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Changes in insomnia as a risk factor for the incidence and persistence of anxiety and depression: a longitudinal community study

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Abstract

Background: The aim of this investigation was to examine the longitudinal association between change in insomnia status and the development of anxiety and depression in the general population.

Methods: A survey was mailed to 5000 randomly selected individuals (aged 18–70 years) in two Swedish counties. After 6 months, a follow-up survey was sent to those ($n = 2333$) who answered the first questionnaire. The follow-up survey was completed by 1887 individuals (80.9%). The survey consisted of questions indexing insomnia symptomatology, socio-demographic parameters, and the Hospital Anxiety and Depression Scale. Change in insomnia status was assessed by determining insomnia at the two time-points and then calculating a change index reflecting incidence (from non-insomnia to insomnia), remission (from insomnia to non-insomnia), or status quo (no change). Multivariate binary logistic regression analyses were used to examine the aim.

Results: Incident insomnia was significantly associated with an increased risk for the development of new cases of both anxiety (OR = 0.32, $p < .05$) and depression (OR = 0.43, $p < .05$) 6 months later. Incident insomnia emerged also as significantly associated with an elevated risk for the persistence of depression (OR = 0.30, $p < .05$), but not for anxiety.

Conclusions: This study extends previous research in that incidence in insomnia was shown to independently increase the risk for the development of anxiety and depression as well as for the maintenance of depression. The findings imply that insomnia may be viewed as a dynamic risk factor for anxiety and depression, which might have implications for preventative work.

Keywords: Insomnia, Anxiety, Depression, Incidence, Persistence, Risk factors

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Background

Previous epidemiological research has clearly shown that current insomnia is associated with an increased risk of developing anxiety and depression. It is, however, rare that studies have investigated whether changes in insomnia over time is predictive of the development of psychiatric conditions. A focus on how the dynamic change in insomnia is related to future psychiatric conditions might provide important information for theory and clinical implications. The current, longitudinal study explored whether changes in insomnia over time was related to the development of anxiety and depression in the general population.

Insomnia is a prevalent health problem and has been suggested to be involved in the incidence and persistence of psychiatric conditions. Several meta-analyses have concluded that insomnia is a risk factor for incident depression (Baglioni et al. 2011; Hertenstein et al. 2019; Li et al. 2016) and incident anxiety (Hertenstein et al. 2019; Pigeon et al. 2017). Less is known about the role of insomnia as a predictor of persistent psychiatric condition. The few studies that have examined the association between insomnia and persistent depression have shown mixed results (Kim et al. 2009; Pigeon et al. 2008; van Mill et al. 2014) and, to the best of our knowledge, only one study has examined the association between insomnia and persistent anxiety (van Mill et al. 2014). The latter study did not find any significant association between insomnia and persistent DSM-IV anxiety disorders.

Expanding the understanding of the predictors for the development of depression and anxiety could provide clues both on how to prevent the onset of psychiatric conditions and to improve the management of patients with these conditions (Baglioni et al. 2011; Ford & Kamerow, 1989; Jansson-Fröjmark and Lindblom 2008; Sivertsen et al. 2014). Also, if insomnia was found to be a predictor for persistence of depression and anxiety, in theory, treatment of insomnia in patients suffering from both insomnia and psychiatric conditions could be a way of improving the treatment of the psychiatric conditions among these patients.

Several issues need to be illuminated to understand the role of insomnia in the development of anxiety and depression. First, although several investigations have explored the link between baseline insomnia and future development of depression and anxiety, it has been rare to explore whether changes in insomnia status over time is predictive of the incidence and persistence of psychiatric conditions (Chen et al. 2017). A focus on changes in insomnia (i.e., incidence or remission) might provide insight into the dynamic development over time. A focus on changes in insomnia might add valuable information for theory and clinical applications. Second, the majority of previous studies have not determined insomnia based on established diagnostic criteria, but based the definition on a few insomnia symptoms items, often neglecting daytime consequences. Third, the lack of screening for other sleep disorders in previous studies poses a potential threat against the control of potential confounding factors since several other sleep disorders have been suggested to be involved in the development of psychiatric conditions, e.g., sleep-related breathing disorder and depression (e.g., Peppard et al. 2006).

The purpose of this study was to investigate the longitudinal association between change in insomnia status and the development of anxiety and depression in the general population. Change in insomnia status was determined by comparing insomnia (yes/no) at two time-points to reflect incidence (from non-insomnia to insomnia), remission (from insomnia to non-insomnia), or status quo (no change). Specifically, the study had two aims. The first aim was to examine whether incident insomnia was associated with the incidence of anxiety and depression. The second aim was to investigate whether incident insomnia was associated with the persistence or remission of anxiety and depression. Thus, the aims were to explore whether change in insomnia status (incidence, remission, or status quo) is associated with an increased risk of developing and maintaining anxiety and depression. Since previous, longitudinal research on the link between insomnia and psychiatric conditions has examined whether insomnia at baseline (yes/no) is associated with an increased risk for other conditions, secondary analyses were also performed to be able to compare our findings with published findings; these analyses used insomnia status at baseline as the predictor variable. Socio-demographic parameters were used as covariates (e.g., Gellis et al. 2005; Morin et al. 2006; Ohayon 2002). Since anxiety and depression act as bidirectional risk factors for one another (Jacobson and Newman 2017), anxiety was used as a covariate when analyzing depression as an outcome and vice versa.

Methods

Overview of the study

This prospective study was carried out in the general population. A survey consisting of questions indexing insomnia symptomatology, socio-demographic parameters, and psychiatric conditions was mailed to the participants on two occasions over 6 months [baseline (T1) and 6-month follow-up T2)]. The study was approved by the Regional Ethics Board in Uppsala, Sweden (2008/157).

Participants and procedure

The survey was sent out to a random sample of 5000 people, aged 18 to 70 years, from two Swedish counties (Örebro and Värmland) during September 2008. The sample was acquired from the national register, and the

randomization was conducted by Statistics Sweden, the national statistics department. In the national register, all residents of Sweden are listed. Simple random sampling (i.e., all residents in the two counties between 18 and 70 years old had an equal probability of being selected) was used in the study. To increase the response rates at the assessment points, several steps (e.g., including a small incentive with the survey and attaching a pre-paid return envelope) were taken in line with a Cochrane review (Edwards et al. 2007).

Of the initial 5000 candidates, 2333 (47.1%) returned the survey at T1. The respondents were compared with register data and proved to be representative for the Swedish population regarding age, gender, relationship status, occupational status, educational level, and reported sleep disturbance (Jansson-Fröjmark et al. 2012). At T2, 1887 participants returned the survey. In all the analyses of the current study, the inclusion criteria required that the participants (1) had returned the surveys at both T1 and T2 and (2) did not fulfill criteria for six primary sleep disorders (for more details, see under Sleep disorders other than insomnia). Of the 1887 responders at T2, 1651 met the criteria for inclusion.

Measures

Data on insomnia symptomology and psychiatric conditions were obtained both at T1 and T2. Information on socio-demographic factors was collected at T1.

Socio-demographic parameters

The following socio-demographic parameters were assessed: age, gender, civil status, vocational status, level of education, and place of birth.

Insomnia symptomology

The questions assessing insomnia symptomatology were based on the DSM-IV-TR and Research Diagnostic Criteria for insomnia (Edinger et al. 2004). To determine sleep disturbance, the participants were asked the question "During the last month: Would you say that you have had any sleep problems? (yes or no)." The participants were also asked to complete categorical questions based on the previous month concerning: average sleep onset latency (< 15 min, 16–30 min, 31–60 min, > 60 min), average wake time after sleep onset (same alternatives as for sleep onset), and average early morning awakening (same alternatives as for sleep onset). The Insomnia Severity Index (ISI; score range 0–28 points) was also used (Bastien et al. 2001). The ISI measures subjective insomnia severity and is a reliable and valid instrument to detect cases of insomnia (Morin et al. 2011). Daytime symptoms and impaired function during the previous month were also assessed. First, the participants were asked: "When your sleep has been disturbed: How have you been affected during the day?" In this question, they were to determine to what degree [not at all (1), somewhat (2), quite much (3), and a lot (4)] they had been affected in the following areas: fatigue/malaise, impairment in attention, concentration or memory, mood disturbance, irritability, daytime sleepiness, reduction in motivation, energy or initiative, proneness for errors or accidents at work or while driving, tension headache, gastrointestinal symptoms, and concerns or worries about sleep (Edinger et al. 2004). Second, the participants were asked: "When your sleep has been disturbed: How have you been affected during the day in the following domains: social and vocational"? The response alternatives for the two functional domains were: not affected (1), some difficulties but not less ability to function (2), less ability to function (3), much less ability to function (4) and could barely function (5).

Sleep disorders other than insomnia.

The questionnaire SLEEP-50 was used to assess six DSM-IV-TR sleep disorders: apnea, narcolepsy, restless legs/periodic limb movement disorder, circadian rhythm disorder, and sleepwalking (Spoormaker et al. 2005). In the questionnaire, the participants are asked to what extent a sleep complaint has applied to them the previous month on a 4-point scale (from 1 = not at all, to 4 = very much). The instrument has good reliability (α = 0.85), acceptable test-retest reliability (r = 0.78), good predictive validity, and a factor structure that matches the DSM-IV-TR sleep disorders. The sensitivity and specificity scores are reasonable for sleep disorders (sensitivity: 0.67–1.00; specificity: 0.69–1.00). The agreement between clinical diagnoses and classification derived from the SLEEP-50 is substantial (kappa = .77).

Sleep group classification.

After each assessment (T1-T2), the participants were classified into distinct groups according to their sleep patterns, daytime impairment, and evidence of sleep disorders other than insomnia. The classification used an algorithm based on a combination of insomnia diagnostic criteria from DSM-IV-TR and Research Diagnostic Criteria for insomnia (Edinger et al. 2004). Two-hundred thirty-six participants who fulfilled criteria for other sleep disorders (i.e., apnea, narcolepsy, restless legs/periodic limb movement disorder, circadian rhythm disorder, and sleepwalking; based on the SLEEP-50) were excluded from the analyses.

Participants in the insomnia group had to confirm a sleep disturbance during the last month, and report initial, middle, or late insomnia (> 30 min awake involuntarily at any stage during an estimated average night). They also had to report some daytime impairment [a minimum of 3 (quite much) on at least one daytime

symptom or a minimum of 4 (much less ability to function) on at least one functional impairment domain)]. The exclusion criteria were: The participants must not meet the criteria for apnea, narcolepsy, restless legs syndrome/periodic limb movement disorder, circadian rhythm disorder, or sleepwalking as assessed with the SLEEP-50. To test if our insomnia definition was able to identify the presence of insomnia, we have investigated the concordance between our definition and two validated Insomnia Severity Index (ISI) cut-offs for insomnia (Jansson-Fröjmark et al. 2012; Morin et al. 2011). The comparison showed high concordance (89.4–99.4%) between the cut-offs on ISI and our definition of insomnia, which indicates that our insomnia definition captures the insomnia construct to an acceptable degree. It should also be underscored that this study's definition of insomnia compares well with other larger investigations (e.g., LeBlanc et al. 2009) while also uniquely assessing other sleep disorders with a validated instrument, the SLEEP-50.

Anxiety and depression.

The Hospital Anxiety and Depression Scale (HADS) was used to measure anxiety and depression (Herrmann 1997; Zigmond and Snaith 1983). The HADS is a self-rating scale and comprises 14 items, each scored on a 4-point scale. Seven of the items are linked to symptoms of anxiety and seven reflect symptoms of depression (score range for each subscale: 0–21). Investigations have shown that the HADS is a psychometrically sound instrument, e.g. good internal consistency and acceptable discriminant validity (Herrmann 1997). An often-used procedure in research to detect a possible case of anxiety or depression has been to dichotomize the two subscales: a score of 7 or less indicates a non-case and a score of 8 or more a definite case (Herrmann 1997). In a review of the studies examining the validity of the HADS, this cut-off score was shown to produce the best balance between sensitivity and specificity for both subscales (Bjelland et al. 2002).

Classification of incident and persistent cases of anxiety and depression

A respondent scoring 8 or higher on either subscale (anxiety or depression) on HADS was defined as a case of anxiety or depression. An incident case of anxiety or depression was defined as a respondent scoring 7 or lower on a HADS subscale (anxiety or depression) at T1, but 8 or higher on the same subscale at T2. A respondent scoring 8 or higher on one of the subscales (anxiety or depression) at both T1 and T2 was defined as a persistent case of anxiety or depression.

Statistical analyses

The analyses were conducted using SPSS for Windows version 24. To examine the role of insomnia as a risk factor for incident and persistent psychiatric conditions, multivariate binary logistic regression analyses were used. The first set of longitudinal analyses were based on those without a specific psychiatric condition (e.g., depression) at T1 and examined the role of insomnia on the incidence of that psychiatric condition, relative to non-incidence of the same condition. In this set, we examined both the role of baseline status (insomnia relative to no insomnia) and the role of change in insomnia status over time (T1-T2). When examining the role of change in insomnia status, change scores for insomnia were calculated by first coding the condition at each time-point as 0 (no) or 1 (yes). Then, the T1 score (0 or 1) was subtracted by the score at T2 [0 (no) or 1 (yes)]. Thus, the change score for each individual take on one out of three possible numbers [i.e., − 1 (incidence), 0 (status quo) or + 1 (remission)]. These numbers were then used to examine group-wise differences over time. The raw data (M and SD) that appear in Tables 2 and 4 are thus the respective groups' average change score for insomnia. The change scores were later included in multivariate logistic regression analyses as independent variables.

The second longitudinal set of analyses was based on those with a specific psychiatric condition at T1 and explored the role of insomnia on the persistence of the same condition, relative to the remission of that condition. As with the analyses concerning incidence, this set both examined the role of baseline status (insomnia relative to no insomnia) and the role of change in insomnia status over time (T1-T2). Change scores were calculated and used in the same way as in the incidence set. To control for possible confounding effects, six sociodemographic parameters, anxiety, and depression were used as covariates in the multivariate analyses. In the current study odds ratios (OR) are presented with 95% confidence intervals. A two-tailed *p*-value smaller than .05 was considered statistically significant.

Results

Study participants

Among the 1651 study participants, the mean age was 47.1 years (SD = 14.5) and 54.9% ($n = 906$) were women. As for civil status, 14.9% ($n = 246$) reported being single, 79.4% ($n = 1311$) being cohabitant, married or having a partner, 4.2% ($n = 69$) being divorced, and 1.5% ($n = 25$) being widowed. Regarding vocational status, 73.3% ($n = 1210$) reported being full or part-time employed or students and 26.7% ($n = 441$) reported being unemployed, on sick leave, on pension or another status. Concerning education, 25.7% ($n = 424$) reported

Table 1 Insomnia at baseline (T1) as a risk factor for the incidence of anxiety and depression at 6-month follow-up (T2): Descriptive statistics, univariate logistic regression analyses and multivariate logistic regression analyses

Health condition (T2)	No insomnia (T1): n (%)	Insomnia (T1): n (%)	Unadjusted model: OR (95% CI)	Adjusted model: OR (95% CI) [a]
Anxiety	40 (3.1)	10 (5.7)	1.90 (0.93–3.89)	1.73 (0.77–3.72)
Depression	39 (3.0)	21 (10.9)	3.93 (2.26–6.84)*	2.94 (1.54–5.58)*

CI Confidence interval, *OR* Odds ratio. * Significant at the .05 level. [a] Odds ratios adjusted for age, gender, civil status, vocational status, educational level, place of birth, anxiety (when depression was the dependent variable), and depression (when anxiety was the dependent variable)

compulsory school as their highest level of education, 45.8% ($n = 756$) high school, and 28.5% ($n = 471$) college or university. The majority (92.1%, $n = 1521$) of the participants were born in Sweden. At T1, 15.9% ($n = 263$) of the study participants fulfilled criteria for insomnia, 7.0% ($n = 116$) for depression, and 9.1% ($n = 150$) for anxiety.

Incidence of anxiety and depression at 6-month follow-up

In the first set of longitudinal analyses, we investigated the role of insomnia at T1 as a risk factor for the incidence of anxiety and depression. Of the 1651 participants, 90.9% ($n = 1501$) did not fulfill the criteria for anxiety at T1. Of the 1501 individuals without anxiety at T1, 50 participants fulfilled criteria for anxiety at T2. As shown in Table 1, insomnia at T1 was not significantly associated with incident anxiety in the unadjusted logistic regression analyses. In the adjusted analysis, age (lower), educational level (compulsory or high school rather than university), and depression (case) at T1 were associated with an increased risk for incident anxiety at T2.

Of the 1651 participants, 93.0% ($n = 1535$) did not meet the criteria for depression at T1. Among the 1535 individuals without depression at T1, 60 participants met the criteria for depression at T2. As detailed in Table 1, insomnia at T1 was significantly associated with the incidence of depression (OR = 3.93) in the unadjusted logistic regression analyses. In the adjusted analysis, insomnia (case; OR = 2.94), civil status (single, divorced, or widowed), place of birth (not born in Sweden), and anxiety (case) were related to an increased risk for incident depression at T2. In all, insomnia at T1 remained a significant risk factor for incident depression in adjusted analyses.

In the main analyses, it was examined whether a change in insomnia status was associated with the incidence of anxiety and depression. As shown in Table 2, incident insomnia was significantly associated with the incidence of anxiety (OR = 0.37) and depression (OR = 0.39) in the unadjusted logistic regression analyses. In the first adjusted analysis, incident insomnia (OR = 0.32) as well as three of the covariates at T1 [age (lower), education level (compulsory or high school rather than university), and depression (case)] were associated with an increased risk for incident anxiety at T2. In the second adjusted analysis, incident insomnia (OR = 0.43) and three covariates [civil status (single, divorced, or widowed), place of birth (not born in Sweden), and anxiety (case)] were related to an increased risk for incident depression at T2. As a whole, incident insomnia remained a significant risk factor for incident anxiety and depression in adjusted analyses.

Persistence of anxiety and depression from baseline to 6-month follow-up

In the second set of longitudinal analyses, we investigated the role of insomnia as a risk factor for the persistence of anxiety and depression, in relation to remission of the conditions. Of the 1651 participants, 9.1% ($n = 150$) fulfilled the criteria for anxiety at T1. Of the 150 individuals with anxiety at T1, 84 participants fulfilled the criteria for anxiety at T2. As can be seen in Table 3, insomnia was not a significant risk factor for persistent anxiety in unadjusted and adjusted logistic regression analyses.

Of the 1651 participants, 7.0% ($n = 116$) met criteria for depression at T1. Of the 116 individuals with depression at T1, 84 participants fulfilled the criteria for depression at T2. Among the 116 individuals with depression at T1, 57 participants met the criteria for

Table 2 Incident insomnia (T1-T2) as a risk factor for the incidence of anxiety and depression at 6-month follow-up (T2): Descriptive statistics, univariate logistic regression analyses and multivariate logistic regression analyses

Health condition (T2)	Changes in insomnia status [M (SD)]: No incident health condition	Changes in insomnia status [M (SD)]: Incident health condition	Unadjusted model OR (95% CI)	Adjusted model OR (95% CI) [a]
Anxiety	.017 (.30)	−.080 (.40)	0.37 (0.16–0.86)*	0.32 (0.13–0.77)*
Depression	.024 (.29)	−.067 (.58)	0.39 (0.18–0.87)*	0.43 (0.18–0.92)*

Observe that a positive mean value for changes in insomnia status indicates remission over time at the group level; a negative value suggests incidence. *CI* confidence interval, *OR* Odds ratio. * Significant at the .05 level. [a] Odds ratios adjusted for age, gender, civil status, vocational status, educational level, place of birth, anxiety (when depression was the dependent variable), and depression (when anxiety was the dependent variable)

Table 3 Insomnia at baseline (T1) as a risk factor for the persistence of anxiety and depression at 6-month follow-up (T2): Descriptive statistics, univariate logistic regression analyses and multivariate logistic regression analyses

Health condition (T2)	No insomnia (T1): n (%)	Insomnia (T1): n (%)	Unadjusted model: OR (95% CI)	Adjusted model: OR (95% CI) [a]
Anxiety	36 (52.2)	48 (62.3)	1.52 (0.78–2.94)	1.78 (0.85–3.73)
Depression	22 (42.3)	35 (61.4)	2.17 (1.01–4.67)*	1.71 (0.63–4.57)

CI Confidence interval, *OR* Odds ratio. * Significant at the .05 level. [a] Odds ratios adjusted for age, gender, civil status, vocational status, educational level, place of birth, anxiety (when depression was the dependent variable), and depression (when anxiety was the dependent variable)

depression at T2. As is depicted in Table 3, insomnia was a significant risk factor for persistent depression in unadjusted logistic regression analyses. Insomnia did, however, not remain significantly associated with persistent depression in adjusted analyses.

In the main analyses, it was investigated if a change in insomnia status was associated with the persistence of anxiety and depression. As displayed in Table 4, incident insomnia was not significantly associated with the persistence of anxiety and depression in the unadjusted logistic regression analyses. In the first adjusted analysis, none of the predictor variables at T1 were associated with an increased risk for persistent anxiety from T1 to T2. In the second adjusted analysis, incident insomnia (OR = 0.30) and two covariates [educational status (college or university) and place of birth (not born in Sweden)] at T1 were related to an increased risk for persistent depression from T1 to T2. As a whole, incident insomnia was only a significant risk factor for persistent depression in adjusted analyses.

Discussion

The purpose of this study was to examine the role of recently developed insomnia as a risk factor for the incidence and persistence of anxiety and depression. This approach is novel, to the best of our knowledge, and the main findings showed that the incidence of insomnia was an independent risk factor associated with a higher likelihood of incident anxiety as well as incident and persistent depression. Another finding was that those who already had insomnia at baseline were more likely to report depression 6 months later. This finding of independent associations between insomnia and incident depression was expected and consistent with thorough reviews of the literature (e.g. Baglioni et al. 2011).

Moving back to one of the main findings, namely how new cases of insomnia were associated with an increased risk for the development of anxiety and depression 6 months later, this points to the possibility that insomnia is not only a static marker for the development of psychiatric problems but also that the incidence of insomnia over time is involved in the development of psychiatric conditions. This might be explained from a cognitive-behavioral perspective in that changes towards increased insomnia symptomatology might trigger cognitions and behaviors that are likely to be detrimental to anxiety and depressive symptoms. For example, a spiral of worrisome and intrusive thoughts triggered by sleep disturbance might influence psychiatric symptoms, such as anxiety symptoms and low mood (Harvey 2002). Also, it is also likely that sleep disturbance acts as a transdiagnostic mechanism for psychiatric disorders in general. For example, it has been proposed that the link between poor sleep quality or quantity and psychiatric conditions might be explained by shared or interacting neurobiology, such as circadian genes and serotonergic systems (Harvey et al. 2011).

Although insomnia has been suggested as a transdiagnostic perpetuating process of mental health problems (Dolsen et al. 2014), less is known regarding its potential role as a precipitating factor. The odds of reporting anxiety or depression after incident insomnia were comparatively small in comparison to the odds of reporting anxiety or depression after reporting insomnia at baseline. As most people with incident insomnia will remit or experience fluctuations in their insomnia severity (Morin et al. 2014), the results could perhaps be explained by how those with insomnia at baseline may

Table 4 Incident insomnia (T1-T2) as a risk factor for the persistence of anxiety and depression at 6-month follow-up (T2): Descriptive statistics, univariate logistic regression analyses and multivariate logistic regression analyses

Health condition (T2)	Changes in insomnia status [M (SD)]: Remission of health condition	Changes in insomnia status [M (SD)]: Persistent health condition	Unadjusted model: OR (95% CI)	Adjusted model: OR (95% CI) [a]
Anxiety	.210 (.48)	.098 (.49)	0.62 (0.31–1.23)	0.68 (0.31–1.44)
Depression	.192 (.44)	.018 (.49)	0.44 (0.18–1.04)	0.30 (0.10–0.93)*

Observe that a positive mean value for changes in insomnia status indicates remission over time at the group level; a negative value suggests incidence. *CI* Confidence interval, *OR* Odds ratio. * Significant at the .05 level. [a] Odds ratios adjusted for age, gender, civil status, vocational status, educational level, place of birth, anxiety (when depression was the dependent variable), and depression (when anxiety was the dependent variable)

have had insomnia for longer than those who developed it some time between baseline and follow-up. Thus, those with insomnia at baseline may have had more severe problems that were more likely to mark or lead to psychiatric problems, Still, if recently developed insomnia means an increased risk of depression or anxiety, this could have groundbreaking clinical implications. If an early intervention for sleep problems, as suggested previously (e.g., Baglioni et al. 2011; Ford & Kamerow, 1989; Jansson-Fröjmark and Lindblom 2008; Sivertsen et al. 2014) could prevent the future development of psychiatric symptoms, much could be saved in terms of healthcare costs and individual sufferings. Recently, Christensen et al. (2016) showed that cognitive-behavioral therapy for insomnia (CBT-I) has a preventative effect on depression, thus producing empirical evidence for the hypothesis. There is also growing evidence showing that, among patients with insomnia comorbid with depression, CBT-I might reduce depressive symptomatology (for reviews, see Cunningham and Shapiro 2018 and Jansson-Fröjmark & Norell-Clarke, 2016). Finally, there is also some evidence suggesting that the improvement in psychiatric conditions following CBT-I goes through the reduction in insomnia symptoms (e.g. Norell-Clarke et al. 2018). These latter empirical findings go hand in hand with the current study's findings in that changes in insomnia might drive alterations in anxiety and depression.

A related direction for future research could be to develop and test methods that can distinguish those at greater risk for persistent insomnia from those who will recover without treatment. One suggestion would be to further test the predictive powers of commonly used insomnia-related questionnaires such as the Dysfunctional Beliefs About Sleep (Morin et al. 1993) or Anxiety and Pre-occupation about Sleep Questionnaire (Tang and Harvey 2004). Scoring high on both questionnaires have respectively been associated with a greater risk of reporting persistent insomnia prospectively (Norell-Clarke et al. 2014) but they have not been tested as early screening tools in clinical settings.

Reporting insomnia at baseline was not significantly associated with the persistence of anxiety and depression in adjusted analyses. This was unexpected, as insomnia has been proposed to be a perpetuating factor involved in the maintenance of depression and anxiety (Taylor et al. 2003). Again, we suspect that the explanation to this finding may be in the characteristics of those that reported insomnia at baseline. In treatment studies, the successful dissemination of cognitive behavioral therapy for insomnia has been associated with less depressive severity as well as less severe anxiety (Cunningham and Shapiro 2018) but this pertains to help-seeking clinical samples who may be more likely to have interconnected problems. Thus, the reasons for improvements in both sleep and psychiatric symptoms in these studies may be because CBT-I affects dysfunctional inner or outer behaviors that drives the vicious cycles of more than one health problem, such as intrusive negative thinking. The problems reported by the participants in our study may have different etiologies. However, incident insomnia was independently related to an increased risk for the persistence of depression. This latter finding extends previous research and provides further evidence for the importance of incident insomnia for the maintenance of depression. This is noteworthy, as previous studies have indicated that insomnia makes depression more difficult to treat (Dew et al. 1997). A patient with depression who develops insomnia may therefore need more targeted treatment for both depression and insomnia.

Even though several prospective studies exploring the role of insomnia as a risk factor for the incidence of psychiatric conditions have been published over the past decade, there are still several knowledge gaps. This study was designed to address some of these limitations. The most important gap that we aimed to address in this study was the lack of prospective studies examining the link between incident insomnia with the incidence and persistence of psychiatric conditions. There were also other strengths with this study, such as a focus on the persistence/remission of anxiety and depression, an assessment of daytime consequences and other sleep disorders, and controlling for socio-demographic parameters.

The current investigation is not without methodological limitations. The first limitation in the current study concerns how the conditions have been measured. A proxy for the insomnia diagnosis was used instead of clinical assessments, but it should be noted that this definition had high concordance with a widely accepted insomnia measure (ISI). Anxiety and depression were measured with a psychometrically sound instrument (HADS) that, although it is fairly accurate at detecting cases of anxiety and depression, it does not provide psychiatric diagnoses. A second shortcoming was the limited response rate (47.1%) at baseline and that the attrition analysis showed that responders were older than non-responders. However, several studies have shown that, when exploring the relations between variables rather than estimates of a single population parameter, high attrition may not have a large impact on the results (Curtin et al. 2000; Keeter et al. 2006). Our sample was also representative of the general Swedish population on demographic variables, including insomnia prevalence and age. A final limitation was that we did not collect information about how the conditions varied over the whole 6-month timespan since we only asked the respondents about the month preceding the surveys.

For this reason, we can, for example, not be certain that persistent depression in the study is equivalent to a period of depression persistent from baseline to follow up. It is likely that variations took place between the time-points, which might have influenced the results of the current study. Future studies could include more frequent measures of sleep and psychiatric problems in order to establish how the symptomatology varied – or persisted – over time.

Conclusions

This study supports the hypothesis that incident insomnia is an independent risk factor for incident anxiety and depression as well as for persistent depression. The overall finding that insomnia is a dynamic marker for psychiatric conditions might have heuristic value for theory and clinical research.

Abbreviations

CBT-I: Cognitive-behavioral therapy for insomnia; CI: Confidence interval; DSM-IV: Diagnostic and Statistical Manual of Mental Disorders, 4th edition; DSM-IV-TR: Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision; HADS: Hospital Anxiety and Depression Scale; ISI: Insomnia Severity Index; M: Mean; OR: Odds ratio; SD: Standard deviation; T1: Time 1; T2: Time 2

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Authors' contributions

MJF, ANC and SJL designed the study and collected the data. MJ and MJF executed the statistical analysis and drafted the paper. MJF, MJ, ANC and SJL revised the manuscript and approved the final version.

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Availability of data and materials

The datasets during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

The study was approved by the Regional Ethics Board in Uppsala, Sweden (2008/157). All the study participants consented to participate.

Consent for publication

All listed authors have approved of the submission of the manuscript to the journal.

Competing interests

The authors declare that they have no conflict of interest.

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References

Baglioni C, Battagliese G, Feige B, Spiegelhalder K, Nissen C, Voderholzer U, et al. Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. J Affect Disord. 2011;135:10–9. https://doi.org/10.1016/j.jad.2011.01.011.

Bastien CH, Vallières A, Morin CM. Validation of the insomnia severity index as an outcome measure for insomnia research. Sleep Med. 2001;2:297–307. https://doi.org/10.1016/S1389-9457(00)00065-4.

Bjelland I, Dahl AA, Haug TT, Neckelmann D. The validity of the hospital anxiety and depression scale: an updated literature review. J Psychosom Res. 2002;52:69–77.

Chen PJ, Huang CLC, Weng SF, Wu MP, Ho CH, Wang JJ, et al. Relapse insomnia increases greater risk of anxiety and depression: evidence from a population-based 4-year cohort study. Sleep Med. 2017;38:122–9.

Christensen H, Batterham PJ, Gosling JA, Ritterband LM, Griffiths KM, Thorndike FP, et al. Effectiveness of an online insomnia program (SHUTi) for prevention of depressive episodes (the GoodNight study): a randomised controlled trial. Lancet Psychiatry. 2016;3:333–41. https://doi.org/10.1016/S2215-0366(15)00536-2.

Cunningham JE, Shapiro CM. Cognitive Behavioural therapy for insomnia (CBT-I) to treat depression: a systematic review. J Psychosom Res. 2018;106:1–12.

Curtin R, Presser S, Singer E. The effects of response rate changes on the index of consumer sentiment. Public Opin Q. 2000;64:413–28. https://doi.org/10.1086/318638.

Dew MA, Reynolds CF, Houck PR, Hall M, Buysse DJ, Frank E, Kupfer DJ. Temporal profiles of the course of depression during treatment: predictors of pathways toward recovery in the elderly. Arch Gen Psychiatry. 1997;54(11):1016–24.

Dolsen MR, Asarnow LD, Harvey AG. Insomnia as a transdiagnostic process in psychiatric disorders. Curr Psychiatry Rep. 2014;16(9):471.

Edinger JD, Bonnet MH, Bootzin RR, Doghramji K, Dorsey CM, Espie CA, et al. Derivation of research diagnostic criteria for insomnia: report of an American Academy of sleep medicine work group. Sleep. 2004;27:1567–96.

Edwards P, Roberts I, Clarke M, DiGuiseppi C, Pratap S, Wentz R, et al. Methods to increase response rates to postal questionnaires. Cochrane Database Syst Rev. 2007;2. https://doi.org/10.1002/14651858.MR000008.pub3.

Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders: an opportunity for prevention?. Jama. 1989;262:1479-84.

Gellis LA, Lichstein KL, Scarinci IC, Durrence HH, Taylor DJ, Bush AJ, Riedel BW. Socioeconomic status and insomnia. J Abnorm Psychol. 2005;114:111–8. https://doi.org/10.1037/0021-843X.114.1.111.

Harvey AG. A cognitive model of insomnia. Behav Res Ther. 2002;40:869–93. https://doi.org/10.1016/S0005-7967(01)00061-4.

Harvey AG, Murray G, Chandler RA, Soehner A. Sleep disturbance as transdiagnostic: consideration of neurobiological mechanisms. Clin Psychol Rev. 2011;31:225–35. https://doi.org/10.1016/j.cpr.2010.04.003.

Herrmann C. International experiences with the hospital anxiety and depression scale-a review of validation data and clinical results. J Psychosom Res. 1997;42:17–41. https://doi.org/10.1016/S0022-3999(96)00216-4.

Hertenstein E, Feige B, Gmeiner T, Kienzler C, Spiegelhalder K, Johann A, et al. Insomnia as a predictor of mental disorders: a systematic review and meta-analysis. Sleep Med Rev. 2019;43:96–105.

Jacobson NC, Newman MG. Anxiety and depression as bidirectional risk factors for one another: a meta-analysis of longitudinal studies. Psychol Bull. 2017;143:1155–200. https://doi.org/10.1037/bul0000111.

Jansson-Fröjmark M, Harvey AG, Norell-Clarke A, Linton SJ. Associations. Between psychological factors and nighttime/daytime symptomatology in insomnia. Cogn Behav Ther. 2012;41:273–87. https://doi.org/10.1080/16506073.2012.672454.

Jansson-Fröjmark M, Lindblom K. A bidirectional relationship between anxiety and depression, and insomnia? A prospective study in the general population. J Psychosom Res. 2008;64:443–9.

Jansson-Fröjmark M, Norell-Clarke A. Cognitive behavioural therapy for insomnia in psychiatric disorders. Curr Sleep Med Rep. 2016;2:233-240.

Keeter S, Kennedy C, Dimock M, Best J, Craighill P. Gauging the impact of growing nonresponse on estimates from a national RDD telephone survey. Public Opin Q. 2006;70:759–79. https://doi.org/10.1093/poq/nfl035.

Kim JM, Stewart R, Kim SW, Yang SJ, Shin IS, Yoon JS. Insomnia, depression, and physical disorders in late life: a 2-year longitudinal community study in Koreans. Sleep. 2009;32:1221–8.

LeBlanc M, Merette C, Savard J, Ivers H, Baillargeon L, Morin CM. Incidence and risk factors of insomnia in a population-based sample. Sleep. 2009;32:1027–37.
Li L, Wu C, Gan Y, Qu X, Lu Z. Insomnia and the risk of depression: a meta-analysis of prospective cohort studies. BMC Psychiatry. 2016;16(1):375.
Morin CM, Belleville G, Bélanger L, Ivers H. The insomnia severity index: psychometric indicators to detect insomnia cases and evaluate treatment response. Sleep. 2011;34:601–8.
Morin CM, LeBlanc M, Daley M, Gregoire JP, Merette C. Epidemiology of insomnia: prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. Sleep Med. 2006;7:123–30. https://doi.org/10.1016/j.sleep.2005.08.008.
Morin CM, LeBlanc M, Ivers H, Bélanger L, Mérette C, Savard J, Jarrin DC. Monthly fluctuations of insomnia symptoms in a population-based sample. Sleep. 2014;37(2):319–26.
Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. Psychol Aging. 1993;8(3):463.
Norell-Clarke A, Jansson-Fröjmark M, Tillfors M, Harvey AG, Linton SJ. Cognitive processes and their association with persistence and remission of insomnia: findings from a longitudinal study in the general population. Behav Res Ther. 2014;54:38–48.
Norell-Clarke A, Tillfors M, Jansson-Fröjmark M, Holländare F, Engström I. Does mid-treatment insomnia severity mediate between cognitive behavioural therapy for insomnia and post-treatment depression? An investigation in a sample with comorbid insomnia and depressive symptomatology. Behav Cogn Psychother. 2018;46:726–37. https://doi.org/10.1017/S1352465818000395.
Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev. 2002;6:97–111. https://doi.org/10.1053/smrv.2002.0186.
Peppard PE, Szklo-Coxe M, Hla KM, Young T. Longitudinal association of sleep-related breathing disorder and depression. Arch Intern Med. 2006;166:1709–15. https://doi.org/10.1001/archinte.166.16.1709.
Pigeon WR, Bishop TM, Krueger KM. Insomnia as a precipitating factor in new onset mental illness: a systematic review of recent findings. Curr Psychiatry Rep. 2017;19(8):44.
Pigeon WR, Hegel M, Unützer J, Fan MY, Sateia MJ, Lyness JM, et al. Is insomnia a perpetuating factor for late-life depression in the IMPACT cohort? Sleep. 2008;31:481–8.
Sivertsen B, Lallukka T, Salo P, Pallesen S, Hysing M, Krokstad S, Øverland S. Insomnia as a risk factor for ill health: results from the large population-based prospective HUNT study in Norway. J Sleep Res. 2014;23:124–32. https://doi.org/10.1111/jsr.12102.
Spoormaker VI, Verbeek I, van den Bout J, Klip EC. Initial validation of the SLEEP-50 questionnaire. Behav Sleep Med. 2005;3:227–46. https://doi.org/10.1207/s15402010bsm0304_4.
Tang NK, Harvey AG. Correcting distorted perception of sleep in insomnia: a novel behavioural experiment? Behav Res Ther. 2004;42(1):27–39.
Taylor DJ, Lichstein KL, Durrence HH. Insomnia as a health risk factor. Behav Sleep Med. 2003;1(4):227–47.
van Mill JG, Vogelzangs N, van Someren EJ, Hoogendijk WJ, Penninx BW. Sleep duration, but not insomnia, predicts the 2-year course of depressive andanxiety disorders. J Clin Psychiatry. 2014;75:119–26.
Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand. 1983;67:361–70. https://doi.org/10.1111/j.1600-0447.1983.tb09716.x.

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