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# The joint effect of sleep duration and insomnia symptoms on the risk of recurrent spinal pain: The HUNT study

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ABSTRACT

Although sleep quantity and quality appear to be interrelated, most previous studies have considered sleep duration and insomnia symptoms as distinct entities. We therefore examined whether there is a joint effect of sleep duration and long-term changes in insomnia symptoms on the risk of recurrent chronic spinal pain. We performed a prospective study of 8,788 participants who participated in three surveys over ~22 years and reported chronic spinal pain at the first, second, or both surveys. Adjusted risk ratios (RRs) were calculated for the risk of recurrent spinal pain at the last survey associated with self-reported sleep duration at the first survey and changes in insomnia symptoms between the two first surveys. Compared to participants with normal sleep duration (7–9 h) and no insomnia symptoms at the two first surveys, participants with insomnia symptoms over the same period had RRs of spinal pain of 1.33 (95% CI 1.26–1.41) in the last survey if they reported normal sleep duration and 1.50 (95% CI 1.34–1.67) if they reported short sleep (≤6 h). The corresponding RRs for spinal pain for participants who improved their sleep symptoms were 1.09 (95% CI 1.00–1.19) for those with normal sleep and 1.13 (95% CI 0.88–1.45) for those with short sleep. In conclusion, people who reported insomnia symptoms over ~10 years in combination with short sleep had a particularly increased risk of recurrent spinal pain. Improvement in insomnia symptoms was associated with a favorable prognosis.



## 1. Introduction

Chronic spinal pain is the leading cause of years lived with disability worldwide, and has vast impact on the society due to reduced work productivity and strain on the welfare system [1]. Despite extensive research on the management of chronic spinal pain, current treatment options are still not capable to fully reverse or hinder a negative progression of this condition [2,3]. This underscores the importance of gaining greater insight into modifiable factors that can improve the long-term prognosis of spinal pain.

More than 50% of people with chronic spinal pain suffer from insomnia symptoms [4–6], and these people often report greater pain intensity [7] and lower pain tolerance [8] along with lower probability of recovery [9,10]. However, these latter studies have assessed insomnia symptoms at one time point and have not considered whether sleep duration contributes to the progression of spinal pain. Although different dimensions of sleep, such as sleep duration and difficulty initiating or maintaining sleep have traditionally been considered as distinct entities, they are likely to be interrelated. This is supported by studies indicating that short and normal sleep duration in people with insomnia are attributed to distinct physiological characteristics [11] where insomnia co-occurring with short sleep duration represent a phenotype associated with the most adverse health outcomes [12]. No previous study has assessed whether specific combinations of variations in insomnia symptoms and sleep duration are associated with a particularly unfavorable prognosis of spinal pain.

It is recommended to use Cognitive Behavioural Therapy for Insomnia (CBT-I) as first-line treatment due to its long-term

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effectiveness [13]. CBT-I usually consists of sleep hygiene, stimulus control training, relaxation training, sleep restriction therapy and cognitive therapy [13]. However, the effect of CBT-I on pain outcomes are inconsistent, possibly explained by studies with insufficient power, short follow-up periods, or lack of CBT components for pain [14]. Increased knowledge about the long-term influence of different dimensions of sleep on the prognosis of spinal pain may be the first step to inform the development of interventions and whether certain phenotypes of insomnia should be specifically targeted. The aim of the current study was therefore to examine the individual and joint effect of sleep duration and ~10-year changes in insomnia symptoms on the risk of recurrent chronic spinal pain.

## 2. Method

### 2.1. Study population

All inhabitants aged 20 years or older in Trøndelag County, Norway, were invited to participate in the Trøndelag Health Study (the HUNT Study) carried out in 1995–97, 2006–08, and in 2017–19. The current study is based on data from 25,908 participants who participated in all three surveys. Of these, 8,788 participants reported chronic spinal pain in either 1995–97, 2006–08, or both, and responded to questions on insomnia symptoms and sleep duration (Fig. 1). Information on lifestyle and health-related factors were collected by questionnaires and clinical examination. Further information regarding the HUNT Study can be found at http://www.ntnu.edu/hunt [15,16].

The study was approved by the Regional Committee for Ethics in Medical Research in Central Norway (project no. 2014/612). The study was carried out according to the Declaration of Helsinki.

### 2.2. Self-reported sleep duration in 1995–97

Sleep duration was assessed by the question: “How many hours do you usually spend sleeping and/or napping during a day?”. We categorized sleep duration into ‘long’ (≥10 h), ‘normal’ (7–9 h), and ‘short’ (≤6 h). Since information on sleep duration was unavailable at the second survey, we could not assess change in sleep duration between the two first surveys.

### 2.3. Insomnia symptoms in 1995–97 and 2006-08

In the first survey, symptoms of insomnia were assessed by the following two questions: 1) "How often during the last month have you had difficulty falling asleep at night?", and 2) "How often during the last month have you woken too early and couldn’t get back to sleep?", with the response options ‘never’, ‘occasionally’, ‘often’, and ‘almost every night’. Participants were classified with insomnia symptoms if they answered ‘often/almost every night’ on one or both questions; otherwise they were classified as having no insomnia.

At the second survey, symptoms of insomnia were assessed by two insomnia questions from the sHUNT-Q sleep screening questionnaire [17] that also were available in 1995–97: 1) "How often during the last three months have you had difficulty falling asleep at night?" and 2) "How often during the last three months have you woken too early and couldn’t get back to sleep?", with each question having three response options ‘never/seldom’, ‘sometimes’, and ‘several times a week’. Participants were classified as having ‘insomnia symptoms’ if they answered ‘several times a week’ on at least one of the questions; otherwise they were classified as having ‘no insomnia symptoms’.

We used the information at the two first surveys to form four categories of change over time: 1) ‘no insomnia at both surveys’, 2) ‘changed from insomnia to no insomnia’, 3) ‘changed from no insomnia to insomnia’, and 4) ‘insomnia at both surveys’.

### 2.4. Chronic spinal pain

The questions on musculoskeletal pain were adopted from the Standard Nordic Questionnaire [18]. At the two first surveys, all participants were asked “During the last year, have you had *pain and/or stiffness* in your muscles and joints that lasted for at least three consecutive months?”. A similar question was given at the last survey, but with some minor adjustments: "During the last year, have you had *pain in your muscles and joints* that lasted for at least three consecutive months?". Participants who answered ‘yes’ to this question were asked to indicate the affected body area(s), including neck, shoulders, upper back, elbows, low back, hips, wrists/hands, knees, and ankles/feet. All participants who answered ’yes’ who also indicated ‘neck’, ‘upper back’, and/or ‘low back’ as affected body area(s) were defined to have chronic spinal pain. Considering the ~10 years gap between the surveys and that chronic pain may fluctuate over time [19], the participants may have had pain free periods between the surveys. Thus, participants who reported chronic spinal pain at the last survey were defined to have a recurrent episode of chronic spinal pain.

### 2.5. Assessment of covariates

All potential confounders were assessed at the second survey, except for i) education that was obtained at the first survey since this information was not collected in the second survey, and ii)

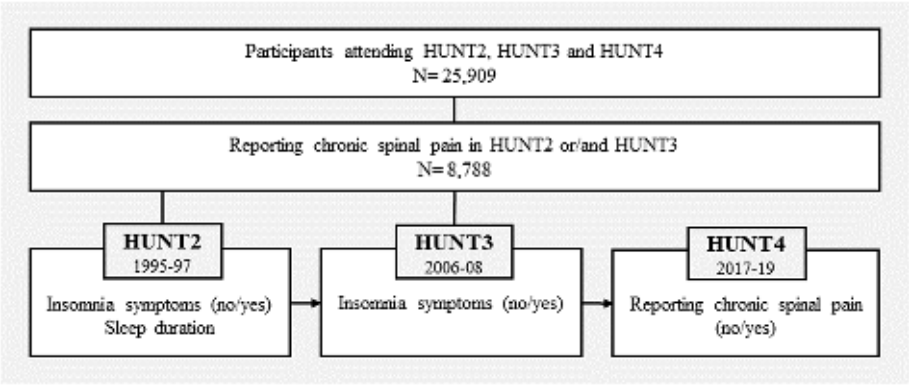


**Fig. 1.** Flow chart showing the selection of participants.

change in body weight between the two first surveys. Educational level was categorized into 'primary school', 'high school', 'college ≤4 years' and 'college >4 years'. The standardized measurements of height (to the nearest centimeter) and weight (to the nearest half kilogram) obtained at the clinical examinations were used to calculate body mass index (BMI) as weight divided by the square of height (kg/m2). Relative change in body weight was calculated as percentage change between the two first surveys. Leisure time physical activity was obtained from questions about frequency, duration and intensity, and participants were classified as 'inactive', 'below recommended' or 'recommended and above' according to public recommendations for physical activity at baseline. Shift work was assessed by the question "Do you work shifts, at night, or on call?", and categorized into 'no', 'yes', and 'unemployed/not working'. Smoking status was divided into five categories ('never smoked', 'former smoker', 'current low-intensity smoker (<10 cigarettes per day)', 'medium-intensity smoker (10–19 cigarettes per day)' and 'high-intensity smokers (20 or more cigarettes per day)'. Symptoms of anxiety and depression were assessed by the Hospital Anxiety and Depression Scale (HADS) with a cut-off score set to ≥8 on both anxiety and depression subscales [20]. Symptoms of anxiety and depression were then categorized into 'no anxiety and depression', 'possible anxiety', 'possible depression', and 'anxiety and depression'. To assess comorbid conditions, participants were asked if they have or have had heart disease, lung diseases, diabetes, or cancer (the response options for each question were 'no' and 'yes').

### 2.6. Statistical analysis

To estimate the relative risk directly and avoid overestimation of the odds ratio [21,22], a modified Poisson regression model was used to estimate risk ratios (RR) of recurrent chronic spinal pain at the last survey associated with sleep duration, changes in insomnia symptoms, and the joint effect of sleep duration at the first survey and change in insomnia symptoms between the two first surveys. The precision of the RRs was assessed by 95% confidence intervals (CIs). Participants who reported short (≤6 h), normal (7–9 h), or long (≥10 h) sleep duration at the first survey in combination with insomnia symptoms at both time points or changes in insomnia symptoms over time were compared with the reference group of participants without insomnia symptoms at both time points and normal sleep duration. All associations were adjusted for sex, age (continuous), BMI (continuous), leisure time physical activity, changes in BMI between the two first surveys, education, and smoking status. Since there is a potential overlap but unclear temporal association between insomnia symptoms and comorbid disorders [23,24], we repeated all the main analyses adjusting for anxiety and/or depression, use of pain medication, and other chronic conditions (heart disease, lung diseases, diabetes, cancer). Moreover, we repeated the analysis only including participants without chronic spinal pain in the first survey to examine if possible pain-induced insomnia symptoms at the first survey influenced the results.

All statistical analyses were performed using Stata for Windows, version 17.0 (StataCorp LP, College Station, Texas).

## 3. Results

Table 1 presents the baseline characteristics stratified by change in insomnia symptoms between the two first surveys. Of the 8,788 participants who participated in all three surveys, 62.5% reported no insomnia symptoms in both surveys; 6% improved their insomnia symptoms; 22.6% developed insomnia symptoms; and 8.8% had persistent insomnia symptoms. At follow-up in the last survey, 57.3% (5,038) reported recurrence of chronic spinal pain. In total, the mean age was 57 years; 64% were females; 22% had higher education; and 20% were current smokers. Compared with participants who were excluded due to missing information at follow-up in HUNT4, the responders tended to be younger (mean age, 57 vs 65), experienced more anxiety and/or depression (25% vs 18%) and a larger proportion experienced at least one comorbid disorder such as heart disease, asthma, chronic obstructive pulmonary disease, diabetes or cancer (40% vs 26%).

Table 2 shows that the RRs of recurrent chronic spinal pain were 1.09 (95% CI 1.01–1.18) for participants who improved their insomnia symptoms, 1.20 (95% CI 1.15–1.25) for participants who worsened their insomnia symptoms, and 1.33 (95% CI 1.26–1.40) for participants who reported insomnia symptoms at both surveys, compared to those without insomnia symptoms at both surveys. Compared to participants with normal sleep (7–9 h), the RRs for recurrent chronic spinal pain were 1.09 (95% CI 1.02–1.18) for those with short sleep (≤6 h) and 1.16 (95% CI 1.08–1.17) for those with long sleep duration (≥10 h).

Table 3 shows the joint effect of sleep duration at the first survey and change in insomnia symptoms between the two first surveys on the risk of recurrent spinal pain at the last survey. Compared to participants without insomnia symptoms at the two first surveys and normal sleep duration (7–9 h) at the first survey, the RRs for recurrent chronic spinal pain for the participants who reported insomnia symptoms over the same period were 1.33 (95% CI 1.26–1.41) for those with normal sleep duration and 1.50 (95% CI 1.34–1.67) for those with short sleep duration (≤6 h). Compared to the same reference category, those who reduced their insomnia symptoms over the same period had RRs of recurrent spinal pain of 1.09 (95% CI 1.00–1.19) and 1.13 (95% CI 0.88–1.45), respectively. In comparison, participants who slept ≥10 h had a RR of recurrent spinal pain of 1.21 (95% CI 1.11–1.31) if they had no insomnia symptoms at both surveys and a RR of 1.31 (95% CI 1.12–1.53) if they reported insomnia symptoms at the two first surveys.

### 3.1. Sensitivity analyses

Additional adjustments for anxiety and/or depression, use of pain medication, comorbid conditions, or physical work demands had negligible influence on the estimates (not reported). Likewise, similar findings were found when we repeated the analysis excluding participants with chronic spinal pain at the first survey, i.e., minimizing the potential influence of reverse causation on these associations.

## 4. Discussion

The current study shows that participants with insomnia symptoms over a ~10-year period have a higher risk of recurrent chronic spinal pain, and that this adverse effect seems to be particularly pronounced among those reporting short sleep. Although the observational design of the current study cannot verify the causal effect of insomnia symptoms and sleep duration on recurrent spinal pain, our results indicate that reducing insomnia symptoms may lead to a more favorable prognosis of chronic spinal pain.

Prospective studies show that insomnia influences the prognosis of chronic spinal pain [9,25–27]. We have in a recent study showed that an increase in number of insomnia symptoms is strongly associated with a poor long-term prognosis of chronic low back pain [9]. This is supported by a study showing that people who developed sleep problems had an increased risk of poor prognosis of low back pain, whereas those who improved their sleep had a more favorable prognosis [25]. The current study expands on these

**Table 1**
Characteristics of the study population stratified by change in insomnia symptoms from 1995–97 to 2006–08.

| | Insomnia symptoms reported in first (1995–97) and second (2006–08) survey | | | |
|---|---|---|---|---|
| | No insomnia symptoms in both surveys | Improved symptoms of insomnia | Developing symptoms of insomnia | Persistent insomnia symptoms |
| Participants, % (no.) | 62.5 (5,496) | 6.0 (531) | 22.6 (1,986) | 8.8 (775) |
| Age, mean (SD) | 56.0 (10.8) | 60.4 (11.0) | 56.3 (10.2) | 59.9 (9.9) |
| Females, % (no.) | 60.0 (3,295) | 64.2 (341) | 72.2 (1,434) | 72.8 (564) |
| Higher education, % (no.)[a] | 23.1 (1,271) | 18.5 (98) | 20.0 (397) | 16.9 (131) |
| Body mass index, mean (SD) | 27.4 (4.2) | 27.6 (4.4) | 27.7 (4.6) | 27.9 (4.7) |
| Percentage change in body mass, mean (SD)[b] | 4.7 (8.5) | 4.1 (9.0) | 5.4 (9.3) | 4.8 (9.7) |
| Moderate-to-high physical activity, % (no.)[c] | 51.9 (2,661) | 46.7 (230) | 52.5 (960) | 48.9 (339) |
| Current smoker, % (no.) | 18.8 (1,032) | 22.4 (119) | 20.2 (401) | 22.1 (171) |
| Shift work, % (no.) | 19.1 (1,047) | 10.9 (58) | 19.4 (385) | 15.7 (121) |
| Anxiety and/or depression, % (no.)[d] | 14.4 (794) | 23.7 (126) | 31.6 (627) | 40.0 (307) |
| Heart disease, % (no.) | 4.8 (263) | 7.7 (41) | 5.6 (111) | 7.2 (56) |
| Lung disease, % (no.) | 12.8 (701) | 19.6 (104) | 15.5 (307) | 17.6 (136) |
| Cancer, % (no.) | 5.0 (276) | 7.3 (39) | 6.1 (121) | 7.4 (57) |
| Self-reported sleep duration, mean (SD)[e] | 7.8 (1.1) | 7.9 (1.4) | 7.8 (1.2) | 7.7 (1.3) |

Abbreviations: SD, standard deviation.
[a] College or higher in 1995–97.
[b] Percentage change from 1995–97 to 2006-08.
[c] At least 150 min/week of moderate intensity physical activity or at least 60 min/week of vigorous intensity physical activity.
[d] Measured by Hospital Anxiety and Depression Scale score ≥8.
[e] Self-reported sleep duration in 1995–97.

**Table 2**
Risk of recurrent spinal pain in 2017–19 associated with sleep duration in 1995–97 and change in insomnia symptoms from 1995–97 to 2006-08.

| Insomnia symptoms and sleep duration | | No. of persons | Recurrent spinal pain in 2017–19 | Crude absolute risk (%) | Age-adjusted, RR[a] | Multi-adjusted, RR (95% CI)[b] |
|---|---|---|---|---|---|---|
| First survey (1995–97) | Second survey (2006–08) | | | | | |
| Sleep duration | | | | | | |
| 7–9 h | N/A | 7,634 | 4,302 | 56.4 | 1.00 | 1.00 (reference) |
| ≥10 h | N/A | 581 | 381 | 65.6 | 1.20 | 1.16 (1.08–1.17) |
| ≤6 h | N/A | 573 | 355 | 62.0 | 1.10 | 1.09 (1.02–1.18) |
| Insomnia symptoms | | | | | | |
| No | No | 5,496 | 2,897 | 52.7 | 1.00 | 1.00 (reference) |
| Yes | No | 531 | 304 | 57.3 | 1.11 | 1.09 (1.01–1.18) |
| No | Yes | 1,986 | 1,287 | 64.8 | 1.23 | 1.20 (1.15–1.25) |
| Yes | Yes | 775 | 550 | 71.0 | 1.38 | 1.33 (1.26–1.40) |

Abbreviations: CI, confidence interval; RR, risk ratio.
[a] Adjusted for age (continuous).
[b] Adjusted for age (continuous), sex (women, men), education (primary school, high school, college ≤4 years, college >4 years), body mass index (continuous), changes in body weight (continuous), leisure time physical activity (inactive, low activity, moderate-to-high), and smoking (never smoked, former smoker, current low-intensity smoker [<10 cigarettes per day], medium-intensity smoker [10–19 cigarettes per day] and high-intensity smokers [20 or more cigarettes per day]).

previous findings by investigating the joint effect of sleep duration and long-term changes in insomnia symptoms on the progression of spinal pain. Our study lends further support to the notion that insomnia with short sleep duration represents a severe phenotype of insomnia associated with increased risk of adverse health outcomes [11]. Our findings are important as they indicate that insomnia symptoms accompanied by short sleep is a phenotype that needs particular attention in people with chronic spinal pain. Sleep duration may be an index of the biological severity of insomnia [11]. The biological mechanism explaining this finding could be related to sustained activation of the stress system [11]. In contrast to insomniacs with normal sleep duration, insomniacs with short sleep duration may have greater levels of physiologically and cognitive-emotionally hyperarousal due to a sustained excessive activation of the hypothalamic-pituitary-adrenal axis and sympathetic nervous system [11]. Long-term disturbed sleep may also alter endogenous pain modulatory pathways known to increase the vulnerability to central sensitization and persistent pain [28], and induce pro-inflammatory responses [29] and stimulate cortical arousal [30] that may act as pain-inducing or pain-facilitating mechanisms [31].

Importantly, the current study assessed the influence of both improving and worsening insomnia symptoms over a 10-year period. Compared to participants without insomnia symptoms and normal sleep duration, the risk of reporting chronic spinal pain was only 13% greater among the short sleepers who reduced their insomnia symptoms. In comparison, short sleeper who increased their insomnia symptoms had 21% higher risk of recurrent spinal pain at the last survey. Similar estimates were found within the category of normal sleep duration for participants who reduced or increased their insomnia symptoms. In contrast, improvement of insomnia symptoms was not clearly associated with lower risk of recurrent spinal pain among the long sleepers (≥10 h). Sleep disturbances are common among people with long sleep duration [32,33] and long sleep duration is not proportionally related to the amount of deep sleep [34]. It is therefore possible that long sleep

**Table 3**
Risk of recurrent spinal pain in 2017–19 associated with the joint effect of sleep duration in 1995–97 and change in insomnia symptoms from 1995–97 to 2006-08.

| Insomnia symptoms stratified by sleep duration | | No. of persons | Recurrent spinal pain in 2017–19 | Crude absolute risk (%) | Age-adjusted, RR[a] | Multi-adjusted, RR (95% CI)[b] |
|---|---|---|---|---|---|---|
| First survey (1995–97) | Second survey (2006–08) | | | | | |
| *7-9h sleep* | | | | | | |
| No insomnia | No insomnia | 4,858 | 2,512 | 51.7 | 1.00 | 1.00 (reference) |
| Insomnia | No insomnia | 432 | 242 | 56.0 | 1.11 | 1.09 (1.00–1.19) |
| No insomnia | Insomnia | 1,740 | 1,124 | 65.6 | 1.25 | 1.22 (1.16–1.27) |
| Insomnia | Insomnia | 604 | 424 | 70.2 | 1.39 | 1.33 (1.26–1.41) |
| *≥10h sleep* | | | | | | |
| No insomnia | No insomnia | 323 | 206 | 63.8 | 1.24 | 1.21 (1.11–1.31) |
| Insomnia | No insomnia | 53 | 36 | 67.9 | 1.36 | 1.28 (1.06–1.54) |
| No insomnia | Insomnia | 123 | 84 | 68.3 | 1.32 | 1.26 (1.12–1.42) |
| Insomnia | Insomnia | 82 | 55 | 67.1 | 1.36 | 1.31 (1.12–1.53) |
| *≤6h sleep* | | | | | | |
| No insomnia | No insomnia | 315 | 179 | 56.8 | 1.09 | 1.09 (0.99–1.20) |
| Insomnia | No insomnia | 46 | 26 | 56.5 | 1.13 | 1.13 (0.88–1.45) |
| No insomnia | Insomnia | 123 | 79 | 64.2 | 1.24 | 1.21 (1.05–1.39) |
| Insomnia | Insomnia | 89 | 71 | 79.8 | 1.57 | 1.50 (1.34–1.67) |

Abbreviations: CI, confidence interval; RR, risk ratio.
[a] Adjusted for age (continuous).
[b] Adjusted for age (continuous), sex (women, men), education (primary school, high school, college ≤4 years, college >4 years), body mass index (continuous), changes in body weight (continuous), leisure time physical activity (inactive, low activity, moderate-to-high), and smoking (never smoked, former smoker, current low-intensity smoker [<10 cigarettes per day], medium-intensity smoker [10–19 cigarettes per day] and high-intensity smokers [20 or more cigarettes per day]).

duration is a proxy for poor sleep continuity or light sleep [35]. It could also reflect poorer physical and mental health among the long sleepers (e.g., depression, underlying disease process) compared to people with normal sleep duration [35], which may lead to poorer back pain prognosis [36,37]. However, our results remained partially unchanged when we adjusted for anxiety and/or depression or comorbid conditions such as heart disease, lung diseases, diabetes, or cancer.

### 4.1. Implications of findings

The increased risk of recurrent spinal pain among people who reported both long-term insomnia symptoms and short sleep indicate that these dimensions should be considered in clinical assessment and treatment planning for chronic spinal pain. This is supported by the result showing the absolute risk difference of recurrent spinal pain was ~28% when comparing people with long-term insomnia symptoms and short sleep (crude absolute risk ~80%) with people without insomnia symptoms and normal sleep duration (crude absolute risk ~52%). It is consensus that pain management should target both physical, psychological, and social modalities [38,39]. However, despite the increasing evidence of a link between insomnia and poor pain prognosis [9,25], sleep is not emphasized as a specific target for pain management in current clinical guidelines or recommendations for managing or preventing recurrence of spinal pain [38–41]. Our findings highlight the importance of a nuanced assessment of sleep behavior in the management of chronic spinal pain, which can be easily implemented as a part of the initial screening and examination of persons with chronic spinal pain. CBT-I has been recommended as first-line treatment for patients with insomnia due to its long-term effectiveness [13], and some evidence indicates that this treatment can be effective also for chronic pain populations [14]. Future intervention studies should aim to investigate whether targeting sleep in management of chronic spinal pain leads to better prognosis for this condition.

### 4.2. Strengths and limitations

The strengths of the current study include the large study population and the prospective design, which enabled us to analyze changes in insomnia symptoms in combination with sleep duration. Moreover, we had the possibility to adjust for several potential confounding factors and conduct several sensitivity analyses. However, we cannot rule out that residual confounding due to unknown or unmeasured factors (e.g., genetics or familial factors) associated with both sleep and chronic pain influenced our results [42–44].

Some limitations must be mentioned. Sleep duration was only measured in the first survey, the symptoms of insomnia were assessed by different questions in the first and second survey, and our definition of insomnia is not aligned with the International Classification of Sleep disorders [45]. We have previously shown that there is a dose-response relationship between number of insomnia symptoms and risk of chronic musculoskeletal pain [46], and that people with insomnia symptoms in all parts of the sleep period have the highest risk of chronic spinal pain among people with insomnia [47]. The results in the present study could therefore be related to specific symptoms or severity of the insomnia disorder. The assessment period for insomnia symptoms in the two surveys were 'the last month' and 'the last three months' in 1995–97 and 2006–08, respectively, and we cannot be sure that this represents the habitual sleep pattern. Similarly, the assessment period for HADS was only 'the last two weeks'. Furthermore, we have no information on fluctuation in insomnia symptoms or other health-related measures between the two surveys; when or why the change in symptoms occurred or if this change was related to changes in pain status. Due to the bidirectional relation between sleep problems and chronic pain [48,49], we cannot exclude the possibility that insomnia symptoms measured at the first and second survey are influenced by acute or chronic pain in the same period or vice versa, potentially overestimating the association with risk of recurrent chronic spinal pain at follow-up. However, when performing a sensitivity analysis excluding participants who reported pain at the first survey to assess risk of recurrent spinal pain among participants without pain-induced insomnia symptoms at the second survey, the results remained largely unchanged. It should also be noted that our study comprises those who participated in three consecutive surveys for more than two decades, which resulted in a particularly healthy study population. In view of this, it is possible that selection into the study, or that we conditioned on sleep duration in our analysis of

joint effect, caused a collider-stratification bias that biased or reversed the association between insomnia symptoms and recurrent spinal pain. Unfortunately, we had no information about why participants dropped out of the study (e.g., disease, death, or other reasons). Finally, the change from no insomnia symptoms in the first survey to insomnia symptoms in the second survey could be mediated through pre-clinical illness [23,24]. Thus, we cannot rule out that the self-reported measure of chronic spinal pain is solely due to musculoskeletal pain or other somatic conditions. However, the results remained largely similar after performing sensitivity analyses adjusted for anxiety and/or depression and other chronic conditions (heart disease, lung diseases, diabetes, cancer).

In conclusion, this study shows that participants reporting short sleep combined with ~10-year insomnia symptoms have a particularly higher risk of recurrent chronic spinal pain, compared to those with normal sleep duration and no insomnia symptoms. Although the causal effect of sleep quantity and quality cannot be clearly established in this study, the result indicates that a reduction in insomnia symptoms is associated with a more favorable prognosis of chronic spinal pain.

## CRediT authorship contribution statement

**Anne Lovise Nordstoga:** study conception and design, The first draft of the manuscript was written, commented on previous versions of the manuscript. **Paul Jarle Mork:** study conception and design, commented on previous versions of the manuscript. **Ingebrigt Meisingset:** study conception and design, commented on previous versions of the manuscript. **Tom Ivar Lund Nilsen:** study conception and design, commented on previous versions of the manuscript. **Eivind Schjelderup Skarpsno:** study conception and design, Data, Formal analysis, were performed, The first draft of the manuscript was written, commented on previous versions of the manuscript, All authors read and approved the final manuscript.

## Declaration of competing interest

None.

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