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Trajectories of insomnia following bereavement

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ABSTRACT

Background: Insomnia symptoms are common following bereavement and may exacerbate severe and protracted grief reactions, such as prolonged grief disorder (PGD). However, typical trajectories of insomnia symptoms and risk factors for having a more chronic insomnia trajectory following bereavement are yet unknown.
Method: In the current investigation, 220 recently bereaved (≤6 months post-loss) participants, completed questionnaires assessing sociodemographic and loss-related characteristics, rumination, experiential avoidance and symptoms of (prolonged) grief and depression, on three time-points (6 months apart). We applied growth mixture models to investigate the typical trajectories of insomnia symptoms following bereavement.
Results: Three insomnia trajectory classes emerged, characterized by a resilient (47 %), recovering (43 %), and a chronic trajectory (10 %). Baseline depression symptoms best predicted the type of insomnia trajectory. At one-year follow-up, 9 %, 27 %, and 60 % of participants met the criteria for probable PGD within the resilient, recovering and chronic trajectory, respectively. A parallel process model showed that temporal changes in insomnia symptoms were strongly related to changes in prolonged grief symptoms.
Conclusion: The results suggest, that targeting insomnia symptoms in the treatment of PGD, particularly with comorbid depression, may be a viable option.

1. Introduction

Bereavement is one of the most stressful life events that a person can experience. Most people adjust to loss using their own coping styles and support sources. However, a small minority develops severe mental health conditions such as depression, post-traumatic stress disorder, and severe and persistent grief, termed prolonged grief [1,2], requiring professional intervention (e.g., [3–5]). A diagnosis characterized by prolonged grief has recently been added to the *International Classification of Diseases*-11th edition (ICD-11; [6]) and the *Diagnostic and Statistical Manual of Mental Disorders*-5th edition text revision (DSM-5-TR; [7]) in the form of prolonged grief disorder (PGD). While there are differences between both forms of PGD within classification systems, both have similar features including the persistence of severe and disabling grief reactions such as yearning for the deceased and cognitive preoccupation with the deceased [8]. Another common mental health problem of the bereaved, associated with severe grief reactions, are sleep disturbances [9,10].

Sleep disturbances, such as insomnia, have long been regarded as secondary symptoms of stress-related and affective disorders. However, there is increasing evidence suggesting that insomnia symptoms not only remain after treating these disorders but are also implicated in their development and persistence (for reviews: [11,12]). For example, studies show that insomnia symptoms contribute to the development and maintenance of post-traumatic stress [13] and depression symptoms [14]. More recently, evidence has emerged indicating a role for insomnia symptoms in the perpetuation of prolonged grief symptoms [9,15,16].

The exact mechanism by which sleep is implicated in stress-related and affective disorders is unclear. However, it has been proposed that the (re)activation, (re)processing, and (re)consolidation of emotional memories that occurs during (REM) sleep decreases the emotional load of these memories [17,18]. A disruption of these sleep-related processes may cause the emotional reaction to memories to persist or even worsen [19–21].

Findings from both longitudinal and experimental studies provide

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converging evidence pointing to the potential perpetuating role of insomnia in PGD. In a longitudinal survey of middle-aged Dutch citizens, self-reported sleep problems and lower overall sleep duration prior to bereavement were related to an increased risk of developing severe prolonged grief symptoms at a six-year follow-up [22]. Additionally, a three-wave study in bereaved adults, employing random intercept cross-lagged panel models, found that changes in insomnia symptoms are prospectively associated with changes in prolonged grief symptoms six months later [15].

Experimentally, two pilot randomized controlled trials have shown that treatments targeting sleep problems, such as internet-based cognitive behavioral therapy for insomnia [16], and function-based therapy (including a focus on sleep; [23]), diminished prolonged grief symptoms more than sleep hygiene and emotion-focused treatment, respectively. The empirical evidence thus seems to point to a causal role for insomnia symptoms in the persistence of prolonged grief symptoms. Understanding which factors best predict the emergence of chronic sleep disturbances following bereavement could thus provide important clues for clinical practice. These predictors can potentially identify those who would most likely benefit from early interventions.

In the present investigation, we will examine the course of sleep problems following bereavement as well as its links with prolonged grief symptoms. Specifically, we will seek to identify the typical trajectories of insomnia symptoms following bereavement. Identifying these potentially distinct trajectories enables us to detect risk and protective factors for (more) chronic insomnia trajectories following bereavement. It has been observed that once sleep problems progress into an insomnia disorder, they are unlikely to be resolved without professional intervention [24,25], putting these individuals at increased risk for other physical and mental health problems, including prolonged grief [12,26]. So far, no study has yet elucidated potentially distinct trajectories of sleep problems following bereavement, their predictors, or their potential relationship with the development of prolonged grief [9].

Generally, studies report a gradual decline of most post-loss mental health problems, including insomnia symptoms, over time [9]. However, studies typically summarize overall tendencies, ignoring the potential for subpopulations that follow distinct trajectories. An influential theory on the development and maintenance of insomnia, the 3P model [27], suggests the existence of at least three different insomnia trajectories following a precipitating event, such as bereavement. The model implies that a person with a predisposition (which can be genetic, socio-economical, etc.), faced with precipitating factors such as a loss, is likely to develop acute insomnia. Perpetuating factors such as worrying about the consequences of not being able to sleep [28] or maladaptive compensatory behaviors such as extending sleep opportunity (e.g., spending more time in bed) [29] can, in turn, lead to chronic insomnia. Thus, following Spielman et al.'s notions, we would first predict the existence of a resilient trajectory, consisting of people with a minimal predisposition for the development of insomnia symptoms. Second, a recovering trajectory, containing people who develop acute insomnia symptoms, which subside after the impact of the stressor diminishes. Third, we would expect a chronic insomnia trajectory, comprised of people who develop acute insomnia and engage in maladaptive compensatory behaviors and cognitive processing, resulting in physical and mental hyperarousal, leading to persistent insomnia. In the general population, various factors such as age, sex, educational level, and having a comorbid mental disorder (e.g., depression) have been identified as risk factors for insomnia (e.g., [30]). Similar factors, as well as certain loss characteristics (e.g., loss of a child or partner, violent cause of death), predict a more severe and protracted grieving process (cf. [3,31]). Furthermore, repetitive negative thinking may be linked to the development of chronic insomnia. In particular, nighttime worry about the consequences of not getting enough sleep appears to negatively affect sleep [28,32]. Similarly, repetitive negative thinking and related emotion regulation strategies are associated with less favorable grief outcomes. In particular, grief rumination and avoidance of loss-related cues and associated internal experiences can lead prolonged grief symptoms to persist (for a review: [33]). Higher (prolonged) grief severity also relates to more severe insomnia symptoms [9]. Accordingly, the present study will consider the abovementioned factors as possible risk factors for chronic insomnia following bereavement.

Using growth mixture models, we investigated the typical trajectories of insomnia symptoms following bereavement. Based on the 3P model, we expected to find three distinct insomnia trajectories. A *resilient* trajectory with no (sub)clinical insomnia symptoms, a *recovering* trajectory with (sub)clinical insomnia symptoms that dissipate over time, and a *chronic* trajectory with clinical insomnia symptoms that do not decline significantly over time. For potential risk factors of a chronic insomnia trajectory, we specifically considered sociodemographic (e.g., age, sex) and loss-related (e.g., cause of death, expectedness of the death) variables, coping strategies (grief rumination and experiential avoidance), and baseline depression and prolonged grief symptoms. Lastly, in line with prior studies suggesting similarities between the (more) chronic trajectories of affective and stress-related symptoms following a loss [34–37], we explored whether trajectories of insomnia and prolonged grief symptoms changed simultaneously over time.

2. Method

2.1. Procedure

The present investigation is part of a larger longitudinal study on psychopathology following bereavement. The study was pre-registered (https://aspredicted.org/y3z2b.pdf) and approved by a local ethical review board. Participants (aged ≥18) recently bereaved of a loved one were recruited through online advertisement on the content network of Google (AdWords) and the website of a national organization for psychologists between May 2019 and September 2020. The advertisements linked to an information page listing the goal, procedure, and a link to the informed consent form and questionnaire. At the end of the baseline measurement (T1), participants could indicate their interest in the longitudinal study. Personal invitations were sent for the second (six-month follow-up; T2) and third waves (12-month follow-up; T3). Reminders were emailed two and three weeks after the initial invitation. All questionnaires were programmed in Qualtrics. Participants could withdraw from the study at any time. No compensation was offered for participation in the study.

2.2. Participants

Five hundred and four participants signed up for the longitudinal study. We used data from 220 participants who experienced a loss in the previous six months and completed at least two questionnaires because we were interested in the trajectories of sleep patterns over time from the acute phase of loss onward. At the six-months follow-up, 208 participants completed the questionnaire, and 194 at the one-year follow-up. Most participants were women (89 %) bereaved of a partner (39 %) or parent (41 %) and experienced a loss due to natural causes (85 %). The mean age was 51.8 (SD = 12.24; range = 20–84) and about half were higher educated (41 %). On average, the loss occurred 2.3 months ago (SD = 1.44; range 1–6). See Table 1 for a summary of sample characteristics.

2.3. Materials

2.3.1. Sociodemographic and loss-related characteristics

Sociodemographic characteristics (age, sex, and education level) and loss-related characteristics (kinship to the deceased, expectedness of the loss (i.e. expected, not expected, neither expected nor unexpected), cause of death and months since loss) were assessed with a self-constructed questionnaire.

Table 1
Baseline Sample Characteristics ($N = 220$).

Demographic variables	
Sex – *valid n* (%)	
Female	195 (89)
Male	25 (11)
Age in years – *M (SD),* range	51.8 (12.24), 20-84
Education level – *valid n* (%)	
High[a]	91 (41)
Low	129 (59)
Loss characteristics	
Months since loss at T1 – *M,* range	2.3, 1-6
Months since loss at T2 – *M,* range	8.3, 7-12
Months since loss at T3 – *M,* range	14.3, 13-18
Deceased is – *valid n* (%)	
Partner	86 (39)
Parent	90 (41)
Sibling	13 (6)
Child	22 (10)
Other	9 (4)
Expectedness of death – *valid n* (%)	
Expected	80 (36)
Unexpected	99 (45)
Both or neither "expected nor unexpected"	41 (19)
Cause of death – *valid n* (%)	
Natural death	187 (85)
Accident	6 (3)
Suicide	15 (7)
Coronavirus (Covid-19)	10 (5)

Note.
[a] High refers to college or university.

2.3.2. Insomnia symptoms

Insomnia symptoms were assessed with the Dutch translation of the Insomnia Severity Index [38]. The seven items cover day and nighttime symptoms as well as worries about sleep in the past two weeks. Items are scored on a scale from 0 to 4 with varying anchors. Total scores can range from 0 to 28. Total scores from 0 to 7, 8 to 14, 15 to 21 and above 21 signify no insomnia, subclinical insomnia, clinical insomnia (of moderate severity), and severe clinical insomnia, respectively [39]. In the current study, internal consistency was good (αs ranged between 0.84 and 0.87 across study waves).

2.3.3. Prolonged grief symptoms

Prolonged grief symptoms were assessed with the Traumatic Grief Inventory Self-Report Plus (TGI-SR+; [40]). The TGI-SR+ is based on the TGI-SR [41] and was specifically designed to cover all criteria of recent grief disorders included in the DSM-5 (persistent complex bereavement disorder), DSM-5-TR and ICD-11 (PGD). The 22 items cover varying grief reactions and are scored on a Likert scale ranging from 1 'never' to 5 'always'. Total scores can range between 22 and 110. Scores of 71 and above indicate probable prolonged grief [40]. In the current study, internal consistency levels were excellent (αs ranged between 0.93 and 0.94 across study waves). To ensure legibility, we use the term prolonged grief symptoms to refer to the scores on the TGI-SR + regardless of the time-point of measurement (thereby ignoring the time criterion of PGD for the first time-point(s)). Furthermore, it is important to note that prolonged grief disorder can only be established in a clinical interview; we use the term 'probable prolonged grief' for notational convenience.

2.3.4. Depression symptoms

Depression symptoms were assessed with the Dutch version of the Quick Inventory of Depressive Symptoms Self-Report [42,43]. The QIDS-SR is a widely used 16-item screening instrument developed to measure depression symptoms per DSM-IV-TR criteria. Items are answered on a 4-point Likert scale ranging from 0 to 3 (varying anchors). Items cover symptoms in several domains such as mood, appetite, weight, psychomotor, and sleep problems in the past week. In the current study, the sleep items were omitted in all main analyses due to potential overlap with the ISI. Total scores are based on a scoring algorithm in which scores of 6–10, 11 to 15, 16 to 20, and 21 to 27 represent mild, moderate, severe, and very severe depression, respectively. The QIDS-SR was administered at the first wave. Baseline internal consistency of the QIDS-SR was acceptable (α = 0.78).

2.3.5. Grief rumination

Grief rumination was measured with the Utrecht Grief Rumination Scale [44]. This 15-item questionnaire measures repetitive and recurrent thoughts about the causes and consequences of the loss in the past month. Items are scored on a 5-point Likert scale ranging from 1 'never' to 5 'very often'. Total scores can range from 15 to 75 with higher scores representing more severe grief rumination. The UGRS was assessed at the first wave and its internal consistency was good (α = 0.85).

2.3.6. Experiential avoidance

Experiential avoidance was measured with the Dutch version of the Acceptance and Action Questionnaire II [45]. The more recent seven-item AAQ-II has not been validated in Dutch. Therefore, we used the ten-item version [46]. The items cover acceptance and experiential avoidance in general, on a scale of 1 'never true' to 7 'always true'. For easier interpretation of the results, we reverse-coded the items such that higher scores reflected more experiential avoidance. The AAQ-II was assessed at the first wave and its internal consistency was excellent (α = 0.90).

2.4. Statistical analysis

The main analyses were performed with Mplus (version 8.6; [47]). Data preparation, descriptive analyses, and initial correlational analyses were performed with IBM SPSS Statistics (version 28). To investigate the typical trajectories of insomnia symptoms following bereavement, we conducted growth mixture modeling (GMM).

Before conducting our main analysis, we checked for univariate (±3 *SD* from the mean) and multivariate (Mahalanobis distance with a probability <.001) outliers. One univariate and one multivariate outlier were detected. We conducted the analyses with and without these outliers. No mentionable differences were found. We therefore only reported the analyses including the outliers.

Before our GMM, we estimated a latent base model by freely estimating the slopes (estimated rate of change) and intercepts (estimated baseline score). The time factors were set to 0 at T1, 0.5 at T2, and free at T3. Hereafter, we estimated a linear latent growth curve model. We set time as 0 at T1, 0.5 at T2, and 1 at T3 to represent time in years. We selected the best-fitting model based on Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), sample size adjusted BIC (ABIC), and parsimony. The best-fitting model was used as the base model for the growth mixture modeling and re-estimated with an increasing number of classes with a maximum of four classes (one more than our hypothesized number of latent classes; [48]), while allowing for within-class variance.

The final model in the growth mixture modeling was selected based on the suggestions of Ram and Grim [48]. First, we checked if AIC, BIC and ABIC levels of a particular class solution were better (lower) compared to the model with one class less (e.g., 3-class vs. 2-class solutions). Hereafter, we inspected entropy, Vuong-Lo-Mendel-Rubin likelihood ratio test (VLRT), and Bootstrapped likelihood ratio test (BLRT). Final decisions were made while keeping interpretability and group size per trajectory in mind.

To examine the risk and protective factors for each of the resulting latent classes, we employed the Bolck–Croon–Hagenaars method [49]. The BCH method uses weights to counteract the possible biases introduced by class membership uncertainty and prevents class shifts in the final model. We employed the BCH method with the final GMM model. First, we predicted class membership from sociodemographic and loss

related variables (age, sex, education level, kinship to the deceased, cause of death, expectedness of the loss, months since loss). Using this as a base model, four separate models were estimated, predicting class membership from baseline grief severity, depression symptoms, grief rumination, and experiential avoidance while statistically controlling for the sociodemographic and loss-related variables. We considered BIC, ABIC, and AIC to check whether adding the baseline predictors added significantly to the model fit. Hereafter, we selected the best predictor (based on model fit) and added the other baseline predictors to the model one by one. Only baseline predictors that increased model fit significantly were kept in the final model. Additionally, we calculated the percentage of probable PGD cases (a score of 71 or higher on the TGI-SR+; [40]) at T3 in each of the insomnia trajectory classes.

Finally, to further our understanding of how insomnia and prolonged grief symptoms move together over time, we conducted a parallel (process) growth curve model analysis. We estimated the average trajectories of insomnia symptoms and prolonged grief symptoms simultaneously and correlated their intercepts and slopes [50]. This model allowed us to look at the interrelation of change in insomnia symptoms with change in prolonged grief symptoms over time (multivariate change).

3. Results

3.1. Preliminary analyses

Overall, insomnia and prolonged grief symptoms declined from baseline to one-year follow-up. At baseline, 27 % of the sample experienced probable clinical insomnia (>14 on ISI), which reduced to 10 % at the one-year follow-up. Probable (moderate) depression (>15 on QIDS) went down from 25 % at baseline to 8 % at one-year follow-up. Probable prolonged grief could only be established at T3 (due to the time criterion of one year for PGD): 20 % of the participants met the cut-off (>70 on TGI-SR+). Thus, post-loss psychopathology symptoms ranged from non-clinical to clinical and declined over time.

3.2. Zero-order correlations

The zero-order correlations between insomnia, prolonged grief, depression symptoms, grief rumination, and experiential avoidance are shown in Table 2. All constructs were related positively both cross-sectionally and longitudinally.

3.3. Growth mixture model (GMM)

The linear model did not fit the data worse than the latent base model, i.e., AIC was comparable and BIC was smaller (see Table 3 for fit indices). Therefore, we continued with the linear model with larger degrees of freedom. Next, we increased the number of classes one by one up to four. The fit statistics for all the models are summarized in Table 3.

Based on ABIC, AIC, interpretability, and entropy, the three-class model fitted the data best. The BIC showed no significant difference between the fit of the two and three-class models and the VLRT and BLRT showed conflicting results. This might be due to the relatively low number of participants that constituted the third theoretically plausible trajectory.

Therefore, we decided to continue with the easier-to-interpret three-class model. Next, to statistically correct for the variability in months since loss at baseline, we added baseline months since loss (at T1, recorded in months) as a covariate to the overall model.

The final model identified three distinct trajectories of insomnia symptoms across the three study waves (see Fig. 1). The first and largest class (n = 103; 47 %), labeled *resilient trajectory,* had no (sub)clinical insomnia symptoms at baseline and showed a significant decrease over time (intercept = 6.89, slope = −4.40, $p < .001$). The second trajectory (n = 94; 43 %), labeled *recovering trajectory,* showed subclinical insomnia symptoms at baseline decreasing significantly over time (intercept = 12.96, slope = −4.05, $p < .001$). The third trajectory (n = 23; 10 %), labeled *chronic trajectory,* showed clinical insomnia symptoms at baseline with no significant reduction from baseline to one-year follow-up (intercept = 16.54, slope = −0.80, $p = .491$). Finally, the slopes of the *resilient* and *recovering* trajectory were not significantly different (*Wald* = 0.14, $df = 1$, $p = .706$), indicating that these trajectories are distinguishable only by their baseline insomnia levels.

Overall, those who started with more severe insomnia symptoms showed greater reduction in insomnia severity over the course of a year ($\beta = -0.97$, $p < .001$). Months since loss (covariate) was unrelated to baseline insomnia severity ($\beta = 0.02$, $p = .778$), but those who experienced the loss more recently had a larger reduction in insomnia severity over time ($\beta = 0.23$, $p = .031$).

3.4. Predictors of trajectory membership

As a base model, we predicted class membership from sociodemographic and loss-related variables (kinship to the deceased (dummy coded as child loss vs. other and partner loss vs. other), expectedness of the loss (dummy coded as expected vs. both expected and unexpected, and unexpected vs. both expected and unexpected), age, sex, education level (dummy coded as high (college or university) vs. low (other education)), months since loss). The odds of belonging to the recovering trajectory vs. resilient trajectory were 4.45 higher in the case of a violent loss vs. a non-violent loss. The cause of death did not differentiate between belonging to the chronic vs. recovering nor chronic vs. resilient trajectory. None of the other sociodemographic or loss-related variables significantly distinguished between the three latent classes in the base model. Full model details are provided in Supplement A.

Starting from this base model, four separate models were evaluated by adding baseline prolonged grief symptoms, depression symptoms, grief-related rumination, and experiential avoidance to the model separately. All baseline symptomatology, with the exception of grief

Table 2
Zero-order correlations between the variables.

Variable	IS T1	IS T2	IS T3	PGS T1	PGS T2	PGS T3	DS T1	GR T1	*M*	*SD*
IS T1	–								10.80	5.79
IS T2	.69	–							8.93	5.50
IS T3	.65	.75	–						7.96	4.96
PGS T1	.44	.38	.35	–					66.73	15.45
PGS T2	.36	.51	.47	.73	–				62.33	15.71
PGS T3	.33	.45	.48	.68	.81	–			57.92	16.55
DS T1	.56	.48	.45	.74	.57	.46	–		12.16	4.91
GR T1	.28	.23	.21	.67	.53	.48	.44	–	43.34	10.64
EA T1	.38	.40	.29	.74	.61	.49	.71	.56	35.35	12.23

Note: All correlations are significant, $p < .001$. IS = insomnia symptoms, PGS = prolonged grief symptoms, DS = depression symptoms, GR = grief rumination, EA = experiential avoidance. All correlations between depression and insomnia symptoms are calculated without the sleep items in the QIDS-SR. All other statistics are calculated with the sleep items in the QIDS-SR.

Table 3
Summary of the fit indices.

Model	Number of classes	AIC	BIC	ABIC	Entropy	VLMR LRT *p*-value	Bootstrapped LRT *p*-value
Latent-base	1	3562.77	3593.31	3564.79			
Linear	1	3563.13	3590.28	3564.93			
Linear	2	3554.93	3592.26	3557.40	.62	.015	0
Linear	3	3544.63	3592.14	3547.78	.76	.044	.667
Linear	4	3543.50	3601.19	3547.32	.81	.344	1.00
Linear with covariate	3	3536.79	3591.09	3540.38	.79	.045	.500

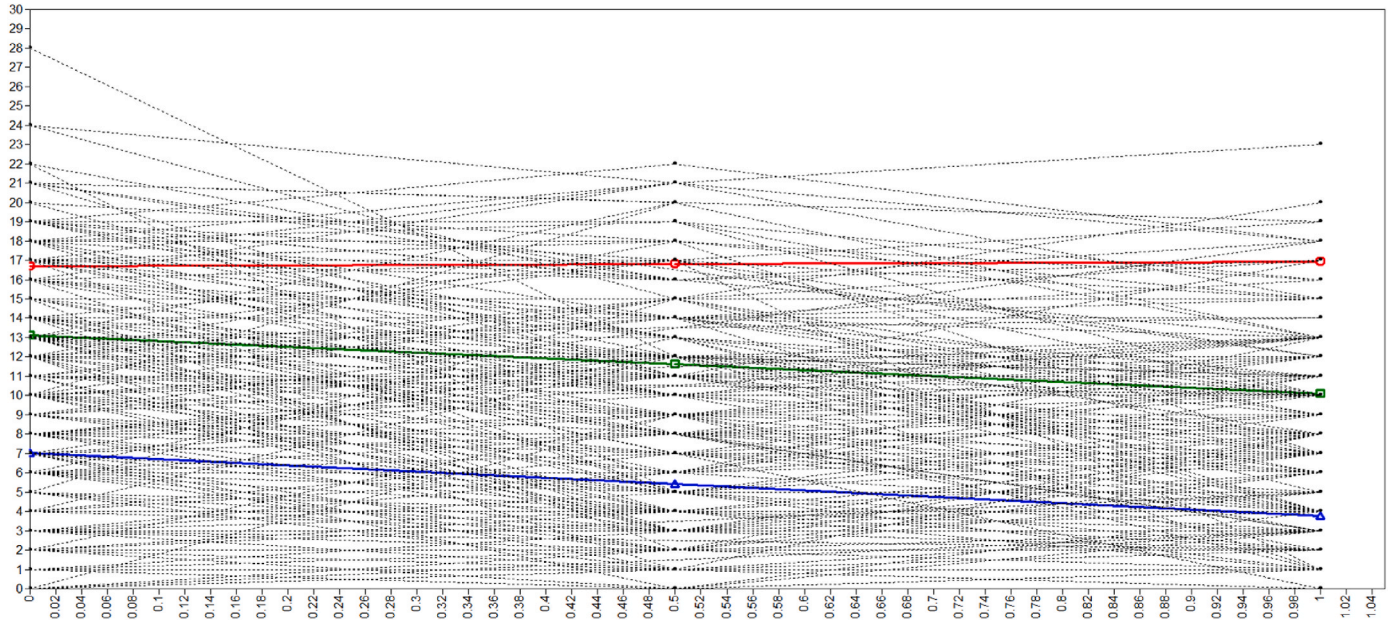

Fig. 1. *Graphical Depiction of the Latent Classes Trajectories*
Note: The blue (triangles), green (squares), and red (circles) lines correspond to the resilient, recovering, and chronic trajectory, respectively. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

rumination, added significantly to the model. More severe prolonged grief symptoms, depression symptoms, and experiential avoidance at baseline increased the odds of belonging to the chronic vs. recovering trajectory (OR's ranged between 1.07 and 1.16), chronic vs. resilient trajectory (OR's ranged between 1.11 and 1.49) and recovering vs. resilient trajectory (OR's ranged between 1.04 and 1.28), respectively (for full model details see Supplement A).

To determine the best combination of baseline symptomatology for predicting trajectory membership we added baseline prolonged grief symptoms and experiential avoidance separately to the model with depression symptoms (the best-fitting model). Neither model increased the model fit significantly. This indicates that prolonged grief symptoms and experiential avoidance do not uniquely add to the variance already explained by baseline depression symptoms and sociodemographic and loss-related variables.

Finally, we calculated the percentage of probable PGD at one-year follow-up (>70 on TGI-SR+) in each of the trajectories (classes). Nine percent of the participants in the *resilient* trajectory experienced probable PGD, 27 % in the *recovering* trajectory, and 60 % in the *chronic* trajectory.

3.5. Parallel process model

The parallel process model did not fit the data well (RMSEA = 0.09). Following the modification indices provided by Mplus, we correlated the residuals of prolonged grief and insomnia symptoms at T2. Hereafter, the model fitted the data well (CFI = 0.99, TLI = 0.99, RMSEA = 0.02, SRMR = 0.02).

On average, participants experienced subclinical insomnia symptoms at (estimated) baseline that decreased over the course of a year (intercept = 10.67; slope = −2.71, $p < .001$). Similarly, mean prolonged grief symptoms at (estimated) baseline could also be considered subclinical (ignoring the time since loss criterion) and decreased over the course of the year (intercept = 66.80; slope = −8.67, $p < .001$). In general, those who reported more severe insomnia symptoms at baseline also reported more severe prolonged grief symptoms ($\beta = 0.56, p < 001$). Furthermore, on average, decreases in insomnia symptoms were more pronounced in those with more severe baseline insomnia ($\beta = -0.47$, $p < .001$) and more severe baseline prolonged grief symptoms ($\beta = -0.29$, $p = .032$). Baseline insomnia severity ($\beta = -0.20$, $p = .119$) nor baseline grief severity ($\beta = -0.06$, $p = .826$) were significantly related to changes in prolonged grief symptoms over time.

Changes in insomnia symptoms were, however, strongly related to changes in prolonged grief symptoms over time ($\beta = 0.81$, $p = .005$). This suggests that insomnia and prolonged grief symptoms follow a similar trajectory. The 95 % confidence interval of this estimated correlation is, however, large, ranging from 0.25 to 1.00.

4. Discussion

The first aim of the study was to distinguish potentially different trajectory classes of insomnia symptoms following bereavement. We used growth mixture modeling to distinguish unobserved subpopulations based on different starting points (intercepts) and differences in change over time (slopes) [48]. In line with our predictions and the 3P model of insomnia [27], we identified three distinct trajectory

classes. The classes were characterized by a resilient, recovering, and chronic trajectory of insomnia symptoms. The second aim of the study was to identify potential risk- and protective factors for a more chronic insomnia trajectory. More severe baseline prolonged grief symptoms, experiential avoidance, and depression symptoms were associated with a higher likelihood of belonging to the chronic vs recovering and recovering vs resilient insomnia trajectory. However, only baseline depression symptoms uniquely distinguished the three trajectories. The third aim of the study was to explore the relationship between insomnia trajectories and prolonged grief. Probable PGD at one-year follow-up was most common in the chronic insomnia trajectory, followed by the recovering and resilient trajectories. Similarly, the parallel process model showed a strong correlation between the slopes of prolonged grief and insomnia symptoms, showing that insomnia and prolonged grief symptoms followed a simultaneous trajectory.

The three-group result for insomnia trajectories following bereavement aligns with the 3P model of insomnia [27] and previous studies showing that most people experience some sleep disturbances following bereavement [9,51]. The resilient and recovering trajectories were different in initial insomnia severity, but showed a similar decline in insomnia symptoms. A likely reason for the differences between these two groups is the difference in the severity of the initial stress reaction to the loss. This hypothesis is supported by our analyses of the relationships between cause of death and trajectories. Those who experienced a (more stressful) violent vs. (less stressful) non-violent loss were more likely to be part of the recovering vs. resilient trajectory. Additionally, those in the recovering trajectory might have had predisposing factors, i.e. characteristics that increase the odds of developing acute insomnia. Thus, it seems that although there were differences in initial insomnia severity between these groups, the insomnia symptoms faded with the dissipation of loss-related distress. Conversely, people in the chronic insomnia trajectory (vs. other trajectories) had higher initial levels of grief, but also showed no significant decline in insomnia symptoms over the course of a year. The latter may be due to the use of maladaptive insomnia coping strategies (e.g., [27,28]). For example, in response to feeling fatigued, these participants might have started napping, spent more time in bed, or started worrying about their sleep. This, in turn, may have perpetuated their insomnia.

The finding that baseline grief severity could differentiate between the different insomnia trajectories corresponds with findings that more severe grief reactions are associated with more severe sleep disturbances [9,52,53]. In further agreement with the existing literature, the relationship between baseline grief severity and insomnia trajectories was better explained by comorbid baseline depression symptoms [9,52,54–56].

One explanation why depression symptoms might be a predictor of a more chronic insomnia trajectory is through effects on activity levels. Depression has been related to sedentary behavior [57] and (especially new) depressive episodes have been related to a decrease in physical activity [58]. Depression symptoms following a loss could thereby lower the homeostatic build-up of sleep pressure and increase insomnia symptoms (cf. [59,60]).

The finding that grief rumination did not differentiate between the identified insomnia trajectories is somewhat puzzling since repetitive negative thinking is thought to play role in the persistence of insomnia (e.g., [28]). However, our observation may be explained by literature suggesting that the content and timing of repetitive negative thought is of importance in relation to insomnia. For example, one study found that insomnia-specific rumination predicted insomnia above depression symptoms but self-focused and depressive rumination did not [61]. Additionally, in a diary study, night-time worrying about sleep related to insomnia, whereas daytime worrying about sleep did not [32]. The tendency to ruminate about the loss may not have the same effects on insomnia symptoms as nighttime repetitive thought about sleep.

Finally, our results suggest that persistent sleep problems are related to more severe post-loss psychopathology. Specifically, probable PGD was most prevalent in the chronic insomnia trajectory followed by the recovering and resilient trajectories, respectively. Furthermore, the parallel process model demonstrated that the intercepts of baseline prolonged grief and insomnia symptoms did not show a significant relationship with the slope of prolonged grief symptoms. This suggests that merely knowing the severity level of both types of symptoms at baseline is insufficient for predicting prolonged grief symptom changes. The trajectory of insomnia and prolonged grief symptoms were however highly correlated. This suggests that prolonged grief and insomnia symptoms move together over time. These findings complement a growing body of research suggesting that insomnia symptoms are implicated in the development and persistence of prolonged grief symptoms (e.g., [15,22]). Furthermore, these results are in line with experimental studies reporting a beneficial effect of treating insomnia symptoms on prolonged grief symptoms [16,23].

Potentially, insomnia symptoms cause the persistence of grief reactions by disrupting the integration of memories about the loss into the existing autobiographical memory base [62–64]. The neurobiological milieu during (REM) sleep, in particular low noradrenaline levels [65], is proposed to facilitate the reduction in the emotional load during the (re)activation and (re)processing of emotional memories [17,18,66]. Under these conditions, REM sleep dreams might also act as a form of exposure (e.g., [66,67]), furthering the reduction of the emotional charge of the memories [66].

Clinically, our results add to a growing knowledge base suggesting that monitoring and targeting insomnia symptoms following bereavement might be valuable, especially in the presence of comorbid depressive symptoms. Early interventions such as sleep hygiene could potentially prevent the development of insomnia syndrome following bereavement. Evidence-based cognitive behavioral therapy for insomnia [68] might help prevent and/or treat PGD [9,15,16].

4.1. Strengths and limitations

Strengths of the current study include a multi-wave design with a large group of recently bereaved participants and the application of advanced statistical techniques. However, some limitations warrant mention. First, the online recruitment led to an overrepresentation of older, Western women who had lost a partner or parent due to natural causes. While this is common in bereavement research [33], a replication in a more representative sample is warranted. Second, the data collection for the current study occurred after bereavement. Therefore, we do not know if insomnia or depressive symptoms were present before the loss.Some of our findings could have been influenced by pre-bereavement psychopathology. Future studies may include a retrospective questionnaire on pre-loss mental health. Third, we included participants who were at baseline in the first six months following a loss. While we statistically corrected for this baseline variance, the differences within this timeframe might have altered our results. Therefore, we recommend cross-validating results from this study using a cohort design.

4.2. Conclusion

Notwithstanding these limitations, our results add to the existing literature, suggesting that therapeutically targeting sleep problems, specifically insomnia symptoms following a loss, could be beneficial for those individuals with a severe grief reaction and insomnia symptoms (e.g., [9,15,16]). Early interventions targeting insomnia symptoms might be especially warranted for those who report comorbid depression symptoms following bereavement. The results warrant a continued scientific focus on the role of disturbed sleep following bereavement [9], including randomized controlled trials on the effect of targeting insomnia symptoms in the bereaved.

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Data availability statement

Due to the sensitive nature of the questions asked in the study, rawdata are only available upon resonable request. Syntaxes for the analyses are available on DataverseNL [69].

CRediT authorship contribution statement

Thomas A. de Lang: Data curation, Formal analysis, Investigation, Writing – original draft. **Asuman Buyukcan-Tetik:** Formal analysis, Writing – review & editing. **Peter J. de Jong:** Funding acquisition, Writing – review & editing. **Marike Lancel:** Conceptualization, Funding acquisition, Writing – review & editing. **Maarten C. Eisma:** Conceptualization, Funding acquisition, Methodology, Project administration, Supervision, Writing – review & editing.

Declaration of Competing interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2023.12.009.

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