Predictors of change over three years of anxiety symptoms of older persons: results from the Longitudinal Aging Study Amsterdam

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Predictors of change in anxiety symptoms of older persons: results from the Longitudinal Aging Study Amsterdam

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ABSTRACT

Background. Data on the course of anxiety in late life are scarce. The present study sets out to investigate the course of anxiety, as measured by the HADS-A (Zigmond & Snaith, 1983) in community dwelling older persons, and to evaluate predictive factors for change over 3 years in anxiety symptoms following the vulnerability/stress model.

Method. Based on the first anxiety assessment, two cohorts were formed: subjects with and subjects without anxiety symptoms. In the non-anxious cohort ($N = 1602$) we studied risk factors for the development of anxiety symptoms; in the anxious cohort ($N = 563$) the same factors were evaluated on their predictive value for restitution of symptoms. Risk factors included vulnerability factors (demographics, health status, personality characteristics and social resources) and stressors (life events occurring in between both anxiety assessments). Logistic regression models estimated the effects of vulnerability factors, stress and their interaction on the likelihood of becoming anxious and chronicity of anxiety symptoms.

Results. It was indicated that the best predictors for becoming anxious were being female, high neuroticism, hearing/eyesight problems and life-events. Female sex and neuroticism also increased the likelihood of chronicity of anxiety symptoms in older adults, but life events were not related to chronicity. The main stressful event in late life associated with anxiety was death of one’s partner. Vulnerability factors and stress added on to each other rather than their interaction being associated with development or chronicity of anxiety.

Conclusion. The vulnerability/stress model offers a useful framework for organizing risk factors for development and chronicity of anxiety symptoms in older persons, but no support was attained for the hypothesis that vulnerability and stress amplify each others effects. Finally, the results indicate to whom preventive efforts should be directed: persons high in neuroticism, women, and those who experience distressing life events.

INTRODUCTION

In recent years interest in the prevalence, course, and aetiology of common mental disorders in older persons is growing. Most research is aimed at depression, but there is an increasing awareness of the importance of anxiety in later life (Salzman & Lebowitz, 1991; Small, 1997). Epidemiological data suggest that anxiety disorders are quite prevalent among older persons, only slightly less prevalent than in younger age groups (Flint & Rifat, 1997; Beekman et al. 1998) and equal to depression and dysthymia. Furthermore, the adverse impact of anxiety on quality of life and use of health-care resources match the consequences of major depression in older persons (de Beurs et al. 1999).
Regarding the aetiology of common mental disorders in older persons, research is scarce and again has focused mainly on depression (e.g. Beekman et al. 1995; Karel, 1997; Bifulco et al. 1998). The present study aims at investigating in a prospective design how anxiety in older persons comes about and why it persists. Utilizing data from a longitudinal study of community dwelling older persons, risk factors for anxiety were investigated, building on the vulnerability-stress model (Goldberg & Huxley, 1992). This model states that psychopathology results from the interplay of environmental stressors and vulnerability to develop psychopathology. Vulnerability factors should account for the considerable differences in susceptibility between individuals to develop an illness in the face of stress. Furthermore, the duration or chronicity of symptoms varies widely across persons. Persistence or remittance of symptoms is also thought to result from vulnerability factors and stressful events. Research shows that factors related to the onset of symptoms overlap to a large extent with factors that determine persistence of symptoms (Goldberg & Huxley, 1992). Vulnerability and protective factors that are best studied are social support (Brown & Harris, 1978; Cohen & Wills, 1985) and personality variables, such as emotional strength or neuroticism (Clark et al. 1994). The negative influence of life events on mental health has been reviewed by Paykel (1994). The utility of the model has been tested in clinical samples as well as community samples, and is generally deemed useful for conceptualizing risk factors for psychopathology. Whether it is also applicable to anxiety in older people has thus far not been tested.

In the present study, symptoms of anxiety were measured twice over an interval of 3 years in a two-wave longitudinal design. Data on anxiety symptoms were recoded following a categorical approach to define subgroups within the sample according to their change in anxiety status. Four groups were distinguished: subjects who became anxious (destabilized ‘cases’), subjects who remained free of anxiety, subjects with a chronic high level of anxiety, and subjects with decreased anxiety (restituted ‘cases’).

It was investigated whether changes in anxiety could be predicted from vulnerability factors or protective factors measured at the start of the time interval and from stressors that had occurred in between both anxiety assessments. Regarding vulnerability/protective factors, four groups of variables were studied: (1) demographic variables such as age, sex, and marital status; (2) variables regarding health status and functioning of the respondent at the first assessment; (3) variables that represent stable psychological traits which may have an aetiological link with the development of anxiety; and (4) social resources. Stressors included the occurrence of events that are expected to have an influence on anxiety, e.g. life events (relocation, retirement, death of family members, etc.), deterioration of health, increase in functional limitations, and deterioration of eyesight or hearing. By comparing destabilized cases with stable low anxious subjects, predictors could be evaluated on their prognostic value for the initiation of anxiety; by comparing restituted cases with stable high anxious subjects, factors predictive of chronicity of anxiety symptoms could be investigated. Finally, the interplay between vulnerability and stress on change in anxiety was studied.

In sum, the study aimed at addressing the following research questions: (1) what are the main effects of vulnerability/protective factors and stressors on destabilization and chronicity of anxiety symptoms in older persons; (2) do the same factors account for destabilization of anxiety and chronicity of symptoms or are different factors involved; (3) do vulnerability factors and stress have an interactive effect on change in anxiety symptoms?

METHOD
Sample and procedure
For the Longitudinal Aging Study Amsterdam (LASA), a random age and sex stratified sample of older (55–85 years) men and women was drawn from the population registers of 11 municipalities in three regions of the Netherlands. Older men were initially oversampled, to ensure sufficient respondents in these strata for a later phase of the study. In the first cycle of LASA (T1), 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 (T2) the respondents were contacted again and 2302 (74%) were still willing and able to partake in the study: 417 respondents (13.4% of the T1 sample) had died before the T2 assessment took place. Of the remaining 2690 respondents, 253 (8.1% of the T1 sample) were interviewed by telephone, which did not include the anxiety assessment, 90 (2.9%) indicated that they were no longer interested in participating in the study, 38 (1.2%) were too ill or cognitively impaired and 17 (0.5%) could not be contacted. Due to item non-response on the main dependent measure in the study, the anxiety subscale of the Hospital Anxiety Depression Scale (HADS-A, Zigmond & Snaith, 1983) at T1 or T2 a further 137 respondents were lost, leaving 2165 subjects for whom an anxiety symptom score was available at both time points (69.7% of the T1 respondents). Older and male respondents were less likely to be included in the second assessment. Non-participants did not differ from the participants in level of anxiety, but did differ on some variables related to change in anxiety (see Results section).

Interviews were conducted in the homes of the respondents by trained and intensively supervised interviewers. All interviews were audio-taped to allow random quality checks. The first cycle of LASA data collection took place between October 1992 and October 1993, the second cycle 3 years later (1995–1996). Some data were collected by means of self-administered questionnaires. After the interview the interviewer left a package of questionnaires for the respondent to complete and send to the study centre. Not all respondents complied: 430 of the 2165 respondents (20%) failed to return completed questionnaires. Self-administered questionnaire data used in the present study are neuroticism and social inadequacy (see below). Non-response on the self-report data was not related to sex of the respondent, but was related on some variables related to change in anxiety (see Results section).

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**Measurements**

**Assessment of change in anxiety status**

Anxiety was measured with the Hospital Anxiety and Depression Scale-Anxiety subscale (HADS-A). This is a 7-item anxiety subscale of the HADS (Zigmond & Snaith, 1983). It is composed of Likert type items, in which the respondent is asked to indicate whether he/she over the past 4 weeks has experienced feelings such as restlessness, tenseness, or panic on a scale scoring from 0 (seldom or never) to 3 (always or almost always). The anxiety subscale score has a theoretical range from 0 to 21.

The HADS-A score at baseline was used to divide the sample into two cohorts of anxious and non-anxious subjects. To distinguish subjects with and without anxiety symptoms, a cut-off point between 3 and 4 on the HADS-A was used. This cut-off was based on a comparison of HADS-A scores of the entire sample with scores of subjects who fulfilled criteria for an anxiety disorder. Part of the sample of the first assessment had been diagnosed with the Diagnostic Interview Schedule (DIS, Robins et al. 1981). A score of ≥ 4 on the HADS-A appeared to be a useful cut-off value to delineate the transition of no anxiety to a diagnosable anxiety disorder (de Beurs et al. 1999). Sensitivity of this cut-off on the HADS-A was 59% and specificity 80%, all for 6-months prevalence of any DSM-III anxiety disorder.

Change in anxiety status was defined as crossing the cut-off value of 4. However, merely crossing the cut-off was deemed insufficient for deciding on whether a subject had changed from non-anxious to anxious or vice versa. Falling on either side of the cut-off point can result from measurement error of the HADS-A. Therefore, the additional requirement of a statistically reliable change on the HADS-A was set, utilizing the Edwards–Nunnally method (Speer, 1992). A respondent’s level of anxiety was considered truly changed, if the score at the second assessment of anxiety fell outside the 5% confidence interval of his or her first assessment. For the Dutch translation of the HADS-A this means on average a difference score of 2.88 (using test–retest reliability and data on scale variance of Spinhoven et al. 1997). Variance of the baseline scores of the present sample was similar to those reported by Spinhoven et al. (1997). Thus, to be considered reliably changed a respondent had to shift 3 or more scale points, which is close to a standard deviation change. According to these two criteria, subjects were assigned to four groups: (1) destabilized cases who became anxious; (2) never anxious respondents; (3) chronically anxious respondents; and (4) no longer anxious or restituted cases.
Risk factors
Following the stress-vulnerability model we selected stable vulnerability factors that had been measured at the time of the first anxiety assessment (female sex, higher age, not/never married, lower socio-economic status (SES), living in Amsterdam, deteriorated physical health). As measure of SES we used a weighted score composed of level of education, occupation and income (range 0–100; van Tilburg et al. 1995). Physical health status was determined in an interview using a detailed questionnaire on presence, duration, principal symptoms, complications, and treatment of common chronic physical diseases (cardiac diseases, peripheral artherosclerosis, stroke, diabetes mellitus, lung diseases, cancer and arthritis). Other chronic diseases were assessed in less detail. The validity of the instrument was supported in a previous study by cross-checking responses with respondents' general practitioners (Kriegsman et al. 1996). For this study, the presence of one or more of these seven chronic diseases was used as indicator of disease burden. Furthermore, self-perceived health was measured with a single question (CBS, 1989); functional limitations were assessed with a previously validated scale developed by the Organization for Economic Collaboration and Development (OECD, van Sonsbeek, 1988; Kriegsman et al. 1997); perception (eyesight and hearing) was assessed by self-report in the T1 and T2 interviews (CBS, 1989). Cognitive functioning was assessed with the Mini-Mental State Exam (MMSE, Folstein et al. 1975). Scores on the MMSE are influenced by level of education. Therefore, education level was controlled for when evaluating the association between cognitive functioning and changes in anxiety.

Four psychological characteristics of the respondents were assessed. Neuroticism (15 items) and Social Inadequacy (dislike and avoidance of social interactions; 10 items) are abbreviated subscales of the Dutch Personality Inventory (validated by Luteijn et al. 1985). Mastery was measured with the abbreviated 5-item ‘mastery’ scale (Pearlin & Scooler, 1978) and self-efficacy with the 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 socialized with regularly and whom they deemed important to them in various domains (relatives, neighbours, work, church, etc.). The validity of the network size index was supported in a previous study (van Tilburg, 1998). Also, respondents were questioned about the exchange of emotional and instrumental support from key members of their social network. Since especially ‘emotional support received’ could serve as an important buffer in the relation between negative life-events and psychopathology, this variable was also included in the analyses. Finally, self-reported loneliness was assessed on an 11-point scale (1 = not lonely to 11 = very lonely, de Jong-Gierveld & Kamphuis, 1985).

Stress was operationalized by assessing events that had occurred in between both anxiety assessments. Respondents were interviewed at T2 about the following life events: death of one’s partner; income loss (at least 100 dfl a month); job loss through retirement, dismissal, or disability; being a victim of crime; relocation; illness of a partner; illness of a relative (father, mother, brother, sister, son, daughter, or grandchild); death of a relative. Other life stressors were derived from the data at both time points: declined physical health (developing a new or additional chronic disease); declined health according to the respondent’s self-report; decreased cognitive functioning ( > 5 points deterioration on the MMSE, Schmand et al. 1995); an increase in functional limitations ( ≥ 1 points decrease on the scale) and hearing or eyesight deterioration (decreased ability on either or both). To reduce the number of variables in analyses regarding life stressors, one variable was composed for stress by differentially weighting life-events and combining them in a composite score representing the impact of the events. Weights for various life-events were derived from Tennant & Andrews (1976). All scales used in the study had been previously validated in the Netherlands or their psychometric properties were evaluated in LASA pilot studies (Deeg et al. 1993).

Statistical analyses
To investigate the prognostic value of vulnerability factors and stress, first the main effects of variables on group membership were assessed
by calculating a series of bivariate associations within the two pairs of groups, comparing destabilized anxious with never anxious respondents and comparing chronically anxious with rested respondents. The strength of the association between dichotomous predictors and change in anxiety status was expressed in odds ratios (ORs). To enable direct comparisons between predictor variables, scores on continuous variables were dichotomized on their median score. Next, the effect of vulnerability, stress, and the interaction between the two on change in anxiety status was investigated with multiple logistic regression analyses. Independent variables were entered in blocks of conceptually related variables and also the most parsimonious set of predictor variables was sought by a stepwise logistic regression procedure. For these analyses, continuous variables were left intact to preserve statistical power.

RESULTS

Sample attrition

In LASA, male respondents of older age were initially oversampled to ensure sufficient respondents in later waves of the longitudinal course of the study. This oversampling resulted in sufficient older men 3 years into the study, but also led to considerable attrition over time. Inevitably, longitudinal data from older respondents suffer from attrition, which is even more so if vulnerable strata are oversampled. T2 anxiety data were available for 70% of the T1 sample. To check for selective attrition we compared the 2165 participating subjects with the 942 non-participants on all variables that were included as predictors with \( \chi^2 \) tests or \( t \) tests. Results indicated that overall, non-participants at T2 were more likely to be male and unmarried. Furthermore, they were older, had lower SES, and were more likely to suffer from one or more chronic disease. Also, non-participants at T2 had reported less mastery and less self-efficacy and their social functioning had been worse at T1. Finally, the cognitive functioning of non-participants had been worse. Thus, the subsample for which longitudinal data were available comprised of individuals who had been the healthier and better functioning part of the larger group of participants at T1. Importantly, participants and non-participants at T2 did not differ in anxiety level at T1. To adjust for the effects of stratified sampling and attrition, a weighting procedure was followed regarding the variables age and sex. Since older male respondents were still over-represented at T2 their data were given a proportionally smaller weight utilizing STATA statistical software for the analyses.

Changes in anxiety

Comparison of scores on the HADS-A at T1 and T2 revealed that, on average, the sample became slightly (but to a statistically significant degree) more anxious over time \( (t(2164) = 2.23; P < 0.05) \). The mean score on the HADS-A at T1 was \( x = 2.51 (\pm 3.28) \) and at T2 was \( x = 2.67 (\pm 3.31) \). Table 1 presents the number of subjects with and without anxiety symptoms at T1 and T2 and information on statistically reliable change. At T1 26.0% of the respondents were anxious; at T2 29.6% scored above the cut-off on the HADS-A. As can be deduced from Table 1, subjects who crossed the cut-off, but whose anxiety level did not change to a statistical reliable degree were considered unchanged and categorized according to their T1 score (77 cases in the never anxious group and 58 cases in the always anxious group).

Vulnerability for anxiety: main effects for demographic variables and health and functioning

Table 2, section A presents bivariate odds ratios (and 95% confidence intervals) for demographic variables measured at the first assessment with change in anxiety status over time. Odds ratios in bold typeface indicate a significantly higher chance of becoming anxious or a higher chance of remaining anxious.

The results reveal that the distribution of the sexes differed over the groups, with women over-represented in the destabilized and the chronically anxious groups. Higher age was associated with destabilization (marginally significant at \( P = 0.05 \)). Marital status, SES and urbanicity were not associated with changes in anxiety.

Section B of Table 2 presents ORs (with 95% CI) for five variables that represent health status at T1. Most variables were associated with destabilization (low perceived health, more functional limitations, and problems with hear-
Table 1. **Number of respondents grouped according to anxiety status at time 1 and time 2**

<table>
<thead>
<tr>
<th>Anxiety symptoms at T1</th>
<th>No (1602 (740%))</th>
<th>Yes (310 (14.3%))</th>
<th>563 (26.0%)</th>
<th>No (233 (10.7%))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety symptoms at T2</td>
<td>No 1292 (59.7%)</td>
<td>Yes 310 (14.3%)</td>
<td>Yes 330 (15.2%)</td>
<td>No 233 (10.7%)</td>
</tr>
<tr>
<td>No reliable change</td>
<td>77 (3.6%)</td>
<td>1369 (63.2%)</td>
<td>233 (10.8%)</td>
<td></td>
</tr>
<tr>
<td>Final categorization</td>
<td>Never anxious</td>
<td>Destabilized</td>
<td>Always anxious</td>
<td>Reinstated</td>
</tr>
</tbody>
</table>

Table 2. **Predictors for change in anxiety status measured at time 1 (N = 2165)**

<table>
<thead>
<tr>
<th>A Demographic variables</th>
<th>Destabilization (N = 233 v. 1369)</th>
<th>Chronicity (N = 388 v. 175)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female sex</td>
<td>N 1148</td>
<td>N 1208</td>
</tr>
<tr>
<td>Age ≥ 70</td>
<td>OR 24</td>
<td>OR 22</td>
</tr>
<tr>
<td>Not/no longer married</td>
<td>1.4†</td>
<td>1.4</td>
</tr>
<tr>
<td>SES &lt; 33</td>
<td>0.8–1.6</td>
<td>0.7</td>
</tr>
<tr>
<td>Living in Amsterdam</td>
<td>0.7–1.5</td>
<td>0.5–1.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>B Health status</th>
<th>Destabilization (N = 233 v. 1369)</th>
<th>Chronicity (N = 388 v. 175)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic disease</td>
<td>1208</td>
<td></td>
</tr>
<tr>
<td>Perceived health</td>
<td>762</td>
<td></td>
</tr>
<tr>
<td>Functional limitations</td>
<td>770</td>
<td></td>
</tr>
<tr>
<td>Hearing/eyesight problem</td>
<td>221</td>
<td></td>
</tr>
<tr>
<td>MMSE &lt; 24</td>
<td>129</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C Personality characteristics</th>
<th>Destabilization (N = 233 v. 1369)</th>
<th>Chronicity (N = 388 v. 175)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuroticism (≥ 5)</td>
<td>800</td>
<td></td>
</tr>
<tr>
<td>Social inadequacy (≥ 5)</td>
<td>819</td>
<td></td>
</tr>
<tr>
<td>Mastery (≥ 12)</td>
<td>1274</td>
<td></td>
</tr>
<tr>
<td>Self-efficacy (≥ 42)</td>
<td>939</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>D Social functioning</th>
<th>Destabilization (N = 233 v. 1369)</th>
<th>Chronicity (N = 388 v. 175)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social netw. size (≤ 12)</td>
<td>1056</td>
<td></td>
</tr>
<tr>
<td>Emot. supp. rec. (≤ 12)</td>
<td>1083</td>
<td></td>
</tr>
<tr>
<td>Loneliness ≤ 2</td>
<td>899</td>
<td></td>
</tr>
</tbody>
</table>

* Marginaly significant (P = 0.05).
† An odds ratio with a confidence interval that does not include 1.0 is statistically significant (P < 0.05).
‡ Controlled for level of education.

Variables were dichotomized as follows: chronic diseases (none or 1 or more); perceived health (excellent or good vs. fair or poor); functional limitations (none vs. 1 or more). Otherwise dichotomization at the median.

Bold typeface indicates a significantly higher chance of becoming anxious or a higher chance of remaining anxious.

Vulnerability for anxiety: main effects of personality characteristics and social functioning

For 1735 of the 2165 subjects (80%) who had completed both anxiety assessments, self-report data on neuroticism and social inadequacy were available. Missing data were proportionally distributed over the four groups and not related to sex. Urbanicity and age of the respondent were related to non-response on the self-report measures, however. Older respondents and residents of Amsterdam were less likely to comply with the instructions to complete and return the questionnaires. By again weighting the data for these analyses we corrected for attrition due to non-response. Table 2, section C presents the ORs (and 95% CI) for neuroticism, social inadequacy, mastery and self-efficacy. High neuroticism, less self-efficacy, and, to a somewhat lesser extent, low mastery at T1 were predictive of destabilization. High neuroticism and low mastery were predictive of chronicity. There was no significant association between social inadequacy at T1 and change in anxiety status. Finally, section D of Table 2 shows the ORs (and 95% CI) for variables regarding social functioning. The results reveal no significant...
association for developing anxiety, but restitution of anxiety was associated with the size of the social network and self-reported loneliness at T1. Subjects who remained anxious had smaller social networks and had reported more loneliness at T1.

Stressors: main effects of life-events and changes in health and functioning

Next, we inspected bivariate associations between stressful events that had occurred in the 3 years in between both assessments and destabilization or restitution of anxiety (Table 3). Two types of stressors were distinguished: personal events and deterioration of health and functioning. The results revealed a significant increase of the incidence of anxiety among the recently widowed. Illness of the partner was also associated with an increase in anxiety. No effect of income loss and job loss was found. Both events were related to age: only in the youngest age group a substantial number of subjects experienced these life events. Therefore, we repeated the bivariate analyses for the age group of 55 to 64 years only. These analyses did not reveal a significant association between loss of job or loss of income and becoming anxious either.

Next, the association between changes in health and functioning and change in anxiety status was analysed. An increase in the number of chronic diseases in the past 3 years was not associated with a higher incidence of anxiety. In addition, we investigated whether developing a first chronic disease was associated with a change in anxiety. Respondents with a ‘new’ disease were compared with those whose somatic condition had remained stable. Again, no relation with anxiety was found. Additional analyses, for each of the seven somatic diseases separately, did not reveal a significant relation with anxiety either (results not shown). However, decreased perceived health, increased functional limitations, and deterioration of hearing and/or eyesight (marginally significant at \( P < 0.07 \)) were associated with change in anxiety. Respondents who rated their health lower at T2 as compared to T1, who reported more limitations in functioning, and more problems with hearing or eyesight were over-represented among those who became anxious. Regarding chronicity of anxiety, no events appeared significantly associated. Similar to the results for developing anxiety, chronicity was associated with decreased subjective health and increased limitations in functioning.

It could not be demonstrated that cognitive decline was a significant risk factor for anxiety. Schmand et al. (1995) propose a drop of > 5 scale points on the MMSE as a meaningful deterioration in cognitive functioning. In the present sample, only very few subjects (\( N = 58; 2.7\% \)) had experienced a decline of such magnitude, a number insufficient for statistical testing. Probably, respondents with a substantial cognitive decline were selectively lost to the second cycle of data gathering. Utilizing a more lenient criterion of > 3 scale points did not reveal a significant association of cognitive decline and anxiety either (results not shown).

For subsequent analyses, stressful life events and deterioration of health and functioning were summed into one variable representing the cumulative distress caused by these event(s). The score was positively skewed, but normalized by a square-root transformation. The transformed distress score had a range of 1–17.3 and the mean score was \( x = 6.19 \ (\pm 3.28) \). The distress score was different for destabilized respondents \( (x = 6.59 \pm 3.29) \) and never anxious respondents \( (x = 6.00 \pm 3.29) \) (\( t(1600) = 2.52; \ P < 0.02; \ d = 0.18 \)), which delineates a small effect size. No difference was found between chronically anxious and restituted respondents \( (t(561) = 0.14; \ P = 0.89) \).

Multiple analyses

A series of logistic regression analyses were performed to investigate the prognostic value of vulnerability factors and distress for change in anxiety while controlling for each other in a multivariate design. For these analyses predictors were selected that were significantly associated with destabilization or restitution according to the bivariate analyses presented in Table 2. Stressful events and changes in health status and functioning were included through their contribution to the composite for distress. First, the anxiety level at T1 was entered in the model to correct for baseline level of anxiety, next demographic variables, variables regarding health and functioning, personality characteristics, social functioning (self-reported loneliness) and, finally, the composite distress score.
Table 3. Bivariate associations between stressful events in-between both assessments and change in anxiety status

<table>
<thead>
<tr>
<th>Events</th>
<th>Destabilization</th>
<th>Chronicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Partner died</td>
<td>132</td>
<td>0.6</td>
</tr>
<tr>
<td>Illness partner</td>
<td>286</td>
<td>0.5</td>
</tr>
<tr>
<td>Death of a relative</td>
<td>676</td>
<td>1.3</td>
</tr>
<tr>
<td>Illness relative</td>
<td>445</td>
<td>1.1</td>
</tr>
<tr>
<td>Income loss</td>
<td>445</td>
<td>0.8</td>
</tr>
<tr>
<td>Job loss</td>
<td>129</td>
<td>0.9</td>
</tr>
<tr>
<td>Victim of crime</td>
<td>80</td>
<td>1.2</td>
</tr>
<tr>
<td>Relocation</td>
<td>283</td>
<td>1.1</td>
</tr>
<tr>
<td>Health and functioning</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increase in chronic diseases</td>
<td>619</td>
<td>0.9</td>
</tr>
<tr>
<td>Developed a chronic disease</td>
<td>272</td>
<td>0.8</td>
</tr>
<tr>
<td>Decreased perceived health</td>
<td>304</td>
<td>3.2</td>
</tr>
<tr>
<td>Increased functional limitations</td>
<td>388</td>
<td>1.9</td>
</tr>
<tr>
<td>Hearing/eyesight deterioration</td>
<td>604</td>
<td>1.6</td>
</tr>
<tr>
<td>Decreased cognitive funct.</td>
<td>58</td>
<td></td>
</tr>
</tbody>
</table>

* Marginaly significant (P < 0.07)  
† An odds ratio with a confidence interval that does not include 1:00 is statistically significant (P < 0.05).  
‡ Cells with N < 5 preclude testing.  
Bold typeface indicates a significantly higher chance of becoming anxious or remaining anxious.

Table 4. Estimated coefficients for models comprising vulnerability factors and distress due to life events for becoming anxious (destabilization) and for remaining anxious (chronicity) in older persons

<table>
<thead>
<tr>
<th></th>
<th>Destabilization (180 v. 1025)</th>
<th>Chronicity (N = 285 v. 134)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimated coefficient</td>
<td>Z</td>
</tr>
<tr>
<td>Block 1</td>
<td>Anxiety at T1 (higher)</td>
<td>−0.01</td>
</tr>
<tr>
<td></td>
<td>Female sex</td>
<td>0.79</td>
</tr>
<tr>
<td></td>
<td>Age (older)</td>
<td>0.00</td>
</tr>
<tr>
<td>Block 2</td>
<td>Perceived health (lower)</td>
<td>0.33</td>
</tr>
<tr>
<td></td>
<td>Functional limitations (more)</td>
<td>−0.07</td>
</tr>
<tr>
<td></td>
<td>Hearing/eyesight problems (more)</td>
<td>0.61</td>
</tr>
<tr>
<td>Block 3</td>
<td>Neuroticism (more)</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>Mastery (less)</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Self-efficacy (less)</td>
<td>0.02</td>
</tr>
<tr>
<td>Block 4</td>
<td>Social network size (smaller)</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td>Loneliness (more)</td>
<td>0.07</td>
</tr>
<tr>
<td>Block 5</td>
<td>Distress (more)</td>
<td>0.07</td>
</tr>
</tbody>
</table>

* P < 0.05; ** P < 0.01; *** P < 0.001.

Estimated and standardized coefficients are given for the final model which includes all variables; the χ² statistic is given for each block of variables entered in the model. A significant χ² indicates a better fit than chance. The cumulative χ² represents the fit of the block plus previous blocks.

Table 4 presents the results of the logistic regression analyses for predicting destabilization and restitution of anxiety: the χ² values for incremental explanatory power for each block of variables and the cumulative χ² are shown. Table 4 also presents the raw and standardized.
coefficients for each variable with change in anxiety status for the final model, which includes all predictor variables listed. Standardized coefficients follow the standard normal distribution.

Regarding destabilization, the results indicate that initial anxiety level and demographic variables have predictive power for destabilization or restitution of anxiety, which is mostly due to the sex variable. Entry of the second block of variables improved the fit of the model for destabilization slightly but significantly: hearing/eyesight problems at T1 (marginally significant at $P = 0.05$) increased the odds for destabilization. The third block improved the fit of the model again. Personality variables, especially neuroticism, predicted destabilization. Adding the next block improved the fit only marginally: little additional predictive power was gained by adding social functioning to the model. Distress added significantly to the explanatory power of the model for destabilization, as results of the fifth block show. According to the standardized coefficients of the final model, becoming anxious was best predicted by female sex, neuroticism and distress.

The results for the model for chronicity of anxiety were quite similar to the model for destabilization. Consecutive blocks had similar explanatory power with the same key variables increasing the odds for chronicity: female sex, and high neuroticism. A difference with the model for destabilization was that low mastery was associated with chronicity and distress was not associated with chronicity.

Next, the interaction between the vulnerability factors and distress was investigated. To limit the number of prognostic variables in the model only two-way interactions were tested between the composite distress score and vulnerability factors. For each of the variables listed in Table 4, first the main effects were entered in the model, followed by the interaction of a predictor with distress. The results revealed that with destabilization as dependent variable, only one interaction added significantly explanatory power to the main effects for the model. The interaction between perceived health and distress appeared significant: (standardized coefficient $z = -2.32; P < 0.02$), indicating that among those who perceived their health as poorer, distress due to life events had a more profound effect on the ratio of destabilized cases. For chronicity, the interactions between neuroticism and distress ($z = -2.32; P < 0.01$) and between loneliness and distress ($z = -1.99; P < 0.05$) were significant. The first interaction meant that for those respondents high in neuroticism at T1, distress and chronicity of anxiety were more strongly associated than for those low in neuroticism. Similarly, the second interaction meant that for those who had reported more loneliness at T1, distress and chronicity were more strongly associated.

Finally, the most parsimonious set of variables for predicting destabilization or restitution for each group was selected with logistic regression utilizing forward stepwise selection (with entry criterion set at $P = 0.20$ (Menard, 1995) and controlling for initial anxiety level). Thus, it could be tested which vulnerability variables and distress would suffice to predict change in anxiety and whether the interaction between vulnerability and distress added predictive power to the model for destabilization and the model for restitution. Destabilization was best predicted (Wald $\chi^2 (4) = 54.8; P < 0.0001$) by neuroticism ($z = 5.70; P < 0.001$), sex ($z = 3.67; P < 0.001$), distress ($z = 2.94; P < 0.01$) and hearing/eyesight problems ($z = 2.06; P < 0.05$) and no interaction term of distress and vulnerability factors could significantly improve the model. This model fits the data well (Hosmer–Lemeshow $\chi^2 (8) = 7.51; P = 0.89$) (Hosmer & Lemeshow, 1989). Regarding restitution of anxiety, we found a model comprising three variables (Wald $\chi^2 (3) = 44.9; P < 0.001$) again high neuroticism ($z = 4.63; P < 0.001$) and female sex ($z = 3.14; P < 0.01$) were predictive of chronicity. Furthermore, mastery ($z = 2.15; P < 0.05$) was entered in the model explaining restitution. The fit of this model was good (Hosmer–Lemeshow $\chi^2 (8) = 7.00; P = 0.54$). Distress or its interaction with vulnerability could not improve the fit.

Since the death of one’s partner is such an important life crisis and highly relevant for older persons, we repeated the multiple stepwise regression analyses, replacing our distress composite with death of one’s partner. This did not improve the fit of the models. For both destabilization and restitution, the composite distress score had better predictive value than death of one’s partner.
DISCUSSION

The present study investigated vulnerability/protective factors and stressors for becoming and remaining anxious in late life. The main findings were that changes in anxiety over time were best predicted by female sex and neuroticism. Hearing and eyesight problems and distress due to life events were associated with becoming anxious, but not with chronicity of anxiety. For few vulnerability factors a significant association was found between their interaction with distress and change in anxiety.

Strengths and limitations of the study

The proportion of respondents suffering from anxiety symptoms at T1 and T2 was 26% and 30%, respectively. First of all, these proportions indicate that the cut-off score on the HADS-A was low, favouring inclusion rather than exclusion of cases. Prevalence studies applying stringent diagnostic interviews, reveal a 6-month prevalence of 10% for anxiety disorders among older persons (Flint, 1994; Beekman et al. 1998).

In the present study anxiety symptoms were assessed, which are of course more prevalent than disorders. The number of subjects in each subgroup gave us ample statistical power (generally $\beta > 0.80$) to test for associations between risk factors and change in anxiety status.

For reliable change, statistically derived and potentially conservative requirements were applied, ensuring that relatively pure groups of destabilized cases (11%), and restituted cases (8%) were formed. Respondents who crossed the cut-off value on the HADS-A, but who did not change to a statistically reliable degree, were considered unchanged and categorized according to their anxiety score at T1. Categorizing these subjects according to their T2 score or omitting them altogether from the study sample did not alter the results in a meaningful way, as additional analyses revealed. However, every cut-off can be criticized for being somewhat arbitrarily chosen. To investigate further the solidity of the present findings, we reid the main analyses while applying a more stringent cut-off of 6/7 on the HADS-A. Of course, the number of destabilized respondents diminished, as did the number of respondents considered anxious at T1, resulting in a decrease of statistical power. Nevertheless, the main conclusion that female sex, neuroticism, and life stress are the main predictors of destabilization, and sex and neuroticism of chronicity still held in these additional analyses.

The strength of the present study is its longitudinal design, allowing for conclusions regarding the direction of associations among variables. Vulnerability factors measured at the first assessment carry a predictive value regarding anxiety status at T2. However, more caution should be applied with stressors, which were based on changes between T1 and T2 (e.g. decreased health and increased functional limitations). These variables should be considered as merely associated with change in anxiety. Furthermore, in a longitudinal study design with respondents of old age, attrition is unavoidable and over-sampling in vulnerable strata increased attrition even further. With regard to age and sex, the sample was made representative of the older Dutch population by weighting cases differentially. Nevertheless, selective attrition is still a potential source of bias in the present data. Comparison of respondents lost at the T2 assessment with those who were included revealed that the former had been worse off on most predictor variables at T1. Their declined health and impaired cognitive functioning may have been the very reason for attrition from the T2 sample. The loss of unhealthy respondents may have resulted in too conservative estimates of associations between vulnerability factors and change in anxiety in the present study.

Another reason for loss of data was non-response on the self-administered questionnaires. Unfortunately, neuroticism was measured in this way and also turned out to be one of the main predictive variables for becoming and remaining anxious. We repeated the multivariate analyses again on the full sample, omitting variables from self-administered questionnaires. The pattern of associations between predictor variables and anxiety did not change in the full sample of 2165 cases versus what had been found with the sample of 1735 cases for whom neuroticism scores had been available.

There is considerable overlap in anxiety and depression symptoms, as is evident from the usually high association between self-report measures for anxiety and depression and high co-morbidity in prevalence studies (Clark &
Watson, 1991). Thus, associations found between vulnerability factors and anxiety, may in part be due to depressive symptomatology. However, since the focus of the present study was on anxiety symptoms in late life, we chose not to include concurrent depression symptoms in the analyses. An investigation by our group comparing risk factors for pure depression, pure anxiety, and both anxiety–depression symptoms in late life is currently under way.

Another limitation to the present findings is the categorical approach to anxiety scores at T1 and T2, which was basic to all analyses. Although not uncommon in psychiatric research and appealing because of the straightforward decomposition of the sample in subgroups, one might criticize this data-analytical strategy, since the dichotomization of a continuous phenomenon leads to loss of information. To accommodate such criticism, we also analysed predictors of change in anxiety in a different manner. For each respondent the change in anxiety over time was expressed in a residual change score. This is the change score adjusted for the respondent’s initial level of anxiety (basically the residuals after regressing the T1 score onto the T2 score (Cronbach & Furby, 1970). Next, correlation coefficients between predictors (vulnerability and stress variables) and residual change in anxiety was calculated. The results matched the findings of logistic regression analyses closely: sex and the personality characteristics, especially neuroticism, were the best predictors of residual change in anxiety. Distress due to life events was not strongly related to changes in anxiety.

Which variables best predict a change in anxiety over time?
The bivariate associations between risk factors and change in anxiety status revealed few strong predictors of becoming anxious or restitution of symptoms, the main exceptions being sex, perceived health and neuroticism. Female sex was strongly and consistently associated with anxiety: in late life women have a higher chance to become more anxious over time and their anxiety symptoms tend to run a more chronic course compared with men. Apart from the main effect of sex, risk profiles for men and women were generally quite similar, as was revealed by additional analyses for men and women separately. Variables reflecting health and functioning were related to developing anxiety symptoms (self-perceived poor health, more functional limitations and eyesight or hearing problems increased the chance of becoming anxious), but not to restitution of anxiety.

With regard to neuroticism, the present findings show that this personality trait was a strong predictor for becoming anxious and for remaining anxious. In many studies an association between neuroticism and psychopathology has been demonstrated, usually in the wider context of investigating the relation between personality characteristics and mood and anxiety disorders (for an extensive review of this research see Clark et al. 1994). However, the present study demonstrated prognostic value of neuroticism for anxiety in a prospective design, which goes beyond demonstrating an association in a cross-sectional study design. Other studies employing a longitudinal design also showed the importance of pre-morbid personality characteristics to later psychopathology. Duncan-Jones and co-workers have reported findings of a longitudinal study in Australia where fluctuations in symptoms of psychopathology were well-predicted by neuroticism scores (see Duncan-Jones, 1987). Ormel & Wohlfart (1991) replicated these findings in a sample of college students. The present findings demonstrate that also in late life neuroticism is the most important vulnerability factor for becoming and remaining anxious.

Do the same factors account for emergence and persistence of anxiety symptoms?
Comparing risk for destabilization with risk for chronicity reveals that in general the same factors are predictive: female sex and high neuroticism put one at risk for developing anxiety symptoms and for remaining anxious as well. Also when looking at specific life events, the similarity of predictors for destabilization and chronicity was striking. The multiple analyses did reveal a difference between destabilization and restitution. Hearing or eyesight problems at baseline were predictive of developing anxiety, but this vulnerability factor was not included in the model for chronicity. Also, for the composite distress due to life events a distinctive pattern of association was found, as it was associated with destabilization but not with chronicity. This last
finding supports the vulnerability/stress model, which states that stress is influential in destabilization, but not in restitution of symptoms (Goldberg & Huxley, 1992).

Interaction of vulnerability and stress

The remaining question posed in the introduction concerns the combined effect of vulnerability factors and stressors on change in anxiety over time. The issue is whether the effects of stressors are modified by vulnerability factors or not. Social functioning variables may illustrate this point. In the literature, a buffering role for social functioning has been proposed (for a review of studies on depression see Paykel, 1994). A large or supportive social network is hypothesized to be a protective factor in the face of stress, whereas older persons with less social resources would be at greater risk for developing psychological problems when stressful events occur. Our findings showed no interaction between social functioning and distress, which does not support a buffering role of social functioning. In fact, the logistic regression analyses revealed few significant interaction terms that were predictive of change in anxiety (only 1 of 11 interaction terms for destabilization and 2 of 11 for chronicity) and the interaction terms did not add significantly to the models in multiple analyses. Thus, the present results favour a model of main effects adding on to each other, rather than an interactive model with multiplicative interaction of variables, where vulnerability factors and stress amplify each other.

Concluding remarks

Generally speaking, the findings regarding predictors of change in anxiety in late life coincide quite well with what is reported in the (scarce) literature on risk factors for anxiety in mixed age groups (Goldberg & Huxley, 1992). However, some disparities are worth mentioning. Although in bivariate analyses health and functioning at T1 were associated with destabilization and chronicity of anxiety, the results of the multivariate analyses indicated that these vulnerability factors did not add substantially to the fit of the models, suggesting that these factors are not that important in late life. Furthermore, development of a first chronic somatic disease did not play a major role in development or persistence of anxiety in older persons either. In younger age groups physical disease is an important vulnerability factor, with psychopathology between 1.5 and 3.0 more likely among those with ill-health (Weyerer, 1990). Possibly, for older persons getting a serious illness is experienced as an on-time event (Neugarten, 1970). These results coincide with findings of Liddell et al. (1991). They reported that fears regarding illness or death of a loved one ranked highest among Fear Survey Schedule items (Geer, 1965) for respondents aged 50 years and over. Fears related to their own death, illness, and injury were seen to decline with age. In discussing this finding, Liddell et al. (1991) speak of ‘adaptive resignation towards the inevitable’.

In total, this study of the waxing and waning of anxiety in a population-based sample of older persons revealed that fluctuations in anxiety were best predicted by sex, neuroticism, and to a lesser extent by distress due to life events. The stress-vulnerability model offered a useful framework to delineate who is at risk for developing and keeping anxiety symptoms: namely, women, older persons with high neuroticism scores and those individuals who experience more than their share of adverse life events. No support for an interaction between vulnerability and stress was found.

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REFERENCES


Predicting change in anxiety in older persons


