5)	1.2 (0.4–3.0)
Personality traits			
Neuroticism	**2.8** (1.6–5.0)	**2.2** (1.1–4.6)	**5.6** (2.5–12.4)
Locus of control	1.6 (0.7–2.4)	1.2 (0.6–2.6)	1.3 (0.6–2.5)
Coping			
Self-efficacy[3]	**2.2** (1.4–3.6)	**2.1** (1.1–3.8)	**2.0** (1.5–3.5)

1. Vulnerability factors were dichotomised on the median to allow for comparison of the size of odds ratios (ORs).
2. Threshold for depression: Center for Epidemiological Studies Depression scale (CES–D) $\geqslant$ 16; threshold for anxiety: Hospital Anxiety and Depression Scale – Anxiety sub-scale (HADS–A) $\geqslant$7.
3. Scoring reversed.
4. Cognitive functioning corrected for educational level.

Table 3 Results of logistic regression analyses for the effect of stressful life events on destabilisation (bivariate analyses)

Life events	%	Depression ($n=73/1638$)	Anxiety ($n=43/1638$)	Depression and anxiety ($n=52/1638$)
		OR (95% CI)	OR (95% CI)	OR (95% CI)
Illness of partner	13.3	1.5 (0.8–2.8)	**2.0** (1.0–4.3)	1.4 (0.7–3.0)
Death of partner	5.8	**2.5** (1.2–5.2)	1.3 (0.4–4.4)	2.3 (1.0–5.6)
Illness of other relative	43.6	1.0 (0.6–1.6)	1.7 (0.9–3.1)	1.2 (0.7–2.1)
Death of other close relative	31.4	1.6 (1.0–2.6)	1.4 (0.7–2.5)	**2.0** (1.1–3.4)
Conflict with others	7.5	1.8 (0.9–3.7)	—[1]	1.2 (0.5–2.9)
Victim of crime	3.6	—[1]	—[1]	**2.9** (1.1–7.4)
Income loss	19.9	1.0 (0.6–1.8)	0.8 (0.4–1.5)	1.2 (0.7–2.2)
Relocation	12.7	1.7 (0.9–3.0)	0.7 (0.3–2.0)	0.9 (0.4–2.2)
Any of the above	73.9	1.7 (0.9–3.1)	1.4 (0.7–2.9)	1.8 (0.9–3.6)

Bold typeface indicates statistically significant results.
1. Insufficient respondents in a category.

DISCUSSION

Limitations

Analyses were performed on a substantially smaller subsample compared with the initial random sample because of attrition from T_1 to T_2. However, attrition for reasons other than mortality was limited. Additional analyses were performed to check for selective attrition. The T_2 sample did differ from the T_1 sample on many of the predictor variables but not in depression or anxiety level. The respondents who were lost had belonged to the older, less healthy, poorest functioning part of the T_1 sample. Furthermore, we selected a subgroup of non-depressed and non-anxious subjects at T_1. Analyses involving neuroticism further diminished the sample size because of non-response on the self-report questionnaires. Loss of subjects through attrition and limiting the sample to emotionally healthy subjects at T_1 limits the range in the risk factors, making it less probable to find significant associations. Furthermore, this reduction in size of the sample diminished the statistical power to find significant results, which is especially relevant for variables with low rates of occurrence, such as being a victim of crime.

Another cautionary remark regarding methodological aspects of the study concerns the status of life events as predictors.

Most life events were assessed retro-spectively at T_2 and therefore are not predictors in the true sense of the word. Our hypothesis was that negative life events would increase the odds for getting symptoms. However, respondents who destabil-ised into depression might have had a better recollection for negative events (mood congruent recall; Bower, 1981), thus biasing the results in favour of the hypo-thesis. On the other hand the most signifi-cant events, such as the death of one's spouse, are unlikely to be influenced by re-call bias. Nevertheless, a cautionary presen-tation of the present findings regarding life events describes them as associations rather than as directional relations. With these limitations in mind we will briefly review the most interesting findings.

Variables predictive of getting depression and/or anxiety symptoms

Important vulnerability factors for getting symptoms were female gender, high neuro-ticism and worse self-rated health. Generally, epidemiological studies report a higher pre-valence of depression and anxiety among women. High neuroticism was predictive for all symptom groups. Of course, neuro-ticism was associated with depression or anxiety level at baseline (cf. Clark *et al*, 1994). The present findings reveal the *prognostic* value of neuroticism for devel-oping emotional problems in a prospective design. Similar findings have been reported for younger age groups. Duncan-Jones (1987) reported the findings of a longitudi-nal study in an Australian sample of mixed-age adults where fluctuations in symptoms of psychopathology were well predicted by neuroticism scores. Ormel & Schaufeli (1991) replicated these findings in a sample of college students. The present findings complement this literature, by demonstrating the importance of premor-bid personality characteristics to emotional problems in late life.

Analysis of the vulnerability factors regarding health and functioning in late life revealed self-perceived poor health as a better predictor for mental health decline than chronic diseases. Apparently, how respondents experience their own health predicts subsequent emotional functioning more strongly than their objective health status. Similar findings were reported by Bath & Morgan (1998). In contrast, with mixed-age groups many studies report a

strong effect of somatic illness on emotional functioning (Viney & Westbrook, 1981). The adverse effect of a physical illness on emotional functioning may diminish with rising age, because in late life chronic diseases are much more common and accepted as a fact of life.

Regarding the specificity of vulnerability factors for either depression or anxiety, the similarity of risk profiles for the groups outweighs dissimilarity. However, some dis-crepancies are worth noting. Higher age was predictive only of depression and remained so after controlling for other factors, such as poorer self-rated health. This finding is in line with the literature documenting that the likelihood of depression increases with older age (Beekman *et al*, 1999). Functional limitations at baseline also were specifically associated with depression and not anxiety. Functional limitations have been impli-cated in depression among older persons previously. Regarding social functioning, network size and amount of emotional sup-port traded places in predicting depression and anxiety. This finding underlines the multi-dimensional nature of social support (Cohen & Wills, 1985).

Recent stressful life events

Partial support for the hypothesised specifi-city of loss *v.* threat events for depression and anxiety was found: loss events (e.g. the death of a family member) were associ-ated with becoming depressed, whereas threat (e.g. a family member getting a major illness) was predictive of becoming anxious. The significant association between death of the partner and depression may in part reflect bereavement rather than depression (Prince *et al*, 1997). However, in additional analyses, leaving out the respondents who lost their spouse in the previous 6 months, death of the partner was still a significant predictor of symptoms of depression. Unexpectedly, being a victim of crime (obviously a threat event) was associated with getting symp-toms of both anxiety and depression. Apparently, this event, although seldom occurring in the life of Dutch citizens, has a quite pervasive influence.

The distinctness of risk profiles for depression and anxiety in older persons underlines the validity of distinguishing these clusters of symptoms. It has been argued that this distinction diminishes in late life, where depression and anxiety are often found as comorbid conditions. The present results show that depression and

anxiety share common vulnerability factors, but also that distinct stressful events produce different outcomes.

Neuroticism, mastery, social support and self-efficacy as effect modifiers

The stress–vulnerability model hypothesises an augmenting effect of high neuroticism and low mastery (Goldberg & Huxley, 1992) and a buffering effect of social support for the effects of life events (Brown & Harris, 1978). Although strong direct effects of neuroticism and distress on destabilisation were found, no evidence for significant interactions with stress were attained. The more specific hypotheses linking neuro-ticism with threat events and mastery with loss events were not supported either. Apparently, the adverse effects of neuro-ticism and life's stress add on to each other, rather than amplify their mutual effects. The results regarding social functioning were similar. Analyses revealed only a marginally significant interaction effect of emotional support and distress for devel-oping anxiety symptoms, indicating that more emotional support buffers the effect of adverse life events. No other interaction terms were significant. The main effects of social support on the deterioration of mental health also were small, which is in line with recent findings (Olstad *et al*, 1999). Overall, the present results do not lend support to the interaction hypothesis of the stress–vulnerability model.

REFERENCES

Barbee, J. G. (1998) Mixed symptoms and syndromes of anxiety and depression: diagnostic, prognostic, and etiologic issues. *Annals of Clinical Psychiatry*, **10**, 15–29.

Baron, R. M. & Kenny, D. A. (1986) The moderator–indicator variable distinction in social psychological research: conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology*, **51**, 1173–1182.

Bath, P. A. & Morgan, K. (1998) Customary physical activity and physical health outcomes in later life. *Age and Ageing*, **27**, 29–34.

Beekman, A. T. F., Deeg, D. J. H., vanTilburg, T., et al (1995) Major and minor depression in later life: a study of prevalence and risk factors. *Journal of Affective Disorders*, **34**, 41–49.

__ , Copeland, J. R. M. & Prince, M. J. (1999) Review of community prevalence of depression in later life. *British Journal of Psychiatry*, **174**, 307–311.

__ , de Beurs, E., van Balkom, A. J. L. M., *et al* (2000) Anxiety and depression in later life: co-occurrence and communality of risk factors. *American Journal of Psychiatry*, **157**, 89–95.

Bosscher, R. J. & Smit, J. H. (1998) Confirmatory factor analysis of the General Self-efficacy Scale. *Behaviour Research and Therapy*, **36**, 339–343.

Bower, G. H. (1981) Mood and memory. *American Psychologist*, **36**, 129–148.

Brown, G. W. & Harris, T. O. (1978) *Social Origins of Depression*. London: Tavistock.

Centraal Bureau voor de Statistiek (1989) *Health Interview Questionnaire*. Heerlen: CBS.

Clark, L. A. & Watson, D. (1991) A tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, **100**, 316–336.

__ , __ & Mineka, S. (1994) Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, **103**, 103–116.

Cohen, S. & Wills, T. (1985) Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, **98**, 310–357.

de Beurs, E., Beekman, A. T. F., van Balkom, A. J. L. M., *et al* (1999) Consequences of anxiety in older persons: its effect on disability, well-being and use of health services. *Psychological Medicine*, **29**, 583–593.

Deeg, D. J. H., Knipscheer, C. P. M. & van Tilburg, W. (1993) *Autonomy and Well-being in the Aging Population: Concepts and Design of the Longitudinal Aging Study Amsterdam*. Bunnik: Netherlands Institute of Gerontology.

Duncan-Jones, P. (1987) Modelling the aetiology of neurosis: long-term and short-term factors. In *The Epidemiology of Psychiatric Disorders* (ed. B. Cooper), pp. 178–191. Baltimore, MD: Johns Hopkins University Press.

Finley-Jones, R. & Brown, G. W. (1981) Types of stressful life event and the onset of anxiety and depressive disorders. *Psychological Medicine*, **168**, 50–57.

Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975) Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, **12**, 189–198.

Goldberg, D. P. & Huxley, P. (1992) *Common Mental Disorders: A Bio-Social Model*. London: Routledge.

Hosmer, D. W. & Lemeshow, S. (1989) *Applied Logistic Regression*. New York, NY: John Wiley & Sons.

Kirby, M., Bruce, I., Radic, A., *et al* (1997) Mental disorders among the community-dwelling elderly in Dublin. *British Journal of Psychiatry*, **171**, 369–372.

Kriegsman, D. M. W., Penninx, B. W. J. H., van Eijk, J. T. M., *et al* (1996) Self-reports and general practitioner information on the presence of chronic diseases in community-dwelling elderly: a study on agreement and determinants of disagreement. *Journal of Clinical Epidemiology*, **49**, 1409–1417.

__ , Deeg, D. J., van Eijk, J. T., *et al* (1997) Do disease specific characteristics add to the explanation of mobility limitations in patients with different chronic diseases? A study in The Netherlands. *Journal of Epidemiology and Community Health*, **51**, 676–685.

Luteijn, F., Starren, J., van Dijk, H. (1985) *Manual for the Dutch Personality Questionnaire* [Handleiding bij de NPV]. Lisse: Swets & Zeitlinger.

MacKinnon, A. J., Christensen, H., Jorm, A. F., *et al* (1994) A latent trait analysis of an inventory designed to detect symptoms of anxiety and depression using an elderly community sample. *Psychological Medicine*, **24**, 977–986.

Menard, S. (1995) *Applied Logistic Regression Analysis*. Thousand Oaks, CA: Sage.

Olstad, R., Sexton, H. & Sogaard, A. J. (1999) The Finnmark study. Social support, social network and mental distress in a prospective population study. *Social Psychiatry and Psychiatric Epidemiology*, **34**, 519–525.

Ormel, J. & Schaufeli, W. B. (1991) Stability and change in psychological distress and their relationship with self-esteem and locus of control: a dynamic equilibrium model. *Journal of Personality and Social Psychology*, **60**, 288–299.

Pearlin, L. J. & Schooler, C. (1978) The structure of coping. *Journal of Health and Social Behavior*, **19**, 2–21.

Prince, M., Harwood, R., Blizard, R., *et al* (1997) Social support deficits, loneliness and life events as risk factors for depression in old age. The Gospel Oak Project VI. *Psychological Medicine*, **27**, 323–332.

Radloff, L. S. (1977) The CES–D scale: a self-report depression scale for research in the general population. *Applied Psychological Measurement*, **1**, 385–401.

Sherer, M., Maddux, J. E., Mercandante, B., *et al* (1982) The self-efficacy scale: construction and validation. *Psychological Reports*, **51**, 663–671.

Speer, D. C. (1992) Clinically significant change: Jacobson and Truax (1991) revisited. *Journal of Consulting and Clinical Psychology*, **60**, 402–408.

Tennant, C. & Andrews, G. (1976) A scale to measure the stress of life events. *Australian and New Zealand Journal of Psychiatry*, **10**, 27–32.

van Sonsbeek, J. L. A. (1988) Methodological and content related aspects of the OECD indicator of chronic functional limitations. *Maandbericht Gezondheid*, **88**, 4–17.

van Tilburg, T. (1994) Losing and gaining in old age: changes in personal network size and social support in a four-year longitudinal study. *Journal of Gerontology*, **53B**, S313–S323.

__ , Dykstra, P., Liefbroer, A. C., *et al* (1995) *Sourcebook of Living Arrangements and Social Networks of Older Adults in The Netherlands*. Amsterdam: Free University Press.

Viney, L. & Westbrook, M. (1981) Psychological reactions to chronic illness-related disability as a function of its severity and type. *Journal of Psychosomatic Research*, **25**, 513–523.

Zigmond, A. S. & Snaith, R. P. (1983) The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, **67**, 361–370.

CLINICAL IMPLICATIONS

■ Important risk factors for getting emotional problems in late life are female gender, bad health, high neuroticism and stressful events.

■ Onset of depression is best predicted by the death of a partner or family member; anxiety is best predicted by having a partner who develops a major illness.

■ Getting symptoms of both depression and anxiety is associated with low socio-economic status, hearing/eyesight problems and falling victim to crime.

LIMITATIONS

■ This analysis was limited to emotionally healthy respondents; factors predictive of recovery were not studied.

■ Although the initial study sample was sizable, the incidence of anxiety and depression symptoms is low, generating relatively small numbers of subjects in the groups that were compared, which limits the power to find significant associations.

■ Because symptoms were assessed with a 3-year interval, short bouts of depression or anxiety may have been missed, thereby ignoring short-lived immediate responses to stressful life events.

EDWIN DE BEURS, PhD, AARTJAN BEEKMAN, MD, SANDRA GEERLINGS, MSc, DORLY DEEG, PhD, RICHARD VAN DYCK, MD, WILLEM VAN TILBURG, MD, Institute for Research in Extramural Medicine & Department of Psychiatry, Vrije Universiteit, Amsterdam

Correspondence: Edwin de Beurs, Leiden University Medical Center, PO Box 9600, 2300 RC Leiden, The Netherlands