The Bidirectional Association Between Depression and Insomnia: The HUNT Study

Borge Sivertsen, PhD, Paula Salo, PhD, Arne Myklebust, PhD, Mari Hysing, PhD, Ståle Pallesen, PhD, Steinar Krokpstad, PhD, Inger Hilde Nordhus, PhD, and Simon Overland, PhD

Objective: Depression and insomnia are closely linked, yet our understanding of their prospective relationships remains limited. The aim of the current study was to investigate the directionality of association between depression and insomnia. Methods: Data were collected from a prospective population-based study comprising the most recent waves of the Nord-Trøndelag Health Study (HUNT) (the HUNT2 in 1995–1997 and the HUNT3 in 2006–2008). A total of 24,715 persons provided valid responses on the relevant questionnaires from both surveys. Study outcomes were onset of depression or insomnia at HUNT3 in persons not reporting the other disorder in HUNT2. Results: Both insomnia and depression significantly predicted the onset of the other disorder. Participants who did not have depression in HUNT2 but who had insomnia in both HUNT2 and HUNT3 had an odds ratio (OR) of 6.2 of developing depression at HUNT3. Participants who did not have insomnia in HUNT2 but who had depression in both HUNT2 and HUNT3 had an OR of 6.7 of developing insomnia at HUNT3. ORs were only slightly attenuated when adjusting for potential confounding factors. Conclusions: The results support a bidirectional relationship between insomnia and depression. This finding stands in contrast to the previous studies, which have mainly focused on insomnia as a risk factor for the onset of depression. Key words: insomnia, depression, directionality, epidemiology, risk factors.

INTRODUCTION

Insomnia has historically been regarded as a symptom of depressive illness. Accordingly, sleep problems are included as a symptom of depression in both major diagnostic classification systems (1,2). Several studies have also shown that depression predicts new onset of insomnia symptoms (3,4). However, the simplistic view of insomnia as a secondary condition to a somatic or psychiatric problem has been challenged by a range of studies, particularly in the case of depression. A large body of evidence has, for example, demonstrated that insomnia is also a significant risk factor for the development of depression. In a recent meta-analysis of longitudinal studies on the topic, Baglioni et al. (5) concluded that nondepressed participants with insomnia have a two-fold risk of later depression, compared with those without insomnia. Insomnia has been found to predict the onset of depression across different age groups, although most studies found a stronger relationship in older adults (4.6–13) compared with both the general population (14–23) and young adults and adolescents (24–29). These studies suggest that insomnia may be prodromal to depression, one of the earlier symptoms of a depressive episode or an independent risk factor for depression.

The sequencing is relevant for how we understand the relationship. It is therefore a severe limitation that only a few studies have examined both potential directions of association. To our knowledge, only three previous studies have investigated the potential bidirectionality between depression and insomnia in the same study, with mixed findings. In a Swedish study of more than 3000 participants from the general population, insomnia symptoms at baseline significantly predicted the onset of depression 1 year later (odds ratio [OR] = 3.5), but the authors also found evidence for the opposite association: depression at baseline was related to subsequent symptoms of insomnia (OR = 2.3) (16). Similarly, a UK prospective study of 2363 adults found that depression predicted the incidence of insomnia at 1-year follow-up (risk ratio, 3.2) (18). However, those studies had limitations because the operationalizations of insomnia did not include daytime impairment, which is a specific insomnia criterion according to the DSM-IV. In one retrospective study of 1014 adolescents, Johnson et al. (30) found clinical insomnia to significantly predict the onset of depression (hazard ratio, 3.8), but no association between prior depression and the onset of DSM-IV insomnia was found. Thus, the present knowledge is not sufficient to draw conclusions concerning the directionality of association between insomnia and depression.

Against this background, we present data from a large prospective population-based study—the two most recent waves of the Nord-Trøndelag Health Study (HUNT2 and HUNT3) with the aim to investigate the bidirectional association between insomnia and depression.

MATERIALS AND METHODS

Participants

In all, 92,100 individuals aged 19 to 89 years received an initial questionnaire and a personal invitation to participate in HUNT2 (1995–1997). Of these, 65,648 individuals (71%) attended a physical examination, where they also
received a second set of questionnaires, which 52,814 individuals (80%) completed. Valid ratings on the variables for the current study were obtained from 43,045 participants (47% of the invited participants). In HUNT3 (2006–2008), 93,010 individuals aged 20 to 89 years were invited with the same procedure as in HUNT2, and 50,625 individuals (54%) attended. Of these, 49,797 individuals received a second set of questionnaires (including sleep questions), which 41,162 individuals (45%) completed. Because of the 11-year lag between the two surveys, participants aged between 20 and 31 years were excluded from the HUNT3, as were also participants older than 67 years in HUNT2 because of age retirement (because one of the insomnia questions assessed impaired work performance). Thus, the final data set for the current article comprised complete, repeatedly measured information from 24,715 individuals.

**Measures**

**Insomnia**

The DSM-IV criteria for insomnia include difficulty initiating or maintaining sleep or experiencing nonrestorative sleep for a period of no less than 1 month (1). In addition, it is a prerequisite that the individual experiences significant impairment in daily functioning. In the HUNT2 study, a proxy for the DSM-IV insomnia diagnosis was based on three items, encompassing persons endorsing sleep onset or terminal/late insomnia (unable to get back to sleep after an awakening) “often” or “almost every night” in the preceding month, in addition to reporting impaired work performance caused by insomnia during the preceding year. This operationalization has also been applied in previous studies (31).

The questionnaire in HUNT3 included the same insomnia items as in HUNT2, with the addition of one item assessing maintenance insomnia. In addition, daytime impairment caused by insomnia was assessed in terms of daytime sleepiness in HUNT3, rather than impaired work performance. The same categories and proxy procedures were used to define insomnia in HUNT3 as in HUNT2. Although not identical, both operationalizations closely resemble the Research Diagnostic Criteria (32) for insomnia syndrome, as specified by the DSM-IV. We also constructed a continuous variable of insomnia by adding the items included in the insomnia proxy.

**Depression**

The Hospital Anxiety and Depression Scale (33) (HADS) was used to assess self-reported symptoms of depression in both HUNT2 and HUNT3. The HADS questionnaire comprises 14 four-point Likert-scaled items, of which 7 are used to assess depression (HADS-D). No items concerning vegetative symptoms or sleep difficulties are included in the HADS. The most recent literature review, covering 31 studies, concluded that the HADS holds good case-finding properties for depression in patient populations in primary care and hospital settings (34). A cutoff score of 8 has been shown to give an optimal balance between sensitivity and specificity at approximately 0.80 for depression according to DSM-III and DSM-IV and the International Classification of Diseases, Eighth and Ninth Editions, and this cutoff has also shown excellent psychometric properties in identifying cases in general practice (35). For the present study, we used both dichotomous and continuous measures of depression.

Anxiety was also assessed using the HADS—anxiety subscale from the same instrument and included as a confounder in the logistic regression analyses.

**Somatic Diagnoses and Symptoms**

Physical health in HUNT2 was assessed in two ways: a) An index for somatic symptoms was computed as the sum of organ systems from which symptoms were reported. Included were questions pertaining to gastrointestinal symptoms (four questions on nausea, heartburn, diarrhea, and constipation), musculoskeletal symptoms (pain in the neck, shoulder, elbow, wrists, back [three areas], hips, knees, and ankles), the head (two questions on headache and migraine), the senses (two questions on hearing and sight), the heart (three areas), hips, knees, and ankles), the head (two questions on headaches and migraines), the senses (two questions on hearing and sight), the heart (one question on palpitations), and respiratory function (one question on respiratory problems). b) An index for self-reported somatic diagnoses comprised asthma, angina pectoris, stroke, myocardial infarction, diabetes, goiter, hypothyroid and hyperthyroid functions, other diseases of the thyroid gland, fibromyalgia, osteoporosis, arthritis, rheumatism, Bektcher disease, cancer, epilepsy, high blood pressure (being treated or monitored), and any other illness (one open item). Both indices were coded continuous by counting the number of symptoms/diagnoses.

**Sociodemographic and Life-Style Variables**

Data on age and sex at the time of the HUNT2 study were obtained from the national population registry. Self-reported educational level was coded as “primary,” “secondary,” or “college/university.” Educational level (three levels), daily cigarette smoking, and being physically active for 1 hour or more in the last week were assessed with self-report. Alcohol abuse was measured by the CAGE questionnaire (36), a four-question test widely used to identify problems with alcohol (score, 0–4). A recent study based on HUNT data indicated good concurrent validity and adequate psychometric properties of the CAGE (37). In the present study, a dichotomous variable was used with the recommended cutoff (two or more affirmative answers).

**Statistics**

IBM SPSS Statistics 20 for Mac (SPSS Inc, Chicago, IL) was used to run blockwise logistic regression analyses to examine the relationship between insomnia and depression at HUNT2 and the onset of depression/insomnia in HUNT3. All analyses were conducted on nondepressed participants or patients without insomnia at HUNT2. Both crude and adjusted analyses were conducted. In the adjusted model, the following covariates were selected based on previous studies showing an association with insomnia, depression, or both (38,39): age, sex, education, anxiety symptoms, somatic symptoms, somatic diagnoses, and life-style factors. They were all entered in the same block to the analyses. We also manually calculated the relative risk ratio, as recommended by Altman and Bland (40), to test for significant differences between the ORs. This is a well-established test of interaction to compare estimates on a log scale because it is not correct to assume that, when two confidence intervals (CIs) overlap, the two estimates are not significantly different (41).

As a sensitivity analysis, we also constructed two continuous variables reflecting insomnia and depression symptoms. Blockwise linear regression analyses were conducted on the standardized sum scores of insomnia and depression (HADS-D) for all participants. For example, depressive symptoms in HUNT2 were used to predict insomnia symptoms in HUNT3, while adjusting for depressive symptoms in HUNT2, and vice versa. Separate analyses were conducted to establish the cross-sectional associations.

**Ethics**

This study was approved by the regional committee for research ethics. After complete description of the study to the participants, written informed consent was obtained.

**RESULTS**

**Sample Characteristics**

In all, 24,715 persons provided valid responses on the relevant questionnaires in both the HUNT2 and HUNT3 studies. The mean ages in the two waves were 45.3 years (range, 19–67 years) and 56.1 years (range, 29–80 years) in HUNT2 and HUNT3, respectively. The sample included more women (56.9%) than men (43.1%). Twenty-five percent had education beyond high school level, and the sample was predominantly white.

**Prevalence and Course of Insomnia and Depression**

Of the persons participating in both HUNT waves, 5.1% \((n = 1268)\) met the criteria for insomnia in HUNT2, compared with 6.5% \((n = 1604)\) in HUNT3 11 years later. For chronicity, 19.2% of the individuals with insomnia in HUNT3 also met the criteria for insomnia 11 years earlier in HUNT2. For depression, 8.9% \((n = 2200)\) scored above the case-level cutoff.
<table>
<thead>
<tr>
<th>Variables</th>
<th>HUNT2 Total Sample</th>
<th>Positive Insomnia</th>
<th>Positive Depression</th>
<th>HUNT3 Total Sample</th>
<th>Positive Insomnia</th>
<th>Positive Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>45.3 (45.1–45.4)</td>
<td>48.1 (47.5–48.7)</td>
<td>&lt;.001</td>
<td>49.0 (48.6–49.4)</td>
<td>&lt;.001</td>
<td>56.1 (56.0–56.3)</td>
</tr>
<tr>
<td>Age, y</td>
<td>45.9 (56.3–57.5)</td>
<td>65.5 (62.8–68.1)</td>
<td>&lt;.001</td>
<td>54.0 (52.0–56.1)</td>
<td>.005</td>
<td>65.6 (63.3–68.0)</td>
</tr>
<tr>
<td>Women, %</td>
<td>24.6 (24.1–25.2)</td>
<td>22.5 (20.2–24.8)</td>
<td>.065</td>
<td>16.3 (14.7–17.8)</td>
<td>&lt;.001</td>
<td>25.6 (23.4–27.8)</td>
</tr>
<tr>
<td>Education beyond high school, %</td>
<td>5.1 (4.9–5.4)</td>
<td>—</td>
<td>—</td>
<td>17.9 (16.3–19.5)</td>
<td>&lt;.001</td>
<td>19.2 (17.3–21.1)</td>
</tr>
<tr>
<td>Depression prevalence, %</td>
<td>8.9 (8.6–9.3)</td>
<td>31.0 (28.4–33.5)</td>
<td>&lt;.001</td>
<td>3.3 (3.1–3.4)</td>
<td>&lt;.001</td>
<td>21.3 (19.3–23.3)</td>
</tr>
<tr>
<td>Anxiety prevalence, %</td>
<td>14.9 (14.5–15.4)</td>
<td>48.6 (45.8–51.3)</td>
<td>&lt;.001</td>
<td>3.2 (3.1–3.4)</td>
<td>&lt;.001</td>
<td>34.6 (32.2–36.9)</td>
</tr>
<tr>
<td>Somatic conditionsc</td>
<td>3.2 (3.2–3.2)</td>
<td>3.5 (3.4–3.6)</td>
<td>&lt;.001</td>
<td>3.2 (3.0–3.3)</td>
<td>&lt;.001</td>
<td>3.4 (3.1–3.4)</td>
</tr>
<tr>
<td>Daily smoking of cigarettes, %</td>
<td>1.8 (1.8–1.9)</td>
<td>4.1 (3.9–4.3)</td>
<td>&lt;.001</td>
<td>3.3 (3.1–3.4)</td>
<td>&lt;.001</td>
<td>3.3 (3.1–3.4)</td>
</tr>
<tr>
<td>Physically active ≥1 hr last week, %</td>
<td>75.9 (75.4–76.5)</td>
<td>69.6 (67.0–72.1)</td>
<td>&lt;.001</td>
<td>65.2 (63.2–67.2)</td>
<td>&lt;.001</td>
<td>72.8 (70.6–75.0)</td>
</tr>
<tr>
<td>Alcohol abuse prevalence, %</td>
<td>8.2 (7.9–8.6)</td>
<td>13.2 (11.1–15.3)</td>
<td>&lt;.001</td>
<td>12.2 (10.6–13.7)</td>
<td>&lt;.001</td>
<td>12.7 (11.0–14.5)</td>
</tr>
<tr>
<td></td>
<td>56.1 (56.0–56.3)</td>
<td>54.1 (53.5–54.7)</td>
<td>&lt;.001</td>
<td>58.5 (58.0–58.9)</td>
<td>&lt;.001</td>
<td>6.5 (6.2–6.8)</td>
</tr>
<tr>
<td>Insomnia prevalence, %</td>
<td>6.5 (6.2–6.8)</td>
<td>—</td>
<td>—</td>
<td>20.8 (19.2–22.5)</td>
<td>&lt;.001</td>
<td>—</td>
</tr>
<tr>
<td>Depression prevalence, %</td>
<td>9.3 (8.9–9.7)</td>
<td>29.8 (27.6–32.0)</td>
<td>&lt;.001</td>
<td>29.8 (27.6–32.0)</td>
<td>&lt;.001</td>
<td>—</td>
</tr>
</tbody>
</table>

HUNT = Nord-Trondelag Health Study.
Data are presented as mean or proportion (95% confidence intervals).

Men and women aged 19 to 67 years at baseline (HUNT2), n = 24,715.

Comparison of positive versus negative insomnia/depression.

Number of self-reported conditions/symptoms.
(HADS-D $\geq 8$) in HUNT2, compared with 9.3% ($n = 2297$) in HUNT3. Among participants who met the criteria for depression in HUNT3, 38% ($n = 876$) also already met the criteria for depression in HUNT2 (see Table 1 for details).

In cross-sectional associations, there was only a moderate level of overlap between insomnia and depression. Of the participants with insomnia in HUNT2, 31.0% ($n = 393$) also scored above the cutoff level for depression. Conversely, 17.9% of the participants with case-level depression also met the criteria for insomnia. A similar pattern was found in HUNT3, with 29.8% ($n = 478$) of the participants with insomnia also being depressed. In HUNT3, 20.8% of the participants with case-level depression met the criteria for insomnia.

Insomnia as a Risk Factor for Developing Depression

To examine the risk of depression onset in HUNT3, we conducted logistic regression analyses including participants below the case-level cutoff for depression in HUNT2. Chronic insomnia—defined as meeting the criteria for insomnia at both HUNT2 and HUNT3—yielded a six-fold increased risk of developing depression in HUNT3 (OR = 6.21, 95% CI = 4.45–8.68) adjusting for age, sex, and education (Table 2). This association was only slightly reduced when additional adjustments were made for somatic symptoms and diagnoses and life-style factors (OR = 5.67, 95% CI = 3.52–9.12). Further adjustment for anxiety reduced the OR to 4.35 (95% CI = 2.67–7.09) but remained significant. In comparison, nondepressed participants with insomnia in HUNT2, irrespective of insomnia status in HUNT3, had an OR of 2.38 (95% CI = 1.94–2.93) for depression onset in HUNT3. This association also remained significant when adjusting for somatic symptoms and diagnoses and life-style factors (OR = 1.66, 95% CI = 1.20–2.29) but not when further adjusting for anxiety (OR = 1.32, 95% CI = 0.95–1.83).

Depression as a Risk Factor for Developing Insomnia

The same approach was used to examine the effect of depression on the development of insomnia. Depression reported at both HUNT2 and HUNT3 (presumably chronic or recurrent depression symptoms) yielded a near seven-fold increased risk of developing insomnia in HUNT3 (OR = 6.71, 95% CI = 5.46–8.26) adjusting for age, sex, and education (Table 3). The OR was reduced to 4.96 (95% CI = 3.65–6.75) when adjusting for somatic symptoms, somatic diagnoses, and life-style factors. Further adjustment for anxiety only slightly reduced the association (OR = 4.34, 95% CI = 3.14–5.99). In comparison, participants with depression in HUNT2, irrespective of depression status in HUNT3, had an OR of 3.27 (95% CI = 2.80–3.82) for insomnia.

### Table 2. Insomnia at HUNT2 as a Risk Factor for the Onset of Depression at HUNT3

<table>
<thead>
<tr>
<th>Adjustment Variables</th>
<th>Insomnia Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HUNT2 Only(^c)</td>
</tr>
<tr>
<td>Age, sex, and education</td>
<td>2.00</td>
</tr>
<tr>
<td>Plus somatic symptoms, somatic diagnoses, and life-style factors</td>
<td>1.66</td>
</tr>
<tr>
<td>Plus anxiety</td>
<td>1.32</td>
</tr>
</tbody>
</table>

\(^a\) Men and women aged 19 to 67 years at baseline (HUNT2), $n = 24,715$.  
\(^b\) Regardless of insomnia status at HUNT3.  
\(^c\) Mutually exclusive groups.

### Table 3. Depression at HUNT2 as a Risk Factor for the Onset of Insomnia at HUNT3

<table>
<thead>
<tr>
<th>Adjustment Variables</th>
<th>Depression Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HUNT2 Only(^c)</td>
</tr>
<tr>
<td>Age, sex, and education</td>
<td>3.27</td>
</tr>
<tr>
<td>Plus somatic symptoms, somatic diagnoses, and life-style factors</td>
<td>2.10</td>
</tr>
<tr>
<td>Plus anxiety</td>
<td>1.72</td>
</tr>
</tbody>
</table>

\(^a\) Men and women aged 19 to 67 years at baseline (HUNT2), $n = 24,715$.  
\(^b\) Regardless of depression status at HUNT3.  
\(^c\) Mutually exclusive groups.
The present findings are in accordance with most of the existing studies concerning insomnia as a significant and independent risk factor for subsequent onset of depression. Although it is difficult to compare effect sizes across studies because of differences in age span, operationalization of exposure and outcome variables, follow-up time, and so on, our result that insomnia increases the risk of later depression by more than four times in the fully adjusted model is stronger than what previously has been reported in samples from the general adult population. In the first meta-analysis in this field, Baglioni et al. (5) found a fixed effect size of 2.1 (95% CI = 1.9–2.4) after excluding study outliers. The stronger effects in our study may be because of the fact that not all studies included in the meta-analysis used a proxy of insomnia including a daytime impairment criterion. It is only to be expected that insomnia symptoms are not as strongly associated with the onset of depression as insomnia syndrome, for which also a daytime criterion is included (1,32,42).

A novel finding of the present study is that depression was an equally strong risk factor for insomnia onset. Although several studies have linked depression with subsequent sleep problems (3,4), only three studies have focused on the bidirectional relationship. This is important given that different methods and samples may affect the findings.
Although Johnson et al. (30) found no association between depression and subsequent insomnia using a retrospective design, a few prospective studies have indicated that depression represents a two- to three-fold increased risk of later insomnia (16,18,43). Compared with our effect sizes of nearly 5 (adjusted model), the contrast between our findings and previous studies is again likely to stem from differences in the impact of insomnia symptoms versus syndrome. As such, the current study supports the bidirectional relationship between insomnia and depression. We conclude that the link does not predominately go from insomnia to depression but, in fact, is equally strong from depression to insomnia.

It should be noted that the effect of chronic insomnia/depression on the onset of the other disorder was only marginally stronger than the cross-sectional relationships at HUNT3, indicating a highly entwined relationship between the two disorders. However, the relatively low comorbidity between insomnia and depression—only 18% to 31% of the participants with one disorder also had the other—can be interpreted because these are also relatively separate entities. It can also be the case that participants with insomnia had coexisting subsyndromal depression (or vice versa), without fulfilling the diagnostic criteria for the other disorder. Firm conclusions about the relationship warrant longitudinal studies with multiple measurements. Still, our results challenge the predominant monotonic causal understanding where insomnia is regarded as a prequel to depression only.

Methodological Issues

This is the first study to explore the suggested bidirectional relationship between insomnia and depression using a prospective design in a large general population sample. A significant strength of the prospective design is that it makes empirical examination of directions of associations possible. The follow-up time between the two data collections was 11 years. The reason for this interval was mainly because the HUNT studies aim to follow the health of the county population approximately every decade, and consequently, the interval was not chosen specifically for the present study. The appropriate length of a follow-up depends on the causal mechanisms and processes between the entities involved; thus, the ideal time for studying the entwined relationship between insomnia and depression is not given. During 11 years, both insomnia and depression might fluctuate, but we have no information about this in the data from the HUNT studies. It has, for example, been suggested that insomnia may be a prodromal symptom of depression, although little is known concerning the time span involved (44). In previous studies, follow-ups have ranged from 12 months to 45 years (20). Although the magnitude of the associations in studies with different follow-ups does not seem to differ significantly (5), longer follow-ups generally require more careful consideration of potential confounding factors that are more likely to influence the outcome over time. In the current study, a rather long list of somatic conditions and complaints was therefore controlled for. Still, adjustments in the final analyses have most likely not captured and fully attenuated all possible confounding effects from chronic somatic or psychiatric conditions. If such diagnoses and symptoms are underreported, the reported relationships may in turn have been overestimated. However, it should be noted that the strong associations between insomnia and depression persisted also when adjusting for anxiety, in addition to the other confounders. Although this clearly involves a risk over overadjustment, these findings emphasize the strong and independent relationship between the two conditions. The participants were also given one open answer category for “other diseases,” this option is not sufficient to detect all relevant diseases and symptoms because it requires personal insight into psychiatric morbidity and syndromes. In addition, we have no information as to the severity of the eventual diseases. However, it is commonly held that severe psychopathology increases risk of nonattendance in epidemiologic studies (45), thus limiting the potential problem of other forms of psychopathology not being registered by the procedures used in the current study. The same mechanisms, allowed to operate twice over an 11-year gap, are likely to result in bias toward a healthier sample. This may result in underestimated prevalence rates but also fewer cases with both depression and insomnia.

Another limitation concerns the difference in the insomnia measures at HUNT2 and HUNT3. In HUNT2, the item for daytime impairment was “impaired work performance,” which leads to the exclusion of old retirees and other nonworking participants and a healthier sample as the result. In HUNT3, this item was changed to daytime sleepiness. Consequently, interpretations of potential differences in prevalence rates of insomnia between the two HUNT waves should be made with caution. Still, we consider it unlikely that these differences in operationalizations should have made a significant difference when investigating the longitudinal associations between insomnia and depression because there were no significant differences between the cross-sectional relationships of the two disorders at the two HUNT waves. Still, it should be noted that neither of these operationalizations included the full spectrum of daytime symptoms of insomnia, such as fatigue, irritability, reduced energy, concentration, memory complaints, and so on. This might have led to some biases in the identification of those with an insomnia syndrome and may also partly explain the moderate level of overlap between the two conditions at baseline.

In addition, depression was assessed by a self-report instrument—the HADS. The lack of clinical interview in confirming a clinical diagnosis of depression is an obvious limitation of the present study. In relation to this, the absence of sleep items or any other vegetative items in the HADS is both a limitation and an asset for the purpose of this study. In a previous publication examining the differential associations of insomnia and depression to medically certified work disability, the HADS was argued to be particularly suitable for this purpose (38). A conventional depression rating scale, including sleep problems as a symptom, would by definition increase circularity and make the interpretation of the results more ambiguous.

Another limitation is that we had no information on the history of previous insomnia or depression before the HUNT2 study. Because some participants may have had early onset of
these disorders during adolescence, there is a risk that we oversampled participants with onset during adulthood. However, Kessler et al. (46) have found that, by the age of 19 years, only 25% will have become depressed (major depressive disorder) of those who ever will. In addition, of those who will become depressed, only half of them do so by the age of 32 years. Still, the current findings should be interpreted with caution with regard to the generalizability to those who first experience clinical-level insomnia or depression beginning in adolescence.

Finally, it should be noted that our observations may relate to common third factors, influencing both insomnia and depression. For example, both insomnia and depressive symptoms increase with age (39,47), and because the insomnia-depression association was approximately equally strong in both directions, there may be common biologic mechanisms involved with increasing impact with higher age.

Clinical Implications

Notwithstanding the mentioned limitations, the key finding was a bidirectional relationship between insomnia and depression over a time span of 11 years. Although causal conclusions based on epidemiological data cannot be made, our results lend some support to previous studies indicating that treatment of the first-occurring disorder may prevent the development of the subsequent condition. Eaton et al. (48) estimated, for example, that eliminating sleep problems at baseline could prevent 47% of new cases with depression during the following year. In addition, studies have shown that treating insomnia with cognitive behavioral therapy indeed eliminates or considerably reduces the level of comorbid depression (49,50). If similar well-controlled studies confirm that early treatment of insomnia prevents the development of depression, this would be of great importance for general practice, where the most individuals with primary insomnia remain undetected and untreated (51). In addition, prevention of new disorders is important because comorbidity in general is associated with poorer prognosis, leaving perhaps both disorders more difficult to treat (52).

The focus in the literature has very much been on insomnia as a risk factor for the onset of depression, with the clinical implication being that treatment of insomnia has preventive value for depression. Bearing in mind the methodological limitations stemming from epidemiological data, our study may well support this perspective, as much as it is indicative evidence for the treatment of depression to prevent the onset of insomnia.

REFERENCES


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