

RESEARCH ARTICLE

# Does physical activity buffer insomnia due to back and neck pain?

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## Abstract

### Introduction

Musculoskeletal pain is highly prevalent and a burden to society, recurrent and persistent low back pain (LBP) and neck pain (NP) being the most common conditions. They are associated with other poor health outcomes such as sleep problems.

Physical activity (PA) prevents LBP and NP, but the direct effect on sleep is unclear. This study explored the effect of pain on insomnia, and examined if adherence to moderate-to-high intensity levels of PA influenced this relationship.

### Methods

In this prospective observational study, 1821 workers were followed over 3 years. Data included self-rated measures of LBP and NP, insomnia and level and amount of PA. Pain variables were used in a “risk profile” for future sick-listing, insomnia was categorized into those with and without such problems, and adherence to PA was defined as reporting moderate-to-high levels in two consecutive years.

In Poisson regression models, individuals with pain risk profiles were analysed according to PA adherence for the outcome insomnia. Repeated measurements allowed control for prior pain.

### Results

In this mainly male working population, individuals with a risk profile for LBP and NP had a significant increased risk (RR = 1.5) of developing insomnia one year later when not adhering to moderate-to-high levels of PA. Among those not reporting prior pain, the risk was even larger (RR = 2.5).

Generalizability may be restricted to relatively healthy males. The individuals who reported a pain risk profile two consecutive years did not get the buffer effect from adhering to moderate-to-high levels of PA in terms of developing insomnia.

## OPEN ACCESS

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**Data Availability Statement:** Data are from the AHA study whose authors may be contacted at the Unit of Intervention and Implementation Research, Institute of Environmental Medicine, Karolinska Institutet. This is due to ethics regulations in Sweden, that are very restrictive in terms of disclosing personal data. Even though each subject would have an identifying ID (no name or social security number), some identification is still possible in principle as we have disclosed the time frame, the work situation and the industries involved. As such, the Swedish authorities would require us to seek additional permission in order to

publish data at this stage. In new projects/studies, this may be applied for when seeking ethical permission, but these data are about 15 years old, and no such permission was sought. Interested, qualified researchers may send data requests to Henrik Jonsson at [henrik.jonsson@ki.se](mailto:henrik.jonsson@ki.se).

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## Introduction

Musculoskeletal pain is a global health burden, the major cause of years lived with disability [1]. This is due to a high prevalence and a high level of recurrence and persistence [2, 3]. On the societal level, the burden is economic and relates to direct costs associated with health care, as well as indirect costs for disability pensions [4, 5]. From the employer level, musculoskeletal pain leads to decreased work productivity, sickness presentism and absenteeism, all are costly for the company [6].

For the individual, the numerous effects are well documented. Aside from decreased work ability and possibly sick leave, pain also affects health in general [7]. People with persistent pain report poor general health [8], comorbidity [9] and poor sleep [10]. Thus, these individuals are caught in a cycle of negative health outcomes, one problem adding insult to the other [9]. As mentioned, musculoskeletal pain tends to be persistent, which means that the individual needs to develop strategies to manage their pain [11]. Preferably, preventive measures should be implemented to minimize recurrence and the level of interference with everyday life.

Sleep has consistently been found to be affected among those suffering from musculoskeletal pain. Reports of decreased sleep quality [12] and disruption of sleep [12] concern patients with low back pain (LBP), neck pain (NP), headache and jaw pain. In a previous prospective study of self-reported bothersome LBP and decreased sleep quality, pain predicted disturbed sleep the following week [13]. In a study with an 11 year follow up, headache and musculoskeletal pain was found to predict insomnia [14]. Indeed, musculoskeletal pain and insomnia seem to be co-existing [15]. A recent critical review suggested that the relationship between pain and sleep may be very complex involving neurotransmitters, psychological affect and sociodemographic factors [16].

The only factor that has been consistently shown to prevent LBP and NP, is physical activity (PA) [17]. However, PA is typically a “perishable” entity [18, 19]; it needs to be performed on a regular basis for the beneficial effects to take place. In today’s society, people are becoming more and more sedentary, and much of the “normal” everyday PA is lost [20]. Further, a recent study found that one of the consequences of LBP is physical inactivity [21], adding further insult to the pain problem. However, a systematic review concluded that the direct effect of exercise on sleep is unclear [22].

In this study, the prospective relationship between LBP, NP and insomnia was explored, and the effect of PA on this relationship. It was a secondary analysis of existing data from a workplace inventory concerning workers’ health, lifestyle and productivity. The organisations that supplied data were large Swedish industrial companies, and data existed from 1821 employees measured at three time points, in 2000, 2001–2 and 2003. The aim of the study was to explore the relationship between LBP/NP and insomnia, and to investigate the influence of adhering to moderate-to-high levels of PA on this relationship.

Specific hypotheses were

1. Future insomnia is predicted by LBP and NP.
2. PA influences this relationship; in such a way that individuals with LBP/NP who adhere to moderate-to-high levels of PA over time experience less insomnia compared to individuals who do not adhere.

## Materials and method

### Design

The data were collected as part of a work place intervention study (the AHA study) in 2000 (T1), 2001–2 (T2) and 2003 (T3) in private companies in Sweden. The methodology has been

described in detail elsewhere [23]. In short, the inventory consisted of a comprehensive validated individual questionnaire about health and work conditions which was sent by mail to all employees, regardless of their work-status (working full- or part-time, being on parental leave or sick leave). The survey resulted in a personal profile for each participant as well as company profiles, both of which highlighted areas with room for improvement concerning health (individual level) and work-environment (organisational level) [24]. The individuals with a risk profile concerning health issues were recommended to seek advice from their occupational health service, and the companies were directed towards improvements in the work environment and leadership if productivity could be improved. After a period of 18 months, the questionnaire was repeated, the improvement was evaluated, and another round of measures towards individual health and productivity was started. After an additional 18 months, the process was repeated a third time. In the original study, the AHA method was shown to improve workers' health and the companies' productivity [23, 25].

## Variables

Demographic and health variables were collected as part of the AHA questionnaire, such as insomnia, the presence and consequences of LBP and NP and patterns of PA.

Concerning sleep, three questions concerning insomnia from the Karolinska Institutet Sleep Questionnaire [26] were asked; about difficulties in falling asleep, in waking up and about having difficulty going back to sleep after waking up. Insomnia is defined by the National Sleep Foundation as “difficulty falling asleep, difficulty staying asleep, waking up too early, and/or having sleep that is not refreshing” [27]. Based on previous research [23], this variable was summarized into three categories: sleep interference on all three dimensions (“severe insomnia”) to no sleep interference on all three dimensions (“no insomnia”) with an intermediate category with “moderate insomnia”.

Questions concerning musculoskeletal pain were based on von Korff et al. [28], and asked about present and past year LBP and NP and present and past year sickness absence in relation to such pain. From these questions, it was possible to create a “high risk pain profile”—a worker with several past episodes and present pain and sick leave more than once the previous year or ongoing for more than 2 months. In a previous study, these individuals were found to be at risk for future sick-leave due to LBP and NP [29]. Likewise, a “no risk pain profile” worker would report no past or recurrent LBP/NP and no long period or repeated sick leave. An intermediate category consisted of individuals with some, but not all, risk factors present [23].

Questions concerning PA were from the modified Swedish “LIV 90” questionnaire [30] and asked about the frequency of performing more than 30 minutes of high, medium and low intensity exercise. From these questions three PA groups were created; those reporting moderate or high intensity exercise more than twice a week or both high/medium intensity exercise once a week were the “high PA” group, those reporting high or medium at a lower frequency were “medium PA”, and those reporting high or medium intensity PA irregularly or never, were termed “low PA”.

Self-Rated Health was measured with a single item question from the Short Form-Questionnaire: “How would you rate your general health?” with five answer options ranging from excellent to poor [31].

## Data analysis

By examining the dependant variable “insomnia”, it was noted that very few individuals reported severe insomnia. As a result, the variable was dichotomized into those without vs those with insomnia (which included the categories moderate and severe). To exclude

**Table 1. Model 1: Examining the effect of PA adherence on subjects with LBP/NP risk profiles at T2 on the risk of developing insomnia at T3, regardless of previous pain at T1 (n = 436).**

TIME	T1	T2		T3
RISK OF LBP & NP	All subjects, both yes/no	Yes	PA adherence T2–T3	Insomnia
INSOMNIA	No	No		

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individuals with persistent insomnia, we chose to examine only those individuals without any “previous” insomnia, at baseline (T1 and T2) (Model 1). This amounted to 436 individuals. As a next step, this group was dichotomized into those with a pain risk profile also at T1 (Model 2, n = 347) and those that did not (Model 3, n = 85).

The independent variable LBP/NP risk profile classified very few subjects in the “high” risk category, and this group was collapsed with the “medium” risk category into a dichotomized index with risk/no risk of LBP/NP.

The independent variable PA was introduced in the model as “PA adherence”. This is a conceptualization of maintaining PA levels over time. Thus, if individuals started reporting moderate or high PA levels at T2 and maintained one of these levels at T3 (individuals with high PA levels could decrease their PA to moderate, but individuals with moderate PA levels needed to maintain this or increase to high PA levels) and individuals who started reporting low PA levels at T2 but increased to moderate or high PA levels at T3 were categorized as having “PA adherence”. All individuals who reported low levels of PA at T3 were classified as “PA non-adherence”.

In regression models, individuals with LBP and NP risk profiles at T2 were analysed according to adherence to their levels of PA (between T2 and T3) for insomnia at T3. Due to the repeated measurements, it was possible to control for prior pain (as reported at T1). Poisson regression was chosen to get estimates of risk ratios. Self-Rated Health was introduced as a covariate in the models.

We explored the following models (Tables 1–3):

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**Table 2. Model 2: Examining the effect of PA adherence on subjects with LBP/NP risk profiles that also reported pain at T1, on the risk of developing insomnia (n = 347).**

TIME	T1	T2		T3
RISK OF LBP & NP	Yes	Yes	PA adherence T2–T3	Insomnia
INSOMNIA	No	No		

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**Table 3. Model 3: Examining the effect of PA adherence on subjects with LBP/NP risk profiles that did not report pain at T1, on the risk of developing insomnia (n = 85).**

TIME	T1	T2		T3
RISK OF LBP & NP	No	Yes	PA adherence T2–T3	Insomnia
INSOMNIA	No	No		

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## Results

Demographic variables of 1821 workers who participated at all three measurements were available for secondary analysis. Of these, subgroups of individuals who did not report insomnia at

**Table 4. Baseline information about the source population and the selected subgroups.**

	Full study n = 1821	Model 1 n = 451	Model 2 n = 361	Model 3 n = 86
<b>Age mean (SD)</b>	43 (10.12)	44 (10.08)	43 (9.91)	44 (10.86)
<b>Males</b>	87.5%	90.2%	89.2%	94.2%
<b>Education</b>				
University-tertiary	9.5%	8.7%	9.0%	8.1%
Secondary	58.1%	58.2%	59.7%	53.5%
Primary	32.4%	33.1%	31.4%	38.4%
<b>Children at home</b>				
0	31.1%	33.0%	30.6%	43.1%
1–2	55.7%	52.8%	54.5%	47.1%
3 or more	14.2%	14.2%	14.9%	9.8%
<b>Work hours/week</b>				
1–20	2.3%	1.1%	1.4%	0%
21–30	2.5%	1.4%	1.0%	3.5%
31–40	63.8%	63.2%	64.6%	57.0%
41 +	31.4%	36.8%	33.1%	39.5%
<b>BMI</b>				
< 24.9- normal	39.3%	39.6%	40.8%	34.9%
25–29.9-overweight	47.4%	47.0%	48.1%	42.4%
30+ obese	13.3%	13.4%	11.1%	22.4%
<b>LBP/NP risk profile</b>	n = 1792			
High + Moderate	1249–69%			
No risk	543–31%			
<b>PA levels</b>	T1	T2	T2	T2
High	827–45%	244–55%	191–54%	51–60%
Moderate	190–10%	47–11%	40–11%	7–8%
Low	794–44%	153–34%	124–35%	27–32%
<b>PA adherence between T2 and T3</b>				
Non-adherence		164 (38%)	125 (36%)	36 (42%)
Adherence		272 (62%)	222 (64%)	49 (58%)
<b>Insomnia</b>				
Severe	147–8%			
Moderate	657–36%			
No	989–55%			
<b>Self-Rated Health</b>		T2	T2	T2
Excellent	236–13%	67–15%	38–11%	28–33%
Very Good	607–33%	187–42%	150–42%	37–43%
Good	705–39%	167–37%	145–40%	20–23%
All right	234–13%	29–6%	27–7%	1–1%
Poor	37–2%	1–0.3%	1–0.3%	

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the initial measurements in T1 and T2 and who reported a LBP and NP risk profile at T2 formed the study sample in Model 1. This subsample was further divided according to pain at T1 into Model 2 (pain risk profile at T1) and Model 3 (no pain risk profile at T1).

The profiles of these individuals are described in Table 4. As mentioned, these were workers from private, mainly industrial companies, which is reflected in the high proportion of males (around 90%) and also in the high proportion of subjects with primary and secondary schooling (around 90%). A majority worked full time (40 hours a week in Sweden), but a fair proportion (over 30%) worked more than that. A relatively high proportion reported moderate-to-high PA levels in all the subsamples. About two-thirds of the cohort adhered to moderate-to-high PA levels during the course of the study, from T2 to T3. Self-Rated Health was higher in

**Table 5. The risk ratios of three models investigating the risk of reporting future insomnia among individuals with a LBP/NP risk profile. \* = statistical significance.**

	PA adherence T2–T3	RR (CI) of Reporting insomnia at T3	p-value
Model 1 (n = 436)	Non-adherence	1.528 (1.01–2.32)	0.046*
	Adherence	1	
Model 2 (n = 347)	Non-adherence	1.332 (0.82–2.16)	0.243
	Adherence	1	
Model 3 (n = 85)	Non-adherence	2.496 (1.02–6.12)	0.046*
	Adherence	1	

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the last subsample, three quarters of individuals reported excellent/very good health, compared to half the original sample. Apart from this difference, the subsamples were similar.

The modified Poisson regression analysis (Model1) showed that individuals with LBP/NP risk profiles at T2 independently of back problems or not at T1 and without insomnia both at T1 and T2, significantly increased the risk of insomnia at T3 (RR = 1.5) if they reported poor adherence to moderate-to-high levels of PA (Table 5).

However, for individuals with LBP/NP risk profiles repeatedly over a longer time period (18 months) i.e. both at T1 and T2 (Model 2), adherence to moderate-to-high PA levels had no impact on reporting insomnia at T3.

Finally, among individuals with LBP/NP risk profile only at T2 (i.e. reported no pain at T1), the risk of insomnia was significantly higher (RR = 2.5) among those with poor adherence to moderate-to-high PA levels. The risk ratios of these models are shown in Table 5.

When introducing Self-Rated Health as a covariate in the model, the estimates were slightly smaller, and PA in Models 1 and 3 were no longer significant (results not shown). No interactions with the independent variables were found. Likely, Self-Rated Health is a construct that is mirrored partly by pain and PA. Therefore, it did not make sense to keep it in the model.

## Discussion

Among a mainly male working population followed over 3 years, those with a LBP/NP risk profile had a significantly increased risk (RR = 1.5) of developing insomnia one year later when not adhering to moderate-to-high PA levels. Among those who had a LBP/NP risk profile two years in a row, the risk of insomnia was not significantly affected by PA adherence. However, among those without a LBP/NP risk profile at T1, i.e. only at T2, the risk of insomnia was significantly higher (RR = 2.5) among those not adhering to moderate-to-high PA levels compared to those who were physically active. Thus, PA seems to buffer insomnia among those with a LBP/NP risk profile, provided this profile does not persist two years in a row.

The main strength of the study was the sample size of workers followed for three years. This enabled subgroup analysis by adherence to PA guideline, and also made the exploration of temporality possible. Thus, the difference between workers with LBP/NP risk profiles at both measuring points (T1 and T2) and those reporting it only later in the study (T2) could be highlighted.

The variables in this study are based on validated questionnaires and resulting indexes for pain, PA and sleep. It could be argued that the measurements on which this study are based were “coincidental moments in time”, reflecting the intermittent nature of LBP and NP. The time points may, however, fail to capture individuals with persistent of pain. This is relevant considering the finding that in subjects with LBP/NP risk profiles at T1 and T2, adherence to the moderate-to-high levels of PA did not influence the risk of insomnia. It may be that these individuals were in fact suffering from “chronic” LBP/NP, but the measurements may just

reflect arbitrary moments in time and may not be adequate to explore temporality. In a previous study from Sweden [32], insomnia was found to only partly explain the development of chronic pain, thus the “direction of risk” may be less important as pain become more widespread and persistent. A systematic review examined the effect of exercise on insomnia in middle-aged women [33] and found that PA did not affect insomnia among these individuals. Possibly, there are also differences between the sexes (our sample was mainly male) concerning the ability of PA to buffer insomnia. Another recent systematic review examined non-pharmacological interventions for insomnia in individuals with persistent pain, but did not include interventions of exercise [34].

The data stem from self-rated measures only. Self-reported PA is known to be over-estimated, and objective measures are recommended. Moreover, little is known regarding the validity of self-reported PA measures among populations with pain. The PA questions in this study were constructed before the World Health Organisation’s recommendations of 150 minutes of activity every week were made [35], and the levels reported herein are therefore difficult to assess in term of adequate PA levels. Adherence to levels of PA was a conceptualization made to illustrate the long-term effects of health enhancing PA as opposed to examining PA levels only in a cross-sectional manner. However, the underlying assumption of this measure was that the variable PA was constant if the same levels were reported at two consecutive time points, which could be an overestimation of the true adherence to PA levels over time. In this case, objective measures would have been preferable. Objective measures can measure total PA over a prolonged period of time, including PA performed in different domains, such as work, leisure time and transport. This would give the opportunity to assess a possible differential impact of PA performed during these different domains.

By selecting only those without insomnia at T1 and T2, we probably selected a slightly healthier subgroup of the source population; indeed 76% rated their health as very good or excellent as opposed to 46% in the full population. These subjects were relatively adherent [36] to moderate-to-high levels of PA, which is a further indication of a healthy worker effect. In a recent American study, adherence to recommended PA levels were found among the slightly older and healthy [37], which is in agreement with these results.

Interestingly, Self-Rated Health changed the estimates just enough to make them non-significant. It may be due to the fact that both pain, sleep and PA are factors contributing to the perceived health of an individual, and controlling for health in our models dilutes the effect of each variable.

In conclusion, PA adherence has the ability to buffer the effect of LBP/NP pain on future insomnia. However, in individuals reporting LBP/NP risk profiles over two consecutive years, PA does not significantly alter this risk.

## Author Contributions

**Conceptualization:** Iben Axén, Lydia Kwak, Irene Jensen.

**Data curation:** Jan Hagberg.

**Formal analysis:** Jan Hagberg.

**Investigation:** Irene Jensen.

**Methodology:** Iben Axén, Lydia Kwak, Irene Jensen.

**Writing – original draft:** Iben Axén.

**Writing – review & editing:** Lydia Kwak, Irene Jensen.

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