Original article

Worry and cognitive control predict course trajectories of anxiety in older adults with late-life depression

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ABSTRACT

Background: Many older adults with depressive disorder manifest anxious distress. This longitudinal study examines the predictive value of worry as a maladaptive cognitive emotion regulation strategy, and resources necessary for successful emotion regulation (i.e., cognitive control and resting heart rate variability [HRV]) for the course of anxiety symptoms in depressed older adults. Moreover, it examines whether these emotion regulation variables moderate the impact of negative life events on severity of anxiety symptoms.

Methods: Data of 378 depressed older adults (CIDI) between 60 and 93 years (of whom 144 [41%] had a comorbid anxiety disorder) from the Netherlands Study of Depression in Older Adults (NESDO) were used. Latent Growth Mixture Modeling was used to identify different course trajectories of six-months BAI scores. Univariable and multivariable longitudinal associations of worry, cognitive control and HRV with symptom course trajectories were assessed.

Results: We identified a course trajectory with low and improving symptoms (57.9%), a course trajectory with moderate and persistent symptoms (33.5%), and a course trajectory with severe and persistent anxiety symptoms (8.6%). Higher levels of worry and lower levels of cognitive control predicted persistent and severe levels of anxiety symptoms independent of presence of anxiety disorder. However, worry, cognitive control and HRV did not moderate the impact of negative life events on anxiety severity.

Conclusions: Worry may be an important and malleable risk factor for persistence of anxiety symptoms in depressed older adults. Given the high prevalence of anxious depression in older adults, modifying worry may constitute a viable venue for alleviating anxiety levels.

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1. Introduction

Anxiety is among the most prevalent and debilitating mental health problems in older adults [1] and often co-occurs with depression [2]. Few studies have traced course trajectories of anxiety disorder and anxiety symptoms and its prognostic course determinants in a cohort of community-dwelling older adults [3,4]. A rather unfavorable long-term outcome of anxiety after 6 years has been observed, predicted by female gender and higher levels of neuroticism at baseline.

Cognitive emotion regulation strategies have never been studied as determinants of the course of anxiety in older adults. Cognitive emotion regulation strategies are cognitive responses to emotion-eliciting events that consciously or unconsciously attempt to modify the magnitude and/or type of individuals’ emotional experience or the event itself [5]. An example of a largely adaptive strategy is cognitive reappraisal, which involves changing the way we are thinking about a situation in order to change how we feel. Maladaptive strategies (such as rumination and suppression) are strongly associated with various forms of psychopathology, such as anxiety and depression [6]. In younger adults, it has been repeatedly shown that worry is a dysfunctional cognitive emotion regulation strategy implicated in onset, maintenance and relapse of anxiety (and depressive) disorders [7].

Inhibitory control is a key mechanism for successful emotion regulation as people are required to inhibit prepotent emotional responses in service of more desirable and appropriate ones. As there are age-related declines in cognitive control, it can be assumed that older adults will have more difficulty to regulate their emotions [8]. Moreover, research in late-life anxiety has
shown that anxiety disorders result in cognitive control deficits arising from disturbances in goal maintenance due to disruption of the ability to inhibit task irrelevant information [9]. Reduced cognitive functioning through aging coupled with late-life anxiety suggest that anxiety and aging pose an enhanced risk for impaired cognitive control of emotion.

Ageing also causes a decrease in parasympathetic tone at rest that is manifested by a decrease in heart rate variability (HRV) [10]. Autonomic control of the heart as measured by HRV is related to attentional control and affective information processing and as such constitutes an important resource for adequate emotion regulation [11]. Individuals with better emotion regulation skills manifest higher levels of resting HRV, and HRV appears to be increased during successful performance on emotion regulation tasks [12]. Moreover, decreases in HRV in response to complex negative situations that affected multiple life domains have been found to be more pronounced in older people than in younger adults [13].

The aim of the present study is to examine whether depressed older adults with higher levels of worry and lower levels of cognitive control and lower levels of HRV will be more likely to show an unfavorable course of anxiety symptoms. Moreover, we studied whether exposure to negative life events possibly overtaxing emotion regulation capacities will result in more severe anxiety symptoms in depressed older adults with higher levels of worry and lower levels of cognitive control and HRV.

2. Methods

2.1. Participants

Data from the Netherlands Study of Depression in Older people (NESDO) [14] were used. NESDO is a multi-site prospective cohort study, including 378 depressed and 132 non-depressed older people (60–93 years). Depressed people were included when they fulfilled the DSM-IV-TR criteria for major depression (95.0%), dysthymia (26.5%) in the previous 6 months or current minor depression (5.0%). Of the depressed older people, 144 (41.0%) also displayed a comorbid anxiety disorder. The population and methods of the NESDO study have been described in detail elsewhere [14]. The study was approved by the ethical boards of the participating institutes and written informed consent was obtained from all participants. In the present study, only depressed people with and without comorbid anxiety were included (n = 378) of whom 285 (75.4%) completed the two-year follow-up assessments [15].

2.2. Measurements

2.2.1. Psychiatric diagnoses

Diagnoses of depression and anxiety at baseline and two-year follow-up were assessed with the Composite International Diagnostic Interview (CIDI; World Health Organization [WHO] version 2.1) according to DSM-IV-TR criteria [16].

2.2.2. Symptom severity

The severity of anxiety symptoms was assessed with the Beck Anxiety Inventory (BAI) [17] at baseline and 6-, 12-, 18-, and 24-months follow-up. Severity of depressive symptoms at baseline was assessed with the self-report version of the Inventory of Depressive Symptomatology (IDS-SR) [18].

2.2.3. Negative life events

The occurrence of recent negative life events (NLE), such as experiencing serious illness or major financial loss, was assessed using the Brugha questionnaire [19,20]. These events reflect the presence of life stressors during the last 12 months before baseline or last six months before the 6-, 12-, 18-, and 24-months follow-up.

2.2.4. Worry

Symptoms of worrying at baseline were assessed with a revised version of the Worry Scale [21]. This questionnaire is especially developed for use with older adults and does not measure the process of worry in general, but comprises subscales that reflect the severity of specific types of worry about financial, health, and social concerns. Based on factor loadings, skewness of mean scores on each item, 15 items were selected for the shortened version [22].

2.2.5. Cognitive control

Cognitive control was measured with the abbreviated version of the Stroop color-word test [23]. This test consisted of three subtasks; the first card (I) had color words (red, blue, green, yellow) printed in black, the second card (II) had colored patches of the same colors, and the last card (III) had color words printed in incongruent colored ink. During the third task, the participants were shown cards displaying names of colors in a non-matching color and asked to name the color of the ink as fast and as accurate as possible. Cognitive control was assessed with the interference score calculated by the following formula: \( (\text{III} – 0.5 \times (\text{I} + \text{II})) / (0.5 \times (\text{I} + \text{II})) \) (t = time in seconds) [24].

2.2.6. Heart rate variability

Physiological measurements were performed with the “Vrije Universiteit Ambulatory Monitoring System” recording the electrocardiogram (ECG) and changes in thorax impedance (dZ) from six electrodes placed on the chest and back of the participants [25]. NESDO participants wore the Vrije Universiteit Ambulatory Monitoring System device during around 116 minutes of the NESDO baseline assessment. The assessment procedure has been described in more detail elsewhere [26]. The interbeat interval (IBI) time series was extracted from the ECG signal to obtain HR. Respiratory sinus arrhythmia (RSA) is a reliable index of cardiac parasympathetic control [27] and was used as a measure of HR. RSA was obtained by combining the IBI time series with the filtered (0.1–0.4 Hz) dZ signal, which corresponds to the respiration signal. RSA was obtained by subtracting the shortest IBI during HR acceleration in the inspiratory phase from the longest IBI during deceleration in the expiratory phase for all breaths, as described in detail elsewhere [25]. As several studies have suggested that research investigating RSA should take respiratory rate (RR) into account as a confounder [28], all RSA analyses were adjusted for RR.

2.3. Statistical analyses

The Stroop interference scores were log-transformed to obtain a near-normal distribution. Moreover, we categorized the number of life events measurements as none, one or two or more life events. The other measures showed a normal distribution. In the moderation analyses, scores for worry, cognitive control and HRV were transformed to z-scores.

As it is unlikely that a single growth trajectory of anxiety symptoms will adequately describe the course of anxiety symptoms among depressed older adults, we first tried to identify different course trajectories of anxiety symptoms, based on repeated BAI scores during 2-year follow-up using Latent Growth Mixture Modeling (LGMM). LGMM assesses whether multiple unobserved latent trajectories are available within an observed overarching group of individuals allowing for different groups of individual growth trajectories to vary around different means. In LGMM, each subgroup trajectory is defined by two latent variables:
intercept and slope [29]. To determine the number of underlying latent trajectories, successive models of one to more subgroups are specified and estimated to find the model that best approximates the subgroups within the data. We first fitted standard models with intercept and linear effects of time. Next, we successively added a quadratic slope factor to allow for curved trajectories. As parameter estimates, we used maximum likelihood with robust standard errors (MLR) that are robust to non-normality and non-independence of observations.

To determine which model best fitted the data, we combined Maximum Likelihood, Bayesian Information Criterion (BIC), sample size adjusted BIC (ssBIC), Lo-Mendell-Rubin likelihood ratio test (LMR), and bootstrapped likelihood ratio test (BLRT). Both the BLRT and LMR provide a P-value, which indicates whether the k=1 class model is rejected in favor of the k class model [30]. After the identification of the number of classes, people were assigned to their most likely class based on model probabilities. Classification quality of the final LGMM model was evaluated based on entropy and average posterior probabilities for latent class membership.

Next, we examined to what extent emotion regulation variables (worry, cognitive control, HRV) were predictive of course trajectories using univariate and multivariate, multinomial logistic regression analyses with the low anxiety group as reference category. In addition to unadjusted analyses, we controlled our multinomial logistic regression analyses for demographic variables (age, gender, education), as well as presence of anxiety disorder and use of antidepressant and anxiolytic medication as we were interested in whether the predictive value of emotion regulation variables is independent of presence of anxiety disorder and medication use is known to affect cognitive control and HRV in particular [31].

Finally, we assessed whether emotion regulation variables modified the effect of exposure to negative life events on severity of anxiety symptoms. Because anxiety severity and exposure to negative life events were measured repeatedly, we used linear mixed models (LMM). This approach allowed us to model the effects of NLEs as time varying variables as well as of time invariant emotion regulation factors on anxiety severity (BAI). In these moderation analyses, the possible additional effect of the interaction of emotion regulation variables with exposure to NLE on severity of anxiety symptoms on top of the direct effect of emotion regulation variables and NLEs could be analyzed.

Statistical analyses were run using SPSS version 23 [32] except the LGMM analyses, which were performed using MPlus v. 7.1 [33]. A P-value <.05 was considered statistically significant.

3. Results

3.1. Descriptives

The overall sample of 378 participants had a mean age of 70.7 years (SD = 7.4) and consisted of 250 (66.1%) women and 138 (33.9%) men. The mean number of years was 10.9 (SD = 3.6). Of the depressed people, 274 (73.1%) used antidepressant medication and 123 (32.5%) used anxiolytic medication. Among the subgroup of depressed people with comorbid anxiety disorder, 25.7% fulfilled the diagnostic criteria for generalized anxiety disorder, 18.1% for panic disorder with or without agoraphobia, 25.0% for agoraphobia, and 45.8% for social anxiety disorder.

3.2. Course trajectories and the external validation of these trajectories

A linear model with an additional quadratic slope factor provided the best representation of the various course trajectories (see Table 1). Although the LMR-test failed to reach significance from the 5-class model onwards, we chose the 3-class model as providing the best fit for the observed data, because the addition of a fourth class served only to split one of the classes into two parallel trajectories with no substantive distinction in symptom levels and the fourth class in this model also consisted of 5 people (1.3%) only precluding meaningful prediction analyses of class membership.

The level of entropy of the 3-class model was high (.87). Moreover, posterior probabilities for class membership were high (class 1: .91; class 2: .97; and class 3: .90), while posterior probabilities for membership to another class were low (ranging from .00 to .10). Fig. 1 shows the course trajectories of the three identified latent classes. As the first class, consisting of 33.5% of our sample (n = 125), showed a moderate level of anxiety and no significant decline in anxiety during follow-up (intercept = 23.4, P < .001; linear slope = -.150, P = .12; quadratic slope = .208, P = .345), we labelled this subgroup as the moderate anxiety group. As the second class, consisting of 57.9% of our sample (n = 216), had low anxiety score which significantly decreased over time (intercept = 10.334, P < .001; linear slope = -.2502, P < .001; quadratic slope = .446, P < .001), we labelled this group as the low anxiety group. As symptoms in the third group, consisting of 8.6% of our sample (n = 32), were severe and persistent (mean intercept = 35.740, P < .001; slope = 1.813, P = .388; quadratic slope = -.427, P = .436), we labelled this group as the severe anxiety group. Participants from both the moderate and severe anxiety group manifested a significantly higher proportion of anxiety disorders and significantly higher levels of depressive and anxiety symptoms at T1 and T5 compared to participants from the low anxiety group (see Table 2). The association of severity of depressive (IDS) with anxiety (BAI) symptoms was high (r = .59, P < .001).

3.3. Emotion regulation variables as predictors of the course of anxiety severity

Univariate multinomial logistic regression analyses showed that worry, cognitive control and HRV predicted course trajectories (see Table 2). The level of worry of both the moderate and severe

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Parameters of fit of Latent Growth Mixture Modelling based on BAI scores at T1 to T5 (n = 375)*.</th>
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<tbody>
<tr>
<td>Linear model</td>
<td>Classes</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>1.1</td>
<td>1</td>
</tr>
<tr>
<td>1.2</td>
<td>2</td>
</tr>
<tr>
<td>1.3</td>
<td>3</td>
</tr>
<tr>
<td>1.4</td>
<td>4</td>
</tr>
<tr>
<td>1.5</td>
<td>5</td>
</tr>
</tbody>
</table>

BIC: Bayesian Information Criterion; ssBIC: sample size adjusted BIC; BLRT: bootstrapped likelihood ratio test; 2LL: 2 log likelihood.

* 375 participants because of missing baseline BAI scores in three people.
anxiety group was significantly higher than of the low anxiety group. Moreover, the severe anxiety group obtained significantly lower scores for cognitive control and higher scores for HRV than the low anxiety group. Repeating the analyses of the emotion regulation variables while controlling for demographic variables, presence of anxiety disorder and use of antidepressant and anxiolytic medication showed that the significant effect of worry (OR = 1.07; 95% CI = 1.03–1.11, respectively OR = 1.09; 95% CI = 1.04–1.15), and cognitive control (OR = 2.22; 95% CI = 1.02–4.85) remained. However, the predictive value of HRV for course trajectories was no longer significant (OR = 1.02; 95% CI = .99–1.05).

3.4. Emotion regulation variables as moderators of the relationship of negative life events with anxiety severity

Participants with or without anxiety disorder reported on average exposure to 1.0 negative life event during each 6-month period. Two negative life events were mentioned on at least three of the five measurements by more than 10% of the respondents: 'being seriously ill, wounded or victim of violence of family member' and 'a friend or family member died'. LMM analyses with a random intercept showed that exposure to 1 (estimate = .62; 95% CI = −.25 to 1.49) or 2 or more negative life events (estimate = .89; 95% CI = −.08–1.86) in comparison to no exposure did not predict severity of anxiety symptoms. Moreover, worry, cognitive control and HRV did not moderate the impact of NLE on course of anxiety severity in unadjusted (see Table 3), as well as adjusted analyses (data not shown).

4. Discussion

We identified a group of participants with mild symptoms of anxiety, which improved during the 2-year follow-up period, a group with moderate and persistent anxiety symptom, and a group with severe and persistent anxiety symptoms. These results concur

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Distribution of putative risk factors at baseline across the identified three course trajectories (n = 375).</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>LA (n=216)</td>
</tr>
<tr>
<td>Demographics</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>70.98 (7.27)</td>
</tr>
<tr>
<td>Gender (female)</td>
<td>134 (62.0%)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>10.68 (3.34)</td>
</tr>
<tr>
<td>Clinical variables</td>
<td></td>
</tr>
<tr>
<td>Anxiety disorder T1</td>
<td>67 (31.0%)</td>
</tr>
<tr>
<td>Anxiety disorder T5*</td>
<td>14 (8.4%)</td>
</tr>
<tr>
<td>BAI T1</td>
<td>11.06 (7.31)</td>
</tr>
<tr>
<td>BAI T5*</td>
<td>7.22 (5.15)</td>
</tr>
<tr>
<td>IDS</td>
<td>24.44 (11.09)</td>
</tr>
<tr>
<td>ADM use (yes)</td>
<td>157 (73.0%)</td>
</tr>
<tr>
<td>Anxietytica use (yes)</td>
<td>57 (26.4%)</td>
</tr>
<tr>
<td>Emotion regulation</td>
<td></td>
</tr>
<tr>
<td>Worry</td>
<td>5.20 (6.58)</td>
</tr>
<tr>
<td>Cognitive control*</td>
<td>1.42 (.79)</td>
</tr>
<tr>
<td>HRV*</td>
<td>18.01 (11.51)</td>
</tr>
</tbody>
</table>

LA: low anxiety group (reference category); MA: moderate anxiety group; SA: severe anxiety group; OR: odds ratio; 95% CI: 95% confidence interval; BAI: Beck Anxiety Inventory; IDS: Inventory of Depressive Symptomatology; ADM: anti-depressive medication; NLEs: number of negative life events (NLE) before baseline.

* Only available for 285 study completers.

* Non-transformed Stroop interference scores.

* Heart rate variability (HRV) analyses controlled for respiratory rate (RR).
with those of previous studies on the course of anxiety symptoms in older adults [3,34] and younger adults [35,36] with anxiety disorder in that they challenge the idea that a ‘chronic’ clinical course is characteristics for all patients with anxiety disorders in general. Also in depressed older adults, those with mild anxiety symptoms (of whom around 30% had an anxiety disorder) tended to improve, while anxiety symptoms were more persistent in those with moderate or severe anxiety symptoms (of whom around 50% had an anxiety disorder).

Our results do not describe the course of pure anxiety in late-life depression. Firstly, the severity of depression was highly associated with severity of anxiety symptoms. Secondly, the three course trajectory groups as identified on the basis of repeated BAI scores also differed regarding depression severity scores at baseline. It would therefore be more accurate to describe these findings as the course of intertwined depressive and anxiety symptoms.

As hypothesized, emotion regulation variables were predictive of course trajectories with higher levels of worry predicting both persistent moderate and severe levels of anxiety symptoms across the 2-year follow-up period. Worry in depressed older adults may predict more persistent levels of anxiety symptoms irrespective of the presence of an anxiety disorder, as worry may function as a cognitive avoidance response attenuating somatic responses to fear related stimuli [37]. Unfortunately, we did not measure rumination, as the predictive value of worry may reflect the effect of repetitive negative thinking traversing a number of different psychiatric phenomena, although rumination may be relatively more pronounced in depression and worry in generalized anxiety disorder [7,38]. There are more similarities than differences across the processes of worry and rumination. These include the fact that they are repetitive, difficult to control, negative in content, predominantly verbal, and relatively abstract [39]. Repetitive negative thinking may therefore constitute a common dimension underpinning rumination and worry [40,41]. As our measure of worry taps into specific types of worry concerns and does not measure the process of worrying, our results remain silent about the relative importance of content-dependent and content-independent aspects of worrying. Consequently, it remains unclear whether the predictive value of worry for the course of anxiety in the present study primarily reflects the content-dependent effect of worrying about future threats or has to be interpreted as an effect of repetitive negative thinking.

These results may have implications for the definition of anxious depression. Anxious depression in late life can be defined based on a syndromal and dimensional approach [42]. In an elderly sample, a syndromal approach seems to identify a completely different group of older adults than using a dimensional approach [22]. Consistent with this, worry predicted moderate and severe symptom levels of anxiety over and above the presence of anxiety disorder. Worry refers to the new specifier of depression ‘with anxious distress’ in the DSM 5 [43], as it relates to symptoms as difficulty concentrating due to worry and fear that something awful may happen. These results suggest that high levels of worry in late-life depression may be of importance for a dimensional approach to anxious depression in late life.

Lower levels of cognitive control (as measured with a higher interference score on the Stroop test) were only predictive of persistent and severe levels of anxiety, but not of persistent and moderate levels of anxiety. The moderate anxiety group even obtained more favorable scores for cognitive control than the low anxiety group, although this difference failed to reach significance. These results concur with previous studies suggesting that the relation of anxiety with cognitive control in late-life anxiety may be non-linear and exhibit the form of an inverted U-shape: in particular, high levels of anxiety may be associated with problems in inhibiting task irrelevant information [9]. Our results extend these finding by showing that diminished cognitive control is also predictive of the course of anxiety in depressed older adults with high levels of anxiety.

Contrary to expectations, we found that higher levels of HRV were predictive of high and persistent symptom levels. However, the effect (OR = 1.02; 95% CI = 1.01–1.05) was marginally significant and awaits future replications. Moreover, the predictive value of HRV for course trajectories of anxiety was no longer significant after taking use of antidepressant medication into account. This result concurs with recent studies showing that depressive and anxiety disorder per se are not associated with cardiac autonomic control, but antidepressants and specifically tricyclic antidepressants are [26,31]. As depressed participants from the high anxious group tended to use less antidepressant medication (56.3%) than from the other two groups (73.0 and 77.2%), it is likely that their high baseline HRV levels primarily expressed their relative non-use of medication and not their symptom severity. Possibly, the predictive value of lower levels of HRV for more favorable course trajectories of anxiety was due to the effectiveness of antidepressant medication as indexed by medication-induced lower levels of HRV at baseline.

Exposure to negative life events did not systematically impact upon the severity of anxiety symptoms. This finding concurs with those of a previous study showing that although negative life events were one of the best predictors of becoming anxious in community-dwelling older adults, negative life events proved to be unrelated to chronicity of anxiety symptoms after three [3] and six years of follow-up [34]. Critically, we found no evidence supporting our hypothesis that the resource demands of negative events may overtax individual cognitive capacities resulting in impaired cognitive control of emotions and enhanced psychological and physiological responding.

There are several explanations for these unexpected findings. The relatively low prevalence of negative life events in the current
study (on average one life event per six months) as well as the relatively poor fit of the type of concerns as measured by the Worry questionnaire with the specific life events covered by the Brugha questionnaire may explain this null-finding. Future studies measuring daily hassles would allow a more meaningful analysis of the question whether worry might moderate the impact of daily hassles (such as financial, health, and social problems) that are the primary focus of habitual worry.

Regarding cognitive control, we assessed whether depressed older adults with high levels of persistent anxiety were less able to inhibit prepotent responses using the classical Stroop test, but we did not measure the extent to which these people also exhibit enhanced attention to threat-related stimuli as would have been possible with an emotional Stroop test [44]. So, it remains undecided whether ‘hot’ cognitive control, which involves emotionally valenced information, could have made people more susceptible to the detrimental effects of exposure to negatively valenced life events. Moreover, according to the selection, optimization, and compensation with emotion regulation (SOC-ER) framework [45], the social-emotional selectivity theory (SST) [46], and strength and vulnerability integration (SAVI) model [47], older adults report high levels of well-being among others because they adapt their use of emotion regulation strategies with aging. To compensate for their age-related decline in cognitive control, they may have been prone to avoid the limitations in negative emotion regulation to preserve a sense of well-being. And it is conceivable that in particular anxious-depressed older adults are likely to show this type of avoidance behavior.

Finally, resting state HRV measurements may not be optimal to examine the effect of HRV on emotional responding, as an ecological momentary assessment study showed that when dealing with complex unpleasant events that affected multiple life domains, psychological and HRV responding to unpleasant events was more pronounced the older the participants were [13].

To overcome the study limitations mentioned above, clinical studies using experiencing sampling methods (ESM) to measure emotion regulation in real life seem warranted [48], and also feasible in aging research [49,50]. Such studies would allow a finer grained analysis of the impact of e.g. state worry, cognitive control and HRV when exposed to negative life events on anxiety.

5. Conclusion

The present study also bears clinical relevance. Depressed people with higher levels of worry may be at risk for an unfavorable course of their anxiety. As worry constitutes a malleable risk factor, worry has been shown to constitute a viable venue for alleviating anxiety using interventions such as mindfulness [51] or cognitive-behavioral therapies targeting perseverative thinking patterns [52]. Given the high prevalence of anxious depression in late life, future studies into the malleability of worry in anxious older people and its therapeutic effects are urgently called for.

Disclosure of interest

The authors declare that they have no competing interest.

References


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