Report Information from ProQuest

17 October 2023 02:25

TABLE OF CONTENTS

Night work, mortality, and the link to occupational group and sex

Åkerstedt, Torbjörn, PhD; Narusyte, Jurgita, PhD; Svedberg, Pia, PhD

ABSTRACT (ENGLISH)

Objective Night shifts are associated with several major diseases. Mortality has been studied only to a limited extent, and the association with night shifts remains unclear. The purpose of the present study was to investigate the association between duration of night shift exposure and mortality in a large sample from the Swedish Twin Registry (the SALT cohort). Results The hazard ratio (HR) for "ever" night shifts for total mortality was 1.07 [95% confidence interval (CI) 1.01-1.15] but 1.15 (95% CI 1.07-1.25) for longer exposure (>5 years). Also, HR for cause-specific mortality due to cardiovascular disease was significant, with higher HR for longer night shift exposure. Mortality due to cancer was significant for longer exposure only. White-collar workers showed significant HR for longer exposure. In particular, male white-collar workers showed a significant HR, with a highest value for longer exposure [HR 1.28 (95% CI 1.09-1.49)]. Heredity did not influence the results significantly. Conclusions Long duration of exposure to night shift work is associated with increased mortality, particularly in male white-collar workers. The lack of effects of accumulated exposure suggests that the results should be interpreted with caution.

FULL TEXT

Headnote

Objective Night shifts are associated with several major diseases. Mortality has been studied only to a limited extent, and the association with night shifts remains unclear. The purpose of the present study was to investigate the association between duration of night shift exposure and mortality in a large sample from the Swedish Twin Registry (the SALT cohort). Methods Cox proportional hazards regression models were used to analyze the data (N=42 731) over a follow-up period of 18 years, with years of night shift work as the exposure variable and adjustment for lifestyle factors and age, and stratification on gender and occupational group.

Methods Cox proportional hazards regression models were used to analyze the data (N=42 731) over a follow-up period of 18 years, with years of night shift work as the exposure variable and adjustment for lifestyle factors and age, and stratification on gender and occupational group.

Results The hazard ratio (HR) for "ever" night shifts for total mortality was 1.07 [95% confidence interval (CI) 1.01-1.15] but 1.15 (95% CI 1.07-1.25) for longer exposure (>5 years). Also, HR for cause-specific mortality due to cardiovascular disease was significant, with higher HR for longer night shift exposure. Mortality due to cancer was significant for longer exposure only. White-collar workers showed significant HR for longer exposure. In particular, male white-collar workers showed a significant HR, with a highest value for longer exposure [HR 1.28 (95% CI 1.09-1.49)]. Heredity did not influence the results significantly.

Conclusions Long duration of exposure to night shift work is associated with increased mortality, particularly in male white-collar workers. The lack of effects of accumulated exposure suggests that the results should be interpreted with caution.

Key terms blue-collar work; cancer; cardiovascular; exposure; gender; night shift; occupation; shift work; shift worker; twin; white-collar work.

Shift work affects 19.8% of the employed population in Sweden (1). Furthermore, shift work that involves night shifts is associated with a higher risk of ischemic heart disease (2), diabetes (3, 4), accidents (5) and breast cancer (6).

Recent studies also found that exposure at young age to night work is related to multiple sclerosis (7) and to rheumatic arthritis (8).

Logically, one would expect the increased risk of disease to be associated also with mortality, at least if the risk of the disease is high and the disease represents a large part of the diseases leading to death. The evidence here is weak, however, and the available studies are few in contrast to the number of studies focusing on diseases, sleep, fatigue, or accident risk. This may be due to difficulties in linking night work exposure to mortality on a national level across occupations. Thiis-Evensen (9) was the first to address the issue of shift work and mortality, but he did not find any difference in mortality in a group of 498 shift versus 212 day workers of a Norwegian company over 31 years of study. Taylor &Pocock (10) studied 8603 workers in England and Wales, finding no excess risk of mortality in shift workers, although a reanalysis of these data concluded that there was an increased risk (11). Karlsson et al (12) also failed to find an association among male paper mill workers, but the study was small and women were not included. But Gu et al (13) found an increased risk of mortality among 78 000 female nurses in the US with rotating shifts (ie, with night work), as did Jörgensen et al (14) among 18 000 Danish nurses.

The studies above investigated particular occupational groups or regions. Among studies of populationbased representative samples, an increased mortality risk was demonstrated in female white-collar night shift workers (15), but number of years of exposure were not available (N=20 000). Nätti et al (16) investigated 4500 individuals in a representative sample of the Finnish population and found a significant doubling of mortality, but only 190 night workers participated. However, a recent meta-analysis including three of the studies listed above did not find a significant effect of night shifts on mortality (17). Furthermore, a study of a representative sample of 159 000 individuals in Denmark did not find a significant association between shift work and mortality (18), but that study did not adjust for lifestyle variables.

A meta-analysis of 5249 men in various occupations (19) did not find a difference in cardiovascular mortality between day and shift work (all types combined, ie, also those without night shifts).

All cited studies above defined exposure at baseline and did not consider years of exposure, which should be an important factor. Only the studies by Akerstedt et al (15), Nätti et al (16), and Hannerz et al (18) used representative samples of the population, and there is a risk that data from particular occupational groups may not be representative of the population. In addition, most studies, thus far, have been quite small. Furthermore, the standard approach in studies of shift work and health is to compare shift to non-shift workers. This leaves room for selection biases with regard to education, physical workload, work environment, socio-economic group and other factors. These are, to a large extent, variables that differ between white- and blue-collar workers (WCW and BCW). It seems reasonable to separate the latter two groups when studying effects of night work on mortality. There is also a possibility that the link between night work and mortality may be influenced by familial (genetic and shared environmental) factors. This has never been addressed before, and the fact that at least diurnal type is a heritable trait (20) makes an association plausible. Also, mortality has shown to be influenced by genetics (21). A twin approach will make it possible to take shared family environmental and genetic factors into account when investigating the association between night work and mortality.

There seems to be a need for a study on night shifts and mortality using a reasonably large representative sample with limited occupational bias and accumulated exposure to night shifts as a key variable. Thus, a relatively old cohort was used to obtain individuals with close to maximum exposure when the study started. The aim of the present study was to investigate the association between night work (with information on the duration of exposure) and mortality, using data from the Swedish Twin Registry. A special focus was on occupation, gender, and the influence of familial factors on the association. Furthermore, cause-specific mortality in cardiovascular disease and cancer was included.

Method

Design

The design was a prospective cohort study. For this study, twins born in Sweden 1900-1958 and who participated in the Screening Across the Lifespan Twin (SALT) study, conducted by the Swedish Twin Registry, were included.

Ages at the time of the interview were 41-99 years. Each individual participated in the SALT computer-assisted, telephone interview once between 1998 and March 2003. The response rate was 74% and the total sample encompassed 42 731 individuals with responses on history of night work. Of these, 12 850 had been exposed to night work. The interview included a number of items regarding different diseases, symptoms, lifestyle, night shifts and sociodemographic factors. The procedure for data collection has previously been described in detail elsewhere (22). Data on death were obtained from the Cause of Death Registry at the National Board of Health and Welfare. Data were linked to the twins by using the unique person identification number available for all Swedish citizens.

Variables

The exposure to night shifts was defined based on the question: "For how many years have you had work hours that included night shifts at least once per month". The exposed group was defined as participants with 1-45 years of night work. This group was further categorized as short (1-5 years) or longer (6-45 years) exposure, since it seemed reasonable to expect the effect of exposure not to be immediate. The selected intervals are compromises between a balanced number of participants and what was considered to be a short-term exposure. For completeness, we also tried one subdivision of 1-5, 6-10, 11-20, and 21-45 years of night shift work for a sensitivity analysis, as well as another subdivision of 1-14 and 15-45 years. The reference group contained those who reported 0 years of night work ("never night work").

The outcome variable was defined as total mortality due to any cause of death. For cause-specific analyses, the main diagnosis behind the cause of death was obtained from the Cause of Death Registry. The mortality was analyzed separately due to cancer (ICD10: C00-D48), circulatory (ICD-10: I00-I99) diseases. In cause-specific mortality analyses, death cases due to those other than the analyzed one were excluded.

The following covariates were included: educational level (0=more than compulsory [reference], ^compulsory); tobacco use (0=no tobacco [reference], 1=tobacco use (includes current or previous regular smoking/snuffing as well as occasional smoking or snuffing)); alcohol consumption (0=no alcohol [reference], 1=alcohol consumption); leisure-time physical activity (0=moderate exercise [reference], 1=low exercise, 2=high exercise); body mass index (BMI) (0=normal weight (>18.5- 25 kg/m2) [reference], 1=underweight (<18.5 kg/m2), 3=overweight (>25-30 kg/m2), 4=obesity (>30 kg/m2)); coffee consumption (0=no coffee [reference], 1-2 cups a day, 3-4 cups a day; >4 cups a day). Severity of disease was assessed by asking SALT participants "Do you have or have you had [here 53 different health problems and conditions were mentioned]" and responses were classified, based on their most severe illness, into categories: 0=no disease, 1=not at all life-threatening (eg, migraine), 2=somewhat life-threatening (such as high blood pressure), and 3=life threatening (eg, stroke, cancer, ischemic heart disease), according to the expected impact of the disease. A more detailed description of this variable and categorization is found elsewhere (23, 24). In addition, self-reported interview data on occupation were coded into occupational groups according to Statistics Sweden (SSYK 96) that were used both as covariates and for stratification. The occupations were grouped into WCW [for example, managers, professionals, office workers, technicians, officers, nurses (codes: 1-4, 011, 021)] and BCW including occupation areas [for example, industrial workers, craftsmen, nurses assistants, drivers, miners, etc (codes 5-9, 031)] according to the criteria of Statistics Sweden.

Statistical analyses

Frequencies were used to describe the background and covariates for groups with 0 and 1-45 years of night work, and for both groups combined. The difference between occupational categories were tested using Chi2 analysis. Exposure was defined as number of years with night shifts. All individuals contributed with time in years until the date of death or the end of follow-up (ie, 31 December 2014). Multiple Cox proportional hazard regression analyses were used to compute hazards ratios (HR) with 95% confidence intervals (CI). After analysis of interaction with exposure, stratification was carried out for BCW and WCW and gender (males/females) across BCW and WCW groups. The analyses were adjusted for within-twin pair dependency by clustering on twin identity. The association was also adjusted for the covariates that were entered stepwise. Only individuals with complete data were included in the analyses of associations. We also made a second analysis with all individuals included.

To assess the influence of familial factors (ie, genetics and family environment), a conditional Cox proportional hazard regression analysis was carried out of same-sex twin pairs that were discordant (different) on mortality. These analyses are based on the fact that twins in a pair are optimally matched on genetics [100% for monozygotic (MZ) or 50% for dizygotic (DZ) twins] and shared (early family) environment (100% for both MZ and DZ twins) as well as age and sex. The familial factors are suggested to be of importance for the studied association if HR computed in conditional (discordant twin) analyses differ from the HR computed in the total sample. Further, a difference between the association estimated for MZ and DZ twins would suggest the importance of genetic factors. All analyses were performed using SAS V.9.4 (SAS Institute, Cary, NC, USA).

Ethical considerations

The Regional ethical committee of Stockholm, Sweden approved this study.

Results

Descriptive statistics of the sample, including background variables, are presented in table 1, and 30.1% of the sample had worked night shifts. Note that these values represent accumulated night shifts over much of a working career. The table shows a higher prevalence of men, overweight, smoking, blue-collar jobs among night shift workers.

The results of the analyses of the association between night work and mortality are presented in table 2. Night work (1-45 years) was significantly associated with future mortality due to all-cause, as well as circulatory death, after full adjustment. Longer duration (>5 years) showed higher HR and mortality due to cancer disease became significant for longer duration only.

In a sensitivity analysis, we divided years of exposure into groups with 1-5, 6-10, 10-20, and 21-45 years, but no trend in HR values was seen for all-cause mortality. We also tested long exposure as 15-45 years to increase the N for that group (with 1-14 years as short exposure). However, the HR for 15-45 years was 1.13(95% CI 1.03-1.26), compared to HR 1.15 (95% CI 1.07-1.25) for 6-45 years. The results for the intermediate exposures did not change markedly, and the HR were non-significant. Specific causes of mortality were not analyzed because of low number of outcomes.

Since it might be argued that smoking, alcohol consumption, BMI, and leisure-time physical activity could be affected by shift work, we also conducted an sensitivity analysis adjusting for background variables only (age, sex, education, and severity of disease at baseline), and in a next step, also for the variables that could be seen as possibly influenced by shift work. After the adjustment for background variables only, we obtained a HR 1.13 (95% CI 1.06-1.21) for 1-45 years of exposure and HR 1.22 (95% CI 1.13-1.32) for 6-45 years. This should be compared with the second model of table 2 (adjusted for all covariates), which yielded HR 1.07 (95% CI 1.01-1.15) and 1.15 (95% CI 1.06-1.24) for 1-45 years and 6-45 years, respectively. The 1-5year exposures retained low and non-significant HR and were not affected.

Since effects of night shifts on mortality may attenuate after retirement, we carried out a sensitivity analysis including only those still at work. For 6-45 years of exposure we obtained HR 1.16 (95% CI 1.07-1.27) for those at work (compared to 1.15 (95% CI 1.07-1.25) for the complete sample. The number of participants was 25 892 for those at work and 30 582 for the complete group). For WCW at work with 6-45 years of exposure, we obtained HR 1.28 (95% CI 1.12-1.47) versus HR 1.25 (95% CI 1.09-1.44) for the complete sample (table 2). For BCW, no HR were significant for either group.

Based on the hypothesis of influence of occupation and gender moderation, we also computed the interactions between exposure (0/1-45 years) and occupational group as well as gender. For the former, model 2 showed a P-value of P=0.06 and, for the latter, P=0.01. Since the first was borderline significant, we stratified for both. Stratification for occupational group did not show any significant association for 1-45 years of exposure for either occupational group (although close to significance), but longer exposure showed a significant HR for WCW (table 3). Interaction analysis or exposure with gender showed P=0.07 in the WCW group and P=0.10 in the BCW group. When gender was added to the analysis, male WCW showed a significant association with mortality for 1-45 years of night work and, particularly, for exposure of 6-45 years (table 4). The association for BCW was not significant. The group of male WCW night shift workers was made up of managers (18% / 23%) in only day working male

(WCW), professionals (MD, teachers, academics, lawyers, psychologists, etc) (28% /0%), college educated (engineers, pilots, nurses, police) (38% / 36%), office employees (11% / 10%), military (3% / 1%). No differences were significant (Chi2). The individual occupations in which night work dominated with >2/3 of the occupation, were police (6.2% of all male WCW), pilots (4.8%), MDs (7.8%), and nurses (1.8%).

In the subsample of discordant twin pairs (table 5), the estimates were adjusted for familial factors as well as sex and age. Significant HR were found for being exposed to night work >5 years. However, after further adjusting for the covariates also used in the analyses of the whole cohort (as in the previous tables), no significant results were seen.

All analyses above were repeated with individuals with missing data retained. This only changed the results marginally.

Discussion

The results showed that 1-45 years of night work was associated with higher risk for all-cause mortality and mortality in circulatory diseases, compared to those not exposed to night work. The effect was stronger among those with longer exposure. The effect was present for both white and blue-collar workers for those with longer exposure. When also gender was considered, a sig- nificant effect was present among male WCW only, and particularly among those with longer exposure. Familial factors and gender seem to be of less importance for the associations studied.

The significant overall effect of 1-45 years of exposure to night shifts was relatively modest. As indicated in the introduction, very few similar studies are available, and the results vary. A recent meta-analysis did not find any significant effects of night work (17). Neither did a recent study of a representative sample of 150 000 individuals (18). However, the (non-significant) risk ratio in that study was 1.07, equal to the HR of 1.07 in the present study. Furthermore, the former study did not adjust for life style factors (smoking, alcohol intake, physical activity, BMI, etc) and this may have attenuated the results.

The present study also considered duration of exposure and the results indicate that longer exposure (>5 years) is associated with mortality (as is 14-45 years). Apart from this observation, there was no clear doseresponse pattern. Thus, the present data suggests a threshold association with a sudden increase in risk after some years of exposure but no accumulation. This is quite unusual in epidemiological studies. The reason for the lack of influence of amount of exposure is not clear, and other studies of night shifts and mortality have not taken duration of exposure into account. However, a similar threshold has been observed for exposure to night work and breast cancer in the same cohort (25). We have no explanation for the apparent threshold effect, but one possibility is the tendency to leave shift work occupations over time (26). The reasons for poor tolerance for shift work seem to be related to fatigue (27). This turnover may prevent serious diseases or mortality to surface in the short run but may eventually result in an accumulation negative effects in those who have remained in night shift work for a long time. This is very speculative, but should be possible to investigate.

The increased mortality due to cardiovascular disease agrees with previous work on cardiovascular morbidity (28, 29). The increased mortality in cancer only occurred for long-term exposure and cancer morbidity, but this is in line with the classification of night shift work as probably cancerogenic in humans by the International Agency for Research on Cancer (30) .

The finding of a significant effect of night work in WCW agrees with the previous finding (15). The latter and the present study contained representative samples of the population, whereas the other available studies have focused on specific groups, like nurses, who are WCW, and found an increased risk (13, 14). Studies of industrial shift workers (BCW) have failed to find any increased risk (12, 31). However, the nurses and the industrial workers are very different in terms of gender, and it is not possible to use results from those studies as support for an association between night work and mortality being stronger among WCW.

The finding of an increased mortality among male WCW only, contrasts with our previous finding of an increased mortality in female WCW (15). The latter study, however, included lower WCW in the BCW group and also included evening work and other non-night work schedules. It is, therefore, not directly comparable to the present cohort. It should be emphasized that increased, but non-significant, HR were seen for longer exposure also in female WCW, as well as in male and female BCW. The reason for the significantly higher mortality for male WCW in the present study is not clear. A number of possible confounders were adjusted for, including alcohol consumption and smoking, while physical workload was handled through the stratification on WCW and BCW. Furthermore, the distribution of major occupational groups did not differ between day and night shift groups among male WCW. Thus, stress should not be an issue, even if we have no possibility to test this possibility. However, it is possible that shift schedules in male WCW might be more demanding than in other shift work groups, as for example, for pilots (time zone shifts), MD (long on-call shifts), and nurses (frequent quick returns, short rests periods). Unfortunately, the present study does not contain information on shift characteristics, and - to the best of our knowledge - there are no available studies of the health burden of different types of night shift schedules in relation to occupational groups. This seems to be an important question for future research.

The association between longer night shift experience and mortality in male BCW was borderline significant, but supports the notion of the link between night shifts and mortality being present mainly in males. Again, we suggest that the characteristics of the shift system may be part of the explanation, but new research focused on these issues is needed.

The lack of hereditary effect somewhat weakens the argument for an effect of night shift work on mortality. Twin comparisons constitute a strong design for interpretations of causality. However, the demonstrated effect for the whole group was small and it is possible that the proportion of discordant twin pairs may have been too small to detect effects.

There are no studies of the pathophysiology behind the link between night work and mortality, but with regard to night work and disease, it has been suggested that the alternation between day and night shifts causes desynchronization of circadian rhythms, and this in turn causes ill health (32). Yet, evidence only derives from animals in simulated night work (33). It is also possible that sleep loss that occurs in connection with night and morning shifts (34) may play a role, since short sleep is associated with mortality (35). However, short sleep here refers to habitual such sleep, not the variation between days of short as long sleep, as found in shift work. Also, alcohol consumption and smoking habits have been suggested as causes of health problems in shift workers (36). The overall impression is that we lack information on the pathophysiology of night work and mortality or health.

The present study has several limitations. A major one is that exposure is only measured once and that we have no information on when exposure occurred. Furthermore, the amount of exposure was based on subjective reports, but objective data on night work exposure are rare in large representative population-based studies, with some exceptions (37). The amount of night work exposure was set at least one night per month. This would have diluted the effects of frequent night work. Probably also factors like frequency of night shifts, shift duration and quick returns (short time between shifts), sleep, age when entering or leaving shift work should be included in studies of night shifts and mortality. In view of this, the present results are restricted to the duration of exposure of night shifts, not night shift work per se.

The strengths of the study are its size, the long follow-up, the possibility to investigate the influence of familial factors on the associations, and the virtually complete information of mortality through national registers.

In conclusion, the present study has shown that accumulated exposure to night shifts is associated with increased mortality among male WCW, possibly also male BCW, and the effect is increased for exposures >5 years. Also cause-specific mortality with cardiovascular diagnoses was increased, possibly also with cancer, but only after long-term exposure. It should be emphasized that the present results only pertain to the duration of the exposure to night shifts, not night shift work per se (which would include, density of night shifts and other characteristics). The results should be interpreted with caution because of the unexplained absence of effects of accumulated exposure.

Acknowledgements

We acknowledge the Swedish Twin Registry for access to data. The registry is managed by Karolínska Institutet and receives funding through the Swedish Research Council under the grant no 2017-00641. The SALT study was funded by the Swedish Council for the Planning and Coordination of Research and by a grant from the National

Institutes of Health, USA (Grant AG 08724). The present work was funded by Nordic Programme on Health and Welfare (grant no 74809) (Nordforsk).
Funding
This study was funded by NordForsk, the Nordic Program on Health and Welfare (74809)

Sidebar

The present study of 42 731 twins showed a significatn hazard ratio for "ever" exposure to night work {HR 1.07 (95% CI 1.00-1.15)] and particularly for >5 years of exposure The increased mortality was seen in both white- and blue-collar workers exposed for >5 years, and particularly in male blue-collar workers. Heredity did not influence the results significantly.

Åkerstedt T, Narusyte J, Svedberg P. Night work, mortality, and the link to occupational group and sex. Scand J Work Environ Health. 2020; 46(5):508-515. doi:10.5271/sjweh.3892

Correspondence to: Torbjörn Åkerstedt, Department of Clinical Neuroscience, Karolinska Institutet, 17177 Stockholm, Sweden. [E-mail: torbjorn.akerstedt@ki.se]

Footnote

1 Division of Psychology, Department of Clinical Neuroscience, Karolínska Institutet, Stockholm, Sweden.
2 Stress Research Institute, Stockholm University, Stockholm, Sweden.
3 Division of Insurance Medicine, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden.

References

References

1. Akerstedt T, Kecklund G. What work schedule characteristics constitute a problem to the individual? A representative study of Swedish shift workers. Appl Ergon. 2017;59(Pt A):320-5. https://doi.org/10.1016/j.apergo.2016.09.007.
2. Cheng M, He H, Wang D, Xu L, Wang B, Ho KM et al. Shift work and ischaemic heart disease: meta-analysis and dose-response relationship. Occup Med (Lond) 2019 May;69(3): 182-8. https://doi.org/10.1093/occmed/kqz020.
3. Knutsson A, Kempe A. Shift work and diabetes-a systematic review. Chronobiol Int 2014 Dec;31(10):114651. https://doi.org/10.3109/07420528.2014.957308.
4. Gan Y, Yang C, Tong X, Sun H, Cong Y, Yin X et al. Shift work and diabetes mellitus: a meta-analysis of observational studies. Occup Environ Med 2015 Jan;72(1):72-8. https:// doi.org/10.1136/oemed-2014-102150.
5. Wagstaff AS, Sigstad Lie JA. Shift and night work and long working hours-a systematic review of safety implications. Scand J Work Environ Health 2011 May;37(3):173-85. https://doi.org/10.5271/sjweh.3146.
6. Hansen J. Night Shift Work and Risk of Breast Cancer. Curr Environ Health Rep 2017 Sep;4(3):325-39. https://doi. org/10.1007/s40572-017-0155-y.
7. Hedström AK, Åkerstedt T, Olsson T, Alfredsson L. Shift work influences multiple sclerosis risk. Mult Scler 2015 Aug;21(9):1195-9. https://doi. org/10.1177/1352458514563592.
8. Hedström AK, Åkerstedt T, Klareskog L, Alfredsson L. Relationship between shift work and the onset of rheumatoid arthritis. RMD Open 2017 Aug;3(2):e000475. https://doi.org/10.1136/rmdopen-2017-000475.
9. Thiis-Evensen E. Skiftarbeid og helse. [Shift work and health] Porsgrunn: Andreas Jakobsens Boktrykkeri; 1949.
10. Taylor PJ, Pocock SJ. Mortality of shift and day workers 1956-68. Br J Ind Med 1972 Apr;29(2):201-7. https://doi. org/10.1136/oem.29.2.201.
11. Knutsson A, Hammar N, Karlsson B. Shift workers' mortality scrutinized. Chronobiol Int 2004;21(6):1049-53. https://doi.org/10.1081/CBI-200035942.
12. Karlsson B, Alfredsson L, Knutsson A, Andersson E, Torén K. Total mortality and cause-specific mortality of Swedish shift- and dayworkers in the pulp and paper industry in 1952-2001. Scand J Work Environ Health 2005 Feb;31(1):30-5. https://doi.org/10.5271/sjweh.845.
13. Gu F, Han J, Laden F, Pan A, Caporaso NE, Stampfer MJ et al. Total and cause-specific mortality of U.S. nurses working rotating night shifts. Am J Prev Med 2015 Mar;48(3):24152. https://doi.org/10.1016/j.amepre.2014.10.018.
14. Jørgensen JT, Karlsen S, Stayner L, Andersen J, Andersen ZJ. Shift work and overall and cause-specific

mortality in the Danish nurse cohort. Scand J Work Environ Health 2017 Mar;43(2): 117-26. https://doi.org/10.5271/sjweh.3612.
15. Åkerstedt T, Kecklund G, Johansson SE. Shift work and mortality. Chronobiol Int 2004;21(6):1055-61. https://doi.org/10.1081/CBI-200038520.
16. Nätti J, Anttila T, Oinas T, Mustosmäki A. Night work and mortality: prospective study among Finnish employees over the time span 1984 to 2008. Chronobiol Int 2012 Jun;29(5):601-9. https://doi.org/10.3109/07420528.2012.675262.
17. Lin X, Chen W, Wei F, Ying M, Wei W, Xie X. Night-shift work increases morbidity of breast cancer and all-cause mortality: a meta-analysis of 16 prospective cohort studies. Sleep Med 2015 Nov;16(11):1381-7. https://doi.org/10.1016/j.sleep.2015.02.543.
18. Hannerz H, Soll-Johanning H, Larsen AD, Garde AH. Night-time work and all-cause mortality in the general working population of Denmark. Int Arch Occup Environ Health 2019 May;92(4):577-85. https://doi.org/10.1007/s00420-018-1394-4.
19. Bøggild H, Knutsson A. Shift work, risk factors and cardiovascular disease. Scand J Work Environ Health 1999 Apr;25(2):85-99. https://doi.org/10.5271/sjweh.410.
20. Toomey R, Panizzon MS, Kremen WS, Franz CE, Lyons MJ. A twin-study of genetic contributions to morningness-eveningness and depression. Chronobiol Int 2015 Apr;32(3):303-9. https://doi.org/10.3109/07420528.2014.9 71366.
21. Åkerstedt T, Narusyte J, Alexanderson K, Svedberg P. Sleep Duration, Mortality, and Heredity-A Prospective Twin Study. Sleep (Basel) 2017 Oct;40(10): https://doi. org/10.1093/sleep/zsx135.
22. Lichtenstein P, De Faire U, Floderus B, Svartengren M, Svedberg P, Pedersen NL. The Swedish Twin Registry: a unique resource for clinical, epidemiological and genetic studies. J Intern Med 2002 Sep;252(3):184-205. https://doi. org/10.1046/j.1365-2796.2002.01032.x.
23. Svedberg P, Bardage C, Sandin S, Pedersen NL. A prospective study of health, life-style and psychosocial predictors of self-rated health. Eur J Epidemiol 2006;21(10):767-76. https://doi.org/10.1007/s10654-006-9064-3.
24. Ropponen A, Narusyte J, Alexanderson K, Svedberg P. Stability and change in health behaviours as predictors for disability pension: a prospective cohort study of Swedish twins. BMC Public Health 2011 Aug;11:678. https://doi.org/10.1186/1471-2458-11-678.
25. Åkerstedt T, Knutsson A, Narusyte J, Svedberg P, Kecklund G, Alexanderson K. Night work and breast cancer in women: a Swedish cohort study. BMJ Open 2015 Apr;5(4):e008127. https://doi.org/10.1136/bmjopen-2015-008127.
26. Knutsson A, Åkerstedt T. The healthy-worker effect: self-selection among Swedish shift workers. Work Stress 1992;6(2):163-7. https://doi.org/10.1080/02678379208260350.
27. Axelsson J, Åkerstedt T, Kecklund G, Lowden A. Tolerance to shift work-how does it relate to sleep and wakefulness? Int Arch Occup Environ Health 2004 Feb;77(2):121-9. https://doi.org/10.1007/s00420-003-0482-1.
28. Knutsson A, Åkerstedt T, Jonsson BG, Orth-Gomér K. Increased risk of ischaemic heart disease in shift workers. Lancet 1986 Jul;2(8498):89-92. https://doi.org/10.1016/ S0140-6736(86)91619-3.
29. Frost P, Kolstad HA, Bonde JP. Shift work and the risk of ischemic heart disease - a systematic review of the epidemiologic evidence. Scand J Work Environ Health 2009 May;35(3): 163-79. https://doi.org/10.5271/sjweh.1319.
30. IARC Monographs Vol 124 group. Carcinogenicity of night shift work. Lancet Oncol 2019 Aug;20(8):1058-9. https:// doi.org/10.1016/S1470-2045(19)30455-3.
31. Bøggild H, Suadicani P, Hein HO, Gyntelberg F. Shift work, social class, and ischaemic heart disease in middle aged and elderly men; a 22 year follow up in the Copenhagen Male Study. Occup Environ Med 1999 Sep;56(9):640-5. https:// doi.org/10.1136/oem.56.9.640.
32. Leger D, Esquirol Y, Gronfier C, Metlaine A. Groupe consensus chronobiologie et sommeil de la Societe francaise de recherche et medecine du sommeil. Le travail posté et de nuit et ses conséquences sur la santé : état des lieux et recommandations [Shift-workers and night-workers' health consequences: State of art and recommendations]. Presse Med 2018;47(11-12 Pt 1):991-9. https://doi.org/10.1016/j. lpm.2018.10.014.

33. Park N, Cheon S, Son GH, Cho S, Kim K. Chronic circadian disturbance by a shortened light-dark cycle increases mortality. Neurobiol Aging. 2012;33(6):1122.e11-1122.e22. https://doi.org/10.1016/j.neurobiolaging.2011.11.005.
34. Åkerstedt T, Kecklund G. Shift work, severe sleepiness and safety. Ind Health 2011;49(2):141-2. https://doi.org/10.2486/indhealth.MS4902ED.
35. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. Sleep 2010 May;33(5):585-92. https://doi.org/10.1093/sleep/33.5.585.
36. Knutsson A. Methodological aspects of shift-work research. Chronobiol Int 2004;21(6):1037-47. https://doi.org/10.1081/CBI-200038525.
37. Cordina-Duverger E, Menegaux F, Popa A, Rabstein S, Harth V, Pesch B et al. Night shift work and breast cancer: a pooled analysis of population-based case-control studies with complete work history. Eur J Epidemiol 2018 Apr;33(4):369-79. https://doi.org/10.1007/s10654-018-0368-x.
Received for publication: 11 December 2019

DETAILS

Subject:	Cancer; Shift work; Population; Exercise; Physical fitness; Heredity; Mortality; Gender; Cardiovascular diseases; Night shifts; Exposure; White collar workers; Confidence intervals; Cardiovascular disease; Nighttime; Variables; Tobacco; Alcohol; Lifestyles; Occupational exposure
Business indexing term:	Subject: Shift work White collar workers
Location:	Sweden
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	508-515,508A
Publication year:	2020
Publication date:	2020
Section:	Original article
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140

e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English
Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3892
ProQuest document ID:	2444686475
Document URL:	https://www.proquest.com/scholarly-journals/night-work-mortality-link-occupational-group-sex/docview/2444686475/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2020-09-22
Database:	Public Health Database

Document 2 of 11

Not just a research method: If used with caution, can job-exposure matrices be a useful tool in the practice of occupational medicine and public health?

Fadel, M; Evanoff, BA; Andersen, JH; d'Errico, A; Dale, AM; Leclerc, A; Descatha, A

ProQuest document link

ABSTRACT (ENGLISH)

The potential usefulness of a JEM as a decision tool for compensation of workrelated musculoskeletal disorders has been examined (3). Because occupational diseases are often under-recognized, another practical application is using a JEM to screen for occupational exposures as part of health surveillance. [...]these JEM developed for research might also be used as a public health tool, provided that their limitations are properly taken into account. Refers to the following texts of the Journal: 2020;46(3):259-267 2020;46(3):231-234 Key terms: JEM; job exposure matrix; job-exposure matrix; method; methodology; occupational health; occupational medicine; public health; tool This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32367143 Marc Fadel, MSc, MD resident,1, Bradley A Evanoff, MD, MPH,2 Johan HAndersen, MD, PhD,3 Angelo d'Errico, MD,4 Ann Marie Dale, PhD,2 Annette Leclerc, PhD,5Alexis Descatha, MD, PhD 1 1 UNIV Angers, CHU Angers, Univ Rennes, Inserm, EHESP, Irset (Institut de recherche en santé, environnement et travail) - UMR_S1085, CAPTV CDC, Angers, France 2 Division of General Medical Sciences, Washington University School of Medicine, St. Louis, Missouri, USA 3 Occupational Medicine, University Research Clinic, Herning, Denmark 4 Unit of Epidemiology, Regional Health Service ASL T03, Grugliasco (Turin), Italy. 5 Inserm, UMS 011 Population-based Epidemiologic Cohorts Unit, Villejuif, France.

FULL TEXT

The recent editorial by Dr Susan Peters "Although a valuable method in occupational epidemiology, jobexposure matrices are no magic fix" ably describes the strengths and limitations of job-exposure matrix (JEM) approaches in occupational epidemiology research (1). In addition to their use in research, we would like to add that JEM may also be of use in compensation and surveillance efforts in occupational health.

JEM could assist the compensation process by supporting the assessment of relevant exposures related to specific health conditions (2). The potential usefulness of a JEM as a decision tool for compensation of workrelated musculoskeletal disorders has been examined (3). Because occupational diseases are often under-recognized, another practical application is using a JEM to screen for occupational exposures as part of health surveillance. Use of JEM to screen for asbestos and wood dust exposure in the clinical setting has shown promising results (4-6). By summarizing multiple exposures at a job level (7), JEM may also assist policy-makers in setting priorities for hazards and controls at work, as well as occupational practitioners to target prevention efforts and direct the conduct of more precise exposure measures to particular jobs.

Sharing JEM across different countries may be useful in providing estimates of exposures across larger populations to calculate global burden of disease related to occupational exposure. The JEMINI (JEM InterNatIonal) initiative was launched to explore the possibility of developing international JEM that could be used across countries (8). Beginning with physical (biomechanical) exposures, this open group has started homogenizing job coding systems and comparing some available JEM. Estimating differences in the level of exposure between countries will require much more work, without guaranteed success.

As Peters mentioned, many limitations exist in the use of JEM. Users of JEM must consider the source of exposure data - expert assessments, data collected from individual workers, or environmental sampling. The coding of occupations is time consuming and can introduce error (9), and more testing of and comparison with automated job coding systems is needed (10). JEM reflect an "average" level of exposure within a job at the expense of individual variation. At population level, JEM can offer a useful estimate of exposures. If used at an individual level in a clinical or compensation setting, JEM cannot replace the professionals involved in exposure assessment but may help them focus their action more effectively on complex situations that require their expertise.

In conclusion, these JEM developed for research might also be used as a public health tool, provided that their limitations are properly taken into account.

Sidebar

Refers to the following texts of the Journal: 2020;46(3):259-267 2020;46(3):231-234

Key terms: JEM; job exposure matrix; job-exposure matrix; method; methodology; occupational health; occupational medicine; public health; tool

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32367143

Marc Fadel, MSc, MD resident,1, Bradley A Evanoff, MD, MPH,2 Johan HAndersen, MD, PhD,3 Angelo d'Errico, MD,4 Ann Marie Dale, PhD,2 Annette Leclerc, PhD,5Alexis Descatha, MD, PhD 1

1 UNIV Angers, CHU Angers, Univ Rennes, Inserm, EHESP, Irset (Institut de recherche en santé, environnement et travail) - UMR_S1085, CAPTV CDC, Angers, France

2 Division of General Medical Sciences, Washington University School of Medicine, St. Louis, Missouri, USA

3 Occupational Medicine, University Research Clinic, Herning, Denmark

4 Unit of Epidemiology, Regional Health Service ASL T03, Grugliasco (Turin), Italy.

5 Inserm, UMS 011 Population-based Epidemiologic Cohorts Unit, Villejuif, France.

Correspondence to: Dr Alexis Decatha, Irset -Inserm UMR 1085, Equipe Ester- CHU/ Université d'Angers, Faculté de Santé, 28 rue Roger Amsler, CS 74521 49045 Angers, France. [e-mail: alexis.descatha@inserm.fr]

References

References

1. Peters S. Although a valuable method in occupational epidemiology, job-exposure-matrices are no magic fix. Scand J Work Environ Health 2020;46:2314. https://doi.org/10.5271/ sjweh.3894

2. Kerbrat J, Descatha A. (The recognition of health consequences of difficult working conditions in France and its evaluation with the use of a job-exposure matrix). Arch Mal Prof Environ. 2018;79:493500. https://doi.org/10.1016/j.admp.2017.12.001
3. Fadel M, Valter R, Quignette A, Descatha A. Usefulness of a job-exposure matrix «MADE »as a decision tool for compensation of work-related musculoskeletal disorders. Eur J Public Health 2019;29:86870. https://doi.org/10.1093/ eurpub/cky274
4. Lorentz E, Despreaux T, Quignette A, Chinet T, Descatha A. (Screening of occupational exposure to asbestos and silica by job-exposure matrix among patients with lung cancer and mesothelioma). Rev Mal Respir. 2019;36:108895. https://doi. org/10.1016/j.rmr.2019.08.006
5. Imbernon E, Goldberg M, Spyckerell Y, Steinmetz J, Bonenfant S, Fournier B. (Use of a job-exposure matrix for the screening of occupational exposure to asbestos). Rev Epidemiol Sante Publique 2004;52:717. https://doi.org/10.1016/S0398-7620(04)99018-9
6. Carton M, Bonnaud S, Nachtigal M, Serrano A, Carole C, Bonenfant S, et al. Post-retirement surveillance of workers exposed to asbestos or wood dust: first results of the French national SPIRALE Program. Epidemiol Prev. 2011;35:31523.
7. Guéguen A, Goldberg M, Bonenfant S, Martin JC. Using a representative sample of workers for constructing the SUMEX French general population based job-exposure matrix. Occup Environ Med. 2004;61:58693. https://doi.org/10.1136/ oem.2003.010660
8. Descatha A, Evanoff BA, Andersen JH, Fadel M, Ngabirano L, Leclerc A, et al. JEMINI (Job Exposure Matrix InterNatIonal) Initiative: a Utopian Possibility for Helping Occupational Exposure Assessment All Around the World? J Occup Environ Med. 2019;61:e3201. https://doi.org/10.1097/ JOM.0000000000001631
9. Petersen SB, Flachs EM, Svendsen SW, Marott JL, BudtzJørgensen E, Hansen J, et al. Influence of errors in job codes on job exposure matrix-based exposure assessment in the registerbased occupational cohort D0C·X. Scand J Work Environ Health 2020;46:25967. https://doi.org/10.5271/sjweh.3857
10. Buckner-Petty S, Dale AM, Evanoff BA. Efficiency of autocoding programs for converting job descriptors into standard occupational classification (SOC) codes. Am J Ind Med. 2019;62:5968. https://doi.org/10.1002/ajim.22928

DETAILS

Subject:	Compensation; Medicine; Public health; Occupational diseases; Epidemiology; Occupational health; Exposure; Asbestos; Occupational exposure; Musculoskeletal diseases; Health surveillance
Business indexing term:	Subject: Compensation Occupational health
Location:	France
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	552-553,552A
Publication year:	2020
Publication date:	2020

Section:	Letter to the Editor
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English
Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3900
ProQuest document ID:	2444686396
Document URL:	https://www.proquest.com/scholarly-journals/not-just-research-method-if-used-with-caution-can/docview/2444686396/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2023-02-27
Database:	Public Health Database

Document 3 of 11

Scandinavian Journal of Work, Environment and Health goes full open access

Rugulies, R [1] ; Burdorf, A [1] National Research Centre for the Working Environment, Department of Public Health, Copenhagen, Denmark

ProQuest document link

ABSTRACT (ENGLISH)

Unlike other major journals in occupational health and safety, SJWEH is published not by a large commercial publishing house but by a not-for-profit organization: the Nordic Association of Occupational Safety and Health (NOROSH) (4). [...]the journal is not published to make a financial profit, and we operate on a relatively small budget

with the constant challenge to balance income and expenses. The five most often cited articles in 2019 among those published in SJWEH in 2018 were all open access articles (11-15). [...]OA also benefits authors. Key terms: editorial; open access; open science This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32852043 Reiner Rugulies, PhD, Editor-in-Chief National Research Centre for the Working Environment, Copenhagen, Denmark [e-mail: rer@nfa.dk] Alex Burdorf, PhD, Editor-in-Chief Department of Public Health, Erasmus Medical Centre, Rotterdam, The Netherlands [e-mail: a.burdorf@erasmuscmc.nl] References 1.

FULL TEXT

As of 1 January 2021, the Scandinavian Journal of Work, Environment and Health (SJWEH) will become a full open access (OA) journal. Under the "gold" OA status, all new articles will be published as "unlocked content" on the journal's website. This makes the final version of an article freely accessible for everyone, and allows for distribution and adaptation as long as the authors are duly acknowledged as they retain the copyright. Online subscriptions will no longer be needed and thus terminated, whereas subscriptions for the printed journal will continue.

Going full OA is a major step in the 45-year history of the journal. With this step, we join the rapidly growing Open Science movement (1). A core aim of this movement is "making full and immediate open access a reality" as proposed by "Plan S" (also known as "cOAlition S"), which has been endorsed among others by the European Commission, the World Health Organization and numerous funding agencies including the Academy of Finland, the Research Council of Norway, and the Swedish Research Council for Health, Working Life and Welfare (Forte) (2).

OA publishing means that research results are immediately available both for critical discussion in the whole scientific community - including researchers in low income countries who may not have access to subscription journals - and for consideration and decision-making by stakeholders and policy-makers. During the current COVID-19 pandemic, many journals have pledged to publish COVID-19 related articles as OA, acknowledging the societal need of getting unrestricted, immediate access to research results (3). At SJWEH, we believe the societal need of getting immediate access to research results is not limited to times of a pandemic.

To become one of the first occupational health and safety journals to go full OA is a double-edged sword. On the one hand, we are proud to be a first mover and support the Open Science movement. On the other hand, there is an economic risk for the journal. Unlike other major journals in occupational health and safety, SJWEH is published not by a large commercial publishing house but by a not-for-profit organization: the Nordic Association of Occupational Safety and Health (NOROSH) (4). Thus, the journal is not published to make a financial profit, and we operate on a relatively small budget with the constant challenge to balance income and expenses. This does not leave much room for error and, therefore, we contemplated full OA for several years before taking the final step (5-7). After continuously increasing the journal's OA content over the years, we now feel we are in a strong position to go 100% OA.

Under the gold OA model, authors are requested to pay an article processing charge (APC) of currently €2300 (NOROSH members: €1150) to publish their papers in the journal. The fee is waived for articles from research institutes in a low income country (according to the definition by the World Health Organization) (8), while articles from research institutes in a middle income country receive an 80% reduction. All articles are published under the Creative Commons CC-BY 4.0 license, the most liberal license that allows for the sharing and adaptation of material as long as proper credit is given to the original source (9).

OA increases the opportunity for article citations (10). The five most often cited articles in 2019 among those published in SJWEH in 2018 were all open access articles (11-15). Thus, OA also benefits authors.

Sometimes a concern is voiced that the gold OA and APC model may result in a decline of quality because journals might be tempted to publish as many articles as possible to generate income (16). Indeed, the ever growing list of predatory journals that send out mass emails to researchers pressing for submission of papers (constituting nowadays a substantial proportion of the daily emails researchers receive), and then publish the articles with no or only a superficial peer-review process, after cashing in on the APC, indicates that publishing poor research might be an attractive business model (17). In SJWEH, though, this will not happen. Our Associ-ate Editors are among the finest scholars in the field of occupational health and safety and they will continue to ensure that only high quality papers are sent out for peer-review, which is - in turn - performed by leading experts in the field. In other words, we can promise our NOROSH members, authors and readers that it will remain difficult to publish papers in the journal and that only the best papers will make it.

The Open Science movement is an exciting development transforming the way research is communicated and shared. We are proud to be a part of this movement, and we ask our authors, reviewers and readers to support the advancing of scientific knowledge in the field by continuing to send high quality papers to the journal, performing rigorous peer-reviews, and reading and discussing our content. Furthermore, qualifying institutions may support the Open Science movement and SJWEH by considering membership in NOROSH (4) (for further information see www.norosh.org/pmwiki.php/Main/Membership).

Sidebar

Refers to the following texts of the Journal: 2019;45(3):215-216 2018;44(3):239-250 2018;44(3):229-238 2018;44(2):134-146 2018;44(1):16-24' ' ' 2015;41(5):42Г424' ' ' 2013;39(1):1-4.

Key terms: editorial; open access; open science

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32852043

Reiner Rugulies, PhD, Editor-in-Chief

National Research Centre for the Working Environment, Copenhagen, Denmark

[e-mail: rer@nfa.dk]

Alex Burdorf, PhD, Editor-in-Chief

Department of Public Health, Erasmus Medical Centre, Rotterdam, The Netherlands

[e-mail: a.burdorf@erasmuscmc.nl]

References

References

1. United Nations Educational, Scientific and Cultural Organization (UNESCO). Open Science [Webpage]. 2020. Available from: https://en.unesco.org/science-sustainable-future/open-science. (Accessed: 9 August 2020).

2. cOAlition S. Plan S. Making full and immediate Open Access a reality [Webpage]. 2020. Available from: https://www.coalition-s. org. (Accessed: 9 August 2020).

3. Wellcome Trust. Sharing research data and findings relevant to the novel coronavirus (COVID-19) outbreak [Webpage]. 2020. Available from: https://wellcome.ac.uk/coronavirus-covid-19/open-data. (Accessed: 9 August 2020).

4. Nordic Association of Occupational Safety and Health (NOROSH). Home page of NOROSH. 2020. Available from: https://www. norosh.org/pmwiki.php/Main/HomePage. (Accessed: 9 August 2020).

5. O'Donoghue-Lindy L. Editorial News. New year, new publisher. Nordic occupational health association takes over reins of Scandinavian Journal of Work, Environment &Health. Scand J Work Environ Health. 2013;39(1):1-4. https://doi.org/10.5271/sjweh.3340

6. Härmä M, Viikari-Juntura E, O'Donoghue-Lindy L. Editorial. Scandinavian Journal of Work, Environment &Health: 40 years of innovative research with societal impact in the field of occupational health. Scand J Work Environ Health. 2015;41(5):421-424. https://doi.org/10.5271/sjweh.3516

7. Burdorf A, Härmä M. Editorial News. The future of Scandinavian Journal of Work, Environment &Health in the era of open science. Scand J Work Environ Health. 2019;45(3):215-216. https://doi.org/10.5271/sjweh.3826

8. World Health Organization. Hinari eligibility. 2020. Available from: https://www.who.inf/hinari/eligibility/en. (Accessed: 9 August 2020).

9. Creative Commons. Attribution 4.0 International (CC BY 4.0). 2020. Available from: https://creativecommons.org/licenses/ by/4.0. (Accessed: 9 August 2020).

10. Tennant JP, Waldner F, Jacques DC, Masuzzo P, Collister LB, Hartgerink CH. The academic, economic and societal impacts of Open Access: an evidence-based review. F1000Res. 2016;5:632. https://doi.org/10.12688/f1000research.8460.3

11. Torquati L, Mielke GI, Brown WJ, Kolbe-Alexander T. Shift work and the risk of cardiovascular disease. A systematic review and metaanalysis including dose-response relationship. Scand J Work Environ Health. 2018;44(3):229-238. https://doi.org/10.5271/sjweh.3700
12. Oakman J, Neupane S, Proper KI, Kinsman N, Nygard CH. Workplace interventions to improve work ability: A systematic review and meta-analysis of their effectiveness. Scand J Work Environ Health. 2018;44(2):134-146. https://doi.org/10.5271/sjweh.3685
13. Koranyi I, Jonsson J, Ronnblad T, Stockfelt L, Bodin T. Precarious employment and occupational accidents and injuries - a systematic review. Scand J Work Environ Health. 2018;44(4):341-350. https://doi.org/10.5271/sjweh.3720
14. Virtanen M, Jokela M, Madsen IEH, Magnusson Hanson LL, Lallukka T, Nyberg ST, et al. Long working hours and depressive symptoms: systematic review and meta-analysis of published studies and unpublished individual participant data. Scand J Work Environ Health. 2018;44(3):239-250. https://doi.org/10.5271/sjweh.3712
15. Wegman DH, Apelqvist J, Bottai M, Ekstrom U, Garcia-Trabanino R, Glaser J, et al. Intervention to diminish dehydration and kidney damage among sugarcane workers. Scand J Work Environ Health. 2018;44(1):16-24. https://doi.org/10.5271/sjweh.3659
16. Else H. Radical open-access plan could spell end to journal subscriptions. Nature. 2018;561(7721): 17-18. https://doi.org/10.1038/ d41586-018-06178-7
17. Grudniewicz A, Moher D, Cobey KD, Bryson GL, Cukier S, Allen K, et al. Predatory journals: no definition, no defence. Nature. 2019;576(7786):210-212. https://doi.org/10.1038/d41586-019-03759-y

DETAILS

Subject:	Research facilities; Working conditions; Journals; Peer review; Public health; Science; Occupational health; Occupational accidents; Occupational safety; Open access; COVID-19; Editorials; Health care facilities; Research centers; Publishing; Low income groups; Pandemics; Medical research; Electronic mail systems; Subscriptions; Coronaviruses; Systematic review
Business indexing term:	Subject: Occupational health Occupational accidents Occupational safety
Company / organization:	Name: World Health Organization; NAICS: 923120
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	455-456,455A
Publication year:	2020
Publication date:	2020
Section:	Editorial
Publisher:	Scandinavian Journal of Work, Environment &Health

Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English
Document type:	Editorial
DOI:	https://doi.org/10.5271/sjweh.3916
ProQuest document ID:	2444686355
Document URL:	https://www.proquest.com/scholarly-journals/scandinavian-journal-work-environment-health-goes/docview/2444686355/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2023-02-27
Database:	Public Health Database

Document 4 of 11

Prevention at work needed to curb the worldwide strong increase in knee replacement surgery for working-age osteoarthritis patients

Kuijer, P Paul F M, PhD [1] ; Burdorf, Alex, PhD [1] Amsterdam Movement Sciences, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

ProQuest document link

ABSTRACT (ENGLISH)

In the upcoming decades, hospitals and clinics around the world face a steep rise in demand from patients seeking knee replacement surgery. An absolute increase in knee replacement surgery of 297%-to 57,893 procedures-is forecasted in The Netherlands between 2005 and 2030. The situation is similar in many countries: Sweden, 163% to 21,700 (2013-2030); Italy, 45% to about 100,000 (2017-2050), the UK, 916% to about 1.2 million (2015-2035); Australia, 276% to 65,569 (2013-2030); and the USA, 673% to 3.48 million (2005-2030). No projections are

available for Asia, however, similar growth percentages have already been seen in Japan of 373% (2007-2014) and Korea (407%, 2001-2010). Here, Kuijer and Burdorf discuss how work might play a significant role in reducing the steep rise in replacement surgery for knee osteoarthritis across the world.

FULL TEXT

Headnote

Key terms: arthroplasty; editorial; knee; knee osteoarthritis; knee replacement surgery; osteoarthritis; prevention

In the upcoming decades, hospitals and clinics around the world face a steep rise in demand from patients seeking knee replacement surgery. An absolute increase in knee replacement surgery of 297% - to 57 893 procedures - is forecasted in The Netherlands between 2005 and 2030 (1). The situation is similar in many countries: Sweden, 163% to 21 700 (2013-2030) (2); Italy, 45% to about 100 000 (2017-2050) (3), the UK, 916% to about 1.2 million (2015-2035) (4); Australia, 276% to 65 569 (2013-2030) (5); and the USA, 673% to 3.48 million (2005-2030) (6). No projections are available for Asia, however, similar growth percentages have already been seen in Japan of 373% (2007-2014) (7) and Korea (407%, 2001-2010) (8). Knee replacement surgery or arthroplasty is the final treatment option for patients suffering from knee osteoarthritis (OA). These increasing numbers are alarming, not only due to the extreme high demands on healthcare provision and budgets, but also for the unforeseen impact on work participation. There is a lack of awareness how work might play a significant role in reducing the steep rise in replacement surgery for knee OA across the world.

Unforeseen impact on work

The largest increase in primary surgery demands is not among the classic knee arthroplasty population of patients aged 70 years and older, but among patients of working age (1-4, 9). For instance, Germany - one of the leading countries in the prevalence of knee arthroplasty - foresees the highest increase in patients aged 5065 years until 2050 (10), and in a similar study using the same database even among patients aged 40-49 years until 2040 (11). In several countries, the current proportion of knee arthroplasty patients under 65 years is already substantial at 30-40%. It is expected in 2030 that the USA will be the first country where the majority of these patients will be younger than 65 years (6), followed by the UK in 2035 (4).

This increase in surgery and shift towards younger age groups can largely be explained by good clinical and cost-effective long-term outcomes, no clear threshold for surgery, and the rising number of younger and more demanding knee OA patients (9). Originally, knee arthroplasty was mainly aimed at reducing pain, improving knee function and thereby enhancing the performance of activities of daily life in the patient population aged 70 years and older. The current success was totally unforeseen by LG Shiers, the founding father of knee arthroplasty (12). In 1954, he concluded: "... few surgeons will ever see 50 patients requiring arthroplasty, let alone operate on them, even in five years". Nowadays, specialized orthopedic surgeons perform this procedure several times per day. Despite the good clinical outcomes, return to work is not that favorable after surgery. About two of every ten working-age patients are dissatisfied with their work ability due to their knee arthroplasty (13). The majority of workers return to their original or other work only after six months (13,14), and about three of every ten patients do not return to their original or other work after a year (13,15). Especially performance of knee-demanding activities like kneeling, crouching and clambering does not improve after surgery (16).

The rapid adaptation of knee arthroplasty as preferred clinical practice for working-age knee OA patients has profound consequences for functioning at work and labor force participation. These consequences receive littleto-no attention in current clinical guidelines and yet are increasingly important outcomes for patients of working age (17, 18). Therefore, occupational health professionals around the world face the urgent challenge to develop evidence of how to improve a patient's ability for sustained employability - as an essential value in life - after surgery (19). Promising initiatives have recently been described although not validated for work participation yet (20-23). More importantly, occupational health professionals should play a primary role in reducing the steep increase in replacement surgery by mitigating the risk of knee OA.

Work to reduce knee osteoarthritis and hospitalization

Until now, little-to-no attention has been given to work as a promising point of engagement for the prevention of knee OA and subsequent hospitalization. A nationwide representative prospective Finnish study showed that high body mass index, prior knee injury, and an intermediate-to-high cumulative physical workload accounted for 70% of hospitalizations due to knee OA (23). Bearing these three modifiable risk factors in mind, preventive strategies at work seem warranted to reduce both the incidence of knee OA and, of course, the associated hospitalization. Primary prevention at work might mitigate all three risk factors. Without question, losing excessive body weight is key (23). Proper &Van Oostrom (24) concluded in their review on health promotion intervention, that strong evidence exists for favorable effects of weight reduction, especially for interventions targeting diet and/or physical activity. Regarding knee injury and cumulative physical workload, reducing the time kneeling and squatting at work appears highly promising to tackle both (25-27).

The upcoming EU-OSHA campaign 2020-2022 'Healthy Workplaces Lighten the Load' starting October 2020 is an excellent opportunity to focus on this specific occupational disease. Intensive interventions tailored to the specific target group are promising to reduce the risk of knee OA (28-30). This approach is in line with the general risk assessment strategies to safeguard workers' health and safety and, therefore, Labor Inspectorates can effectively enforce compliance with the occupational safety and health regulations (31). Moreover, the proposed combined interventions on lifestyle and workplace adaptations are also in line with the stepped-care strategy for secondary prevention of knee OA (32) and of course tertiary prevention by bringing the rising hospital admission curve to its knees and thereby contributing to better health and prolonged working lives of workers at risk of "running out of cartilage".

Sidebar

Refers to the following texts of the Journal: 2019;45(4):321-323 2019;45(6):546-559 2019;45(2):103-113

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32780145

P. Paul F.M Kuijer, PhD

Netherlands Center for Occupational Diseases, Department of Public and Occupational Health, Amsterdam Public Health Research Institute, Amsterdam Movement Sciences, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands.

[e-mail: p.p.kuijer@amsterdamumc.nl]

Alex Burdorf, PhD

Department of Public Health, Erasmus Medical Center, Rotterdam, The Netherlands

[Email: a.burdorf@erasmusmc.nl]

References

References

1. Otten R, van Roermund PM, Picavet HS. Trends in aantallen knie- en heupartroplastieken: de vraag naar knie- en heupprotheses blijft voorlopig toenemen [Trends in the number of knee and hip arthroplasties: considerably more knee and hip prostheses due to osteoarthritis in 2030]. Ned Tijdschr Geneeskd. 2010;154:A1534. pubmed.ncbi.nlm.nih.gov/20619009/

2. Nemes S, Rolfson O, W-Dahl A, et al. Historical view and future demand for knee arthroplasty in Sweden. Acta Orthop. 2015;86(4):426-431. doi.org/10.3109/17453674.2015.1034608

3. Romanini, E, Decarolis F, Luzi I, Zanoli G, Venosa M, Laricchiuta P, et al. Total knee arthroplasty in Italy: reflections from the last fifteen years and projections for the next thirty. Int Orthop. (SICOT). 2019;43:133-138. doi.org/10.1007/s00264-018-4165-7

4. Culliford D, Maskell J, Judge A, Cooper C, Prieto-Alhambra D, Arden NK, et al. Future projections of total hip and knee arthroplasty in the UK: results from the UK Clinical Practice Research Datalink. Osteoarthr Cartilage. 2015;23(4):594-600. doi. org/10.1016/j.joca.2014.12.022

5. Ackerman IN, Bohensky MA, Zomer E, Tacey M, Gorelik A, Brand CA, et al. The projected burden of primary total knee and hip replacement for osteoarthritis in Australia to the year 2030. BMC Musculoskelet Disord. 2019;20(1):90. doi.org/10.1186/s12891019-2411-9

6. Kurtz S, Ong K, Lau E, Mowat F, Halpern M. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. J Bone Joint Surg Am. 2007;89(4):780-785. doi.org/10.2106/JBJS.F.00222
7. Kawata M, Sasabuchi Y, Inui H, Taketomi S, Matsui H, Fushimi K, et al. Annual trends in knee arthroplasty and tibial osteotomy: Analysis of a national database in Japan. Knee. 2017;24(5):1198-1205. doi.org/10.1016/j.knee.2017.06.005
8. Koh IJ, Kim TK, Chang CB, Cho HJ, In Y. Trends in use of total knee arthroplasty in Korea from 2001 to 2010. Clin Orthop Relat Res. 2013;471(5):1441-1450. doi.org/10.1007/s11999-012-2622-y
9. Price AJ, Alvand A, Troelsen A, Katz JN, Hooper G, Gray A, et al. Knee replacement. Lancet. 2018;392(10158):1672-1682. doi. org/10.1016/S0140-6736(18)32344-4
10. Klug A, Gramlich Y, Rudert M, Drees P, Hoffmann R, Weißenberger M, et al. The projected volume of primary and revision total knee arthroplasty will place an immense burden on future heath care systems over the next 30 years. [published online ahead of print, 2020 Jul 15] Knee Surg Sports Traumatol Arthrosc. 2020;1-12. doi.org/10.1007/s00167-020-06154-7
11. Rupp M, Lau E, Kurtz SM, Alt V. Projections of primary TKA and THA in Germany from 2016 through 2040. Clin Orthop Relat Res. 2020; 478(7):1622-1633. doi: 10.1097/CÜRR.0000000000001214
12. Shiers LG. Arthroplasty of the knee; preliminary report of new method. The Journal of Bone and Joint surgery. British Volume. 1954;36-B(4):553-560. pubmed.ncbi.nlm.nih.gov/13211730/
13. Kievit AJ, van Geenen RC, Kuijer PP, Pahlplatz TM, Blankevoort L, Schafroth MU. Total knee arthroplasty and the unforeseen impact on return to work: a cross-sectional multicenter survey. J Arthroplasty. 2014;29(6):1163-1168. doi.org/10.1016/j. arth.2014.01.004
14. Hylkema TH, Brouwer S, Stewart RE, van Beveren J, Rijk PC, Brouwer RW, et al. Two-year recovery courses of physical and mental impairments, activity limitations, and participation restrictions after total knee arthroplasty among working-age patients. Disabil Rehabil. 2020;1-10. doi.org/10.1080/09638288.2020.1766583
15. Leichtenberg CS, Tilbury C, Kuijer PPFM, Verdegaal SHM, Wolterbeek R, Nelissen RGHH, et al. Determinants of return to work 12 months after total hip and knee arthroplasty. Ann R Coll Surg Engl. 2016;98(6):387-395. doi.org/10.1308/rcsann.2016.0158
16. Kievit AJ, Kuijer PPFM, de Haan LJ, Koenraadt KLM, Kerkhoffs GMMJ, Schafroth MU, et al. Patients return to work sooner after unicompartmental knee arthroplasty than after total knee arthroplasty. [published online ahead of print, 2019 Aug 30] Knee Surg Sports Traumatol Arthrosc. doi.org/10.1007/s00167-019-05667-0
17. Bardgett M, Lally J, Malviya A, Deehan D. Return to work after knee replacement: a qualitative study of patient experiences. BMJ Open 2016;6(2):e007912. doi.org/10.1136/bmjopen-2015-007912
18. Witjes S, van Geenen RC, Koenraadt KL, van der Hart CP, Blankevoort L, Kerkhoffs GMMJ, et al. Expectations of younger patients concerning activities after knee arthroplasty: are we asking the right questions?. Qual Life Res. 2017;26(2):403-417. doi. org/10.1007/s11136-016-1380-9
19. Coenen P, Hulsegge G, Daams JG, van Geenen RC, Kerkhoffs GM, van Tulder MW, et al. Integrated care programmes for sport and work participation, performance of physical activities and quality of life among orthopaedic surgery patients: a systematic review with meta-analysis. BMJ Open Sport &Exercise Medicine 2020;6:e000664. doi.org/10.1136/bmjsem-2019-000664
20. Hoorntje A, Waterval-Witjes S, Koenraadt KLM, Kuijer PPFM, Blankevoort L, Kerkhoffs GMMJ, et al. Goal attainment scaling rehabilitation improves satisfaction with work activities for younger working patients after knee arthroplasty: results from the randomized controlled ACTION trial. J Bone Joint Surg Am. 2020;00:1-9doi.org/10.2106/JBJS.19.01471
21. Straat AC, Coenen P, Smit DJM, Hulsegge G, Bouwsma EVA, Huime JAF, et al. Development of a Personalized m/eHealth Algorithm for the Resumption of Activities of Daily Life Including Work and Sport after Total and Unicompartmental Knee Arthroplasty: A Multidisciplinary Delphi Study. Int. J. Environ. Res. Public Health 2020;17(14): 4952. https://doi.org/10.3390/ ijerph17144952
22. Coole C, Baker P, McDaid C, Drummond A. Using intervention mapping to develop an occupational advice

intervention to aid return to work following hip and knee replacement in the United Kingdom. BMC Health Serv Res 2020;20:523 https://doi. org/10.1186/s12913-020-05375-3
23. Kontio T, Heliövaara M, Viikari-Juntura E, Solovieva S. To what extent is severe osteoarthritis preventable? Occupational and non-occupational risk factors for knee and hip osteoarthritis. Rheumatology (Oxford). 2020;keaa238. doi.org/10.1093/rheumatology/ keaa238
24. Proper KI, van Oostrom SH. The effectiveness of workplace health promotion interventions on physical and mental health outcomes - a systematic review of reviews. Scand J Work Environ Health. 2019;45(6):546-559. doi.org/10.5271/sjweh.3833
25. Hong CY, Lee CG, Kim DH, Cho YS, Kim KY, Ryu SY, Song HS, Workrelated risk factors of knee meniscal tears in korean farmers: a cross-sectional study. Saf Health Work. doi.org/10.1016/j.shaw.2020.05.008.
26. Rytter S, Jensen LK, Bonde JP, Jurik AG, Egund N. Occupational kneeling and meniscal tears: a magnetic resonance imaging study in floor layers. J Rheumatol. 2009;36(7):1512-1519. doi.org/10.3899/jrheum.081150
27. Verbeek J, Mischke C, Robinson R, Ijaz S, Kuijer P, Kievit A, et al. Occupational Exposure to Knee Loading and the Risk of Osteoarthritis of the Knee: A Systematic Review and a Dose-Response Meta-Analysis. Saf Health Work. 2017;8(2):130-142. doi. org/10.1016/j.shaw.2017.02.001
28. van der Beek AJ, Kunst AE. How can we break the vicious circle between poor health and exit from paid employment?. Scand J Work Environ Health. 2019;45(4):321-323. doi.org/10.5271/sjweh.3838
29. Visser S, van der Molen HF, Kuijer PP, Sluiter JK, Frings-Dresen MH. Stand up: comparison of two electrical screed levelling machines to reduce the work demands for the knees and low back among floor layers. Ergonomics. 2016;59(9):1224-1231. doi.org/ 10.1080/00140139.2015.1122233
30. Mikkelsen S, Pedersen EB, Brauer C, Møller KL, Alkjaer T, Koblauch H, Simonsen EB, Thygesen LC. Knee osteoarthritis among airport baggage handlers: A prospective cohort study. Am J Ind Med. 2019;62(11):951-960. doi.org/10.1002/ajim.23044
31. Andersen JH, Malmros P, Ebbehoej NE, Flachs EM, Bengtsen E, Bonde JP. Systematic literature review on the effects of occupational safety and health (OSH) interventions at the workplace. Scand J Work Environ Health. 2019;45(2):103-113. doi. org/10.5271/sjweh.3775
32. Smink AJ, van den Ende CH, Vliet Vlieland TP, Swierstra BA, Kortland JH, Bijlsma WJ, et al. "Beating osteoARThritis": development of a stepped care strategy to optimize utilization and timing of non-surgical treatment modalities for patients with hip or knee osteoarthritis. Clin Rheumatol. 2011;30(12):1623-1629. doi.org/10.1007/s10067-011-1835-x

DETAILS

Subject:	Osteoarthritis; Surgeons; Knee; Clinical medicine; Trends; Occupational health; Medical personnel; Joint replacement surgery; Surgery; Workloads; Hospitalization; Patients; Arthritis; Age; Biomedical materials; Prevention; Cartilage; Public health; Joint surgery; Surgical implants
Business indexing term:	Subject: Occupational health Workloads
Location:	Italy; United Kingdom--UK; Australia; Sweden; Netherlands; United States--US; Germany; Japan
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46

Issue:	5
Pages:	457-460,457A
Publication year:	2020
Publication date:	2020
Section:	Editorial
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English
Document type:	Editorial
DOI:	https://doi.org/10.5271/sjweh.3915
ProQuest document ID:	2444686341
Document URL:	https://www.proquest.com/scholarly-journals/prevention-at-work-needed-curb-worldwide-strong/docview/2444686341/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2020-09-22
Database:	Public Health Database

The mediating role of sleep, physical activity, and diet in the association between shift work and respiratory infections

Loef, Bette, MSc; Van der beek, Allard J, PhD; Hulsegge, Gerben, PhD; van Baarle, Debbie, PhD; Proper, Karin I, PhD

ABSTRACT (ENGLISH)

Objectives Shift work may be associated with an increased incidence of respiratory infections. However, underlying mechanisms are unclear. Therefore, our aim was to examine the mediating role of sleep, physical activity, and diet in the association between shift work and respiratory infections. Methods This prospective cohort study included 396 shift and non-shift workers employed in hospitals. At baseline, sleep duration and physical activity were measured using actigraphy and sleep/activity diaries, sleep quality was reported, and frequency of meal and snack consumption was measured using food diaries. In the following six months, participants used a smartphone application to report their influenza-like illness/acute respiratory infection (ILI/ARI) symptoms daily. Mediation analysis of sleep, physical activity, and diet as potential mediators of the effect of shift work on ILI/ARI incidence rate was performed using structural equation modeling with negative binomial and logistic regression. Results Shift workers had a 23% [incidence rate ratio (IRR) 1.23, 95% CI 1.01-1.49] higher incidence rate of ILI/ARI than non-shift workers. After adding the potential mediators to the model, this reduced to 15% (IRR 1.15, 95% CI 0.94-1.40). The largest mediating (ie, indirect) effect was found for poor sleep quality, with shift workers having 29% more ILI/ARI episodes via the pathway of poorer sleep quality (IRR 1.29, 95% CI 1.02-1.95). Conclusions Compared to non-shift workers, shift workers had a higher incidence rate of ILI/ARI that was partly mediated by poorer sleep quality. Therefore, it may be relevant for future research to focus on perceived sleep quality as an underlying mechanism in the relation between shift work and increased infection susceptibility.

FULL TEXT

Headnote

Objectives Shift work may be associated with an increased incidence of respiratory infections. However, underlying mechanisms are unclear. Therefore, our aim was to examine the mediating role of sleep, physical activity, and diet in the association between shift work and respiratory infections.

Methods This prospective cohort study included 396 shift and non-shift workers employed in hospitals. At baseline, sleep duration and physical activity were measured using actigraphy and sleep/activity diaries, sleep quality was reported, and frequency of meal and snack consumption was measured using food diaries. In the following six months, participants used a smartphone application to report their influenza-like illness/acute respiratory infection (ILI/ARI) symptoms daily. Mediation analysis of sleep, physical activity, and diet as potential mediators of the effect of shift work on ILI/ARI incidence rate was performed using structural equation modeling with negative binomial and logistic regression.

Results Shift workers had a 23% [incidence rate ratio (IRR) 1.23, 95% CI 1.01-1.49] higher incidence rate of ILI/ARI than non-shift workers. After adding the potential mediators to the model, this reduced to 15% (IRR 1.15, 95% CI 0.94-1.40). The largest mediating (ie, indirect) effect was found for poor sleep quality, with shift workers having 29% more ILI/ARI episodes via the pathway of poorer sleep quality (IRR 1.29, 95% CI 1.02-1.95).

Conclusions Compared to non-shift workers, shift workers had a higher incidence rate of ILI/ARI that was partly mediated by poorer sleep quality. Therefore, it may be relevant for future research to focus on perceived sleep quality as an underlying mechanism in the relation between shift work and increased infection susceptibility.

Key terms acute respiratory infection; influenza-like illness; lifestyle behavior; mediation; mediation analysis; occupational health; shift worker.

Workers employed in healthcare and various other occupational sectors regularly work in shifts around the clock (1). Together, this group of shift workers makes up a considerable part of the workforce, with 21% of European workers, and as much as 41% of healthcare workers, working in shifts (1). Nonetheless, shift work can be considered an occupational hazard because it has been linked to an increased risk of multiple diseases (2), such as cardiovascular diseases (3), metabolic syndrome (4), and diabetes mellitus type 2 (5). In addition, we recently found in a prospective cohort study that shift workers in healthcare had 20% more respiratory infections than non-shift workers, indicating that shift workers may also be more susceptible to infections (6). As respiratory infections, such as common cold and influenza-like illness, are responsible for a high burden of disease and substantial productivity loss (7), insight into possibilities to reduce this increased susceptibility in shift workers is needed.

Several possible explanations for the health effects of shift work have been proposed, including the behavioral pathways sleep, physical activity, and diet (8-10). Previous studies have shown shift workers to experience more sleep disturbances and have poorer dietary and physical activity habits (11-13). Engaging in these unhealthy behaviors has been found to be associated with increased infection susceptibility (14-16), and may, therefore, mediate the association between shift work and respiratory infections. However, research is currently lacking on the mediating role of behavioral pathways for health in general and infection susceptibility in particular. Studying this mediating role could contribute to the understanding of how shift work is linked to increased susceptibility to infection. Therefore, the aim of the current study was to examine the mediating role of sleep, physical activity, and diet in the association between shift work and respiratory infections.

Methods

Study design

The Klokwerk+ study is a prospective cohort study that aims to examine the effects of shift work on infection susceptibility and body weight gain, and the mechanisms underlying these health effects (17). In Klokwerk+, healthcare workers from different hospitals in The Netherlands used a smartphone application to report their influenza-like illness/acute respiratory infection (ILI/ ARI) symptoms on a daily basis between September 2016 and June 2017. At the baseline measurement in September-December 2016, participants received the smartphone application, two accelerometers and a sleep/ activity diary to measure sleep and physical activity for seven days, and a food diary to keep for three days. Furthermore, participants completed a questionnaire with questions about shift work status, lifestyle, and health. At the follow-up measurement in April-June 2017, another questionnaire was completed (17).

Measures

Shift work, In the baseline and follow-up questionnaire, healthcare workers completed questions about their shift work status. Participants reported their current work schedule and whether they ever and currently worked night shifts (00.00-06.00 hours) and rotating shifts. Rotating shifts were defined as any work schedule rotating between day, evening, night and/or sleep shifts. Participants were considered shift workers if they worked rotating and/or night shifts (both at baseline and follow-up), and non-shift workers if they did not work rotating and night shifts for at least six months before baseline. For participants who changed their shift work status during follow-up (N=7), only the data collected up to that point in time was included.

Respiratory infections, The incidence rate of ILI/ARI episodes was used as measure for infection susceptibility (6). The occurrence of ILI/ARI episodes was measured using a smartphone application in which participants reported daily the presence or absence of the following symptoms: cough, sore throat, shortness of breath, runny/blocked nose, fever, malaise, hoarseness, and coughed-up mucus. An ILI/ARI episode was defined as having >2 symptoms on the same day or >1 symptom on two consecutive days (6). An episode ended when no symptoms were reported for two consecutive days. Sneezing and wheezing were also included in the symptoms list, but these symptoms were excluded from the definition of ILI/ARI to prevent classifying allergy symptoms as ILI/ARI. The application was initially developed to detect parent-reported cases of ILI in children and it appeared to be a useful tool for prospective studies. For the Klokwerk+ study, it was further adjusted to make it applicable for the measurement of ILI/ARI among adults (6).

Potential mediators: sleep, physical activity and diet, To determine sleep and physical activity levels, participants were instructed to wear triaxial accelerometers (Actigraph GT3X devices, Actigraph, Pensacola, FL, USA) that were taped to their thigh, for 24 hours/day, for seven consecutive days (18). During these days, participants kept a diary in which work, sleep, and non-wear times were reported. Data from the accelerometers was analyzed using Acti4 software (NRCWE, Copenhagen, Denmark, and BAuA, Berlin, Germany). This software uses validated algorithms to estimate the time spent in the following body postures and physical activity types: sedentary (sitting/lying), standing, walking, running, stairclimbing, and cycling (19). First, periods not covered in the diary and non-wear time were excluded from data analysis. Next, based on sleep onset and offset times reported in the diary and corresponding sedentary periods in the accelerometer data, sleep duration was calculated. For every participant, the number of days with short (<7 hours/days) and long (>9 hours/ day) sleep duration were assessed (20, 21). Because not all participants wore the accelerometers for the same amount of time, the percentage of days with short and long sleep duration was calculated. Sleep quality was measured using one question from the Pittsburgh Sleep Quality Index (PSQI) in which participants were asked to indicate how they would rate their overall sleep quality in the past month (very good, fairly good, fairly poor, very poor) (22). This measure was dichotomized as fairly/very poor versus fairly/very good sleep quality. To determine physical activity levels, time spent in the different physical activity types during waking hours was assessed with the accelerometer data and expressed as percentage of the total time (18). Percentages of walking, running, stairclimbing, and cycling were combined to form one measure for physical activity during leisure and one measure for physical activity at work.

To assess dietary behaviors, participants kept a food diary for three consecutive days (23). In the food diary, the type and amount of consumed foods and drinks were reported, as well as the time of day at which these were consumed. Within the Klokwerk+ study, the focus was on the frequency of eating episodes in order to compare meal patterns and meal balance between shift and non-shift workers. Therefore, the validated Food-Based Classification of Eating Episodes (FBCE) was used to classify the eating episodes of the participants (24). In short, based on the combination of products (eg, animal protein, starch, vegetables, fruits, fats, sugars) consumed at one moment in time, the number of consumed meals and snacks per day was determined (23).

Covariates

The following factors were important covariates in the association between shift work and respiratory infections based on earlier analyses (6) and were therefore included in the mediation model as potential confounders: age, gender, occupation (nurse versus other healthcare worker), influenza vaccination status (ie, whether participants received the seasonal influenza vaccine), and general perceived health [measured on a 5-point Likert scale (excellent-bad)].

Statistical analysis

Differences between shift and non-shift workers in ILI/ ARI incidence rate, potential mediators, and covariates were calculated using independent t- and chi-square tests.

Because the continuous mediators did not show a linear relation with ILI/ARI incidence rate, they were dichotomized based on the median. For consistency, the same cutoffs were used for the same lifestyle behaviors. The cutoffs were >33% versus <33% of days having a short (<7 hours/day) and long (>9 hours/day) sleep duration, >12% versus <12% of leisure/working time being physically active (ie, walking, running, stairclimbing, and cycling), and >3 versus <3 meals/snacks per day.

The mediation analysis of sleep, physical activity, and diet as mediators of the effect of shift work on ILI/ ARI incidence rate was conducted using structural equation modeling (SEM). SEM was chosen to construct the multiple mediation model because it is an efficient method to examine the pathways of different potential mediators simultaneously. The upper part of figure 1 shows the model of the total effect (c) of shift work on ILI/ARI incidence rate. The lower part shows the multiple mediation model of the indirect effects of sleep (a1-3, b1-3), physical activity (a4-5, b4-5), and diet (a6-7, b6-7), as well as the direct effect of shift work (c') on ILI/ARI incidence rate that is independent of the included mediators and other covariates.

To calculate the estimates for the different paths, SEM was conducted with negative binomial regression for the c-path, b-paths, and c'-path to ILI/ARI incidence rate with the number of completed diaries as an offset variable (25), and with logistic regression for the a-paths to the dichotomous mediators. The indirect effect of each mediator was calculated as the product of the a-path and b-path (26). Next, a 95% bootstrap confidence interval (CI) using 5000 bootstrap resamples was calculated for each indirect effect to determine whether mediation was statistically significant (26, 27). The indirect effects and bootstrap CI were calculated using the untransformed regression coefficients of the logistic (a-paths) and negative binomial (b-paths) regression analyses. Subsequently, the results were back-transformed to create incidence rate ratios (IRR) by taking e (base of the natural logarithm) raised to the power of the regression coefficients.

In total, 396 participants (67%) had complete data on all potential mediators, ILI/ARI incidence rate, and covariates, and were therefore included in the current mediation analysis. Because there was a limited number of accelerometers available to measure sleep and physical activity, most cases of missing data were due to the fact that participants did not receive an accelerometer. To avoid possible bias due to exclusion of participants, missing values on sleep, physical activity, and diet were also imputed using multiple imputation with 33 imputation datasets (33% of participants had incomplete data) (28) in a sensitivity analysis.

SEM analyses were performed using Stata/SE, version 14.2 (StataCorp LLC, College Station, TX, USA) and multiple imputation was conducted using IBM SPSS Statistics, version 24.0 (IBM Corporation, Armonk, NY, USA).

Results

Study population

Of the 611 healthcare workers who enrolled in the Klokwerk+ study, 589 participants - who neither changed shift work status during follow-up nor stopped working in shifts in the 6 months before baseline - were included in the analysis of the association between shift work and ILI/ARI incidence rate in our earlier study (6). In the current study, 396 participants with complete data on sleep, physical activity, and diet were included for the multiple mediation analysis based on complete cases. Shift workers were younger [40.4 (standard deviation (SD) 12.1) years versus 47.0 (SD 10.4) years] and more often nurses (81.3% versus 33.3%) than non-shift workers (table 1). Shift workers reported on average more ILI/ARI episodes [3.5 (SD 2.4) episodes] than non-shift workers [2.9 (SD 1.8) episodes]. The largest differences between shift and non-shift workers in sleep, physical activity, and diet were found in short sleep duration and physical activity at work. Table 1 shows that 30.3% of shift workers frequently had a short sleep duration compared to 13.0% of non-shift workers. Furthermore, 53.5% of shift workers were highly physically active at work, compared to 24.6% of non-shift workers.

Multiple mediation model

Similar as reported previously for the total study population (6), shift workers had a higher incidence rate of ILI/ARI than non-shift workers in this subpopulation of Klokwerk+. The total effect of shift work on ILI/ ARI incidence rate was 0.205 (95% CI 0.010-0.401), adjusted for covariates. This indicates that shift workers had a 23% [incidence rate ratio (IRR)=e°.205=1.23, 95% CI 1.01-1.49] higher incidence rate of ILI/ARI than non-shift workers [figure 2, table 2, supplementary material (www.sjweh.fi/show_abstract.php?abstract_ id=3896) table S1]. After adding the potential mediators to the model, the direct effect of shift work on ILI/ARI incidence was 0.138 (95% CI -0.062-0.338) (IRR 1.15, 95% CI 0.94-1.40). Compared to non-shift workers, shift workers had a 2.84 times higher odds of frequently having a short sleep duration (95% CI 1.26-6.39) and a 3.19 times higher odds of having poor sleep quality (95% CI 1.27-8.01) (table 2). The odds ratio (OR) of having a high physical activity level at work was also higher in shift workers compared to non-shift workers (OR 2.80, 95% CI 1.47-5.34). Regarding the associations between the potential mediators and ILI/ARI incidence rate (b-paths), table 2 shows that self-reported poor sleep quality (IRR 1.25, 95% CI 1.05-1.47) was associated with a statistically significantly higher ILI/ ARI incidence rate.

The indirect effects of sleep, physical activity, and diet on the association between shift work and ILI/ARI incidence rate are presented in table 3. Shift workers had a 29% higher ILI/ARI incidence rate than non-shift workers via self-reported poorer sleep quality (IRR 1.29, 95% CI 1.02-1.95). The indirect effects of short (IRR 1.05, 95% CI 0.89-1.31) and long (IRR 0.99, 95% CI 0.91-1.06) sleep duration, physical activity during leisure (IRR 1.00, 95% CI 0.95-1.06) and at work (IRR 1.07, 95% CI 0.92-1.28), and number of meals (IRR 1.01, 95% CI 0.91-1.13) and snacks (IRR 1.04, 95% CI 0.97-1.16) were small and not statistically significant.

Missing data on sleep, physical activity, and diet was imputed to conduct the multiple mediation model on the total study population (N=589). Supplementary figure S1 and table S2 show similar path coefficients and indirect effects for imputed data analysis as for complete case analysis.

Discussion

The aim of the current study was to examine the mediating role of sleep, physical activity, and diet in the association between shift work and respiratory infections, defined as ILI/ARI incidence rate. Healthcare shift workers had a 23% higher incidence rate of ILI/ ARI than non-shift workers. After including the potential mediating factors, this higher incidence rate of shift workers compared to non-shift workers reduced to 15%, and did not remain statistically significant. The association between shift work and ILI/ARI incidence rate was mediated by poorer sleep quality among shift workers.

Among this group of healthcare workers, shift work was associated with a higher incidence rate of ILI/ ARI than non-shift workers. Correspondingly, evidence from a recent mechanistic study in mice suggests that shift workers who experience circadian disruption may be at increased risk for respiratory infections (29). Two epidemiological studies that examined the relation between shift work and infectious diseases also found an increased incidence of infectious diseases among shift workers (30, 31), but one study reported that infectious diseases were more common among non-shift workers (32). Nonetheless, these three previous studies had cross-sectional designs, and more prospective studies are needed to give further insight into the association between shift work and infectious diseases.

The largest indirect effect was found for self-reported poor sleep quality, with shift workers having 29% more ILI/ARI episodes via a poorer sleep quality. Shift workers more often reported a very or fairly poor sleep quality in the past month than non-shift workers, and poor sleep quality was statistically significantly associated with an increased ILI/ARI incidence rate. Similarly, although no mediation analysis was performed, in an earlier study among employees with different work schedules, shift workers had worse sleep quality and a higher prevalence of common infections compared to day workers (31). Together, these results indicate that sleep quality may be an important mechanistic factor of increased infection susceptibility among shift workers. Immunological mechanisms could potentially explain the connection between sleep and infection risk, as sleep is likely to modulate immune function (14, 33). A study among Japanese workers also concluded that sleep quality, and not just quantity, may be associated with alterations in white blood cell count (34). Based on the results of the current study, a focus on improving perceived sleep quality among shift workers may be recommended. Nonetheless, to increase the impact of a focus on sleep quality in prevention strategies, future research should confirm the findings of the current study and examine if sleep quality also mediates the association between shift work and other health outcomes.

In the current study, the perceived quality of sleep seemed to play a larger role in infection susceptibility of shift workers than objectively measured sleep duration. Both short and long sleep duration were not mediators in the association between shift work and respiratory infections. Prior reviews have concluded that shift work is associated with a decrease in sleep duration (35, 36). We also found that shift workers more often had a short sleep duration. Because a long sleep duration was also more frequently observed among shift workers in table 1, it becomes apparent that especially a regular sleep duration of 7-8 hours/day is often lacking in shift workers. With respect to the association between mediator and outcome, short as well as long sleep duration was not associated with ILI/ARI incidence rate. Nonetheless, earlier studies among healthy adults found an increased susceptibility to common cold and pneumonia among short sleepers (37-39). Regarding long sleep duration, a recent review concluded that there is currently not enough evidence to link long sleep duration to infection risk (33). An explanation for the fact that we did not observe a relation between short sleep duration and ILI/ARI may be that, in two of these earlier studies, the occurrence of infection was experimentally induced and sleep was monitored prior to administration of the virus (37, 39), while our approach was to measure shift workers' general sleep duration and monitor subsequent naturally occurring infections. As general sleep duration may not be very representative for sleep duration prior to naturally occurring infections, we may have underestimated the effect of short sleep duration on ILI/ARI incidence. Therefore, it would be interesting to study also more acute effects of engaging in shift work, its immediate effects on sleep duration, and the possible subsequent increase in infection episodes.

Based on the results of the current study, we found no evidence that physical activity is an underlying mediator linking shift work and ILI/ARI incidence rate. As found earlier, physical activity levels of shift and non-shift workers during leisure were similar, but shift workers were more physically active at work (18). However, an association between physical activity and ILI/ARI incidence rate, and thus the association between mediator and outcome, was lacking. Correspondingly, a Cochrane systematic review did not find an association between moderate-intense physical activity and ARI (40). Diet (ie, number of meals and snacks) also did not mediate the association between shift work and ILI/ARI incidence rate. However, meal and snack frequency is only one aspect of dietary habits. As different micronutrients (eg, vitamins) are required for an efficient immune response (15), it may be relevant for future studies to take into account these dietary factors as potential mediators in the association between shift work and infectious diseases. Nonetheless, snacking behavior and meal consumption may still be important mediators in those health effects of shift work that more strongly depend on the energy balance, such as obesity and cardiovascular diseases (3).

Strengths and limitations

In the current study, a daily diary application on a smartphone was used to measure ILI/ARI incidence during an entire winter season, resulting in 92% of all possibly completed diaries being completed (6). The reported incidence of ILI/ARI in this study was considerably higher than incidences derived from the traditional Dutch surveillance method, because an infection episode did not have to be confirmed by a clinician (6). Nonetheless, for the aim of comparing incidence rates between shift workers and non-shift workers, we believe our data are suitable.

Another strength is that multiple potential behavioral mediators were included, because the relation between shift work and health is likely to be multifactorial. Yet, sleep quality was the only relevant mediator in the association between shift work and ILI/ARI incidence rate, and, although not statistically significant, the ILI/ARI incidence rate of shift workers compared to non-shift workers was still 15% higher after including potential mediators. Therefore, studies with larger sample sizes that include more and different potential mediators (eg, psychosocial and physiological factors, such as stress, light exposure, and immunological factors) may be needed to better understand underlying mechanisms linking shift work and infection susceptibility. In addition, an important assumption of the statistical model used in this study is that there is no unmeasured confounding or interaction of the exposure-mediator, mediator-outcome, and exposure-outcome relations. Currently, new mediation methodology is developing in which these assumptions can be further explored, which can be used in future work (41). As this is the first study using mediation analysis to study the role of lifestyle behaviors in the association between shift work and respiratory infections, more research is needed to replicate our findings.

To conduct mediation analysis, a longitudinal design is preferred, in which the exposure is measured before the mediator, and the mediator is measured before the outcome (41). Although in the current study, exposure and mediators were both measured at baseline, shift work exposure referred to performing shift work for >6 months prior to baseline. Therefore, we feel confident that shift work exposure was already present before the measurements of the current lifestyle mediators at baseline. Furthermore, our aim was to measure shift work as an exposure that is long-term, and more chronic in nature, and we think it is unlikely that physical activity, sleep, and diet had a significant effect on this chronic measure of shift work exposure. A strength of the current study is its prospective design in which the outcome was measured after the exposure and mediators. Nonetheless, the measurement of the outcome started immediately after the baseline measurements of the mediators, without any time-lag. To address this issue, we conducted a sensitivity analysis in which we have only included episodes of the outcome measure ILI/ ARI that occurred more than 30 days after the measurement of the mediators (supplementary figure S2 and table S3). This analysis provided the same results as the main analysis, which makes us more confident about the robustness of the findings.

In the current study, the objective measurements of the mediators were collected in a one-week time window. The use of objective instead of subjective measurements of sleep and physical activity is a strength of our study and provides us with valuable information to compare lifestyle behaviors between shift and non-shift workers on a group level. However, on the individual level, lifestyle behaviors may vary from one week to the next. For future research, it would therefore be useful to collect more repeated measurements of these lifestyle behaviors. Our aim was to study the effect of long-term, chronic shift work exposure, via general lifestyle behaviors, on ILI/ARI incidence. Nonetheless, collecting more repeated measurements of lifestyle as well as specific work schedule prior to the measurements of lifestyle, may give an opportunity to study acute effects of working night shifts on lifestyle and possibly subsequent immediate changes in respiratory infection risk. This would require a different design to take into account possible reversed causality, in which the occurrence of the infection episodes may influence lifestyle behaviors. To do so, a within-subject design comparing the effects of working night shifts and day shifts on lifestyle and infection risk within the same subjects may be suitable.

Approximately 20% of values on sleep, physical activity, and diet variables were missing. Missing data were primarily due to the fact that participants did not wear an accelerometer as there were not enough accelerometers available for all participants. There were no statistically significant differences between participants with missing data on one or more of the potential mediators (N=193) and participants without missing data (N=396) in age, gender, or any of the other covariates. Furthermore, as the results based on multiple imputed data did not differ from the complete case analysis, the impact of missing data on the results of this study is considered limited.

Lastly, the results of the current study apply to healthcare workers, and results may be different for other occupational groups.

Concluding remarks

Compared to non-shift workers, shift workers had a higher incidence rate of ILI/ARI that was partly mediated by poorer sleep quality. Although shift work was also associated with short sleep duration and more physical activity at work, these factors were not mediators in the association with ILI/ARI incidence. For future research, it may be relevant to focus on perceived sleep quality as underlying mechanism in the relation between shift work and increased infection susceptibility.

Acknowledgements

This study was funded by the Strategic Program project 24/7 Health of the Dutch National Institute for Public Health and the Environment (RIVM). The funding bodies had no role in the study design; collection, analysis, and interpretation of data; writing of the manuscript; or the decision to submit the manuscript for publication. The authors would like to thank Judith Rijnhart for her support in the statistical analysis.

Conflict of interest

The authors declare no conflicts of interest.

Protection of research participants

Approval of the study was obtained from the institutional review board of the University Medical Center Utrecht, Utrecht, The Netherlands on March 15, 2016 (study protocol number 16-044/D, NL56022.041.16). Informed consent was obtained from all participants.

Sidebar

In this study among healthworkers, the association between shift work and an increased incidence rate of respiratory infection episodes was mediated by pooprer perceived sleep quality among shift workers. To prevent respiratory infections in shift workers, it may therefore be relevant for future intervention research to focus on stategies to improve perceived sleep quality.

Loef B, van der Beek AJ, Hulsegge G, van Baarle D, Proper KI. The mediating role of sleep, physical activity, and diet in the association between shift work and respiratory infections. Scand J Work Environ Health. 2020;46(5):516-524. doi:10.5271/ sjweh.3896

Correspondence to: Karin I Proper, PhD, Center for Nutrition, Prevention and Health Services, National Institute for Public Health and the Environment; P.O. Box 1, 3720 BA Bilthoven, The Netherlands. [E-mail: karin.proper@rivm.nl]

Footnote

1 Center for Nutrition, Prevention and Health Services, National Institute for Public Health and the Environment, Bilthoven, The Netherlands.

2 Department of Public and Occupational Health, Amsterdam UMC, Vrije Universiteit Amsterdam, Amsterdam Public

Health Research Institute, Amsterdam, The Netherlands.
3 The Netherlands Organization for Applied Scientific Research, TNO, Leiden, The Netherlands.
4 Center for Immunology of Infectious Diseases and Vaccines, National Institute for Public Health and the Environment, Bilthoven, The Netherlands.
5 Department of Immunology, Laboratory for Translational Immunology, University Medical Center Utrecht, Utrecht University, Utrecht, The Netherlands.

References

References

1. European Foundation for the Improvement of Living and Working Conditions (Eurofound). Sixth European Working Conditions Survey - Overview report. Luxembourg: Publications Office of the European Union; 2016.
2. Papantoniou K, Vetter C, Schernhammer ES. Shift work practices and opportunities for intervention. Occup Environ Med 2017 Jan;74(1):2-3. https://doi.org/10.n36/ oemed-2016-103904
3. Torquati L, Mielke GI, Brown WJ, Kolbe-Alexander T. Shift work and the risk of cardiovascular disease. A systematic review and meta-analysis including doseresponse relationship. Scand J Work Environ Health 2018 May;44(3):229-38. https://doi.org/10.5271/sjweh.3700.
4. Wang F, Zhang L, Zhang Y, Zhang B, He Y, Xie S et al. Meta-analysis on night shift work and risk of metabolic syndrome. Obes Rev 2014 Sep;15(9):709-20. https://doi. org/10.1111/obr.12194.
5. Gan Y, Yang C, Tong X, Sun H, Cong Y, Yin X et al. Shift work and diabetes mellitus: a meta-analysis of observational studies. Occup Environ Med 2015 Jan;72(1):72-8. https:// doi.org/10.1136/oemed-2014-102150.
6. Loef B, van Baarle D, van der Beek AJ, Sanders EA, Bruijning-Verhagen P, Proper KI. Shift Work and Respiratory Infections in Health-Care Workers. Am J Epidemiol 2019 Mar;188(3):509-17. https://doi. org/10.1093/aje/kwy258.
7. Tsai Y, Zhou F, Kim IK. The burden of influenza-like illness in the US workforce. Occup Med (Lond) 2014 Jul;64(5):341-7. . https://doi.org/10.1093/occmed/kqu022
8. Knutsson A. Health disorders of shift workers. Occup Med (Lond) 2003 Mar;53(2):103-8. https://doi.org/10.1093/ occmed/kqg048.
9. Merkus SL, Holte KA, Huysmans MA, van Mechelen W, van der Beek AJ. Nonstandard working schedules and health: the systematic search for a comprehensive model. BMC Public Health 2015 Oct;15:1084. https://doi. org/10.1186/s12889-015-2407-9.
10. Puttonen S, Härmä M, Hublin C. Shift work and cardiovascular disease - pathways from circadian stress to morbidity. Scand J Work Environ Health 2010 Mar;36(2):96-108. https://doi.org/10.5271/sjweh.2894.
11. Atkinson G, Davenne D. Relationships between sleep, physical activity and human health. Physiol Behav 2007 Feb;90(2-3):229-35. https://doi.org/10.1016/j. physbeh.2006.09.015.
12. Boivin DB, Boudreau P. Impacts of shift work on sleep and circadian rhythms. Pathol Biol (Paris) 2014 Oct;62(5):292301. https://doi.org/10.1016/j.patbio.2014.08.001.
13. Souza RV, Sarmento RA, de Almeida JC, Canuto R. The effect of shift work on eating habits: a systematic review. Scand J Work Environ Health 2019 Jan;45(1):7-21. https:// doi.org/10.5271/sjweh.3759.
14. Almeida CM, Malheiro A. Sleep, immunity and shift workers: A review. Sleep Sci 2016 Jul-Sep;9(3):164-8. https://doi.org/10.1016/j.slsci.2016.10.007.
15. Calder PC, Yaqoob P. Nutrient regulation of the immune response. In: Erdman Jr JW, MacDonald IA, Zeisel SH, editors. Present knowledge in nutrition. 10th ed. Hoboken, USA: John Wiley &Sons; 2012. p. 688-708.
16. Shephard RJ. Special feature for the Olympics: effects of exercise on the immune system: overview of the epidemiology of exercise immunology. Immunol Cell Biol 2000 Oct;78(5):485-95. https://doi. org/10.1111/j.1440-1711.2000.t01-1-.x.
17. Loef B, van Baarle D, van der Beek AJ, van Kerkhof LW, van de Langenberg D, Proper KI. Klokwerk + study protocol: an observational study to the effects of night-shift work on body weight and infection susceptibility and the mechanisms underlying these health effects. BMC Public Health 2016 Aug;16:692. https://doi.org/10.1186/s12889-016-3317-1.

18. Loef B, van der Beek AJ, Holtermann A, Hulsegge G, van Baarle D, Proper KI. Objectively measured physical activity of hospital shift workers. Scand J Work Environ Health 2018 May;44(3):265-73. https://doi.org/10.5271/sjweh.3709.
19. Skotte J, Korshøj M, Kristiansen J, Hanisch C, Holtermann A. Detection of physical activity types using triaxial accelerometers. J Phys Act Health 2014 Jan;11(1):76-84. . https://doi.org/10.1123/jpah.2011-0347.
20. Hulsegge G, Loef B, van Kerkhof LW, Roenneberg T, van der Beek AJ, Proper KI. Shift work, sleep disturbances and social jetlag in healthcare workers. J Sleep Res 2019 Aug;28(4):e12802. https://doi.org/10.1111/jsr.12802.
21. Watson NF, Badr MS, Belenky G, Bliwise DL, Buxton OM, Buysse D et al.; Consensus Conference Panel. Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society on the Recommended Amount of Sleep for a Healthy Adult: methodology and Discussion. Sleep (Basel) 2015 Aug;38(8):1161-83. https:// doi.org/10.5665/sleep.4886.
22. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989 May;28(2): 193-213. https://doi.org/10.1016/0165-1781(89)90047-4.
23. Hulsegge G, Loef B, Benda T, van der Beek AJ, Proper KI. Shift work and its relation with meal and snack patterns among healthcare workers. Scand J Work Environ Health 2020 Mar;46(2):143-51. https://doi.org/10.5271/sjweh.3829.
24. Lennernäs M, Andersson I. Food-based classification of eating episodes (FBCE). Appetite 1999 Feb;32(1):53-65. https://doi.org/10.1006/appe.1998.0196.
25. Christensen KB, Andersen PK, Smith-Hansen L, Nielsen ML, Kristensen TS. Analyzing sickness absence with statistical models for survival data. Scand J Work Environ Health 2007 Jun;33(3):233-9. https://doi.org/10.5271/sjweh.1132.
26. Sobel ME. Asymptotic confidence intervals for indirect effects in structural equation models. Sociol Methodol 1982;13:290-312. https://doi.org/10.2307/270723.
27. Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav Res Methods 2008 Aug;40(3):879-91. https://doi.org/10.3758/BRM.40.3.879.
28. White IR, Royston P, Wood AM. Multiple imputation using chained equations: issues and guidance for practice. Stat Med 2011 Feb;30(4):377-99. https://doi.org/10.1002/ sim.4067.
29. Sengupta S, Tang SY, Devine JC, Anderson ST, Nayak S, Zhang SL et al. Circadian control of lung inflammation in influenza infection. Nat Commun 2019 Sep;10(1):4107. https://doi.org/10.1038/s41467-019-11400-9.
30. Boden K, Brasche S, Straube E, Bischof W. Specific risk factors for contracting Q fever: lessons from the outbreak Jena. Int J Hyg Environ Health 2014 Jan;217(1):110-5. https://doi.org/10.1016/j.ijheh.2013.04.004.
31. Mohren DC, Jansen NW, Kant IJ, Galama J, van den Brandt PA, Swaen GM. Prevalence of common infections among employees in different work schedules. J Occup Environ Med 2002 Nov;44(11):1003-11. https://doi.org/10.1097/00043764-200211000-00005.
32. Puttonen S, Viitasalo K, Härmä M. Effect of shiftwork on systemic markers of inflammation. Chronobiol Int 2011 Jul;28(6):528-35. https://doi.org/10.3109/07420528.2011.5 80869.
33. Besedovsky L, Lange T, Haack M. The Sleep-Immune Crosstalk in Health and Disease. Physiol Rev 2019 Jul;99(3):1325-80. https://doi.org/10.1152/ physrev.00010.2018.
34. Nishitani N, Sakakibara H. Subjective poor sleep and white blood cell count in male Japanese workers. Ind Health 2007 Apr;45(2):296-300. https://doi.org/10.2486/ indhealth.45.296.
35. Pilcher JJ, Lambert BJ, Huffcutt AI. Differential effects of permanent and rotating shifts on self-report sleep length: a meta-analytic review. Sleep 2000 Mar;23(2):155-63. . https://doi.org/10.1093/sleep/23J2.1b
36. Sallinen M, Kecklund G. Shift work, sleep, and sleepiness - differences between shift schedules and systems. Scand J Work Environ Health 2010 Mar;36(2):121-33. https://doi. org/10.5271/sjweh.2900.
37. Cohen S, Doyle WJ, Alper CM, Janicki-Deverts D, Turner RB. Sleep habits and susceptibility to the common cold. Arch Intern Med 2009 Jan;169(1):62-7. https://doi. org/10.1001/archinternmed.2008.505.

38. Patel SR, Malhotra A, Gao X, Hu FB, Neuman MI, Fawzi WW. A prospective study of sleep duration and pneumonia risk in women. Sleep (Basel) 2012 Jan;35(1):97-101. https://doi.org/10.5665/sleep.1594.
39. Prather AA, Janicki-Deverts D, Hall MH, Cohen S. Behaviorally Assessed Sleep and Susceptibility to the Common Cold. Sleep (Basel) 2015 Sep;38(9):1353-9. https://doi.org/10.5665/sleep.4968.
40. Grande AJ, Keogh J, Hoffmann TC, Beller EM, Del Mar CB. Exercise versus no exercise for the occurrence, severity and duration of acute respiratory infections. Cochrane Database Syst Rev 2015 Jun;(6):CD010596. https://doi. org/10.1002/14651858.CD010596.pub2.
41. Oude Groeniger J, Burdorf A. Advancing mediation analysis in occupational health research. Scand J Work Environ Health 2020 Mar;46(2):113-6. https://doi.org/10.5271/ sjweh.3886.
Received for publication: 3 December 2019

DETAILS

Subject:	Infections; Shift work; Behavior; Software; Physical activity; Food; Workers; Medical personnel; Diaries; Questionnaires; Influenza; Diet; Sleep; Mediation; Regression analysis; Multivariate statistical analysis; Illnesses; Accelerometers; Sleep deprivation; Smartphones; Respiratory tract infection
Business indexing term:	Subject: Shift work Workers Mediation Smartphones
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	516-524,516A
Publication year:	2020
Publication date:	2020
Section:	Original article
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English

Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3896
ProQuest document ID:	2444686312
Document URL:	https://www.proquest.com/scholarly-journals/mediating-role-sleep-physical-activity-diet/docview/2444686312/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2023-08-04
Database:	Public Health Database

Metalworking fluids and cancer mortality in a US autoworker cohort (1941–2015)

Costello, Sadie, PhD; Chen, Kevin, MPH; Picciotto, Sally, PhD; Lutzker, Liza, MPH; Eisen, Ellen, ScD

ABSTRACT (ENGLISH)

Objectives This report describes the extended follow-up (1941-2015) of a cohort of 38 549 automobile manufacturing workers with potential exposure to metalworking fluids (MWF). The outcomes of interest were mortality from cancers of the esophagus, stomach, intestine, rectum, bladder, liver, pancreas, larynx, lung, skin, prostate, brain, and female breast, as well as leukemia. This report includes 5472 deaths from cancer, more than ten times the numbers of deaths in our last summary report published 20 years ago. Methods Standardized mortality ratios were computed for the entire study period. Adjusted hazard ratios (HR) were estimated in Cox proportional hazard models with categorical variables for cumulative exposure to each type of MWF. Results Exposure-response patterns are consistent with prior mortality reports from this cohort. We found increased risk of skin and female breast cancer with straight fluids. For the first time, we found elevated risk of stomach cancer mortality. Overall, many of the exposure-response results did not suggest an association with MWF. Conclusions Mortality is a poor proxy for cancer diagnosis for treatable cancers and not the optimal outcome measure in etiological studies. Although the HR presented here handle bias from the healthy worker hire effect and left truncation, they do not handle bias from healthy worker survivor effect, which likely results in underestimates of the health impacts of MWF. Although this updated summary provides some information on the risk of cancer from MWF, targeted future analyses will help clarify associations.

FULL TEXT

Headnote

Objectives This report describes the extended follow-up (1941-2015) of a cohort of 38 549 automobile manufacturing workers with potential exposure to metalworking fluids (MWF). The outcomes of interest were mortality from cancers of the esophagus, stomach, intestine, rectum, bladder, liver, pancreas, larynx, lung, skin,

prostate, brain, and female breast, as well as leukemia. This report includes 5472 deaths from cancer, more than ten times the numbers of deaths in our last summary report published 20 years ago.
Methods Standardized mortality ratios were computed for the entire study period. Adjusted hazard ratios (HR) were estimated in Cox proportional hazard models with categorical variables for cumulative exposure to each type of MWF.
Results Exposure-response patterns are consistent with prior mortality reports from this cohort. We found increased risk of skin and female breast cancer with straight fluids. For the first time, we found elevated risk of stomach cancer mortality. Overall, many of the exposure-response results did not suggest an association with MWF.
Conclusions Mortality is a poor proxy for cancer diagnosis for treatable cancers and not the optimal outcome measure in etiological studies. Although the HR presented here handle bias from the healthy worker hire effect and left truncation, they do not handle bias from healthy worker survivor effect, which likely results in underestimates of the health impacts of MWF. Although this updated summary provides some information on the risk of cancer from MWF, targeted future analyses will help clarify associations.
Key terms auto manufacturing; cohort analysis; cohort study; Cox model; exposure; standardized mortality ratio.
Metalworking fluids (MWF) are complex mixtures of oils and chemical additives widely used to cool and lubricate metal machining operations. MWF are aerosolized when sprayed, generating airborne particulate matter (PM) at concentrations up to two orders of magnitude higher than allowable by the US ambient air pollution standards (1). Classified as straight (mineral oils), soluble (oils emulsified in water), or synthetic (without oils), MWF continue to pose a potential hazard to millions of workers in automobile manufacturing as well as other metal machining jobs related to electronics manufacturing, new technologies, and alternative energy. Some MWF constituents are carcinogenic in animals, including N-nitrosamines (2) found in water-based synthetic fluids and some polycyclic aromatic hydrocarbons (PAH) (3) found in the oil-based fluids. Efforts to reduce exposures to these potentially carcinogenic MWF have been ongoing for decades. Removal of PAH from MWF began in the 1950s when large industrial users began shifting to more refined oils, and US Environmental Protection Agency (EPA) regulations during the 1980s were directed at reducing nitrosamine exposures (4). In 1998, the National Institute of Occupational Safety and Health (NIOSH) released a criteria document with a recommended exposure limit (REL) for occupational exposure to MWF of 0.5 mg/m3 for total PM (TPM) and 0.4 for respirable PM (5).
Several reviews of the evidence on MWF and cancer followed the NIOSH Criteria Document (4, 6-9). Calvert et al (4) summarized the evidence basis of the NIOSH report on cancer risk among workers exposed to MWF, concluding that there was substantial evidence for increased risk at several sites, including larynx, rectum, pancreas, skin, scrotum, and bladder, associated with at least some MWF. Savitz (6) concluded that evidence was strongest for associations between cancers of the larynx and rectum in relation to the oil-based fluids. Mirer (7, 8) noted positive results for stomach cancer in older studies and internal analyses of labor union [United Autoworkers (UAW)] data without quantitative exposure information, as well as for lung, liver, pancreatic, and laryngeal cancer, and for leukemia. In all reviews, attention was focused on air and skin exposure to the oil-based MWF in use before the oils became more highly refined in the mid-1970s. Most of the quantitative evidence cited in all the reviews came from the ongoing UAW-General Motors (GM) cohort study (10).
The UAW-GM study was jointly funded by labor and management as a cancer mortality study with an extensive exposure assessment component, motivated by worker concerns about digestive and respiratory cancers in relation to MWF exposure. Standardized mortality ratios (SMR) have been reported twice for this cohort, the first based on the original end of follow-up in 1985 and the second based on extended follow-up to 1995 (10, 11). SMR for the two outcomes of original interest, stomach and lung cancer, were not elevated in either report. A series of results from exposure-response analyses have also been reported based on the extensive historical exposure assessment for straight, soluble and synthetic MWF. Results based on Cox proportional hazard models for digestive and respiratory cancer mortality in relation to MWF exposures have been largely null (11-15). However, results based on cancer incidence in this cohort have been more mixed. There is modest evidence that exposure to straight, oil-based MWF increases the risk of laryngeal (14, 16), bladder (17), melanoma (18), breast (19), and colon (20), cancer incidence.

Limited evidence was also reported for increased risk of cervical cancer (21) and breast cancer in younger women (19).

In 2003, the UAW petitioned the Occupational Safety and Health Administration (OSHA) for a temporary standard for MWF with an exposure limit of 0.5 mg/m3 TPM. The petition was based on the evidence for nonmalignant respiratory health effects of MWF, asthma and hypersensitivity pneumonitis, rather than for cancer. OSHA ultimately denied the petition (6). The UAW's decision to petition for regulatory efforts based on nonmalignant health effects suggests that in 2003, there was insufficient evidence that MWF are carcinogenic at concentrations found in the workplace. Yet, a recent risk assessment for cancer and MWF based entirely on published results from the UAW-GM cohort study, concluded that substantial risk exists at 0.1 mg/m3 respirable PM, one quarter of the current NIOSH REL (and the internal GM limit) (9). The cancer sites contributing the most attributable cases were larynx, esophagus, brain, breast and cervix.

In summary, the literature to date suggests that oil- and water-based MWF may indeed cause increased risk of several specific cancers, although none of the evidence is conclusive. In this context, we report results from an extended vital status follow-up, from 1941 to 2015 - 20 years beyond the last reported mortality follow-up of the UAW-GM cohort.

Methods

Details regarding the UAW-GM cohort mortality study have been described extensively in previous publications (10, 11, 22-24). Here, we describe the methods in brief.

Study population

The present study of the UAW-GM cohort includes all hourly workers identified through company records at three automobile manufacturing plants in Michigan who worked for >3 years and were hired between 1 January 1938 and 31 December 1981. After excluding the 4% of subjects missing more than half of their employment history, 38 549 were included in this analysis. Follow-up for mortality now extends from 1941 to 2015, 21 years longer than the previous update (11) and includes more than 1.5 million person-years. Over the 74 years of follow-up, 53% of the study population has died. Subjects were considered lost to follow-up upon reaching the oldest observed age at death (106 years). By this definition, <0.5% of the participants were lost to follow-up.

Covariates

Subject characteristics, including year of birth, sex (male or female), race (white, black, or unknown), and worksite (plant 1, 2, or 3) were obtained from company records. Subjects with unknown race (22%) were assumed to be white in this analysis based on available demographics (10). In a sensitivity analysis, subjects at plant 1 with missing race were assumed to be black.

Exposure

Exposure assessment has been described in previous publications (23-25). Quantitative exposure to MWF was based on several hundred personal and area sizeselective samples for PM (mg/m3) collected across jobs and departments by the research team, in combination with historical industrial hygiene records. Scale factors were applied to estimate historical levels of exposure relative to baseline measurements made by the research industrial hygienists (mid 1980s) (23). These scale factors reflect the dramatic decreases in exposure concentrations over the second half of the 20th century, particularly in the early 1970s with the passage of the Occupational Safety and Health Act.

MWF exposures were assigned to individuals according to job and department and calendar time, weighted by work time. Missing exposure data were interpolated for those missing less than half of their work history. The exposure-response models considered exposure to straight, soluble, and synthetic MWF measured as cumulative exposure to TPM. The work history records were initially collected in 1985 and extended up to 1995. Exposure-response models for this analysis are based on cumulative MWF exposure (mg/m3-years) lagged by 21 years; lagging accounts for disease latency and is necessitated by the available data.

Outcome

Data on vital status and cause of death were obtained through the Social Security Administration, the National Death

Index, company records, death certificates, and state mortality files (10). Causes of death were selected for exposure-outcome modeling based on the previous report on cancer mortality in this cohort in 2001 (11).

Analytic methods

Person-years were accumulated from three years after hire until death, end of follow-up, or the maximum observed age at death. Causes and dates of death were obtained from company records, the Social Security Administration, death certificates, state mortality files, and the National Death Index. Underlying causes of death were coded conforming to the International Classification of Diseases, revisions 9 and 10 [ICD-9 and ICD-10, respectively; see the supplementary material (www.sjweh.fi/show_abstract.php?abstract_id=3898) for ICD coding). Where possible, these ICD codes were mapped to cause of death descriptions according to the keys used in the Lifetable Analysis System (LTAS) (2628). SMR were computed for cancer outcomes, as well as several chronic diseases and external causes of death. Reference rates for deaths prior to 2010 were extracted from LTAS; reference rates for deaths in or after 2010 were obtained through the CDC Underlying Cause of Death database (29, 30) and SMR were calculated using R version 3.6.1 (R Core Team, Vienna Austria).

We estimated associations between cumulative exposure to straight, soluble, and synthetic MWF and each cancer outcome as adjusted hazard ratios (HR) in Cox proportional hazards models with age as the timescale. In addition to age, all models included year of hire, race, sex, and plant, as well as time-varying calendar year and the other MWF exposures to adjust for potential confounding. Cumulative exposures to the three MWF were categorized with a pre-determined reference group. Zero exposure was the reference group for straight and synthetic fluid. For soluble exposures, a more ubiquitous exposure in this cohort, the upper bound of the reference group was set to 0.05 mg/m3 to avoid extremely small numbers of cancer cases in the reference group and thereby increase stability of the HR estimates. This cut-off is approximately 1% of what cumulative exposure would be after ten years at the NIOSH REL. To maximize statistical efficiency, we used the distribution of exposure to each fluid type among the cases of each cancer to determine the cut points for the exposed categories.

Results

A summary of the study population characteristics is presented in table 1. Over half of this predominantly white and male cohort had died by the end of follow up in 2015. While at work, approximately half of the workers had been exposed to straight fluids, a third to synthetics, and a majority (82%) were exposed to soluble fluids. Although only a quarter of the workforce was employed at plant 1, most of the cohort members categorized as black worked at this urban plant (data not shown). Results are presented as SMR as well as adjusted HR, estimated in Cox models based on quantitative exposure estimates for each fluid type.

Standardized mortality ratios

SMR are presented for specific cancers and other major causes of death in table 2. The SMR for all causes of death combined was <1.0. This was driven by the low SMR for all heart disease (SMR=0.75) as well as nonmalignant respiratory diseases (SMR=0.84) and cerebrovascular disease (SMR=0.83). The SMR for all cancers was also <1.0. Although the majority of the SMR for specific cancers were <1.0, the SMR for some digestive and respiratory cancers was elevated including for esophageal (SMR=1.06), stomach (SMR=1.10), pancreatic (SMR=1.05), laryngeal (SMR=1.17) and lung (SMR=1.07) cancers. The SMR for lung cancer was the only one that was positive and statistically significant.

Proportional hazards models

Our primary focus was on cancers of the digestive and respiratory systems: esophageal, stomach, rectal, lung, and larynx cancers; we also present models for pancreas, prostate, female breast, and skin cancers and for leukemia, based on previously elevated SMR. The adjusted HR for these cancers and cumulative exposure to straight, soluble, and synthetic MWF are presented in figures 1, 2, and 3, respectively. (See supplementary tables S2-4.)

The estimated exposure-response pattern for cumulative straight fluid was non-monotonic for all cancers except skin and breast cancer. In the highest exposure categories, skin cancer rose to a HR of 1.32 [95% confidence interval (CI) 0.67-2.58] and breast cancer to 2.13 (95% CI 1.04-4.39). Notably, the HR for stomach cancer was also highest in the highest category and rose to 1.86 (95% CI 1.17-2.97). The HR were mostly elevated for esophageal, liver, pancreatic, and prostate cancer in response to straight fluid exposure, but generally below the null for lung, colon, rectal, bladder, and brain cancers and leukemia.

The exposure-response patterns for exposure to cumulative soluble fluid were non-monotonic for all cancers. The HR for rectal cancer rose to 2.18 (95% CI 1.07-4.48) in the middle category. The HR were mostly elevated for esophageal, bladder, skin, and brain cancers in relation to soluble fluid, but generally below the null for laryngeal, lung, stomach, colon, liver, pancreatic, prostate, and breast cancers.

The exposure-response patterns for exposure to cumulative synthetic fluid were monotonic for esophageal, rectal, and prostate cancers and leukemia. The HR in the highest category was 1.39 (95% CI 0.84-2.30) for esophageal cancer, 1.64 (0.79-3.41) for rectal cancer, 1.30 (95% CI 0.89-1.89) for prostate cancer, and 1.37 (95% CI 0.86-2.19) for leukemia. In addition, the HR were generally elevated in response to cumulative synthetic fluid for laryngeal and brain cancers and below the null for colon, pancreatic, bladder, skin, and breast cancers.

Results did not change when we classified people with unknown race as either white or black in plant 1 (data not shown).

Discussion

This updated report includes almost 5500 deaths from cancer, more than ten times the number of cancer deaths in our last summary report published almost 20 years ago. Most of the patterns reported here are consistent with that previous summary, as well as with results of cancer-specific papers published from this cohort during the interim and suggest that a malignancy-based OHSA standard for MWF would be appropriately health protective. For example, increasing straight fluid exposure was associated with increased risk of skin and female breast cancers. Interestingly, for the first time in this cohort, we report an increase in stomach cancer mortality with increasing straight fluid exposure, which was the original hypothesis motivating this cohort study. Although there are some suggestions of increased risk that we will explore in targeted analyses, many exposure-response results do not suggest any association. It is certainly possible that MWF simply do not predict many of these cause-specific cancers; however, there are also limitations which can lead to attenuation, including using mortality as a surrogate outcome for cancer diagnosis, a lack of data on potential confounders, such as smoking, and the healthy worker survivor effect.

Mortality may be a reasonable proxy for diagnosis for cancers with a poor 5-year survival rate, such as lung or pancreatic cancer. However, many cancers have become more highly treatable over the 75-year study period. Thus, cancer mortality is a measure that is bound to (i) be less sensitive for cancers with better 5-year survival and (ii) disproportionately include cancers that were diagnosed at later stages, were more aggressive or treated less effectively. Given the known social and racial disparities in medical care (31) and cancer survival (32), we assume that the cause-specific cancer deaths identified in this analysis are a non-random subset of all occurrences of cancer in this cohort. Mortality outcomes can also obfuscate a time-window or lagged analysis since date of death can be years after the first date of diagnosis. For these reasons, incidence is generally preferred to mortality as an outcome measure for cancer etiology studies.

Mortality does, however, offer some advantages as an outcome over incidence. The Michigan Cancer Registry started in 1985, and linkage can identify cancer incidence in the cohort, but limited to diagnoses in the state of Michigan that occurred after the initiation of the registry. This data structure can lead to increased potential for misclassified outcomes and survivor bias due to left truncation. Thus, although mortality may not be the best outcome for studying the increased risk of cancer from an occupational exposure, it does allow us to leverage the full cohort of almost 40 000 workers followed for up to 75 years.

There are known risk factors for many of the cancers presented in this paper that were not measured in this cohort, for example, Helicobacter pylori infection for stomach cancer, sun exposure for skin cancer, diet for rectal cancer, and parity for breast cancer. Clearly, not all risk factors need to be adjusted for, however, those that are also associated with exposure need to be. Given the lack of association between most of the cancers and MWF, we considered whether we were missing information on a ubiquitous risk factor that might be inversely associated with increased MWF exposure. That is, is there a risk factor for mortality from several cancers that is more likely to occur among the unexposed? In this cohort, assembly workers were classified as unexposed to each specific type of MWF and comprise a large portion of the reference group for all fluid types, but especially soluble fluid. If assembly workers were more likely to be exposed to other occupational chemicals, smoke cigarettes, or have less favorable socio-economic status than machine operators or machinists, our results could be globally attenuated due to confounding. Unfortunately, we are not able to test this theory since we do not have smoking or socio-economic data for members of our cohort.

Due to the quantitative exposure assessment of MWF, this UAW-GM cohort study has contributed substantially to our understanding of the health effects of MWF. However, any exposure assessment based on a job exposure matrix will result in some non-differential exposure misclassification which would likely result in attenuation of results. Additionally, the necessary use of a 21-year lag may also lead to attenuation, especially for cancers with shorter latency.

Our final area of concern is attenuation from the healthy worker effect (33). We present both SMR, using an external reference group, and Cox models, using an internal reference group. The SMR is known to suffer from the healthy worker hire effect because people who are hired into physically demanding jobs are healthier at baseline than the general population. Thus, SMR can mask a harmful effect of occupational exposures. Cox models avoid this well-known bias by using unexposed workers as the reference group. Even internal analyses can be attenuated from the healthy worker survivor effect, however, because workers who are the least susceptible to the ill effects of an occupational exposure stay at work the longest and accrue the most exposure. The use of a 21-year lagged exposure metric diminishes the problem, but does not account for any self-selection out of the work force that occurred prior to 21 years before cancer mortality. Of note, we avoided a portion of healthy worker survivor effect, known as left truncation bias (34), by only including workers who were hired after the start of follow up in 1941. However, eligibility into the study required three years of work prior to entering follow up. We expect that those who survived the first three years of work may be different from those that left earlier and therefore note that there is built-in left truncation bias by study design. Other than restricting to those hired after the start of follow up, we did not address the healthy worker survivor effect in this manuscript.

We report elevations in skin, breast and stomach cancer mortality from long term occupational exposure to MWF. Several excess cancer risks previously reported in this cohort have become closer to the null with extended follow-up. Before concluding that MWF exposures are not associated with other cancers, possible attenuation by the healthy worker survivor effect should be excluded. If leaving work is a time dependent confounder of future exposure and the outcome and caused by previous exposure, then the Cox model is not adequate (35). Despite the extensive exposure assessment, large sample size and long follow-up, causal inference methods such as g-methods (36) may also be necessary to avoid underestimation.

Acknowledgements

This work was supported by NIOSH/CDC grant number R01OH011092. The University of California at Berkeley Center for the Protection of Human Subjects approved this research. The authors declare no conflicts of interest.

Sidebar

We present an extended cancer mortality follow-up of an autoworkers cohort potentially exposed to metalworking fluids. Patterns are consistent with previous reports, providing further evidence of increased risk of skin and female breast cancer with straight fluids, as well as new evidence for stomach cancer. Overall, there continues to be enough evidence to support a federal standard for metalworking fluid.

Costello S, Chen K, Picciotto S, Lutzker L, Eisen E. Metalworking fluids and cancer mortality in a US autoworker cohort (1945-2015). Scand J Work Environ Health. 2020;46(5):525-532. doi:10.5271/sjweh.3898

Correspondence to: Sadie Costello, PhD, Environmental Health Science, School of Public Health, University of California, Berkeley, 2121 Berkeley Way #5302, Berkeley, CA 94720-7360, USA. [E-mail: Sadie@berkeley.edu]

Footnote

1 Environmental Health Science, School of Public Health, University of California, Berkeley CA, USA.

References

References

1. EPA. National ambient air quality standards for ozone. Fed Regist 2014;79(242).

2. IARC. N-nitrosodiethanolamine. In "IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Some N-Nitroso Compounds." Lyon, France; 1978.

3. IARC. Monograph 34: polynuclear aromatic compounds, Part3, industrial exposures in aluminum production, coal gasification, coke production, and iron and steel founding. Genevia: World Health Organization, 1984.

4. Calvert GM, Ward E, Schnorr TM, Fine LJ. Cancer risks among workers exposed to metalworking fluids: a systematic review. Am J Ind Med 1998 Mar;33(3):282-92. https://doi. org/10.1002/(SICI)1097-0274(199803)33:3<282:AIDAJIM10>3.0.CO;2-W.

5. NIOSH. Criteria for a Recommended Standard: Occupational Exposure to Metalworking Fluids. US; 1998 p. 98-102.

6. Savitz DA. Epidemiologic evidence on the carcinogenicity of metalworking fluids. Appl Occup Environ Hyg. Taylor &Francis; 2003;18(11):913-20.

7. Mirer FE. New evidence on the health hazards and control of metalworking fluids since completion of the OSHA advisory committee report. Am J Ind Med 2010 Aug;53(8):792-801. https://doi.org/10.1002/ajim.20853.

8. Mirer F. Updated epidemiology of workers exposed to metalworking fluids provides sufficient evidence for carcinogenicity. Appl Occup Environ Hyg 2003 Nov; 1 8(1 1): 902-12. https://doi. org/10.1080/10473220390237511.

9. Park RM. Risk assessment for metalworking fluids and cancer outcomes. Am J Ind Med 2018 Mar;61(3):198-203. https://doi.org/10.1002/ajim.22809.

10. Eisen EA, Tolbert PE, Monson RR, Smith TJ. Mortality studies of machining fluid exposure in the automobile industry I: A standardized mortality ratio analysis. Am J Ind Med 1992;22(6):809-24. https://doi.org/10.1002/ajim.4700220604.

11. Eisen EA, Bardin J, Gore R, Woskie SR, Hallock MF, Monson RR. Exposure-response models based on extended follow-up of a cohort mortality study in the automobile industry. Scand J Work Environ Health. 2001;27(4):240-9. https://doi.org/10.5271/sjweh.611.

12. Mehta AJ, Malloy EJ, Applebaum KM, Schwartz J, Christiani DC, Eisen EA. Reduced lung cancer mortality and exposure to synthetic fluids and biocide in the auto manufacturing industry. Scand J Work Environ Health 2010 Nov;36(6):499-508. https://doi.org/10.5271/sjweh.3088.

13. Garcia E, Picciotto S, Neophytou AM, Bradshaw PT, Balmes JR, Eisen EA. Lung cancer mortality and exposure to synthetic metalworking fluid and biocides: controlling for the healthy worker survivor effect. Occup Environ Med 2018 Oct;75(10):730-5. https://doi.org/10.1136/ oemed-2017-104812.

14. Zeka A, Eisen EA, Kriebel D, Gore R, Wegman DH. Risk of upper aerodigestive tract cancers in a case-cohort study of autoworkers exposed to metalworking fluids. Occup Environ Med 2004 May;61(5):426-31. https://doi. org/10.1136/oem.2003.010157.

15. Malloy EJ, Miller KL, Eisen EA. Rectal cancer and exposure to metalworking fluids in the automobile manufacturing industry. Occup Environ Med 2007 Apr;64(4):244-9. https://doi.org/10.1136/oem.2006.027300.

16. Eisen EA, Tolbert PE, Hallock MF, Monson RR, Smith TJ, Woskie SR. Mortality studies of machining fluid exposure in the automobile industry. III: A case-control study of larynx cancer. Am J Ind Med 1994 Aug;26(2):185-202. https://doi. org/10.1002/ajim.4700260205.

17. Friesen MC, Costello S, Eisen EA. Quantitative exposure to metalworking fluids and bladder cancer incidence in a cohort of autoworkers. Am J Epidemiol 2009 Jun;169(12):1471-8. https://doi.org/10.1093/aje/kwp073.

18. Costello S, Friesen MC, Christiani DC, Eisen EA. Metalworking fluids and malignant melanoma in autoworkers. Epidemiology 2011 Jan;22(1):90-7. https:// doi.org/10.1097/EDE.0b013e3181fce4b8.

19. Garcia E, Bradshaw PT, Eisen EA. Breast cancer incidence and exposure to metalworking fluid in a cohort of female autoworkers. Am J Epidemiol 2018 Mar;187(3):539-47. https://doi.org/10.1093/aje/kwx264.

20. Izano MA, Sofrygin OA, Picciotto S, Bradshaw PT, Eisen EA. Metalworking fluids and colon cancer risk. Environ Epidemiol. 2019;3(1):e035. https://doi.org/10.1097/ EE9.0000000000000035.

21. Betenia N, Costello S, Eisen EA. Risk of cervical cancer among female autoworkers exposed to metalworking fluids. Scand J Work Environ Health 2012 Jan;38(1):78-83. https:// doi.org/10.5271/sjweh.3193.
22. Tolbert PE, Eisen EA, Pothier LJ, Monson RR, Hallock MF, Smith TJ. Mortality studies of machining-fluid exposure in the automobile industry. II. Risks associated with specific fluid types. Scand J Work Environ Health 1992 Dec;18(6):351-60. https://doi.org/10.5271/sjweh.1562.
23. Hallock MF, Smith TJ, Woskie SR, Hammond SK. Estimation of historical exposures to machining fluids in the automotive industry. Am J Ind Med 1994 Nov;26(5):621-34. https://doi.org/10.1002/ajim.4700260505.
24. Woskie SR, Smith TJ, Hallock MF, Hammond SK, Rosenthal F, Eisen EA et al. Size-selective pulmonary dose indices for metal-working fluid aerosols in machining and grinding operations in the automobile manufacturing industry. Am Ind Hyg Assoc J 1994 Jan;55(1):20-9. https:// doi.org/10.1080/15428119491019221.
25. Woskie SR, Virji MA, Hallock M, Smith TJ, Hammond SK. Summary of the findings from the exposure assessments for metalworking fluid mortality and morbidity studies. Appl Occup Environ Hyg 2003 Nov;18(11):855-64. https://doi. org/10.1080/10473220390237377.
26. NIOSH. Lifetable Analysis System (LTAS) Manual 3.0.0. 2014.
27. Schubauer-Berigan MK, Hein MJ, Raudabaugh WM, Ruder AM, Silver SR, Spaeth S et al. Update of the NIOSH life table analysis system: a person-years analysis program for the windows computing environment. Am J Ind Med 2011 Dec;54(12):915-24. https://doi.org/10.1002/ajim.20999.
28. Waxweiler RJ, Beaumont JJ, Henry JA, Brown DP, Robinson CF, Ness GO et al. A modified life-table analysis system for cohort studies. J Occup Med 1983 Feb;25(2):115-24.
29. Underlying Cause of Death 1999-2017 [Internet]. 2014. Available from: https://wonder.cdc.gov/wonder/help/ucd.html.
30. Friede A, Reid JA, Ory HW. CDC WONDER: a comprehensive on-line public health information system of the Centers for Disease Control and Prevention. Am J Public Health 1993 Sep;83(9):1289-94. https://doi.org/10.2105/AJPH.83.9.1289.
31. Nelson A. Unequal treatment: confronting racial and ethnic disparities in health care. J Natl Med Assoc 2002 Aug;94(8):666-8.
32. Bradley CJ, Given CW, Roberts C. Disparities in cancer diagnosis and survival. Cancer2001 Jan;91(1): 178-88. https:// doi.org/10.1002/1097-0142(20010101)91:1<178:AID-CNCR23>3.0.CO;2-S.
33. Eisen EA, Picciotto S, Robins J. Healthy worker effect. Encycl Environmetrics. 2012. p. 1-7.
34. Applebaum KM, Malloy EJ, Eisen EA. Left truncation, susceptibility, and bias in occupational cohort studies. Epidemiology 2011 Jul;22(4):599-606. https://doi. org/10.1097/EDE.0b013e31821d0879.
35. Robins J, Hernan M. Estimation of causal effects of time-varying exposures. In: Fitzmaurice G, Davidian M, Verbeke G, Molenberghs G, editors. Advances in Longitudinal Data Analysis. Chapman &Hall; 2009. p. 553-9.
36. Naimi AI, Cole SR, Kennedy EH. An introduction to g methods. Int J Epidemiol 2017 Apr;46(2):756-62.
Received for publication: 21 January 2020

DETAILS

Subject:	Leukemia; Larynx; Bias; Bladder; Mortality; Employment; Esophagus; Risk; Metalworking fluids; Pancreas; Intestine; Exposure; Petitions; Statistical models; Rectum; Fatalities; Health risks; Skin; Etiology; Cancer; Health hazards; Dose-response effects; Occupational safety; Breast cancer; Workers; Stomach; Stomach cancer; Gastric cancer; Computational fluid dynamics; Prostate; Occupational exposure; Manufacturing; Ratios
Business indexing term:	Subject: Employment Occupational safety Workers Manufacturing Ratios

Location:	United States--US
Company / organization:	Name: Environmental Protection Agency--EPA; NAICS: 924110; Name: Occupational Safety &Health Administration--OSHA; NAICS: 926150; Name: United Automobile Aerospace &Agricultural Implement Workers of America--UAW; NAICS: 813930; Name: National Institute for Occupational Safety &Health; NAICS: 926150
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	525-532,525A
Publication year:	2020
Publication date:	2020
Section:	Original article
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English
Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3898
ProQuest document ID:	2444686310
Document URL:	https://www.proquest.com/scholarly-journals/metalworking-fluids-cancer-mortality-us/docview/2444686310/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2023-08-04
Database:	Public Health Database

Psychosocial working characteristics before retirement and depressive symptoms across the retirement transition: a longitudinal latent class analysis

Åhlin, Julia K, PhD [1] ; Peristera, Paraskevi, PhD [1] ; Westerlund, Hugo, PhD [1] ; Hanson, Linda L Magnusson, PhD [1] [1] The Division for Epidemiology, Stress Research Institute, Department of Psychology, Stockholm University, Stockholm, Sweden

ProQuest document link

ABSTRACT (ENGLISH)

Objectives Retirement is a major life transition. However, previous evidence on its mental health effects has been inconclusive. Whether retirement is desirable or not may depend on pre-retirement work characteristics. We investigated trajectories of depressive symptoms across retirement and how a number of psychosocial working characteristics influenced these trajectories. Methods We included 1735 respondents from the Swedish Longitudinal Occupational Survey of Health (SLOSH), retiring during 2008-2016 (mean retirement age 66 years). They had completed biennial questionnaires reporting job demands, decision authority, workplace social support, efforts, rewards, procedural justice and depressive symptoms. We applied group-based trajectory modelling to model trajectories of depressive symptoms across retirement. Multinomial logistic regression analyses estimated the associations between psychosocial working characteristics and depressive symptom trajectories. Results We identified five depression trajectories. In four of them, depressive symptoms decreased slightly around retirement. In one, the symptom level was initially high, then decreased markedly across retirement. Perceptions of job demands, job strain, workplace social support, rewards, effort-reward imbalance and procedural justice were associated with the trajectories, while perceptions of decision authority and work efforts were only partly related to the trajectories. Conclusions We observed a rather positive development of depressive symptoms across retirement in a sample of Swedish retirees. For a small group with poor psychosocial working characteristics, symptoms clearly decreased, which may indicate that a relief from poor working characteristics is associated with an improvement for some retirees. However, for other retirees poor working characteristics were associated with persistent symptoms, suggesting a long-term effect of these work stressors.

FULL TEXT

Headnote

Key terms: depression; depressive symptom; effort-reward imbalance; job control; job demand; job strain; latent class analysis; longitudinal study; mental health; older worker; psychosocial; psychosocial working characteristic; retirement; SLOSH; stress; Swedish Longitudinal Occupational Survey of Health; work stress
Åhlin JK, Peristera P, Westerlund H, Magnusson Hanson LL. Psychosocial working characteristics before retirement and depressive symptoms across the retirement transition: a longitudinal latent class analysis. Scand J Work Environ Health. 2020;46(5): 488-497 . doi:10.5271/sjweh.3889

Objectives Retirement is a major life transition. However, previous evidence on its mental health effects has been inconclusive. Whether retirement is desirable or not may depend on pre-retirement work characteristics. We investigated trajectories of depressive symptoms across retirement and how a number of psychosocial working characteristics influenced these trajectories.

Methods We included 1735 respondents from the Swedish Longitudinal Occupational Survey of Health (SLOSH), retiring during 2008-2016 (mean retirement age 66 years). They had completed biennial questionnaires reporting job demands, decision authority, workplace social support, efforts, rewards, procedural justice and depressive symptoms. We applied group-based trajectory modelling to model trajectories of depressive symptoms across retirement. Multinomial logistic regression analyses estimated the associations between psychosocial working characteristics and depressive symptom trajectories.

Results We identified five depression trajectories. In four of them, depressive symptoms decreased slightly around retirement. In one, the symptom level was initially high, then decreased markedly across retirement. Perceptions of job demands, job strain, workplace social support, rewards, effort-reward imbalance and procedural justice were associated with the trajectories, while perceptions of decision authority and work efforts were only partly related to the trajectories.

Conclusions We observed a rather positive development of depressive symptoms across retirement in a sample of Swedish retirees. For a small group with poor psychosocial working characteristics, symptoms clearly decreased, which may indicate that a relief from poor working characteristics is associated with an improvement for some retirees. However, for other retirees poor working characteristics were associated with persistent symptoms, suggesting a long-term effect of these work stressors.

Key terms depression; effort-reward imbalance; job control; job demand; job strain; longitudinal study; mental health; older worker; SLOSH; stress; Swedish Longitudinal Occupational Survey of Health; work stress.

With increasing life expectancy in developed countries and population aging, many governments move towards increased retirement ages (1), stressing the importance of health promotion for healthy and active aging. However, the timing of retirement has been debated (2). Retirement itself is a major life transition and may be important for maintained well-being. However, previous research regarding mental health across retirement has been inconclusive. Beneficial effects of retirement have been observed (3, 4). For example, purchases of antidepressants have been found to decrease after retirement (5). Others have found unchanged purchases of psychotropic drugs/antidepressants, and self-reported depressive symptoms following retirement (6-8). Conversely, retirement has also been associated with increasing depressive symptoms (9). As depression in older adults is a major public health concern - associated with large costs in terms of premature mortality, morbidity and lower quality of life (10) - a greater understanding of mental health across retirement is important for identifying effective intervention strategies.

It has been suggested that retirees' well-being across retirement does not follow a uniform pattern, (11, 12) which may explain the heterogeneity in findings. The association between retirement and health likely depends on the reasons for retirement, eg, statutory and early vol- untary retirement has been found to be associated with improved health, while the findings for retirement due to ill-health have been the opposite, suggesting a health selection into retirement (4). Whether retirement is desirable or not can also depend on work characteristics (13-16). It is well known that poor work characteristics could affect subsequent mental health negatively (17), but it is uncertain whether there are long-term effects. Very few studies have investigated the role of (psychosocial) working conditions in relation to mental health or depression following retirement (16, 18, 19). One study found that poor psychosocial work environment in midlife was associated with higher depressive symptoms during retirement, suggesting chronic effects of work stress on mental health (18). On the other hand, relief from work-related stress/strain has been suggested to explain why retirement affects health (20). A poor work environment before retirement has been associated with higher prevalence of suboptimum health while in work, but a greater retirement-related improvement in health (13). A recent study found that individuals with more disadvantageous working conditions experienced more substantial improvements in mental health following retirement, especially in the short term (16).

In the present study, we investigate trajectories of depressive symptoms across retirement, and how a number of psychosocial working characteristics in the end of working life may influence these trajectories. This study adds to the rather inconsistent literature regarding mental health across the transition from work to retirement, by considering depressive symptoms specifically and that symptoms may develop differently over time for different groups of individuals. In addition, this study investigates the role of a number of specific psychosocial working characteristics in depressive symptom development across the retirement transition, some of which have not been examined in the previous literature on mental health across retirement.

Methods

Data and study population

We used data from the Swedish Longitudinal Occupational Survey of Health (SLOSH), a cohort survey of individuals aged 16-64 years from across the entire country and fairly representative of the Swedish working population (21). SLOSH participants have been followed-up by postal self-completion questionnaires biennially, since 2006 until 2018 (waves 1-7) thus far, with response rates of 48-65%. Some participants have been followed up since 2006, while others have been followed up since 2008, 2010, or 2014. One version of the questionnaire is for people in paid work, defined as gainful employment of >30% of full-time on average during the past three months ('workers'), and another version for people working less, or who have left the labor force temporarily or permanently ('non-workers'). More details of the SLOSH study can be found elsewhere (21).

Inclusion and exclusion criteria

The present study is based on all currently available waves (1-7) in SLOSH. In total, 29 676 individuals (73% of the total cohort) responded to at least one survey in 2006-2018. We selected only participants for whom we could observe a retirement transition, defined as going from paid work in one wave (completed the questionnaire for 'workers') to being retired (completed a questionnaire for 'non-workers' and reported being retired) in the following wave during waves 2-6. Individuals were classified as retired if they reported being old age retirees or receiving another type of pension (eg, contractual pension) on a full-time basis. Retirement due to ill-health (like disability pension, or early retirement on health grounds) was not classified as retirement since health trajectories are likely to differ from those of individuals going through old age retirement/other type (12). We excluded participants who reported transitions from retirement back to paid work, so-called unretirement (N=103), and 621 individuals who had not reported depressive symptoms in >4 waves. Thus, our sample included 1735 individuals (figure 1). A sensitivity analysis of the 621 individuals compared to the 1735 included individuals is presented in the supplementary material (www.sjweh.fi/show_abstract. php?abstract_id=3889) table S1 and showed no statistically significant differences between the excluded and included individuals. Among those included, 31% had data on depressive symptoms in seven waves, 41% in six waves, 17% in five waves, and 11% in four waves. The majority had data on depressive symptoms both before and after retirement (N=1639).

Psychosocial working characteristics

We included a number of psychosocial working characteristics from several dominating theoretical work stress models in waves 1-6 assessed by self-reports. Job demands, job control and workplace social support were measured by the Demand-Control-Support Questionnaire (22, 23). We specifically analyzed the subdimension decision authority of job control, since the subdimension skill discretion may be of less relevance in the modern working life. We used median split for classifying high and low demands, decision authority and social support. In addition, a variable combining demands and control was created according to the Job Strain Model (24). High demands and low control was defined as job strain, while the other three combinations were defined as no job strain. Moreover, we used the short version of the effortreward imbalance (ERI) questionnaire, which has shown to have satisfactory psychometric properties (25-27), to assess work efforts and rewards. Median split was used to classify high/low efforts and rewards, in order to facilitate the interpretation and comparison of the exposure variables. We also calculated the ERI ratio, where a ratio >1 was classified as ERI.

Procedural justice was measured with a seven-item scale (28). Median split was used to classify high/low procedural justice.

We assessed the following covariates as potential confounders: sex, age, civil status, occupational position, physical inactivity, excessive alcohol consumption, smoking, cardiovascular disease and diabetes, in line with similar studies (18, 19). The working characteristics and covariates were derived from the wave prior to reporting retirement, but if the variable was missing in that wave, data from two waves prior to retirement was used. A detailed description of the exposure and covariate variables and depressive symptoms is available in the supplementary material.

Depressive symptoms

Depressive symptoms were assessed in waves 1-7 using the subscale Symptom Checklist-core depression (SCL-CD6) (29) of the (Hopkins) Symptom Checklist (SCL-90) (30). Participants reported on a five-point Likert scale to what extent during the last week they had experienced: feeling blue, feeling no interest in things, feeling lethargic or low in energy, worrying too much about things, blaming oneself for things, and feeling everything is an effort. We used a sum scale serving as an indicator of depressive symptoms severity, ranging from 0-24. A score between 0-6 has been suggested to indicate no depression, 7-9 doubtful depression, 10-11 mild depression, 12-15 moderate depression and 16-24 severe depression, similar to the ICD-10 diagnostic system (31).

Statistical analyses

First, we conducted descriptive analyses to investigate how the level of depressive symptoms changed across retirement. Second, we applied group-based trajectory modelling (GBTM) to model trajectories of depressive symptoms across retirement using the plugin STATA TRAJ (32). GBTM identifies subgroups of individuals who follow a similar developmental course over time or age, in terms of a repeatedly measured behavior or phenomena (33). Time was years before and after retirement, ranging from nine years (corresponding to five waves) before retirement, to eleven years (corresponding to six waves) after retirement. The first wave a participant reported being retired was assigned +1 year, as the retirement transition took place sometime between years -1 to +1.

To decide the optimal number of trajectory groups and their complexity level (ie, the polynomial shape) that best described the trajectories, we followed the main principles as described more in detail previously (3335). Briefly, we proceed by comparing lower number of trajectory groups to higher, after also identifying the most appropriate shape of the trajectories in that group (starting from cubic to linear). The models were compared through model fit using Bayesian Information Criterion (BIC) (36, 37) with lower BIC indicating a better fitting model. However, BIC can sometimes continue to decrease as more trajectory groups are added (33). Therefore, we considered a model with more groups (and thus lower BIC) inferior than a model with less groups, if a trajectory group in that larger model contained <1% of the sample, when the model no longer captured new distinctive features of the data, or when entropy (index of classification accuracy ranging from 0-1 with values closer to 1 indicating better precision) (38), or average posterior probabilities of assignment (APPA; preferably >0.7) (33) declined. We assumed a censored normal distribution (39).

Once the optimal trajectory model for depressive symptoms was identified, we investigated the distribu- tion of pre-retirement characteristics and psychosocial working characteristics in the trajectory groups. Then, we examined how pre-retirement levels of job demands, decision authority, job strain, workplace social support, efforts, rewards, ERI and procedural justice were associated with membership in the depression trajectory groups by fitting multinomial logistic regression models. First, crude models were fitted for each predictor separately. Second, the models were adjusted for sex, age, civil status and occupational position pre-retirement. Third, the models were additionally adjusted for physical inactivity, excessive alcohol consumption, smoking, cardiovascular disease and diabetes.

Results

Average depressive symptoms

When investigating the mean scores of depressive symptoms across retirement, the mean symptom level was 5.9 [standard deviation (SD) 5.1] nine years before retirement and successively decreased until retirement. Symptoms were lowest the first time point after retirement (mean 2.9, SD 3.8) (see figure 2). The mean change in depressive symptom scores between the wave before and the wave individuals reported being retired was -1.5, indicating a reduction in symptoms. Symptoms decreased in 53.5% of the sample, while it remained unchanged in 23.7% and increased in 22.9% when comparing the wave before with the wave of retirement.

Trajectories of depressive symptoms across retirement

To assess different patterns of depressive symptoms across retirement in the study population, we tested trajectory models with up to seven trajectory groups. However, we considered the five-group model as the best because this model provided new distinctive features of the data compared to four groups, entropy was second best (0.82), APPA was satisfactory (0.88), and both entropy and APPA decreased when adding a sixth group. The five trajectories were labelled according to symptom level at baseline and stability/change across the retirement (figure 3). Group 1 - no depression, stable (very low) (N=471) - symptoms were very low across the time period, indicating no depression. Group 2 - no depression, stable (low) (N=838) - the largest group, had a slightly higher symptom level than group 1. Group 3 - moderate depression, considerably decreasing (N=38) - was small and had relatively high symptoms prior to retirement that decreased to no depression at the end of the period. Group 4 - mild depression, decreasing (N=326) - had symptoms of mild depression initially, which decreased to doubtful depression. Finally, group 5 - moderate depression, stable (N=62) - with symptoms of moderate depression remained on a similar level over time. As shown in figure 3, the points, which represent the average symptom score for each trajectory at each timepoint, are slightly above the fitted polynomial curves -1 year before retirement and below the lines in +1 year following retirement. This accords to the findings depicted in figure 2.

Characteristics of the trajectories

Distribution of some demographic variables prior to retirement in the study sample as well as stratified by trajectory group are presented in table 1. Average retirement age was 66 years. There were significant differences in the distribution of sex, pre-retirement age, civil status and occupational position between the trajectory groups. In the group 3, 4 and 5 trajectories, there were, eg, larger proportions of women, singles and unskilled workers, compared to the proportions in the group 1 trajectory. Distribution of some health variables and the psychosocial working characteristics in the study sample and stratified by trajectory group are presented in table 2. There were significant differences in the distribution of physical inactivity, excessive drinking, smoking and cardiovascular disease and these factors were most common in the group 4 and 5 trajectories. In the group 3, 4 and 5 trajectories, there were, eg, larger proportions of individuals who perceived high demands, low decision authority, job strain, low social support, high efforts, low rewards, ERI and low procedural justice, compared to the proportions in the group 1 trajectory.

Associations between pre-retirement psychosocial working characteristics and trajectories of depressive symptoms

Table 3 shows the results from the multinomial logistic regression analyses to predict membership in the depression trajectories. In the crude models, perceiving high job demands, job strain, low social support, low rewards, ERI and low procedural justice prior retirement was associated with all trajectories of depressive symptoms with higher symptom level compared to the reference trajectory of group 1. If exposed to a "risky" level of psychosocial working characteristics, the risk estimates of belonging to (especially) the group 3 and 5 trajectories were large. On the other hand, perceiving low decision authority was not associated with a higher risk of belonging to the group 3 trajectory and high efforts were not significantly associated with the group 2 and 5 trajectories compared to the reference trajectory. After adjustments in models 1 and 2, all estimates remained statistically significant except for the group 2 and 5 trajectories associated with low decision authority. In general, the RR were slightly attenuated comparing the crude models with models 1. When comparing model 1 with model 2, some RR were attenuated, while others increased or remained unchanged (see table 3).

Discussion

Main findings

Our results seem to support a beneficial effect of retirement, albeit quite modest, in terms of depressive symptoms. This is in line with a review concluding that retirement has beneficial effects on mental health (3), and other studies observing decreasing depressive symptoms in relation to retirement (14). Our results were also in line with those by Fleischmann et al (16), which showed that mental health improves already before retirement. However, we adopted a different analytic strategy than many previous studies on this topic and supported a heterogenous development of depressive symptoms across retirement (11, 12).

We further found that perceptions of job demands, job strain, workplace social support, rewards, ERI and procedural justice were associated with all the trajectories of depressive symptoms, while perceptions of decision authority and work efforts were only related to some of the trajectories. Interestingly, increased risks of belonging to even the no depression, stable (low) (group 2) trajectory for those with worse working characteristics when compared to the lowest reference trajectory were observed, indicating that poor working conditions may be associated with worse mental health, even in individuals with a low symptom level. In a previous paper, we similarly found that high demands and low social support predicted trajectories with higher levels of depressive symptoms while in working life (35). The fact that most of the psychosocial working characteristics were associated with a higher probability of belonging to the moderate depression, considerably decreasing (group 3) trajectory, may indicate that the retirement-related relief from exposure to these work stressors could have a beneficial effect on depressive symptoms. Fleischmann et al (16) also found support for such a relief where positive changes in mental health were more explicit for those retiring from poorer working conditions including high job demands, lower social support, or lower decision authority. This was in line with our findings except for the results regarding decision authority. Westerlund et al (13) similarly found that high (physical and psychological) job demands were associated with a more pronounced retirement-related improvement regarding self-rated health, suggesting that perceived health problems are relieved by retirement among older workers with poor working conditions. However, since the group 3 trajectory included so few individuals, more evidence is needed to substantiate this finding. Several psychosocial working characteristics were on the other hand, also associated with the moderate depression, stable (group 5) trajectory with highest depressive symptom scores, which seem to support previous findings suggesting that job demands or psychosocial stress at work (operationalized with the job demand-control and ERI models) during midlife were associated with post-retirement mental health (18, 19). This implies that job demands/efforts may have long-term effects on mental health, and our results also suggest that this may be true for workplace social support, rewards and organizational justice.

Strengths and limitations

This study contributed to the field by addressing a need for longitudinal studies regarding potentially influencing factors like work characteristics in relation to mental health effects of retirement (3). In contrast to a similar study treating all retirees as one group using piecewise trajectories (16), we applied group-based trajectory modelling to identify subgroups of retirees with different patterns of depressive symptoms across retirement. We thereby considered that retirees do not constitute an homogenous group (11, 12, 40), with individuals possibly experiencing deteriorating, improved or stable mental health. This study thus contributes to the current literature by supporting that mental health across retirement is heterogenous and that factors like work characteristics may play a role. Furthermore, we analyzed a rather large sample, approximately representative of the Swedish working population, thereby increasing the study's generalizability. Many previous studies have been relying on cross-sectional designs or only two waves, and moreover, few previous studies have used designs that are effective in terms of catching the effect of retirement on health and vice versa (8). Latent class growth analysis may also be suitable to capture different patterns such as recurrence and remission in depressive symptoms, which are known to exist in depression and may have different consequences (41). Our associations were generally robust when controlling for sex, age, occupational position, civil status, health risk behaviors, cardiovascular disease and diabetes, indicating that these factors did not explain the associations to a large extent. However, we cannot exclude that other unmeasured factors unrelated to work co-occurred with the retirement transition and thus may explain the decreased levels of depressive symptoms.

Some limitations include that only small proportions of the retirees followed the patterns of symptoms in the group 3 (2.2%) and 5 (3.6%) trajectories. This reduces the reliability of the findings regarding patterns of symptom development and leads to wide CI for the risk estimates of the relationship between work characteristics and these trajectories. We used dichotomous exposure variables and thereby lost some information. We only included individuals who had responded to several SLOSH questionnaires, who are possibly healthier and thus depressive symptom levels could be underestimated. A limited exposure contrast is also possible, which may result in underestimation of the associations.

When investigating the trajectories visually, there seemed to be a tendency for the symptom level to slightly increase around 9-11 years following retirement. It should be noted that there were relatively few observations so many years before and after retirement and that the selection of the shapes of trajectories based on polynomial functions of time/age is known to generate patterns unsupported by the data, such as uplifts at each end of the time axis (42). These patterns should therefore be interpreted with caution. It should also be acknowledged that the trajectories may not fully capture individual change, it can be difficult to identify trajectories with different shapes (43) and that we cannot draw causal conclusions. However, if causal, these results stress the value of workplace interventions targeting these types of work characteristics for healthy and active aging. Improved working environment may also enable increased retirement age.

Concluding remarks

Our findings generally indicated a modest, yet positive effect of retirement on depressive symptoms in a sample of Swedish retirees, with variation between groups and a small group showing a clear improvement. Furthermore, a relief from poor psychosocial working characteristics seemed to be associated with a more significant improvement. However, poor working characteristics were also associated with persistent symptoms suggesting a long-term effect of psychosocial working characteristics on depressive symptoms.

Acknowledgments

We would like to thank Loretta Platts who contributed with valuable comments regarding the analyses and interpretation of results. We also would like to thank all participants in the SLOSH study. In addition, we would like to thank AFA Insurance, the Swedish Research Council for Health, Working Life and Welfare (FORTE) and the Swedish Research Council for funding.

Funding

This work was supported by AFA Insurance (grant number 140323). The Swedish Longitudinal Occupational Survey of Health has also been supported by the FORTE (grant numbers 2005-0734 and 2009-1077) and the Swedish Research Council (VR) (grant numbers 2009-6192, 825-2013-1645, 821-2013-1646 and 201700624). FORTE also supports research based at the Stress Research Institute through the Stockholm Stress Center (grant number 2009-1758). The funders had no involvement in the conduction of this study.

Competing interests

None declared.

Ethics approval

The study was approved by the Regional Research Ethics Board in Stockholm, Sweden (2006/158-31, 2008/240-32, 2010/0145-32, 2012/373-31/5, 2013/217332 and 2015/2187-32). Participants gave informed consent by responding to the questionnaires.

Sidebar

Affiliation: The Division for Epidemiology, Stress Research Institute, Department of Psychology, Stockholm University, Stockholm SE-106 91, Sweden. julia.ahlin@su.se

Refers to the following texts of the Journal: 2012;38(5):409-417 2006;32(6):443-462

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32091111

Additional material

Please note that there is additional material available belonging to this article on the Scandinavian Journal of Work, Environment &Health -website.

Correspondence to: Julia Åhlin, The Division for Epidemiology, Stress Research Institute, Department of Psychology, Stockholm University, Stockholm SE-106 91, Sweden. [E-mail: julia.ahlin@su.se]

References

References

1. Christensen K, Doblhammer G, Rau R, Vaupel JW. Ageing populations: the challenges ahead. The Lancet. 2009;374(9696): 1196-208. https://doi.org/10.1016/S01406736(09)61460-4.

2. Burdorf A. Is early retirement good for your health? BMJ 2010;341. https://doi.org/10.1136/bmj.c6089.

3. van der Heide I, van Rijn RM, Robroek SJW, Burdorf A, Proper KI. Is retirement good for your health? A systematic review of longitudinal studies. BMC public health. 2013;13. https://doi.org/10.1186/1471-2458-13-1180.

4. Jokela M, Ferrie JE, Gimeno D, Chandola T, Shipley MJ, Head J, et al. From Midlife to Early Old Age Health

Trajectories Associated With Retirement. Epidemiology. 2010;21(3):28490.
https://doi.org/10.1097/EDE.0b013e3181d61f53.
5. Oksanen T, Vahtera J, Westerlund H, Pentti J, Sjosten N, Virtanen M, et al. Is Retirement Beneficial for Mental Health? Antidepressant Use Before and After Retirement. Epidemiology. 2011 ;22(4):553-9. https://doi.org/10.1097/EDE.0b013e31821c41bd.
6. Nyberg A, Peristera P, Magnusson Hanson LL, Westerlund H. Socio-economic predictors of depressive symptoms around old age retirement in Swedish women and men. Aging and Ment Health. 2019;23(5):558-65.
https://doi.org/10.1080/1360786 3.2018.1430741.
7. Leinonen T, Lahelma E, Martikainen P. Trajectories of antidepressant medication before and after retirement: the contribution of socio-demographic factors. Eur J Epidemiol. 2013;28(5):417-26. https://doi.org/10.1007/s10654-0139792-0.
8. Laaksonen M, Metsa-Simola N, Martikainen P, Pietilainen O, Rahkonen O, Gould R, et al. Trajectories of mental health before and after old-age and disability retirement: a registerbased study on purchases of psychotropic drugs. Scand J Work Environ Health. 2012;38(5):409-17. https://doi.org/10.5271/ sjweh.3290.
9. Dave D, Rashad I, Spasojevic J. The Effects of Retirement on Physical and Mental Health Outcomes. South Econ J. 2008;75(2):497-523.
10. Chapman DP, Perry GS. Depression as a major component of public health for older adults. Preventing chronic disease. 2008;5(1).
11. Pinquart M, Schindler I. Changes of life satisfaction in the transition to retirement: a latent-class approach. Psychol Aging. 2007;22(3):442-55. https://doi.org/10.1037/08827974.22.3.442.
12. Wang M. Profiling retirees in the retirement transition and adjustment process: Examining the longitudinal change patterns of retirees' psychological well-being. J Appl Psychol. 2007;92(2):455-74.
https://doi.org/10.1037/00219010.92.2.455.
13. Westerlund H, Kivimaki M, Singh-Manoux A, Melchior M, Ferrie JE, Pentti J, et al. Self-rated health before and after retirement in France (GAZEL): a cohort study. Lancet. 2009;374:1889-96.
https://doi.org/10.1016/S01406736(09)61570-1.
14. Westerlund H, Vahtera J, Ferrie JE, Singh-Manoux A, Pentti J, Melchior M, et al. Effect of retirement on major chronic conditions and fatigue: French GAZEL occupational cohort study. BMJ. 2010;341.
https://doi.org/10.1136/bmj.c6149.
15. Wang M, Shi JQ. Psychological Research on Retirement. Annu Rev Psychol. 2014;65:209-33.
https://doi.org/10.1146/ annurev-psych-010213-115131.
16. Fleischmann M, Xue B, Head J. Mental Health Before and After Retirement-Assessing the Relevance of Psychosocial Working Conditions: The Whitehall II Prospective Study of British Civil Servants. J Gerontol B Psychol Sci Soc Sci. 2019. https://doi.org/1G.1093/geronb/gbz042.
17. Stansfeld SA, Candy B. Psychosocial work environment and mental health - a meta-analytic review. Scand J Work Environ Health. 2006;32(6):443-62. https://doi.org/10.5271/ sjweh.1050.
18. Wahrendorf M, Blane D, Bartley M, Dragano N, Siegrist J. Working conditions in mid-life and mental health in older ages. Adv Life Course Res. 2013; 18(1): 16-25. https://doi. org/10.1016/j.alcr.2012.10.004.
19. Virtanen M, Ferrie JE, Batty GD, Elovainio M, Jokela M, Vahtera J, et al. Socioeconomic and Psychosocial Adversity in Midlife and Depressive Symptoms Post Retirement: A 21-year Follow-up of the Whitehall II Study. Am J Geriatr Psychiatry. 2015;23(1):99-109. https://doi.org/10.1016/j. jagp.2014.04.001.
20. Eibich P. Understanding the effect of retirement on health: Mechanisms and heterogeneity. J Health Econ. 2015;43:1-12. https://doi.org/10.1016/jjhealeco.2015.05.001.
21. Magnusson Hanson LL, Leineweber C, Persson V, Hyde M, Theorell T, Westerlund H. Cohort Profile: The Swedish Longitudinal Occupational Survey of Health (SLOSH). Int J Epidemiol. 2018;47(3):691.
https://doi.org/10.1093/ije/ dyx260.
22. Sanne B, Torp S, Mykletun A, Dahl AA. The Swedish Demand-Control-Support Questionnaire (DCSQ): Factor

structure, item analyses, and internal consistency in a large population. Scand J Public Health. 2005;33(3):166-74. https:// doi.org/10.1080/14034940410019217.
23. Fransson EI, Nyberg ST, Heikkila K, Alfredsson L, Bacquer D, Batty GD, et al. Comparison of alternative versions of the job demand-control scales in 17 European cohort studies: the IPD-Work consortium. BMC Public Health. 2012;12:62. https://doi.org/10.1186/1471-2458-12-62.
24. Karasek R, Theorell T. Healthy Work: Stress, Productivity, and the Reconstruction of Working Life. New York: Basic Books; 1990.
25. Siegrist J, Wege N, Pühlhofer F, Wahrendorf M. A short generic measure of work stress in the era of globalization: effort-reward imbalance. Int Arch Occup Environ Health. 2009;82(8):1005-13. https://doi.org/10.1007/s00420-0080384-3.
26. Leineweber C, Wege N, Westerlund H, Theorell T, Wahrendorf M, Siegrist J. How valid is a short measure of effort-reward imbalance at work? A replication study from Sweden. Occup and Environ Med. 2010;67(8):526-31. https://doi. org/10.1136/oem.2009.050930.
27. Siegrist J, Li J, Montano D. Psychometric properties of the Effort-Reward Imbalance Questionnaire. Department of Medical Sociology, Faculty of Medicine, Duesseldorf University, Germany, 2014.
28. Moorman RH. Relationship between organizational justice and organizational citizenship behaviors - Do fairness perceptions influence employee citizenship. J Appl Psychol. 1991;76(6):845-55. https://doi.org/10.1037/00219010.76.6.845.
29. Magnusson Hanson LL, Westerlund H, Leineweber C, Rugulies R, Osika W, Theorell T, et al. The Symptom Checklist-core depression (SCL-CD6) scale: psychometric properties of a brief six item scale for the assessment of depression. Scand J Public Health. 2014;42(1):82-8. https:// doi.org/10.1177/1403494813500591.
30. Lipman RS. Depression Scales Derived from the Hopkins Symptom Checklist. In: Sartorius N, Ban TA, editors. Assessment of depression. Berlin: Springer-Verlag; 1986: 232-48. https://doi.org/10.1007/978-3-642-70486-4_22.
31. Bech P. Klinisk psykometri. Denmark: Munksgaard Danmark; 2011.
32. Jones BL, Nagin DS. A Stata Plugin for Estimating GroupBased Trajectory Models. [Working Paper]. Carnegie Mellon University 2012.
33. Nagin DS. Group-based modeling of development. Cambridge, MA.: Harvard University Press; 2005. https://doi. org/10.4159/9780674041318.
34. Andruff H, Carraro N, Thompson A, Gaudreau P, Louvet B. Latent Class Growth Modelling: A Tutorial. Tutor Quant Methods Psychol. 2009;5(1):11-24. https://doi.org/10.20982/ tqmp.05.1.p011.
35. Åhlin JK, Rajaleid K, Jansson-Fröjmark M, Westerlund H, Magnusson Hanson LL. Job demands, control and social support as predictors of trajectories of depressive symptoms. J Affect Disord. 2018;235:535-43. https://doi.org/10.1016/j. jad.2018.04.067.
36. Schwarz G. Estimating dimension of a model. Ann Stat. 1978;6(2):461-4. https://doi.org/10.1214/aos/1176344136.
37. Raftery AE. Bayesian model selection in social research. In: Marsden PV, ed. Sociol Methodol 1995:25:111-63. https://doi. org/10.2307/271063.
38. Jung T, Wickrama KAS. An Introduction to Latent Class Growth Analysis and Growth Mixture Modeling. Soc Personal Psychol Compass. 2008;2(1):302-17. https://doi.org/10.1111/ j.1751-9004.2007.00054.x.
39. Nagin DS. Analyzing developmental trajectories: A semiparametric, group-based approach. Psychol Methods. 1999;4(2):139-57. https://doi.org/10.1037/1082989X.4.2.139.
35. Schwarz G. Estimating dimension of a model. Ann Stat. 1978;6(2):461-4. https://doi.org/10.1214/aos/1176344136.
40. Henning G, Lindwall M, Johansson B. Continuity in Well-Being in the Transition to Retirement. Geropsych. 2016;29(4):225-37. https://doi.org/10.1024/1662-9647/ a000155.
41. Colman I, Ataullahjan A. Life Course Perspectives on the Epidemiology of Depression. Can J Psychiat. 2010;55(10):622-32. https://doi.org/10.1177/07067437100 5501002.

42. Francis B, Elliott A, Weldon M. Smoothing Group-Based Trajectory Models Through B-Splines. J Dev Life Course Criminol. 2016;2(1):113-33. https://doi.org/10.1007/s40865016-0025-6.
43. Twisk J. Is It Necessary to Classify Developmental Trajectories over Time? A Critical Note. Ann Nutr Metab. 2014;65(23):236-40. https://doi.org/10.1159/000362506.
Received for publication: 18 October 2019

DETAILS

Subject:	Occupational stress; Mental health; Older workers; Work environment; Social support; Age; Retirement; Retirees; Psychotropic drugs; Working conditions; Questionnaires; Mental depression; Regression analysis; Occupational health; Social interactions; Longitudinal studies; Reinforcement; Latent class analysis
Business indexing term:	Subject: Occupational stress Older workers Work environment Retirees Working conditions Retirement
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	488-497,488A
Publication year:	2020
Publication date:	2020
Section:	Original article
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English
Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3889

ProQuest document ID:	2444686162
Document URL:	https://www.proquest.com/scholarly-journals/psychosocial-working-characteristics-before/docview/2444686162/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2023-02-27
Database:	Public Health Database

Persistent and changing job strain and risk of coronary heart disease. A population-based cohort study of 1.6 million employees in Denmark

Rugulies, R [1] ; Framke, E; Sørensen, J K; Svane-Petersen, A C; Alexanderson, K; Bonde, J P; Farrants, K; Flachs, E M; Hanson, L L Magnusson; Nyberg, S T; Kivimäki, M; Madsen, I E H [1] National Research Centre for the Working Environment, Lersø Parkallé 105, DK-2100 Copenhagen, Denmark

ProQuest document link

ABSTRACT (ENGLISH)

Objectives This study aimed to examine the association between job strain and incident coronary heart disease (CHD) in Denmark, while accounting for changes of job strain. Methods We included all employees residing in Denmark in 2000, aged 30-59 years with no prevalent CHD (N=1 660 150). We determined exposure to job strain from 1996-2009 using a job exposure matrix (JEM) with annual updates. Follow-up for incident CHD was from 2001-2010 via linkage to health records. We used Cox regression to calculate hazard ratios (HR) and 95% confidence intervals (CI) for the association between job strain and incident CHD. Results During 16.1 million person-years, we identified 24 159 incident CHD cases (15.0 per 10 000 person-years). After adjustment for covariates, job strain in 2000 predicted onset of CHD during a mean follow-up of 9.71 years (HR 1.10, 95% CI 1.07-1.13). When analyzing changes in job strain from one year to the next and CHD in the subsequent year, persistent job strain (HR 1.07, 95% CI 1.03-1.10), onset of job strain (HR 1.20, 95% CI 1.12-1.29) and removal of strain (HR 1.20, 95% CI 1.12-1.28) were associated with higher CHD incidence compared to persistent no job strain. Associations were similar among men and women. Conclusions Job strain is associated with a higher risk of incident CHD in Denmark. As we used a JEM, we can rule out reporting bias. However, under- or overestimation of associations is possible due to non-differential misclassification of job strain and residual confounding by socioeconomic position.

FULL TEXT

Headnote

Objectives This study aimed to examine the association between job strain and incident coronary heart disease (CHD) in Denmark, while accounting for changes of job strain.

Methods We included all employees residing in Denmark in 2000, aged 30-59 years with no prevalent CHD (N=1 660 150). We determined exposure to job strain from 1996-2009 using a job exposure matrix (JEM) with annual

updates. Follow-up for incident CHD was from 2001-2010 via linkage to health records. We used Cox regression to calculate hazard ratios (HR) and 95% confidence intervals (CI) for the association between job strain and incident CHD.
Results During 16.1 million person-years, we identified 24 159 incident CHD cases (15.0 per 10 000 personyears). After adjustment for covariates, job strain in 2000 predicted onset of CHD during a mean follow-up of 9.71 years (HR 1.10, 95% CI 1.07-1.13). When analyzing changes in job strain from one year to the next and CHD in the subsequent year, persistent job strain (HR 1.07, 95% CI 1.03-1.10), onset of job strain (HR 1.20, 95% CI 1.12-1.29) and removal of strain (HR 1.20, 95% CI 1.12-1.28) were associated with higher CHD incidence compared to persistent no job strain. Associations were similar among men and women.
Conclusions Job strain is associated with a higher risk of incident CHD in Denmark. As we used a JEM, we can rule out reporting bias. However, under- or overestimation of associations is possible due to non-differential misclassification of job strain and residual confounding by socioeconomic position.
Key terms cardiovascular disease; CHD; epidemiology; JEM; job control; job exposure matrix; psychological demand; psychosocial work environment; stress; work stress.
Meta-analyses of prospective cohort studies suggest that adverse psychosocial working conditions may contribute to onset of coronary heart disease (CHD) (1-5). The combination of high psychological demands and low decision latitude at work, denoted "job strain", has been most extensively studied. An individual participant data (IPD) meta-analysis of 13 European cohort studies showed that job strain was associated with approximately 20% excess risk of CHD [pooled hazard ratio (HR) of 1.23, 95% confidence interval (CI) 1.10-1.37] (1). Another meta-analysis summarizing 31 studies, including the IPD-Work consortium data, showed a slightly higher excess risk (pooled HR 1.33, 95% CI 1.19-1.49) (3). The underlying mechanisms linking job strain to CHD may include activation and dysregulation of the sympatho-adrenal medullary and the hypo- thalamic-pituitary-adrenal stress axes, inflammatory processes and increase in hazardous health behaviors, such as smoking intensity and leisure-time physical inactivity (6, 7).
However, there are important limitations to the evidence, calling for further research. First, job strain has predominantly been measured by self-reported data, raising concerns about reporting bias. Undetected pre-clinical CHD may cause individuals experiencing work as more strenuous, yielding spurious associations between baseline self-reported job strain and subsequent onset of clinical CHD.
Second, job strain has predominantly been assessed only once, at baseline, and changes in job strain over time usually have not been accounted for, likely resulting in imprecise measurement and exposure misclassification. Furthermore, measuring job strain repeatedly would allow analyzing if job strain over several years is more harmful to health than job strain measured at a single point in time.
Third, it is unknown whether onset of job strain is associated with a higher risk of CHD and that removal of job strain is associated with a lower risk. Identifying such associations would strengthen the interpretation of job strain as a causal factor in the etiology of CHD.
We address these limitations by analyzing the association between job strain and CHD in the Danish workforce while (i) measuring job strain with a job exposure matrix (JEM) (rather than individual-level self-reports) with annual updates, (ii) examining whether number of years with job strain is associated with risk of CHD, and (iii) analyzing the association between persistent job strain, as well as job strain onset and removal, and risk of CHD.
Methods
Study design and population
We used data from the JEMPAD (Job Exposure Matrix Analyses of Psychosocial Factors and Healthy Ageing in Denmark) study, a nationwide register-based study on work environment and health. A JEMPAD study on the association between educational attainment and risk of cardiovascular morbidity and mortality, and the role of household income and job strain for this association, was recently published (8).
The study population was drawn from the Integrated Database for Labor Market Research (IDA) at Statistics Denmark (9). We included all individuals residing in Denmark (independent of their nationality), aged 30-59 years

and employed (excluding the self-employed) in the year 2000. We excluded 1323 individuals with missing data on age, sex, or migration background, yielding 1 680 214 individuals. Using their unique Danish civil registration number, we linked these individuals to other population-based registers providing information on socio-demographic variables and health.

We excluded 20 064 individuals diagnosed with CHD (ICD 8: 410-414; ICD 10: 120-125; ICD 9 was never used in Denmark) from 1 January 1977 [when the diagnosis first became available in the National Patient Register (10)] to 31 December 2000 (the day before start of follow-up period), yielding a study population of 1 660 150 individuals. To identify incident CHD during follow-up, we linked these individuals to records from national health registers until 31 December 2010. The duration of follow-up was motivated by keeping the job strain measure consistent across the follow-up period, as this measure was job-group-specific, and the registration of job groups had changed from 2010 onwards.

Job strain

We estimated the predicted probability of job strain with a JEM based on information from the Danish Work Environment Cohort Study (DWECS) (11, 12). DWECS was a survey on working conditions and health, conducted in a randomly selected sample of the Danish workforce from 1990-2010. We included DWECS data from the years 2000 and 2005 (N=10 749) with information on job strain. In accordance with previous research (1), we measured job strain by combining three items on psychological demands at work and five items on job control, and defined job strain as simultaneously scoring psychological demands above the median and job control below the median. We constructed the JEM in DWECS as the predicted probability of job strain given job group, sex, age, and year of data collection (2000, 2005). Job group was coded according to the four-digit level of DISCO-88, the Danish version of the International Standard Classification of Occupations (ISCO)-88 system (13).

We assigned the predicted probabilities of job strain to each individual from the JEMPAD cohort annually from 1996-2009. We categorized individuals into groups with and without job strain based on the median split of the annual distribution of the predicted probability of job strain. Individuals predominantly non-employed in a given year (eg, due to unemployment, self-employment, disability retirement, or statutory retirement) were assigned a separate category ["not applicable (NA) job strain"] during these years. See the supplementary material (www.sjweh.fi/show_abstract.php?abstract_ id=3891), appendix 1 for a detailed description of the JEM.

Incident coronary heart disease

We ascertained incident CHD by retrieving both main and secondary diagnoses from the National Patient Register (10) and underlying and contributing causes from the Danish Register of Causes of Death (14) from 1 January 2001 to 31 December 2010. The two registers are valid tools for studying CHD at the population level (15). Incident CHD was defined as either incident non-fatal myocardial infarction (ICD-10: I21, I22) or death due to CHD (I20-I25).

Covariates

As potential confounders, we considered age, sex, migration background, family type, health services use in the year before exposure ascertainment (as an indicator of health status, including possible undiagnosed prevalent CHD) and socioeconomic position, measured by household disposable income. We further presented individuals' general occupational position (based on the first digit DISCO-88 code) and educational attainment [based on the International Standard Classification of Education (ISCED) (16)] to describe the study population but did not use these two variables for statistical adjustment in the main analyses. Adjusting for occupational position was inadvisable because both occupational position and job strain were based on DISCO-88 codes. Adjusting for education may have resulted in overadjustment because educational attainment is intertwined with job group, as certain levels of education are a necessary prerequisite for entering specific jobs (eg, becoming a lawyer, engineer or a physician requires a university degree). However, not adjusting for education could lead to underadjustment. To get the most complete picture, we therefore reported estimates unadjusted for education in the main analyses and estimates adjusted for education in a supplementary analysis. Further, we conducted separate analyses by level of education to explore if the association between job strain and risk of CHD differed by educational attainment.

Information on age, sex and migration background was retrieved from the Population Register (17). Age was included with piecewise linear splines accounting for nonlinear association between age and CHD (knots at 30-35, 36-42, 43-50 and 51-59 years, respectively). For migration background, we used the classification by Statistics Denmark (17) distinguishing between (i) individuals with no migration background, (ii) immigrants, and (iii) descendants of immigrants. Data on family type was retrieved from the Population Register (17) and the Family Relation Register (18) and categorized into six groups, combining information on marriage/cohabitation with presence of children in the household (see table 1 for details). Information on annual household disposable income, that is the sum of earned income and social transfer payments of all household members after deduction of taxes and interest expenses, was retrieved from registers on personal income and transfer payments (19) and categorized into deciles based on the distribution of individuals within each year. We retrieved information on health services use, provided by primary healthcare professionals, such as general practitioners, from the Danish National Health Service Register (20) and categorized the number of health services used into deciles based on the annual distributions.

All covariates were updated annually from 20002009, except sex and migration background that were included based on the status in 2000.

Statistical analysis

All analyses were conducted using SAS 9.4 (SAS Institute, Cary, NC, USA). Individuals were followed from year 2000 until the first CHD event, censoring (emigration from Denmark, non-CHD death), or end of follow-up, whichever came first. Using Cox proportional hazard models with calendar time as the underlying time axis we calculated HR and 95% CI for the association between job strain and incident CHD.

We performed three main analyses, depicted in supplementary appendix 2, figure S1. First, we performed a "traditional" analysis with job strain measured at baseline (year 2000) and first CHD event during follow-up (2001-2010) (analysis #1).

Second, we examined the association between number of years exposed to job strain in the five years before start of follow-up (1996-2000) and first CHD event during follow-up (2001-2010). This analysis was restricted to individuals who were employed in all years from 1996-2000 (N=1 353 249) (analysis #2).

Third, we analyzed the association between persistent job strain, as well as job strain onset and removal, and first CHD event during follow-up, ie, the association between changes in job strain from year t-1 to year t (1999-2000, 2000-2001, ... 2008-2009) and first CHD event in year t+1 (2001, 2002, ... 2010) (analysis #3). Individuals contributed with exposure information in each exposure period until they were removed from the analysis because of a CHD event or censoring. We further coded if an individual moved from job strain or no job strain to non-employment and vice versa or if an individual remained in non-employment.

We incrementally adjusted analyses for sex, age, family type, migration background, health services use, and household disposable income. In analyses #1 and #2, all covariates were treated as time-invariant based on year 2000 values. In analysis #3, sex and migration background were treated as time-invariant, whereas age, family type and household income were treated as time-varying with a one-year time lag between the measurement of the covariates and the measurement of the outcome. Health service use was treated as time-varying, with a two-year time lag between the measurement of the covariate and the measurement of the outcome to ensure that health service use was measured before job strain and, therefore, was not a mediator of the association between job strain and incident CHD.

We conducted four supplementary analyses. First, to examine if associations differed when enhancing exposure contrast, we repeated analysis #1 analyzing job strain categorized into quartiles instead of dichotomized by median split. Second, to examine if associations were different for men and women, we repeated the main analyses separately for men and women. Third, we repeated the main analyses with further adjustment for education. Fourth, we repeated the main analyses conducted separately by educational level.

Results

Characteristics of the study population at baseline

Table 1 shows the characteristics of the study population in 2000. Men and women were nearly equally represented and mean age was about 44 years. The most prevalent occupational groups were technicians and associate professionals (19.0%), followed by professionals (15.2%), clerks (11.7%), and service workers and shop and market sales workers (11.5%).

Job strain in 2000 and incident CHD (analysis #1)

Table 2 shows the association between job strain, measured in 2000, and incident CHD from 2001-2010. Mean time of follow-up was 9.71 years. During 16 117 512 person-years, we identified 24 159 incident cases of CHD (15.0 cases per 10 000 person-years), 11 032 in the no job strain group (13.7 per 10 000 person-years) and 13 127 in the job strain group (16.3 per 10 000 personyears). The HR for comparing individuals with job strain to individuals without job strain was 1.16 (95% CI 1.13-1.19) after adjustment for age, sex, family type, migration background and health service use (model 2) and 1.10 (95% CI 1.07-1.13) after further adjustment for household disposable income (model 3).

Years with job strain and incident CHD (analysis #2)

Table 3 shows the association between number of years with job strain from 1996-2000 and incident CHD from 2001-2010. Mean time of follow-up was 9.74 years. This analysis was restricted to individuals who were employed in all years from 1996-2000 (N=1 353 249 providing 13 176 222 person-years). Compared to individuals with no exposure to job strain, there was a higher risk of CHD for those with exposure during 1-2 years (HR 1.21, 95% CI 1.16-1.26), 3-4 years (HR 1.20, 95% CI 1.15-1.26), or all 5 years (HR 1.14, 95% CI 1.11-1.18) in the most adjusted analysis. There was no indication of a dose-response association, ie, after 1-2 years of job strain, the risk did not increase further with increasing number of years with job strain.

Persistent job strain, onset of job strain and removal of job strain and incident CHD (analysis #3)

Table 4 shows the association between persistent job strain, onset of job strain, and removal of job strain from year t-1 to year t and incident CHD in year t+1. Compared to the reference group of persistent no job strain, there was a higher risk of incident CHD among individuals with persistent job strain (HR 1.07, 95% CI 1.03-1.10), individuals moving from no job strain to job strain (onset) (HR 1.20, 95% CI 1.12-1.29), and individuals moving from job strain to no job strain (removal) (HR 1.20, 95% CI 1.12-1.28) after adjustment for covariates.

Compared to individuals with persistent no job strain, exiting employment was associated with an increased risk of CHD, both for those exiting from a job with job strain (HR 1.23, 95% CI 1.14-1.33) and those exiting from a job without job strain (HR 1.19, 95% CI 1.11-1.29). Entering employment was associated with a higher risk of CHD if individuals entered a job with job strain (HR 1.23, 95% CI 1.08-1.40), but not if individuals entered a job without job strain (HR 1.04, 95% CI 0.91-1.18).

Supplementary analyses

To enhance exposure contrast, we repeated analysis #1 with a job strain variable categorized by quartiles instead by median split. Compared to the group with low job strain in 2000, HR for CHD were 1.20 (95% CI 1.15-1.24), 1.18 (95% CI 1.14-1.23) and 1.24 (95% CI 1.19-1.29) for the groups with medium-low, medium-high and high job strain, respectively (appendix 3, table S2).

Repeating the main analyses separately for men and women did not reveal any major differences between the sexes (appendix 4, tables S3-5).

When we repeated the main analyses, while adjusting for education, estimates became attenuated with some estimates (eg, onset of job strain) remaining considerable (appendix 5, tables S6-8). When we conducted analyses separately by educational level, associations between job strain and risk of CHD were strongest for individuals with a high level of education, however CI overlapped (appendix 6, tables S9-11).

Discussion

In this population-based study of the Danish workforce, persistent and changing exposure to job strain - compared to persistent absence of job strain - was associated with a higher risk of CHD, defined as non-fatal myocardial infarction or CHD mortality. These results suggest that the psychosocial work environment in general, and job strain in particular, may contribute to the etiology of CHD. Using repeated measures of exposure and assessing job strain not by self-report but with a JEM, this study addressed important limitations of most previous studies.

The main results are summarized in figure 1. Job strain predicted CHD when measured (i) at baseline, (ii) as the number of years with job strain in a five-year period before start of follow-up, (iii) as persistent job strain in two subsequent years, (iv) as onset of job strain, defined as moving from the no job strain category to the job strain category, and (v) as removal of job strain, defined as moving from the job strain category to the no job strain category. We found no clear dose-response patterns between years with job strain and risk of CHD.

Comparison with previous studies on job strain and CHD

With more than 1.6 million individuals and more than 16 million person-years of follow-up, this is the largest prospective study so far on job strain and risk of CHD. The second-largest study was the IPD-Work consortium analysis that reported a pooled HR of 1.23 in 13 harmonized European cohort studies with 197 473 participants providing 1.49 million person-years (1). Although we used a different methodological approach in JEMPAD compared to IPD-Work, the JEMPAD estimates were either similar to, or only slightly lower than, the estimates from IPD-Work.

To our knowledge, no large-scale study has previously analyzed the association of onset and removal of job strain and risk of CHD. In an analysis of 7253 civil servants from the British Whitehall II cohort, Kivimäki et al (21) examined job strain at two points, three years apart, and reported elevated HR of CHD for job strain score at both phase 1 (HR 1.23, 95% CI 1.10-1.38 per one standard deviation increase) and phase 2 (HR 1.15, 95% CI 1.03-1.29). Changing job strain score from phase 1 to phase 2 was not related to risk of CHD. As these analyses were based on a continuous job strain score, estimates on onset and removal of job strain were not available.

To analyze the association between job strain and risk of CHD using a JEM is not novel. Case-control studies using JEM were the method of choice in the beginning of job strain research in Sweden in the 1980s (22-25). From the mid-1990s, when an increasing number of cohort studies with individual-level job strain exposure data became available (3), JEM analyses of job strain went out of fashion. Recently, there has been a revival of interest in psychosocial job exposure matrices, with the development of new matrices for different psychosocial work environment factors in The Netherlands (26), France (27), Finland (28), Denmark (29, 30), and Australia (31). To our knowledge, the most recently published study on job strain and CHD using a JEM was a study with 6070 Swedish men that reported a HR of 1.31 (95% CI 1.01-1.70) among those exposed to job strain (32). Unlike our study, though, the Swedish study did not examine changes of job strain over time.

Interpretation

There has been a concern with previous studies that estimates of the association between job strain and CHD may be inflated due to reporting bias, as individuals with undetected pre-clinical CHD might experience and report working conditions as more strenuous than individuals without pre-clinical CHD (33). Because we used a JEM to ascertain job strain, the measurement of the exposure to job strain was not dependent on the individuals' report of working conditions, and we can rule out that reporting bias has inflated our estimates. However, a weakness of JEM is their inability to detect differences in job strain levels within occupational groups, likely resulting into non-differential exposure misclassification and bias towards an underestimation of associations.

We defined job strain as a dichotomous variable, comparing job strain with no job strain, as this was also the definition used by the IPD-Work consortium (1). Another often used operationalization is the quadrant model, with the four groups of no job strain (low demands and high control), passive work (low demands and low control), active work (high demand and high control), and job strain (high demands and low control). The quadrant model may have yielded different results, and we encourage further research on this model.

To assess job strain in the register data, we used the median split of the predicted probabilities of job strain, resulting in 50% exposed and 50% unexposed and a HR of 1.10 in the analysis on job strain in 2000 and CHD during 2001-2010. When we enhanced exposure contrast by categorizing job strain into quartiles, the HR for the quartile with the highest predicted probability of job strain was 1.24. This result indicates that our estimates may have been conservative and would have been stronger if we had defined job strain with a higher exposure contrast.

There was no indication that chronic job strain over a five-year period was more hazardous than exposure of 1-2 years only during a 5-year period. It is possible that this estimate for chronic job strain was affected by healthy worker selection, with employees remaining in high strain jobs throughout the 5-year period being healthier or more resilient than those changing to lower strain jobs.

That individuals moving from the no job strain to the job strain category had a HR of CHD of 1.20 - compared to individuals remaining in the no job strain category - may provide the strongest argument for the case that there is a causal effect of job strain on risk of CHD. The interpretation of a causal effect is further strengthened by the result that individuals entering employment were -compared to the persistent no job strain group - at higher risk of CHD when they entered a job with job strain but not when they entered a job without job strain.

Removal of job strain was associated with an increased risk of CHD of the same magnitude as onset of job strain when comparing the two groups to persistent no job strain. One explanation may be reverse causation, ie, individuals with pre-clinical CHD moving to jobs without job strain shortly before manifest a clinical CHD event. A similar association has been observed in a study on working hours, where myocardial infarction was more common when the participant either worked long working hours or short hours as a part-timer (34). We tried to account for health selection by adjusting for health-services use, but a residual selection effect might have remained.

Staying persistently out of employment and moving from employment to out of employment were both associated with an increased risk of CHD. This may reflect health selection, ie, that individuals with preclinical CHD are no longer able to stay in employment, or a causal effect of unemployment on the risk of CHD (35), or both.

Because of the large sample size, it is possible that small and clinical unimportant estimates may become "statistically significant". Thus, the interpretation of the estimates should not primarily focus on statistical significance but should focus on the magnitudes of the point estimate and the estimates within the CI. Considering that the HR of CHD associated with onset of job strain (1.20, 95% CI 1.12-1.29) was of about the same magnitude as the HR of cardiovascular disease associated with obesity in the literature (36), we conclude that job strain was associated with a moderate, but clinically important, excessive risk of CHD.

Strengths and limitations

The strengths of this study are its population-based design, the large study population, the number of cases yielding precise estimates with narrow CI, the registerbased outcome ascertainment, and the repeated measure of exposure and confounders. To our knowledge, this is the first study analyzing the association between onset and removal of job strain and risk of CHD. Given the large study population, we were able to conduct analyses separately for men and women, demonstrating that associations were similar in both sexes. Using a JEM to assess job strain ensured that estimates were not affected by reporting bias.

The study also had several important limitations. Because we measured job strain with a JEM, we do not know if the individuals were indeed exposed to job strain but rather only that they worked in a job with a certain exposure probability. Consequently, there may have been non-differential exposure misclassification, likely biasing the estimates towards an underestimation of associations. Exposure misclassification may also have been caused by the fact that we measured job strain not with a standard instrument but with items approximating such an instrument and by selective nonresponse in the DWECS survey.

We defined persistent job strain as exposure to job strain in two subsequent years. We acknowledge that alternative definitions of persistence, using longer time periods, may have yielded different results.

It is possible that we overestimated the association between job strain and CHD due to residual confounding by socioeconomic position. Some studies have reported a higher prevalence of job strain in employees of low socioeconomic position (37) and it is well documented that low socioeconomic position is strongly associated with a higher risk of CHD in high-income countries (38), including Denmark (8). Consequently, we adjusted the estimates for an indicator of socioeconomic position, ie, household disposable income, and these adjustments resulted in attenuated estimates (as can be seen in tables 2-4 when comparing model 2 with model 3). In contrast to most other studies, we measured socioeconomic position not only at baseline but also during follow-up, increasing precision in the measurement of the potential confounder and allowing us to treat socioeconomic position as a time-varying covariate in some of the analyses. While this is a strength of our study, residual confounding by socioeconomic position remains a possibility and adjustments for additional indicators of socioeconomic position would have strengthened the study. However, we refrained from doing so in the main analyses as the two other measures of socioeconomic position in JEMPAD - occupational status and educational attainment, - were intertwined with our job-group-based measure of job strain. For the sake of completeness, we provided estimates adjusted for education in the supplementary analyses. As expected, the estimates were attenuated, but some remained of a considerable magnitude (eg, onset of job strain). When exploring the association between job strain and CHD across educational levels, we found a tendency for stronger associations among those with a high level of education. Previously, we had reported that the association between low education and cardiovascular morbidity and mortality was attenuated after accounting for both household income and job strain (8). Further studies examining the interplay of socioeconomic position and job strain with regard to risk of CHD are recommended.

We had no information on behavioral risk factors of CHD such as smoking, unhealthy diet or lack of physical activity (36). This can be considered a limitation, as many studies on job strain and CHD have routinely adjusted for these variables (1). However, smoking, diet and lack of physical activity may represent mediators rather than potential confounders for the association between job strain and CHD (39). Adjustment for factors on the causal pathway does not inform about confounding but would rather lead to underestimation of the association between the exposure and the outcome.

There are several other psychosocial work environment conditions that, either alone or in interplay with job strain, may affect coronary health. A recent metaanalysis showed that exposure to either job strain or effort-reward imbalance was associated with a 1.16-fold higher risk of CHD, whereas simultaneous exposure to both job strain and effort-reward imbalance yielded a 1.41-fold higher risk (4). Other recent studies suggest that exposure to job insecurity (2), workplace bullying (5), and workplace violence (5) may also contribute to risk of cardiovascular disease. We plan to address some of these potential risk factors in future analyses in JEMPAD.

Concluding remarks

Job strain, ascertained by a JEM, was associated with a 7-21% higher risk of CHD in the Danish workforce. This association was similar for men and women and was seen across different types of analyses, including job strain at baseline, number of years with job strain, persistent job strain, and onset of job strain. Removal of job strain in the year preceding the CHD event also yielded a higher risk of CHD, compared to persistent no job strain. One possible explanation for this result could be health selection.

It has been suggested that causal inference from observational epidemiological studies can be strengthened by triangulation, ie, combining results from different approaches with different methodological strengths and limitations, ideally with potential biases that are in opposite directions (40). We suggest that our results have strengthened the evidence that job strain may be a causal factor in the etiology of CHD. Our estimates were similar to the estimate of the IPD-Work consortium analyses that measured job strain not with a JEM but with individual-level data, an approach that was more vulnerable to reporting bias but less vulnerable to nondifferential misclassification than the approach taken in this study.

Acknowledgement

This study was supported by grants from the NordForsk Nordic Program on Health and Welfare (grant #75021), from the Danish Working Environment Research Fund (grants #17-2014-03 and #27-2017-03) and the Swedish Research Council of Health, Working Life and Welfare (grant #2017-01943). Mika Kivimäki was supported by NordForsk, the Academy of Finland (grant #311492) and Helsinki Institute of Life Science. The funders of the study had no role in study design, data collection, data analysis, data interpretation, writing of the report or decision to submit for publication.

Conflict of interest

The authors declare no conflicts of interest.

Protection of research participants

This study complies with the Declaration of Helsinki. No patients were involved. In Denmark, studies that are based on questionnaire and register-data only do not require approval from the National Committee on Health Research Ethics. The Danish Data Protection Agency approved this study through the joint notification of the National

Research Centre for the Working Environment (#2015-57-0074). All data are stored in a protected server environment hosted by Statistics Denmark.

Sidebar

In the Danish workforce, job strain at baseline, job strain within a five-year period before baseline, persistent job strain, as well as onset of job strain and removal of job strain were associated with a 7-21% higher risk of incident coronary heart disease compared to being unexposed to job strain.

Refers to the following texts of the Journal: 1993;19(1):21-28 2006;32(6):473-481 2003;29(4):2 7 072 79

Key terms: cardiovascular disease; CHD; cohort study; coronary heart disease; Denmark; employee; epidemiology; JEM; job control; job exposure matrix; job strain; psychological demand; psychosocial work environment; strain; stress; work stress

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32202306

Additional material

Please note that there is additional material available belonging to this article on the Scandinavian Journal of Work, Environment &Health -website.

Rugulies R, Framke E, Sørensen JK, Svane-Petersen AC, Alexanderson K, Bonde JP, Farrants K, Flachs EM, Magnusson Hanson LL, Nyberg ST, Kivimäki M, Madsen IEH. Persistent and changing job strain and risk of coronary heart disease. A populationbased cohort study of 1.6 million employees in Denmark. Scand J Work Environ Health. 2020;46(5):498-507. doi:10.5271/ sjweh.3891

Correspondence to: Reiner Rugulies, National Research Centre for the Working Environment, Lersø Parkallé 105, DK-2100 Copenhagen, Denmark. [E-mail: rer@nrcwe.dk]

Footnote

1 National Research Centre for the Working Environment, Copenhagen, Denmark.
2 Section of Epidemiology, Department of Public Health, University of Copenhagen, Denmark.
3 Department of Psychology, University of Copenhagen, Denmark.
4 Division of Insurance Medicine, Department of Clinical Neuroscience, Karolínska Institutet, Stockholm, Sweden.
5 Department of Occupational and Environmental Medicine, Bispebjerg and Frederiksberg Hospital, Denmark.
6 Stress Research Institute, Stockholm University, Stockholm, Sweden.
7 Clinicum, Faculty of Medicine, University of Helsinki, Helsinki, Finland.
8 Helsinki Institute of Life Sciences, Helsinki, Finland.
9 Department of Epidemiology and Public Health, University College London, London, UK.

References

References

1. Kivimäki M, Nyberg ST, Batty GD, Fransson EI, Heikkilä K, Alfredsson L, et al. Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis of individual participant data. Lancet. 2012;380(9852):1491-7. https://doi. org/10.1016/S0140-6736(12)60994-5.

2. Virtanen M, Nyberg ST, Batty GD, Jokela M, Heikkilä K, Fransson EI, et al. Perceived job insecurity as a risk factor for incident coronary heart disease: systematic review and metaanalysis. Br Med J. 2013;347:f4746. https://doi.org/10.1136/ bmj.f4746.

3. Kivimäki M, Kawachi I. Work stress as a risk factor for cardiovascular disease. Curr Cardiol Rep. 2015; 17(9):630. https://doi.org/10.1007/s11886-015-0630-8.

4. Dragano N, Siegrist J, Nyberg ST, Lunau T, Fransson EI, Alfredsson L, et al. Effort-reward imbalance at work and incident coronary heart disease: a multi-cohort study of 90,164 individuals. Epidemiology. 2017;28(4):619-26. https://doi. org/10.1097/EDE.0000000000000666.

5. Xu T, Magnusson Hanson LL, Lange T, Starkopf L, Westerlund H, Madsen IEH, et al. Workplace bullying and workplace violence as risk factors for cardiovascular disease: a multi-cohort study. Eur Heart J. 2018;40(14):1124-34. https:// doi.org/10.1093/eurheartj/ehy683.

6. Brotman DJ, Golden SH, Wittstein IS. The cardiovascular toll of stress. Lancet. 2007;370(9592):1089-100.

https://doi. org/10.1016/S0140-6736(07)61305-1.
7. Kivimäki M, Steptoe A. Effects of stress on the development and progression of cardiovascular disease. Nat Rev Cardiol. 2018;15(4):215-29. https://doi.org/10.1038/ nrcardio.2017.189.
8. Framke E, Sørensen JK, Andersen PK, Svane-Petersen AC, Alexanderson K, Bonde JP, et al. Contribution of income and job strain to the association between education and cardiovascular disease in 1.6 million Danish employees. Eur Heart J. 2020;41(11):1164-1178. https://doi.org/10.1093/eurheartj/ ehz870.
9. Petersson F, Baadsgaard M, Thygesen LC. Danish registers on personal labour market affiliation. Scand J Public Health. 2011;39(7 Suppl):95-8. https://doi. org/10.1177/1403494811408483.
10. Lynge E, Sandegaard JL, Rebolj M. The Danish National Patient Register. Scand J Public Health. 2011;39(7 Suppl):30-3. https://doi.org/10.1177/1403494811401482.
11. Burr H, Bjorner JB, Kristensen TS, Tüchsen F, Bach E. Trends in the Danish work environment in 1990-2000 and their associations with labor-force changes. Scand J Work Environ Health. 2003;29(4):270-9. https://doi.org/10.5271/ sjweh.731.
12. Feveile H, Olsen O, Burr H, Bach E. Danish Work Environment Cohort Study 2005: From idea to sampling design. Statistics in Transition. 2007;8(3):441-58.
13. International Labour Organization (ILO). ISCO-88. 2004. Available from: http://www.ilo.org/public/english/bureau/ stat/isco/isco88/. [Accessed: 22 May 2019].
14. Helweg-Larsen K. The Danish Register of Causes of Death. Scand J Public Health. 2011;39(7 Suppl):26-9. https://doi. org/10.1177/1403494811399958.
15. Madsen M, Davidsen M, Rasmussen S, Abildstrom SZ, Osler M. The validity of the diagnosis of acute myocardial infarction in routine statistics: a comparison of mortality and hospital discharge data with the Danish MONICA registry. J Clin Epidemiol. 2003;56(2):124-30. https://doi.org/10.1016/ S0895-4356(02)00591-7.
16. United Nations Educational Scientific and Cultural Organization (UNESCO). International Standard Classification of Education (ISCED 1997) 1997. Available from: http:// www.unesco.org/education/information/nfsunesco/doc/ isced_1997.htm. [Accessed: 22 May 2019].
17. Statistics Denmark. Documentation of statistics: Population and elections 2019. Available from: https://www.dst.dk/en/ Statistik/dokumentation/documentationofstatistics?subje ct=02. [Accessed: 22 May 2019].
18. Statistics Denmark. Documentation of statistics: Households, families and children 2019. Available from: https://www.dst. dk/en/Statistik/dokumentation/documentationofstatistics/ households-families-and-children. [Accessed: 22 May 2019].
19. Baadsgaard M, Quitzau J. Danish registers on personal income and transfer payments. Scand J Public Health. 2011;39(7 Suppl):103-5. https://doi.org/10.n77/14034948n405098.
20. Andersen JS, Olivarius Nde F, Krasnik A. The Danish National Health Service Register. Scand J Public Health. 2011;39(7 Suppl):34-7. https://doi.org/10.n77/1403494810394718.
21. Kivimäki M, Head J, Ferrie JE, Brunner E, Marmot MG, Vahtera J, et al. Why is evidence on job strain and coronary heart disease mixed? An illustration of measurement challenges in the Whitehall II study. Psychosom Med. 2006;68(3):398-401. https://doi.org/10.1097/01.psy.0000221252.84351.e2.
22. Alfredsson L, Karasek R, Theorell T. Myocardial infarction risk and psychosocial work environment: an analysis of the male Swedish working force. Soc Sci Med. 1982;16(4):463-7. https://doi.org/10.1016/0277-9536(82)90054-5.
23. Johnson JV, Stewart WF. Measuring work organization exposure over the life course with a job-exposure matrix. Scand J Work Environ Health. 1993;19(1):21-8. https://doi. org/10.5271/sjweh.1508.
24. Johnson JV, Stewart W, Hall EM, Fredlund P, Theorell T. Long-term psychosocial work environment and cardiovascular mortality among Swedish men. Am J Public Health. 1996;86(3):324-31. https://doi.org/10.2105/AJPH.86.3324.
25. Hammar N, Alfredsson L, Johnson JV. Job strain, social support at work, and incidence of myocardial infarction. Occup Environ Med. 1998;55(8):548-53. https://doi. org/10.1136/oem.55.8.548.
26. Rijs KJ, van der Pas S, Geuskens GA, Cozijnsen R, Koppes LL, van der Beek AJ, et al. Development and

validation of a physical and psychosocial job-exposure matrix in older and retired workers. Ann Occup Hyg. 2014;58(2):152-70.
27. Niedhammer I, Chastang JF, Levy D, David S, Degioanni S, Theorell T. Study of the validity of a job-exposure matrix for psychosocial work factors: results from the national French SUMER survey. Int Arch Occup Environ Health. 2008;82(1):87-97. https://doi.org/10.1007/s00420-008-0311-7.
28. Solovieva S, Pensola T, Kausto J, Shiri R, Heliovaara M, Burdorf A, et al. Evaluation of the validity of job exposure matrix for psychosocial factors at work. PLoS One. 2014;9(9):e108987. https://doi.org/10.1371/journal. pone.0108987.
29. Madsen IEH, Gupta N, Budtz-Jorgensen E, Bonde JP, Framke E, Flachs EM, et al. Physical work demands and psychosocial working conditions as predictors of musculoskeletal pain: a cohort study comparing self-reported and job exposure matrix measurements. Occup Environ Med. 2018;75(10):752-8. https://doi.org/10.1136/oemed-2018-105151.
30. Flachs EM, Petersen SEB, Kolstad HA, Schlünssen V, Svendsen SW, Hansen J, et al. Cohort profile: DOC·X: a nationwide Danish occupational cohort with eXposure data - an open research resource Int J Epidemiol. 2019;48(5):1413-1413k. https://doi.org/10.1093/ije/dyz110.
31. Milner A, Niedhammer I, Chastang JF, Spittal MJ, LaMontagne AD. Validity of a job-exposure matrix for psychosocial job stressors: Results from the Household Income and Labour Dynamics in Australia Survey. PLoS One. 2016;11(4):e0152980. https://doi.org/10.1371/journal. pone.0152980.
32. Torén K, Schiöler L, Giang WK, Novak M, Söderberg M, Rosengren A. A longitudinal general population-based study of job strain and risk for coronary heart disease and stroke in Swedish men. BMJ Open. 2014;4(3):e004355. https://doi. org/10.1136/bmjopen-2013-004355.
33. Bonde JP, Munch-Hansen T, Agerbo E, Suadicani P, Wieclaw J, Westergaard-Nielsen N. Job strain and ischemic heart disease: a prospective study using a new approach for exposure assessment. J Occup Environ Med. 2009;51(6):732-8. https:// doi.org/10.1097/JOM.0b013e3181a826f6.
34. Sokejima S, Kagamimori S. Working hours as a risk factor for acute myocardial infarction in Japan: case-control study. Br Med J. 1998;317(7161):775-80. https://doi.org/10.1136/ bmj.317.7161.775.
35. Lundin A, Falkstedt D, Lundberg I, Hemmingsson T. Unemployment and coronary heart disease among middle-aged men in Sweden: 39 243 men followed for 8 years. Occup Environ Med. 2014;71(3):183-8. https://doi.org/10.1136/ oemed-2013-101721.
36. Cheng S, Claggett B, Correia AW, Shah AM, Gupta DK, Skali H, et al. Temporal trends in the population attributable risk for cardiovascular disease: the Atherosclerosis Risk in Communities Study. Circulation. 2014;130(10): 820-8. https:// doi.org/10.1161/CIRCULATIONAHA.113.008506.
37. Hoven H, Siegrist J. Work characteristics, socioeconomic position and health: a systematic review of mediation and moderation effects in prospective studies. Occup Environ Med. 2013;70(9):663-9. https://doi.org/10.1136/ oemed-2012-101331.
38. Schultz WM, Kelli HM, Lisko JC, Varghese T, Shen J, Sandesara P, et al. Socioeconomic status and cardiovascular outcomes: Challenges and interventions. Circulation. 2018;137(20):2166-78. https://doi.org/10.1161/ CIRCULATIONAHA.117.029652.
39. Siegrist J, Rödel A. Work stress and health risk behavior. Scand J Work Environ Health. 2006;32(6):473-81. https://doi. org/10.5271/sjweh.1052.
40. Munafô MR, Davey Smith G. Robust research needs many lines of evidence. Nature. 2018;553(7689):399-401. https:// doi.org/10.1038/d41586-018-01023-3.
Received for publication: 27 August 2019

DETAILS

Subject:	Occupational stress; Socioeconomic factors; Confidence intervals; Heart diseases; Cardiovascular diseases; Cardiovascular disease; Regression analysis; Occupational health; Exposure; Statistical analysis; Population studies; Coronary artery disease; Health risks; Mentoring programs; Cohort analysis
Business indexing term:	Subject: Occupational stress
Location:	Denmark
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	498-507,498A
Publication year:	2020
Publication date:	2020
Section:	Original article
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English
Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3891
ProQuest document ID:	2444686089
Document URL:	https://www.proquest.com/scholarly-journals/persistent-changing-job-strain-risk-coronary/docview/2444686089/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2023-03-06

The mediating effect of work-life interference on the relationship between work-time control and depressive and musculoskeletal symptoms

Albrecht, S C [1] ; Kecklund, G; Leineweber, C [1] Stress Research Institute, Stockholm University, 106 91 Stockholm, Sweden

ProQuest document link

ABSTRACT (ENGLISH)

Objectives Evidence shows that work-time control (WTC) affects health but underlying mechanisms are still unclear. Work-life interference (WLI) might be a step on the causal pathway. The present study examined whether WLI mediates effects on mental and physical health and contrasted these to other causal pathways. Methods Four biennial waves from the Swedish Longitudinal Occupational Survey of Health (SLOSH, N=26 804) were used. Cross-lagged analyses were conducted to estimate if WLI mediated effects from WTC (differentiating between control over daily hours and time off) to subsequent depressive and musculoskeletal symptoms. Other causal directions (reversed mediation, direct and reversed direct effects) and robustness of mediation (by including covariates) were examined. Results WLI partially mediated the relationship of WTC (control over daily hours/time off) with both health outcomes. Indirect effect estimates were small for depressive symptoms (-0.053 for control over time off and -0.018 for control over daily hours) and very small for musculoskeletal symptoms (-0.007 and -0.003, respectively). While other causal directions were generally weaker than causal mediational pathways, they played a larger role for musculoskeletal compared to depressive symptoms. Estimates relating to control over time off were in general larger than for control over daily hours. Conclusions Our results suggest that WLI mediates part of the effect from WTC to mental/musculoskeletal symptoms, but small estimates suggest that (i) WTC plays a small but consistent role in effects on health and (ii) particularly regarding musculoskeletal disorders, other causal directions and mediators need to be further examined.

FULL TEXT

Headnote

Objectives Evidence shows that work-time control (WTC) affects health but underlying mechanisms are still unclear. Work-life interference (WLI) might be a step on the causal pathway. The present study examined whether WLI mediates effects on mental and physical health and contrasted these to other causal pathways.

Methods Four biennial waves from the Swedish Longitudinal Occupational Survey of Health (SLOSH, N=26 804) were used. Cross-lagged analyses were conducted to estimate if WLI mediated effects from WTC (differentiating between control over daily hours and time off) to subsequent depressive and musculoskeletal symptoms. Other causal directions (reversed mediation, direct and reversed direct effects) and robustness of mediation (by including covariates) were examined.

Results WLI partially mediated the relationship of WTC (control over daily hours/time off) with both health outcomes. Indirect effect estimates were small for depressive symptoms (-0.053 for control over time off and -0.018 for control over daily hours) and very small for musculoskeletal symptoms (-0.007 and -0.003, respectively). While other causal

directions were generally weaker than causal mediational pathways, they played a larger role for musculoskeletal compared to depressive symptoms. Estimates relating to control over time off were in general larger than for control over daily hours.
Conclusions Our results suggest that WLI mediates part of the effect from WTC to mental/musculoskeletal symptoms, but small estimates suggest that (i) WTC plays a small but consistent role in effects on health and (ii) particularly regarding musculoskeletal disorders, other causal directions and mediators need to be further examined.
Key terms autonomy; flexible work hour; psychosocial factor; working hour; work-life balance; mental health; depression; depressive symptom; musculoskeletal pain; physical health; cohort study; mediation; Swedish Longitudinal Occupational Survey of Health.

An increasing body of research presents evidence that workers' control over their working hours - work-time control (WTC) - is associated with health outcomes over time (1-3). Adapted from Knauth (4), WTC can be described as an individual's autonomy regarding duration and distribution of working hours. This definition points to a two-dimensional structure of WTC (5, 6): While control over daily working hours reflects daily length and starting and ending times of work, control over time off relates to taking time off from work in the form of taking breaks, running private errands during work, and scheduling vacation and other types of leave. Although WTC enables workers to structure working hours, this inevitably also affects non-work-time (7). WTC is conceptually related but not identical to the jobcontrol dimension in the Job-Demands-Control Model (8). Job control refers to autonomy regarding content of work and how work tasks are performed, whereas WTC describes the temporal aspect of autonomy at work (9). By allowing workers greater control over their working hours, positive effects on health, work-life balance, well-being and even performance are predicted. Evidence shows that while lower levels of WTC are associated with deteriorating health outcomes over time (10, 11), high WTC may prevent ill-health. In several longitudinal studies, higher levels of WTC were associated with fewer depressive symptoms, lower levels of fatigue, and lower risk of disability pension due to musculoskeletal and mental disorders (3, 12, 13). Some studies observed stronger effects of control over time off on health (in contrast to control over daily hours) suggesting that taking breaks and scheduling vacation is particularly important for workers (3, 14, 15). These results highlight that both mental and physical health are related to WTC, but the underlying mechanisms behind these associations are still not well understood.

On the one hand, effects of WTC on health can be explained by the effort-recovery model (16) stating that efforts spent at work need to be balanced with recovery time. Insufficient recovery can lead to adverse health outcomes. WTC allows to manage workload from a temporal perspective (control over daily hours) as well as ensure workers can take opportunities to recover both in and outside of working hours (control over time off) (1). On the other hand, WTC enables workers to manage (and reduce) strain due to conflicting responsibilities stemming from work and private/family life. By adapting working hours and time off from work, time between work and personal interests can be better balanced-which in turn affects health and well-being (1). To advance our understanding of causal pathways and mediator variables linking WTC with health, the present paper focused on the mediating role that work-family conflict may play in this relationship.

Work-family conflict can emerge when workers experience difficulties in balancing responsibilities from work and family life. It is defined as the conflict that arises when demands from work and family roles are to some extent mutually incompatible (17). This concept has been broadened to include non-worktime roles and responsibilities other than family - as individuals without dependant children can still experience conflicts between private life and work (18). In a modern world, individuals often have a multitude of responsibilities, interests and social groups, all of which need to be balanced with work commitments. This is better captured by the term work-life interference (WLI) (19). While the relationship of work and non-work is bidirectional, this contribution focusses on work interfering with private life, and not vice versa since this direction is generally found to be stronger and more common (20).

A theory was previously proposed for why WLI could be an intermediate step on the causal pathway between WTC and subsequent health/well-being. If workers can self-determine working hours and regulate time to match individual needs and align them with family and other responsibilities, perceiving WLI should be less likely and work/non-work balance would be promoted (1). In turn, this balance facilitates maintaining well-being and good health-related behavior (21). In contrast, lower levels of WTC increase the likelihood to experience WLI while higher levels are associated with a reduction in WLI (22). Moreover, one study found differential effects between the two sub-dimensions of WTC. While control over time off was directly associated with WLI, control over daily hours only buffered against negative effects from long contractual working hours on WLI (23). High WLI is linked to unfavorable consequences for health, for example major depression (24), emotional exhaustion (25), headaches, and sleep problems (26). Moreover, ill-health can aggravate WLI and decrease WTC; research on reversed causal relationships is scarce but particularly mental health has been found to affect different work characteristics, either by changing perceptions of the respective factor or changing the job and work environment (27).

So far, only two studies have looked at the mediating role of WLI in the WTC-health relationship. In a natural experiment, researchers found that an increase in WTC at time 1 decreased interference of work with private life (time 2), which was (at the same time point) associated with longer sleep duration, better sleep quality, less emotional exhaustion, and slightly more frequent physical exercise (2). Drawing conclusions on mediation from this study however is problematic as the design was not fully longitudinal (28).

A more recent study based on a sample of workers in the healthcare sector concluded that WLI did play a mediating role in the relation between WTC and emotional exhaustion (29). The author also found evidence that emotional exhaustion mediated effects of WTC on WLI, meaning WTC affected emotional exhaustion which in turn influenced WLI. Although this study made an important contribution, it has limitations: a brief time span of one year, WTC being measured only at baseline, and a narrow set of covariates (age, gender and sleep time). The study did not allow for detecting effects of WTC that unfold over a longer period of time, effects on other health outcomes or in other population groups, nor did it sufficiently address confounding bias.

Aims

To extend evidence from past research, the present study investigated whether WLI mediates the relationship between the two sub-dimensions of WTC (control over daily hours and control over time off) with depressive and musculoskeletal symptoms. Particularly, we were interested in potential differential results between mental and physical health outcomes as well as the two WTC sub-dimensions. Additionally, we aimed to assess reversed mediational effects, direct, and reversed direct effects in these relationships.

Methods

Study design and population

The data come from the Swedish Longitudinal Occupational Survey of Health (SLOSH), which is a biennial postal survey. An open cohort of participants, SLOSH is based on the 2003-2011 Swedish Work Environment Survey (SWES), which consisted of a sample of gainfully employed Swedish residents (aged 16-64 years). A full cohort profile can be found elsewhere (30). Different questionnaires are completed by those in paid work (>30% full-time) and those who are temporarily or terminally outside of paid work. The sample is approximately representative in terms of gender and distribution of labor market sectors.

The present study sample is based on participants who responded to at least one SLOSH questionnaire (for those in work) between 2010-2016 with a total sample size of 26 804 (response rates 2010: 56.4%, N=11 525; 2012: 56.8%, N=9880; 2014: 52.6%, N=20 316; 2016: 50.9%, N=19 360). The Regional Research Ethics Board in Stockholm ethically approved both SLOSH (2012/373-31/5) and the present study (2014/696-31/5).

Measures

Work-time control. A 5-item scale adapted from Ala-Mursula et al (5) measured perceived control over working hours, rated on a 5-point Likert scale from 1 (very little) to 5 (very much). Items differentiate between two subdimensions of WTC: control over daily hours (items regarding length and starting and ending time of work) and control over time off (items on taking breaks, running private errands, taking vacation/leave) (5, 6). A discussion of properties of the scale can be found elsewhere (6). We calculated means for each sub-dimension and each of the four waves between 2010-2016. Cronbach's alphas for control over daily hours were 0.92 (2010), 0.93 (2012), 0.93 (2014) and 0.93 (2016), and for control over time off 0.75, 0.75, 0.77, and 0.77.

Work-life interference. WLI was measured four times between 2010-2016 using a 4-item scale (19), with items such as "I come home from work too tired to do things I would like to do". Responses were rated as "not at all", "rarely", "sometimes", "often" or "almost all the time"; means were calculated. Cronbach's alphas were 0.89 (2010), 0.89 (2012), 0.90 (2014), and 0.91 (2016).

Depressive symptoms. A 6-item subscale of the Symptom Checklist (SCL-CD) measured core depressive symptoms with items regarding feeling blue, having no interest in things, feeling low in energy, worrying too much, blaming oneself, and perceiving everything as effortful. The scale's validity and unidimensionality has been previously confirmed (31). Respondents rated how troublesome symptoms were during the last week from "not at all" (0) to "extremely" (4). Sum scores were calculated for each wave from 2010-2016. Cronbach's alphas were 0.92 (2010), 0.91 (2012), 0.91 (2014), and 0.89 (2016).

Musculoskeletal symptoms. Between 2010-2016, respondents were asked if they had been diagnosed with or experienced a disease in their back, joints or muscles during the last two years. Responses were rated as "no", "yes, but doesn't affect my life", "yes, affects my life a little", and "yes, affects my life a lot".

Covariates

Covariate selection was led by theoretical considerations and previous knowledge on which variables were related to at least two of the main constructs. In a second step, covariates were selected based on directed acyclic graphs (DAG). Participant's gender and socio-economic position (manual, lower-manual, and medium-to-high non-manual work) were available from register data throughout the study period. Self-reported data were used for the following covariates in 2010-2016: (i) age, (ii) highest level of education, (iii) shift-work status (defined as those regularly working shift/rostered hours in- or excluding nights or exclusively night hours), (iv) weekly working hours (<10->55 hours per week), (v)civil (cohabiting yes/no), and (vi) parental status (yes/no).

Statistical analysis

Based on a path analysis model in structural equation modelling, we used cross-lagged panel models for mediation analyses which take stability and correlation of measurements over time into account. The two health outcomes - depressive and musculoskeletal symptoms - were analyzed separately to limit model complexity and allow for detecting differential effects. Data preparation was performed in SPSS Statistics for Windows, version 22.0 (IBM Corp, Armonk, NY, USA) while SEM was executed in Mplus 7 (32). Maximum likelihood estimates are reported and full information maximum likelihood (FIML) was utilized to reduce bias due to missing data (33).

To test for full longitudinal mediation, we used a step-wise analytic approach that was adapted from Cole &Maxwell (28) and Little (34, 35). It needs to be established first whether including mediational paths to a baseline model improves model fit and second, whether any other causal processes above and beyond the mediational pathways are important (such as reversed mediation, direct effects or reversed direct effects). Evidence of other directed pathways does not automatically contradict mediation - the purpose is rather to evaluate the relations between constructs (34). Lastly, robustness of the mediation model was tested by entering all relevant directed paths, allowing different lags between time points within constructs, and adding covariates into the model. Mediation pathways should remain significant in this final model. Regarding covariates, gender, age, and highest educational level were included as time-stable variables and correlated with each construct at the first time point. The remaining covariates (socio-economic position, shift-work status, weekly working hours, civil and parental status) were measured at the first and last time point and allowed to correlate with all constructs at the respective time (eg, civil status 1<->WTC1; civil status 4<->WTC4).

In the modelling process, paths with the same time lag were held constant in all models, respectively (eg, WTC1->WTC2 was fixed to the same coefficient as WTC2->WTC3). Specifically, the following models (all of which including auto-regressive paths, eg, WTC1->WTC2) were estimated and compared against each otherwhere applicable (figure 1): (i) a null-model (model 0) that allowed only cross-sectional covariance between constructs while omitting any cross-lagged relationships over time; (ii) a causal-mediation-only model (model 1) with additional cross-lagged pathways [eg, WTC1->WLI2->Health3 (H3)]; (iii) a fullmediation model (model 2) including causal mediational pathways and reversed mediational directions (eg, HI->WLI2->WTC3); (iv) a direct-effects model (model

3) where direct effects were additionally entered into the previous model (eg, WTC1->H2); (v) a reverseddirect effects model (model 4) where reversed directs effects were added (eg, HI->WTC2); and (vi) a final model (model 5), step-wise including directed paths that improved model fit in previous tests (eg, causal mediation, reversed mediation, direct and reversed direct effects), lags >1 within constructs, and covariates that was pruned (ie, non-significant paths removed). If mediation remained significant in this final model, the total indirect effect was calculated (based on a model letting other causal pathways be correlated) as the sum of the products (see figure 1, model 1) of all paths from WTC at the first time point to health at the last time point going through the mediator WLI (eg, WTC1^WLI2^H3^H4) (35). Significance/confidence intervals (CI) for the total indirect effect were assessed with bootstrap estimation (5000 samples).

Considering the amount of tests included in the analysis, the significance level was set to 0.001 for all tests (36). Models were evaluated both on model fit indices and the Chi2 statistics - the latter one needing to be treated cautiously as it is affected by large sample sizes and trivial disturbances easily become significant (37). Acceptable absolute fit is suggested by the root mean square error of approximation (RMSEA) <0.08 (38). Incremental fit is regarded as good with values >0.95 on the comparative fit index (CFI) (39). Relative model fit of nested models is indicated by lower values on the Bayesian information criterion (BIC) (40). Chi2 difference tests were used to compare nested models (model 0-4) with a significant result indicating that the reduced, simpler model may be too parsimonious and essential variables might be missing (28, 34).

Results

Descriptives

Sample characteristics are presented in table 1. Participants were on average 49 years old at baseline (SD=11.77). Lower levels of WTC (both sub-dimensions) were reported by those who were women, older, lower educated (particularly control over daily hours), single, without children, manual workers, fewer weekly working hours and working shift-work (particularly control over daily hours). WFC was experienced more often by those who were women, highly educated, single or without children (both small differences), non-manual workers, working more weekly hours, and working shifts. Those perceiving more depressive symptoms were women, older, better educated, without children, single, and working few or many weekly hours. More musculoskeletal symptoms were perceived by those who were women, younger, lower educated, cohabiting, working shift-work, manual workers, and working fewer weekly hours.

Cross-sectional inter-correlations and means/standard deviations are found in the supplementary material (www.sjweh.fi/show_abstract.php?abstract_id=3887), table S1. Correlation coefficients of control over daily hours/time off varied between -0.03- -0.20 for WLI, -0.04- -0.14 for depressive symptoms, and -0.06- -0.12 for musculoskeletal symptoms. Coefficients for control over time off were generally larger than for control over daily hours. WLI correlated with depressive symptoms (0.33-0.51) and with musculoskeletal symptoms (0.100.16). All coefficients became in general smaller with a larger interval between measurements.

Depressive symptoms

For control over daily hours we found improved model fit by adding causal mediational pathways via WLI (model 1, WTC^WLI ß=-0.011, P<0.001; WLI^H ß=0.647, P<0.001) to a model allowing only crosssectional covariances (model 0). Entering reversed mediation pathways (model 2, H^WLI ß=0.021, P<0.001; WLI^WTC ß=-0.009, P=0.119) fit data better than causal mediation pathways only as indicated by model fit indexes and Chi2 difference tests. Adding direct (model 3, WTC^H ß=-0.033, P=0.035) and reversed direct pathways (model 4, H^WTC ß=-0.003, P=0.010) however did not substantially improve model fit (table 2). Thus, the final model (model 5, standardized estimates in supplementary figure S1) included causal mediation and initially also reversed mediation paths; since a part of the reversed mediation pathways became non-significant (WLI^WTC), these paths were pruned in model 5. Pathways from WTC to subsequent WLI were non-significant before inclusion of covariates (WTC^WLI ß=-0.010, P=0.001). In the final model (including adjustment for potential confounders), all coefficients of causal mediation were significant (WTC^WLI ß=-0.013, P<0.001, WLI^H ß=0.391, P<0.001). The total indirect effect estimate was -0.018 (95% CI -0.026- -0.010, P<0.001, table 3); that is, for every 1-unit increase of the mean score of

control over daily hours (range 1-5) a decrease in the sum score of depressive symptoms (range 0-24) of 0.018 is attributable to WLI.
For control over time off, causal mediational pathways (model 1, WTC^WLI ß=-0.033, P<0.001, WLI^H ß=0.639, P<0.001) fit data better than crosssectional covariances only (model 0). Reversed mediation paths (model 2, H^WLI ß=0.021, P<0.001, WLI^WTC ß=-0.018, P<0.001) were retained in the final model (model 5) as those paths seemed to explain variance above and beyond causal mediational pathways. Direct effects (model 3, WTC^H ß=-0.040, P=0.046) and reversed direct effects (model 4, H^WTC ß=-0.003, P=0.001) did not substantially improve model fit (table 2). In the final model (supplementary figure S2), inclusion of covariates did not substantially change results and causal mediation pathways remained significant (WTC^WLI ß=-0.035, P<0.001, WLI^H ß=0.387, P<0.001). The total indirect effect was estimated at -0.053 (95% CI -0.065- -0.042, P<0.001), meaning that for every 1-unit increase in the mean score of control over time off, a decrease in the sum score of depressive symptoms of 0.053 is attributable to WLI.
Musculoskeletal symptoms
For control over daily hours, model fit improved by entering causal mediation paths (model 1, WTC^WLI ß=-0.013, P<0.001, WLI^H ß=0.069, P<0.001) to the null model. Reversed mediation (model 2, H^WLI ß=0.035, P=0.001, WLI^WTC ß=-0.009, P=0.110), direct (model 3, WTC^H ß=-0.025, P<0.001) and reversed direct paths (model 4, H^WTC ß=-0.031, P<0.001) were retained in the final model (table 4). Covariate inclusion attenuated estimates only slightly. Reversed mediation paths from WLI to WTC became non-significant (thus were removed from the model), while causal mediation pathways from WTC to musculoskeletal symptoms via WLI remained significant (WTC^WLI ß=-0.019, P<0.001, WLI^H ß=0.053, P<0.001) in the final model (standardized estimates in supplementary figure S3). The total indirect effect was -0.003 (95% CI -0.004- -0.002, P<0.001).
For control over time off, model fit improved by entering causal mediation paths (model 1, WTC^WLI ß=-0.036, P<0.001, WLI^H ß=0.068, P<0.001) to the null model. Model fit further improved when adding reversed mediation (model 2, H^WLI ß=0.030, P<0.001, WLI^WTC ß=-0.018, P=0.110), direct (model 3, WTC^H ß=-0.035, P<0.001), and reversed direct paths (model 4, H^WTC ß=-0.030, P<0.001). Again, reversed indirect pathways from WLI to subsequent WTC became non-significant in the final model (model 5, supplementary figure S4) and were removed. Including covariates slightly attenuated most estimates, but causal mediation paths remained significant (WTC^WLI ß=-0.045, P<0.001, WLI^H ß=0.048, P<0.001). The total indirect effect was estimated at -0.007 (95% CI -0.008- -0.005, P<0.001).
Discussion
Using a large, prospective sample of the Swedish workforce, this panel study found that WLI partially mediated effects of WTC (control over daily hours and time off) on two different health indicators: higher WTC led to less WLI, which in turn benefitted health to a small degree. Indirect effects were slightly larger for depressive symptoms and very small for musculoskeletal symptoms. These effects were stronger than those of other causal directions, with the exception of effects from depressive symptoms to subsequent WLI. Reversed mediation, direct, and reversed direct effects generally played a larger role for musculoskeletal compared to depressive symptoms. Out of the two sub-dimensions of WTC, control over time off consistently showed larger effects than control over daily hours.
Even though indirect effect estimates were significant but small-to-very small in size, this needs to be put in relation to the overall effect WTC has on WLI and health. In results presented in this study, cross-sectional correlations between concepts were very small but con- sistently significant. A comprehensive systematic review on the topic showed that effects of WTC on work-life balance were fairly small but evidence was deemed as strong and consistent (1). Evidence is slightly less consistent but increasing regarding health outcomes which may be due to lack of power to detect small effects in studies with smaller samples - larger studies tend to find effects (3, 12, 41) as opposed to smaller ones (42, 43). WTC is one small part of an individual's psychosocial work environment and describes a very specific area. Therefore, small effect sizes are not unexpected and not unusual for other factors of the work environment either (44).
Results from this study are in line with previous research. Based on an occupational cohort with one year follow-up,

a study found that WLI mediated the relationship between a general measure of WTC and emotional exhaustion (29). Our findings support these results but additionally highlight several points: (i) the two sub-dimensions of WTC differed consistently in size; (ii) indirect effect estimates varied for different health outcomes; (iii) reciprocal mediation, direct and reversed direct effects explained a smaller part of the effect; and (iv) effects remained robust even after controlling for a number of covariates.

Sub-dimensions of WTC

In results presented here, control over time off (such as taking breaks and vacation) was associated more strongly with health indicators as well as WLI, and WLI better explained prospective effects of control over time off in contrast to control over daily hours. Previous research found that workers reported the highest need (even though prevalence was also high) for controlling when to take leave/vacation while control over daily hours was less required (and less common) (15). Self-determining daily starting and ending times of work might depend to a larger degree on cultural and organizational norms (45) and may therefore be less important to workers. Some research suggests that despite flexibility to determine starting and ending times of work, employees may not make the best choice in terms of recovery and sleep, particularly regarding shiftwork (46). On the other side, control over time off may prevent WLI by allowing individuals to take breaks/run private errands when needed and to plan days off from work longer in advance.

In the literature, WTC is often used as one global measure (1) but the current study and previous research (3, 15) highlight that different aspects of WTC might affect health differently and vary in their mechanisms. Research on flexible working hours should take potential differential effects into account and consider measuring and analyzing sub-dimensions of WTC.

Comparing the two health outcomes

We compared two health indicators regarding mental and physical health - depressive and musculoskeletal symptoms. Mediational pathways via WLI were stronger for depressive symptoms, particularly between WLI and depressive symptoms. This suggests that WTC may affect mental health to a larger extent than physical health. Few studies have directly compared mental and physical health regarding WTC. Findings from a Finnish study showed that while disability pension due to musculoskeletal disorders was more consistently associated with WTC, effects on disability pension due to mental disorders were stronger (with a smaller sample and wider confidence intervals) (12). Moreover, WLI may be more strongly related to mental health (26) and hence mediate effects of WTC on health more for mental than physical health. Other mediating variables might play a larger role in explaining effects of WTC on physical health, as indicated by the presence of other causal directions.

Other causal directions

Even though causal mediation coefficients (WTC^WLI and WLI^H) were an important contributor to explained variance, we still found evidence for reversed mediational effects: health affected subsequent levels of WLI, which in turn influenced perceived WTC. We found similar results in a previous study where, although reciprocal effects were found, causal effects from WTC to subsequent depressive symptoms explained data better (3). Our results extend these findings and suggest that while WLI mostly acts as mediating variable in effects of WTC on health, there are some reversed causal processes: sub-optimal health increases perceived conflict between work and private life, which in turn decreases ratings of WTC. However, in all final models, we found that pathways from WLI to subsequent levels of WTC became non-significant, indicating that reversed effects were predominantly from health to subsequent WLI. Pathways from depressive symptoms to WLI were particularly strong. These changes could be "objective" decreases in WLI, but it is more likely that a decrease in health changes perceptions of stressors and resources, which in turn affects rating of WLI.

In our results, direct pathways explained part of the effect from WTC to subsequent musculoskeletal symptoms but did not substantially improve model fit regarding depressive symptoms. This means either that WTC directly affects physical health - eg, by promoting physical relaxation or facilitating healthy behavior - or, more likely, that other mediating variables play an important role within the effect chain (summarized as the direct effect in our models), eg, a sense of autonomy, reduced stress, increased muscular relaxation and reduced physical strain. In line with the

effort-recovery model (16), recovery may be of particular interest to investigate as potential mediator. WTC allows workers both to recover (mentally and physically) by taking breaks when needed at work and to schedule time off from work. If workers utilize WTC, especially control over time off, to increase recovery opportunities, this could buffer against work overload and chronic physical strain but also WLI, fatigue and other negative health effects.
In our study, direct and reversed direct effects played a smaller role for depressive symptoms. This might indicate that WLI is a more important link in the chain of causation for depressive symptoms, meaning that more of the effect from WTC to depression goes via WLI than for musculoskeletal symptoms. This notion is supported by results from Hämmig et al (26) showing that work-life conflict was more strongly associated with mental than physical health. Reversed direct effects (but also reversed mediational paths) could indicate that results regarding musculoskeletal symptoms may be slightly biased by unmeasured symptoms of health before the study. Overall, our results suggest the relationship between WTC and musculoskeletal disorders is less clear, and WLI seems to explain a smaller part of this effect.

Strengths and limitations

The present study has some key strengths. We used a prospective design with panel data spanning six years, which is approximately representative of the Swedish working population. The analysis accounted for stability and correlation of measurements over time of all variables. We examined two different health indicators and considered a number of covariates.
However, several limitations need to be highlighted. This study is almost exclusively based on self-reported data and known issues with this type of data apply. As with all observational studies, we cannot rule out that unmeasured confounding and especially intermediate confounders could have biased mediational estimates. To minimize validity concerns, we utilized DAG and included several relevant covariates, but estimates might still be biased due to not-included variables. The statistical method used here comes with strong assumptions, among them the one of ergodicity, which allows to generalize from population processes to the individual (47). While especially WTC may be less affected by stable, trait-like differences, mixing within- and between-person variance may still have created bias in our results (specifically considering reversed causation). Attrition could have been a problem in our study. As a number of baseline data are available for non-responders after the first included wave, it was possible to utilize FIML estimation to fill in missingness under the missing at random (MAR) assumption (48). Musculoskeletal symptoms were measured with one categorical item only. We repeated analyses with diagonally weighted least squares (WLSMV) estimation and results did not differ to maximum likelihood estimation regarding model decisions and general direction. Longitudinal mediation analysis relies heavily on using data with an optimal lag between time points, often without knowing what the optimal lag is for effects to fully unfold (34). In our study, repeated measurements were available every other year allowing us to examine potential effects that develop over a longer period of time and become manifest in mental and physical health. However, we cannot be certain that two years is the optimal lag to reach maximum effect size and we were unable to investigate effects unfolding over a shorter (or longer) period of time.

Practical implications

Even though effects of WTC via WLI on subsequent health were comparably small, results presented here have implications for employers. Addressing factors of work environment can be useful as it reaches the majority of, if not all employees with an intervention. As WTC is a modifiable factor in most occupations (at least to some degree), an increase in autonomy regarding working hours could help employees to align work and private life better and lessen build-up of health problems, particularly regarding mental health. Especially control over time off appears to buffer against work-life conflict, which in turn can prevent ill-health. At the same time, our results suggest that ill-health, especially mental health issues, can negatively impact levels of perceived WLI and WTC. If baseline health within a group of workers is already deteriorating, it is most likely helpful to not only increase control over working hours but also address health issues which in turn may improve perceived WLI and WTC.

Concluding remarks

We found evidence that WLI partially mediates effects from WTC to subsequent health - particularly regarding depressive symptoms and to a lesser degree for musculoskeletal pains. Our results highlight that one subdimension of WTC (ie, control over time off) seemed to buffer more against WLI and in turn was associated with a decrease in depressive and musculoskeletal symptoms. Reversed mediational and direct effects still played a role, indicating both reversed causality and remaining unexplained mechanisms, especially regarding physical health. Future research needs to further advance our understanding of the causal pathway between WTC and long-term health effects and different potential mediators should be investigated.

Acknowledgements

We would like to thank all SLOSH participants. This work was supported by the Swedish Research Council for Health, Working Life and Welfare (2013-0448) and NordForsk, the Nordic Programme on Health and Welfare (74809).

Conflict of interest

The authors declare no conflicts of interest.

Sidebar

This is the first full panel study including four waves over 6 years to investigate causal pathways behind work-time control affecting health. Results imply that increased control over time off and (to a lesser degree) control over daily hours buffer against work-life interference which in turn can prevent particularly depressive and to some extent musculoskeletal symptoms.

Refers to the following texts of the Journal: 2004;30(2):139-148 2005;31(1):3-14 2006;32(6):502-514 2012;38(1):56-64. 2012;38(4):299-313 2014;40(1):66-73 2015;41(4):347-355

Key terms: autonomy; depression; depressive symptom; flexible work hour; mediating effect; mediation; mental health; musculoskeletal; musculoskeletal pain; musculoskeletal symptom; physical health; psychosocial factor; Swedish Longitudinal Occupational Survey of Health; work-life balance; work-life interference; work-time control; working hour

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32118284

Additional material

Please note that there is additional material available belonging to this article on the Scandinavian Journal of Work, Environment &Health -website.

Albrecht SC, Kecklund G, Leineweber C. The mediating effect of work-life interference on the relationship between work-time control and depressive and musculoskeletal symptoms. Scand J Work Environ Health. 2020;46(5):469-479. doi:10.5271/ sjweh.3887

Correspondence to: Sophie C Albrecht, Stress Research Institute, Stockholm University, 106 91 Stockholm, Sweden. [E-mail: sophie. albrecht@su.se]

Footnote

1 Stress Research Institute, Stockholm University, Stockholm, Sweden.

References

References

1.Nijp HH, Beckers DG, Geurts SA, Tucker P, Kompier MA. Systematic review on the association between employee worktime control and work-non-work balance, health and well-being, and job-related outcomes. Scand J Work Environ Health 2012 Jul;38(4):299-313. https://doi. org/10.5271/sjweh.3307.

2. Moen P, Kelly EL, Tranby E, Huang Q. Changing work, changing health: can real work-time flexibility promote health behaviors and well-being? J Health Soc Behav 2011 Dec;52(4):404-29. https://doi. org/10.1177/0022146511418979.

3. Albrecht SC, Kecklund G, Rajaleid K, Leineweber C. The longitudinal relationship between control over working hours and depressive symptoms: results from SLOSH, a population-based cohort study. J Affect Disord 2017 Jun;215:143-51. https://doi.org/10.1016/jjad.2017.03.010.

4. Knauth P. Innovative worktime arrangements. Scand J Work Environ Health 1998;24 Suppl 3:13-7.

5. Ala-Mursula L, Vahtera J, Linna A, Pentti J, Kivimäki M. Employee worktime control moderates the effects of job

strain and effort-reward imbalance on sickness absence: the 10-town study. J Epidemiol Community Health 2005 Oct;59(10):851-7. https://doi.org/10.1136/ jech.2004.030924.
6. Albrecht SC, Kecklund G, Tucker P, Leineweber C. Investigating the factorial structure and availability of work time control in a representative sample of the Swedish working population. Scand J Public Health 2016 May;44(3):320-8. https://doi.org/10.1177/1403494815618854.
7. Fagan C. The temporal reorganization of employment and the household rhythm of work schedules. Am Behav Sci 2001;44(7): 1199-212. https://doi. org/10.1177/00027640121956728.
8. Karasek RA, Theorell T. Healthy Work: Stress, Productivity, and the Reconstruction of Working Life. New York: Basic Books; 1990.
9. Härmä M. Workhours in relation to work stress, recovery and health. Scand J Work Environ Health 2006 Dec;32(6):502-14. https://doi.org/10.5271/sjweh.1055.
10. Ala-Mursula L, Vahtera J, Kivimäki M, Kevin MV, Pentti J. Employee control over working times: associations with subjective health and sickness absences. J Epidemiol Community Health 2002 Apr;56(4):272-8. https://doi. org/10.1136/jech.56.4.272.
11. Salo P, Ala-Mursula L, Rod NH, Tucker P, Pentti J, Kivimäki M et al. Work time control and sleep disturbances: prospective cohort study of Finnish public sector employees. Sleep (Basel) 2014 Jul;37(7):1217-25. https://doi. org/10.5665/sleep.3842.
12. Vahtera J, Laine S, Virtanen M, Oksanen T, Koskinen A, Pentti J et al. Employee control over working times and risk of cause-specific disability pension: the Finnish Public Sector Study. Occup Environ Med 2010 Jul;67(7):479-85. https://doi.org/10.1136/oem.2008.045096.
13. Takahashi M, Iwasaki K, Sasaki T, Kubo T, Mori I, Otsuka Y. Sleep, fatigue, recovery, and depression after change in work time control: a one-year follow-up study. J Occup Environ Med 2012 Sep;54(9):1078-85. https://doi. org/10.1097/JOM.0b013e31826230b7.
14. Takahashi M, Iwasaki K, Sasaki T, Kubo T, Mori I, Otsuka Y. Worktime control-dependent reductions in fatigue, sleep problems, and depression. Appl Ergon 2011 Jan;42(2):244-50. https://doi.org/10.1016/j.apergo.2010.06.006.
15. Nijp HH, Beckers DG, Kompier MA, van den Bossche SN, Geurts SA. Worktime control access, need and use in relation to work-home interference, fatigue, and job motivation. Scand J Work Environ Health 2015 Jul;41(4):347-55. https://doi.org/10.5271/sjweh.3504.
16. Meijman TF, Mulder G. Psychological aspects of workload. In: Drenth PJD, Thierry H, de Wolff C, editors. A Handbook of Work and Organizational Psychology: Volume 2: Work Psychology. 2nd ed. Hove (UK): Psychology Press; 1998. p. 5-33.
17. Greenhaus J, Beutell NJ. Sources of conflict between work and family roles. Acad Manage Rev 1985;10(1):76-88. https://doi.org/10.5465/amr.1985.4277352.
18. Hämmig O, Gutzwiller F, Bauer G. Work-life conflict and associations with work- and nonwork-related factors and with physical and mental health outcomes: a nationally representative cross-sectional study in Switzerland. BMC Public Health 2009 Nov;9:435. https:// doi.org/10.1186/1471-2458-9-435.
19. Fisher GG, Bulger CA, Smith CS. Beyond work and family: a measure of work/nonwork interference and enhancement. J Occup Health Psychol 2009 Oct;14(4):441-56. https://doi. org/10.1037/a0016737.
20. Frone MR, Russell M, Cooper ML. Prevalence of work-family conflict: are work and family boundaries asymmetrically permeable? J Organ Behav 1992;13:723-9. https://doi.org/10.1002/job.4030130708.
21. Greenhaus JH, Allen TD, Spector PE. Health consequences of work-family conflict: the dark side of the work-family interface. Empl Heal Coping Methodol. 2015;5:61-98. https://doi.org/10.1016/S1479-3555(05)05002-X.
22. Jansen NW, Kant I, Nijhuis FJ, Swaen GM, Kristensen TS, et al. Impact of worktime arrangements on work-home interference among Dutch employees. Scand J Work Environ Health 2004 Apr;30(2):139-48. https://doi. org/10.5271/sjweh.771.
23. Geurts SA, Beckers DG, Taris TW. Kompier M a J, Smulders PGW. Worktime demands and work-family interference: does worktime control buffer the adverse effects of high demands? J Bus Ethics 2009;84:229-41.

https://doi.org/10.1007/s10551-008-9699-y.
24. Hanson LL, Leineweber C, Chungkham HS, Westerlund H. Work-home interference and its prospective relation to major depression and treatment with antidepressants. Scand J Work Environ Health 2014 Jan;40(1):66-73. https://doi. org/10.5271/sjweh.3378.
25. Demerouti E, Geurts SA, Bakker AB, Euwema M. The impact of shiftwork on work-home conflict, job attitudes and health. Ergonomics 2004 Jul;47(9):987-1002. https:// doi.org/10.1080/00140130410001670408.
26. Hämmig O, Gutzwiller F, Bauer G. Work-life conflict and associations with work- and nonwork-related factors and with physical and mental health outcomes: a nationally representative cross-sectional study in Switzerland. BMC Public Health 2009 Nov;9(1):435. https://doi. org/10.1186/1471-2458-9-435.
27. de Lange AH, Taris TW, Kompier MA, Houtman IL, Bongers PM. Different mechanisms to explain the reversed effects of mental health on work characteristics. Scand J Work Environ Health 2005 Feb;31(1):3-14. https://doi. org/10.5271/sjweh.843.
28. Cole DA, Maxwell SE. Testing mediational models with longitudinal data: questions and tips in the use of structural equation modeling. J Abnorm Psychol 2003 Nov;112(4):558-77. https://doi.org/10.1037/0021-843X.112.4.558.
29. Yu K. Schedule Control, Work Interference With Family, and Emotional Exhaustion : A Reciprocal Moderated Mediation Model. J Pacific Rim Psychol. 2017; 11 e3:1-10. https://doi.org/10.1017/prp.2017.3.
30. Magnusson Hanson LL, Leineweber C, Persson V, Hyde M, Theorell T, Westerlund H. Cohort Profile: The Swedish Longitudinal Occupational Survey of Health (SLOSH). Int J Epidemiol 2018;47(3):691-92i. https://doi.org/10.1093/ ije/dyx260.
31. Magnusson Hanson LL, Westerlund H, Leineweber C, Rugulies R, Osika W, Theorell T et al. The Symptom Checklist-core depression (SCL-CD6) scale: psychometric properties of a brief six item scale for the assessment of depression. Scand J Public Health 2014 Feb;42(1):82-8. https://doi.org/10.1177/1403494813500591.
32. Muthén LK, Muthén BO. Mplus User's Guide. Seventh Ed. Los Angeles, CA: Muthén &Muthén; 2012.
33. Enders CK, Bandalos DL. The relative performance of full information maximum likelihood estimation for missing mata in structural equation models. Struct Equ Model A Multidiscip J. 2001;8(3):430-57.
34. Little TD. Mediation and Moderation. In: Longitudinal Structural Equation Modeling. New York/London: Guilford Press; 2013. p. 286-322.
35. Little TD. Specifying and interpreting a longitudinal panel model. In: Longitudinal structural equation modeling. New York/London: Guilford Press; 2013. p. 180-208.
36. Sterne JA, Davey Smith G. Sifting the evidence-what's wrong with significance tests? BMJ 2001 Jan;322(7280):226-31. https://doi.org/10.1136/bmj.322.7280.226.
37. Jöreskog KG, Sörbom D. LISREL 8: Structural Equation Modeling with the SIMPLIS command language. Chicago: Scientific Software International; 1993.
38. Little TD. Model Fit, Sample Size, and Power. In: Longitudinal Structural Equation Modeling. New York/London: Guilford Press; 2013. p. 106-36.
39. Bentler PM. Comparative fit indexes in structural models. Psychol Bull 1990 Mar;107(2):238-46. https://doi. org/10.1037/0033-2909.107.2.238.
40. Schwarz G. Estimating the dimension of a model. Ann Stat 1978;6(2):461-4. https://doi.org/10.1214/aos/n76344136.
41. Nakata A, Takahashi M, Irie M. Association of overtime work with cellular immune markers among healthy daytime white-collar employees. Scand J Work Environ Health 2012 Jan;38(1):56-64. https://doi.org/10.5271/sjweh.3183.
42. Nabe-Nielsen K, Garde AH, Diderichsen F. The effect of work-time influence on health and well-being: a quasiexperimental intervention study among eldercare workers. Int Arch Occup Environ Health 2011 Aug;84(6):683-95. https://doi.org/10.1007/s00420-011-0625-8.
43. Tucker P, Rutherford C. Moderators of the relationship between long work hours and health. J Occup Health

Psychol 2005 Oct;10(4):465-76. https://doi.org/10.1037/10768998.10.4.465.
44. Magnusson Hanson LL, Chungkham HS, Åkerstedt T, Westerlund H. The role of sleep disturbances in the longitudinal relationship between psychosocial working conditions, measured by work demands and support, and depression. Sleep (Basel) 2014 Dec;37(12):1977-85. https:// doi.org/10.5665/sleep.4254.
45. Galinsky E, Stein PJ. The impact of human resource policies on employees. J Fam Issues 1990; 11 (4):368-83. https://doi. org/10.1177/019251390011004002.
46. Kecklund G, Eriksen CA, Åkerstedt T. Police officers attitude to different shift systems: association with age, present shift schedule, health and sleep/wake complaints. Appl Ergon 2008 Sep;39(5):565-71. https://doi. org/10.1016/j.apergo.2008.01.002.
47. Ellen L. Hamaker. Why researchers should think "withinperson": A paradigmatic rationale. In: Mehl MR, Conner TS, editors. Handbook of Research Methods for Studying Daily Life. New York: The Guilford Press; 2012. p. 43-61.
48. Graham JW, Cumsille PE, Elek-Fisk E. Methods for Handling Missing Data. In: Handbook of Psychology. Hoboken, NJ, USA: John Wiley &Sons, Inc.; 2003. p. 87-114.
Received for publication: 18 March 2019

DETAILS

Subject:	Sleep; Work environment; Working hours; Interference; Work life balance; Estimates; Signs and symptoms; Mental disorders; Mediation; Mental health; Mental depression; Musculoskeletal diseases
Business indexing term:	Subject: Work environment Working hours Work life balance Mediation
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	469-479,469A
Publication year:	2020
Publication date:	2020
Section:	Original article
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X

Source type:	Scholarly Journal
Language of publication:	English
Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3887
ProQuest document ID:	2444685975
Document URL:	https://www.proquest.com/scholarly-journals/mediating-effect-work-life-interference-on/docview/2444685975/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2023-09-12
Database:	Public Health Database

Association of long working hours with accidents and suicide mortality in Korea

Lee, Hye-Eun, MD, PhD [1] ; Kim, Inah, MD, MPH, PhD [2] ; Kim, Hyoung-Ryoul, MD, PhD [3] ; Kawachi, Ichiro, MD, PhD [4] [1] Korea Institute of Labor Safety and Health, Seoul, Republic of Korea [2] Department of Occupational and Environmental Medicine, College of Medicine, Hanyang University, Seoul, Republic of Korea [3] Department of Occupational and Environmental Medicine, College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea [4] Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, Boston, MA, United States

ABSTRACT (ENGLISH)

Objectives The deleterious health effects of long working hours have been previously investigated, but there is a dearth of studies on mortality resulting from accidents or suicide. This prospective study aims to examine the association between working hours and external-cause mortality (accidents and suicide) in Korea, a country with some of the longest working hours in the world. Methods Employed workers (N=14 484) participating in the Korean National Health and Nutrition Examination Survey (KNHANES) were matched with the Korea National Statistical Office's death registry from 2007-2016 (person-years = 81 927.5 years, mean weighted follow-up duration = 5.7 years). Hazard ratios (HR) for accident (N=25) and suicide (N=27) mortality were estimated according to weekly working hours, with 35-44 hours per week as the reference. Results Individuals working 45-52 hours per week had higher risk of total external cause mortality compared to those working 35-44 hours per week [HR 2.79, 95% confidence interval (CI) 1.22-6.40], adjusting for sex, age, household income, education, occupation, and depressive symptoms. Among the external causes of death, suicide risk was higher (HR 3.89, 95% CI 1.06-14.29) for working 45-52 hours per week compared to working 35-44 hours per week. Working >52 hours per week also showed increased risk for suicide (HR 3.74, 95% CI 1.03-13.64). No statistically significant associations were found for accident mortality. Conclusions Long working hours are associated with higher suicide mortality rates in Korea.

FULL TEXT

Headnote

Key terms: accident; depression; injury; karoshi; KNHANES; Korea; Korean National Health and Nutrition Examination Survey; long working hour; mental health; occupational; overwork; suicide; work hour; working hour; working time; worktime

Lee H-E, Kim I, Kim H-R, Kawachi I. Association of long working hours with accidents and suicide mortality in Korea. Scand J Work Environ Health. 2020;46(5):480-487. doi:10.5271/sjweh.3890

Objectives The deleterious health effects of long working hours have been previously investigated, but there is a dearth of studies on mortality resulting from accidents or suicide. This prospective study aims to examine the association between working hours and external-cause mortality (accidents and suicide) in Korea, a country with some of the longest working hours in the world.

Methods Employed workers (N=14 484) participating in the Korean National Health and Nutrition Examination Survey (KNHANES) were matched with the Korea National Statistical Office's death registry from 2007-2016 (person-years = 81 927.5 years, mean weighted follow-up duration = 5.7 years). Hazard ratios (HR) for accident (N=25) and suicide (N=27) mortality were estimated according to weekly working hours, with 35-44 hours per week as the reference.

Results Individuals working 45-52 hours per week had higher risk of total external cause mortality compared to those working 35-44 hours per week [HR 2.79, 95% confidence interval (CI) 1.22-6.40], adjusting for sex, age, household income, education, occupation, and depressive symptoms. Among the external causes of death, suicide risk was higher (HR 3.89, 95% CI 1.06-14.29) for working 45-52 hours per week compared to working 35-44 hours per week. Working >52 hours per week also showed increased risk for suicide (HR 3.74, 95% CI 1.03-13.64). No statistically significant associations were found for accident mortality.

Conclusions Long working hours are associated with higher suicide mortality rates in Korea.

Key terms depression; injury; karoshi; KNHANES; Korean National Health and Nutrition Examination Survey; mental health; occupational; overwork; work hour; working time; work time.

Among the Organization for Economic Cooperation and Development (OECD) countries, Korea ranked as one of the top nations for longest working hours between 2008 and 2018 (1). In 2018, the annual working hour average in Korea was 1993 hours, while the working hour average for the OECD countries collectively was 1734 hours per year (1). Previous studies have established an association between long working hours and adverse outcomes, including coronary heart disease (2), stroke (3), mental health disorders (4, 5), reproductive health problems (6), and accidents (7). As working hours in East Asian countries (Japan, Korea, and Taiwan) are generally longer than those of western countries, deaths related to overwork (called karoshi), usually from cardiovascular disease, represent a growing social concern (8). Recently, suicide among overworked employees has drawn urgent attention in both Japan and Korea (9, 10). However, studies on working hours and suicide are limited to descriptive case series (11, 12), with one notable exception of a longitudinal study in the UK (13). Although the mechanism linking long working hours and suicide is not yet fully understood, a number of studies have examined the association between long working hours and depressive symptoms or suicide ideation (4, 5, 14). The deleterious impact of long working hours on mental health status is an obvious pathway connecting long working hours and suicide.

Besides cardiovascular disease and suicide, accidents are another potentially fatal outcome associated with long working hours. Fatigue and sleep loss potentially mediate the association between long working hours and accidents both in and out of the workplace (15, 16). However, the majority of studies on working hours and accidents have remained cross-sectional and/or used selfreported accidents as the outcome (7). On the contrary, a recent prospective study using national registers to assess accidents concluded there was no association between long working hours and accidents (17).

Thus, although some previous studies support an adverse impact of long working hours on suicide and accident mortality, this association is not well established by longitudinal data. In addition, to our knowledge, the association between long working hours and suicide or accident-related deaths has not been previously reported in the East Asian context.

Accordingly, the aim of this prospective study was to investigate the relationship between long working hours and accident mortality/suicide in a Korean working population based on nationwide longitudinal data.

Methods

Study population

Our data were derived from the Korean National Health and Nutrition Examination Survey (KNHANES) conducted by the Korea Centers for Disease Control and Prevention (KCDC) between 2007-2015. These data were then matched with death registry data compiled by the Korea National Statistical Office (KNSO) from 2007-2016. The survey used a multi-stage, clustersampling design based on the National Census Registry; hence, statistical analyses of this survey were based on sample weights assigned to sample participants. Among the 73 353 participants in KNHANES, 66 384 participants provided consent to link their data to the death registry. We restricted the subjects to employed workers by excluding the economically inactive population (37 702), employers and self-employed workers (8965), and unpaid family workers (2105). Employers and selfemployed workers were excluded due to their ability to control their working hours; despite their working hours being even longer than employed workers, they are not subject to working hour regulations (18). Additionally, we excluded the following individuals: those <18 years, individuals with <15 work hours per week or missing information on working hours, and covariates. After these exclusions, our analytic cohort comprised 14 484 men and women. The selection process of the study population is presented in figure 1.

Ascertainment of outcomes

The cohort dataset was matched with the death registry of the KNSO from 2007-2016 with the use of a unique identification number. As all deaths in Korea are reported to the KNSO by law, coverage of the death registry can be considered complete. Information on the specific cause of death according to the Korean Classification of Disease (KCD) and date of death was provided by KNSO. The KCD is compatible with the International Classification of Diseases-10 (ICD-10). Deaths from total external cause (V01-Y98), subsets including accidents (V01-V99; transport accidents, and W00-X59; other external causes of accidental injury), and intentional self-harm (X60X84) were used as our outcomes. During an average 5.2 person-years of follow-up, 56 participants died from total external causes. Among them, 25 individuals died from accidents (13 from transport accidents and 12 from the other accidents) and 27 died from suicide.

Assessment of working hours

Working hours were measured by responses to a question on the KNHANES asking: "How many hours do you usually work per week, including overtime?" Working hours were classified into four groups: (i) 15-34, (ii) 35-44, (iii) 45-52, and (iv) >52 hours per week. The top code of >52 hours per week was based on the maximum permitted working hours according to the Labor Standard Act in Korea (19). This Act has defined standard working hours as 40 hours per week, with extensions up to 52 hours per week permitted with the worker's consent. However, working on weekends was not subject to regulation until 2018, therefore enabling workers to work >52 hours per week legally if they worked on a Saturday or Sunday.

Covariates

Age, sex, household income, education, occupation, and depressive symptoms were included in our regression models as possible confounders. Socioeconomic status (SES), including occupation, is associated with both accident and suicide mortality (20). Depressive symptoms are a well-established risk factor for suicide and could be related to accidents as well (21). These covariates were collected during interviews in the KNHANES.

Monthly household income was equalized for household size (gross monthly household income divided by the square root of household size) and participants were divided into four groups according to quartile of standardized household income by survey year. Occupation was coded into nine categories according to the Korean Standard

Classification of Occupation (22), and we collapsed these into six groups (managers and professionals; office workers; service and sales workers; agricultural, forestry, and fishery workers; plant and machine operators and assemblers; and elementary occupations). The response to the question, "Have you experienced serious sadness or hopelessness that restricts your daily life continuously for >2 weeks in the last year?" was used to define depressive symptoms, with an affirmative response indicating a positive for depressive symptoms.

Statistical analysis

Cox proportional hazards models were developed to estimate hazard ratios (HR) with 95% confidence intervals (CI) for the association between working hours and deaths from accidents and suicide. In the Cox models, person-days were calculated from the initial date of participation in the KNHANES until either the date of death (including deaths from non-accidental causes) or 31 December 2016, whichever occurred first. The analytic model included age, sex, education, occupation, household income, and depressive symptoms as covariates. We applied the integrated survey weights, calculated by averaging weights over sampled years, because we used data from multiple waves of the survey. The sampling weights for each wave of the survey was calculated and provided by the KCDC to ensure the survey data could be inflated to the population level from which the sample was derived. More KNHANES sampling weight details can be found elsewhere (23).

Ethical approval

The Institutional Review Board of the Korea Center for Disease Control and Prevention reviewed and approved the pilot study of the KNHANES-linked cause of death data (IRB No. 2018-07-01-P-A).

Results

The distribution of working hours according to sample characteristics is presented in table 1. Of these participants, 35.6% worked 35-44 hours per week, 24.7% worked 45-52 hours per week, and 24.9% worked >52 hours per week. Working for >52 hours per week was prevalent among men (30.9%), those with middle lower household income (29.1%), those with middle school education (30.5%), and plant and machine operators and assemblers (39.1%).

The number of cases and participant mortality rates are shown in table 2. The accident mortality rates were 16.4, 21.1, 46.7, and 36.3 per 100 000 in the <35, 35-44, 45-52, and >52 hours/week groups, respectively. Suicide rates of 12.5, 12.0, 51.2, and 52.8 per 100 000 were observed in the <35, 35-44, 45-52, and >52 hours/week groups, respectively. The majority of deaths from accidents (24 cases) were among men; there was only one case among women. Suicide rates were also higher among men (46.8 per 100 000) than women (10.2 per 100 000).

Table 3 shows the results from the Cox regressions examining the association between working hours and mortality due to accidents and suicide. Proportional hazards assumptions were met. In the model adjusting for sex, age, household income, education, occupation, and depressive symptoms, participants working 45-52 hours/week showed elevated total external cause mortality risk (HR 2.79, 95% CI 1.22-6.40) compared to the reference group reporting 35-44 hours/ week. Men and women working >45 hours/week showed higher suicide mortality risk (45-52 hours: HR 3.89, 95% CI 1.06-14.29; >52 hours: HR 3.74, 95% CI 1.03-13.64) compared to the reference group. No statistically significant associations were found for accident mortality.

Discussion

Total external causes

We found that individuals working 45-52 hours per week have a higher statistically significant risk of external cause mortality compared to those working 35-44 hours per week. Those working >52 hours showed a higher HR, but the result was not statistically significant. The risk of total external cause mortality is mainly driven by the excess risk of suicide because suicide showed a significantly elevated HR in the groups working 45-52 and >52 hours. On the contrary, those working >52 hours showed a lower risk of accidents compared to the standard working hour group. This opposite direction of association between accidents and suicide among the >52 hours group suggests that mortality from these causes might have different pathways.

Accidents

In previous studies (24, 25), an adverse impact of long working hours among hospital workers (including young doctors) on traffic accidents have been reported [odds ratio 2.3 for extended shift (>24 hours), 95% CI 1.63.3]. In one case-crossover study (16), there was a strong trend in increased rate ratios (RR) for traffic accidents and shift duration (RR 0.92, 95% CI 0.52-1.62 for >8 hours/day, RR 4.00, 95% CI 0.45-35.8 for >12 hours/ day). For work-related accidents, several studies have also revealed the association between long working hours and increased self-reported or objectively confirmed work-related injury (26, 27). One case-crossover study showed that the risk for work-related injury in workers who worked >64 hours per week was 1.88 times greater than among those who worked <40 hours (28). A probable explanation for the association between long working hours and accidents is fatigue due to lack of sleep (24, 29).

The current study's results were not consistent with these previous findings. The HR for accident mortality was lower among the >52 working hour group than the standard working hour group, although the differences were not statistically significant, and the CI was wide. A number of reasons could underlie this discrepancy. First, in the current study, there was a wide time gap between the assessment of working hours and accidents, while previous works measured working hours at the time of accidents (16, 24, 25). Sleep loss and fatigue can be more related to long working hours immediately preceding the accidents. Second, the outcome of the current study was mortality from accidents, while most previous studies used experiences of accidents as an outcome. As we used an extreme end of an accident outcome, the results could not be compared directly. In fact, a previous study using a similar design to ours (census-based longitudinal study in UK) found lower or similar risk of all accidental mortality for men working >55 compared to 35-40 hours/week among professional/ managers, self-employed, and routine occupations (13).

Suicide

Although extensive research has been conducted on the association between long working hours and mental health (including depressive symptoms and suicidal ideation), very few studies have focused specifically on completed suicide. In Korea and Japan, where overwork-related suicide is a growing social concern, descriptive characteristics of suicide cases (compensated as work-related mortality) have been reported (11, 12). The daily working hours of 22 work-related suicide cases in Japan ranged from 10-16 hours (11). In a Korean report, "chronic long working hours" was the second most prevalent reason, following "acute stressful events", for approved cases of compensable work-related mental disease, which included suicide (12). One UK-based longitudinal study examining the association of long working hours and completed suicide showed a 1.23-1.24 times higher risk in the >55 hour/ week group compared to the 35-40 hour/week group among professionals/managers, but the results were not statistically significant (13).

Elevated risk of suicide might be due to the wellestablished association between long working hours and poor mental health (4, 5). However, suicide rate was not associated with depressive symptoms at our data baseline. This could be caused by the time gap between the survey and the events of suicide or depression. Indeed, a longitudinal study in the UK, which reported no depressive symptoms at baseline, showed a higher risk of incident depression among participants with long working hours after a 5-year follow-up (4).

A second explanation for the association between long working hours and suicide could be the deleterious effects these long hours have on relationships with family and friends. Social isolation and family conflict are widely reported risk factors for suicide (30), and long working hours have been shown to increase work-life conflict (31, 32). According to a 2018 psychological autopsy report of the Korean Psychological Autopsy Center, among the 103 suicide cases, occupational stress was second only to mental health issues as a primary stressor. (33). In this report, qualitative analysis of 52 employed workers' pathways to suicide revealed that their main occupational stressors included change of work, work demands, and relationships in the workplace (33). Long working hours are closely related to work demands, and work demands could affect the relationships between supervisors and coworkers.

Low SES is also a risk factor for suicide. A previous study in Korea revealed that suicide risk is 2.28 times higher in Medicaid recipients than in 10th-decile highest income individuals (34). In the current study, the lowest household income group showed markedly higher suicide rates (97.9 per 100 000) than other groups (20.1-37.7 per 100 000). Since working hours could be confounded by SES, we built analytic models adjusting for SES. However, we found that the association persisted in the adjusted model, suggesting that long working hours are associated with suicide risk, regardless of SES.

Strengths and limitations

The present study has significant strengths and limitations. Its strengths are that the subjects were drawn from a nationwide sample rather than from selected subgroups. Additionally, cause of death was determined from validated records. To our knowledge, this is the first study investigating the impact of long working hours on accident and suicide mortality in Korea.

Limitations of our study must be mentioned as well: the number of cases was relatively small; therefore, the CI for HR remained wide. Especially for women, accident mortality was extremely rare. Due to the small number of the cases, caution is warranted in generalizing the results of the current study. Working hours were measured based on self-report and collected only once at baseline. Since working hours are time-dependent variables, we cannot rule out the misclassification of exposure during follow-up. This possibility of nondifferential working hour misclassification could have biased the results toward the null. Other time-varying covariates such as depressive symptoms could have changed during follow-up as well. However, due to the scarcity of repeated assessments of mental health status, we were unable to conduct a mediation analysis (ie, to check whether changes in depressive symptoms mediated the association between long working hours and suicide). Nevertheless, the lack of mediation analysis does not affect overall risk estimates of working hours for outcomes. Further analysis of working hour effects on mortality from accidents and suicide with a sufficient number of cases with longer follow-up periods, larger cohorts, and additional measures of working hours and covariates may follow in the future.

Concluding remarks

In conclusion, our study shows that workers who work long hours (>44 hours per week) have a higher risk of suicide in Korea.

Acknowledgments

This work was supported by the pilot study of the KNHANES-linked cause of death data, conducted by the KCDC, and the research fund of Hanyang University (HY-2015).

Conflicts of interest

The authors declare no conflicts of interest.

Sidebar

This is the first study to investigate working hours and accidents and suicide mortality in Korea. The results highlight the significant association between long working hours and suicide risk, even among workers working 45-52 working hours/week, which is within the limit of the working hour by legislation.

Affiliation: Department of Occupational and Environmental Medicine, College of Medicine, Hanyang University, 222 Wangsimni-ro, Seongdong-gu, Seoul 04763, Korea. inahkim@hanyang.ac.kr

Refers to the following texts of the Journal: 2005;31(5):329-335 2011;37(3):173-185 2013;39(5):515-520 2017;43(6):578-586

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32096547

Correspondence to: Inah Kim, Department of Occupational and Environmental Medicine, College of Medicine, Hanyang University, 222 Wangsimni-ro, Seongdong-gu, Seoul 04763, Korea. [E-mail: inahkim@hanyang.ac.kr]

References

References

1. OECD. Average annual hours actually worked. 2014; Available from: https://www.oecd-ilibrary.org/content/data/data00303-en.

2. Virtanen M, Heikkila K, Jokela M, Ferrie JE, Batty GD, Vahtera J, et al. Long working hours and coronary heart disease: a systematic review and meta-analysis. Am J Epidemiol. 2012 Oct 1;176(7):586-96. https://doi.org/10.1093/aje/kws139.

3. Kivimaki M, Jokela M, Nyberg ST, Singh-Manoux A, Fransson EI, Alfredsson L, et al. Long working hours and risk of coronary heart disease and stroke: a systematic review and meta-analysis of published and unpublished data for

603,838 individuals. Lancet. 2015 Oct 31;386(10005):1739-46. https:// doi.org/10.1016/S0140-6736(15)60295-1.
4. Virtanen M, Ferrie JE, Singh-Manoux A, Shipley MJ, Stansfeld SA, Marmot MG, et al. Long working hours and symptoms of anxiety and depression: a 5-year follow-up of the Whitehall II study. Psychol Med. 2011 Dec;41(12):2485-94. https://doi.org/10.1017/S0033291711000171.
5. Kim I, Kim H, Lim S, Lee M, Bahk J, June KJ, et al. Working hours and depressive symptomatology among full-time employees: Results from the fourth Korean National Health and Nutrition Examination Survey (2007-2009). Scand J Work Env Health. 2013 Sep 1;39(5):515-20. https://doi. org/10.5271/sjweh.3356.
6. Takeuchi M, Rahman M, Ishiguro A, Nomura K. Long working hours and pregnancy complications: women physicians survey in Japan. BMC Pregnancy Childbirth. 2014;14:245. https:// doi.org/10.1186/1471-2393-14-245.
7. Wagstaff AS, Sigstad Lie JA. Shift and night work and long working hours-a systematic review of safety implications. Scand J Work Env Health. 2011 May;37(3):173-85. https:// doi.org/10.5271/sjweh.3146.
8. Cheng Y, Park J, Kim Y, Kawakami N. The recognition of occupational diseases attributed to heavy workloads: experiences in Japan, Korea, and Taiwan. Int Arch Occup Environ Health. 2012 Oct;85(7):791-9. https://doi. org/10.1007/s00420-011-0722-8.
9. Takahashi M. Sociomedical problems of overwork-related deaths and disorders in Japan. J Occup Health. 2019 Jul;61(4):269-77. https://doi.org/10.1002/1348-9585.12016.
10. Kim I, Koo MJ, Lee H-E, Won YL, Song J. Overwork-related disorders and recent improvement of national policy in South Korea. J Occup Health. 2019 Jul;61(4):288-96. https://doi. org/10.1002/1348-9585.12060.
11. Amagasa T, Nakayama T, Takahashi Y. Karojisatsu in Japan: characteristics of 22 cases of work-related suicide. J Occup Health. 2005 Mar;47(2):157-64. https://doi.org/10.1539/ joh.47.157.
12. Lee J, Kim I, Roh S. Descriptive study of claims for occupational mental disorders or suicide. Ann Occup Environ Med. 2016;28:61. https://doi.org/10.1186/s40557-016-01477.
13. O'Reilly D, Rosato M. Worked to death? A census-based longitudinal study of the relationship between the numbers of hours spent working and mortality risk. Int J Epidemiol. 2013 Dec;42(6):1820-30. https://doi.org/10.1093/ije/dyt211.
14. Yoon JH, Jung PK, Roh J, Seok H, Won JU. Relationship between Long Working Hours and Suicidal Thoughts: Nationwide Data from the 4th and 5th Korean National Health and Nutrition Examination Survey. PLoS One. 2015;10(6):e0129142. https://doi.org/10.1371/journal. pone.0129142.
15. Dembe AE, Erickson JB, Delbos RG, Banks SM. The impact of overtime and long work hours on occupational injuries and illnesses: new evidence from the United States. Occup Env Med. 2005 Sep;62(9):588-97. https://doi.org/10.1136/ oem.2004.016667.
16. Valent F, Di Bartolomeo S, Marchetti R, Sbrojavacca R, Barbone F. A case-crossover study of sleep and work hours and the risk of road traffic accidents. Sleep. 2010 Mar;33(3):34954. https://doi.org/10.1093/sleep/33.3349.
17. Larsen AD, Hannerz H, MÃjler SV, Dyreborg J, Bonde JP, Hansen J, et al. Night work, long work weeks, and risk of accidental injuries. A register-based study. Scand J Work Environ Health. 2017 Nov 1;43(6):578-86. https://doi. org/10.5271/sjweh.3668.
18. Park J, Kwon OJ, Kim Y. Long Working Hours in Korea. Ind Health. 2012;50(5):458-62. https://doi.org/10.2486/ indhealth.MS1353.
19. Labor Standard Act [Internet]. Sect. 50 Feb 1, 2012. Available from: https://elaw.klri.re.kr/eng_service/lawView. do?hseq=25437&lang=ENG.
20. Lee H-E, Kim H-R, Chung YK, Kang S-K, Kim E-A. Mortality rates by occupation in Korea: a nationwide, 13-year follow-up study. Occup Environ Med. 2016;73(5):329-35. https://doi. org/10.1136/oemed-2015-103192.
21. Laursen TM, Musliner KL, Benros ME, Vestergaard M, MunkOlsen T. Mortality and life expectancy in persons with severe unipolar depression. J Affect Disord. 2016 Mar 15;193:203-7. https://doi.org/10.1016/jjad.2015.12.067.
22. Statistics Korea. The Korean Standard Classification of Occupations [Internet]. Korean Standard Statistical Classification. [cited 2020 Jan 3]. Available from: http://kssc. kostat.go.kr/ksscNew_web/ekssc/main/main.do#.
23. Kim Y. The Korea National Health and Nutrition Examination Survey (KNHANES): current status and challenges.

Epidemiol Health. 2014 Apr 30;36:e2014002. https://doi.org/10.4178/ epih/e2014002.
24. Barger LK, Cade BE, Ayas NT, Cronin JW, Rosner B, Speizer FE, et al. Extended work shifts and the risk of motor vehicle crashes among interns. N Engl J Med. 2005 Jan 13;352(2):125-34. https://doi.org/10.1056/NEJMoa041401.
25. Kirkcaldy B, Trimpop R, Cooper C. Working hours, job stress, work satisfaction, and accident rates among medical practitioners and allied personnel. Int J Stress Manag. 1997 Apr 1;4(2):79-87.
26. Dong X. Long workhours, work scheduling and work-related injuries among construction workers in the United States. Scand J Work Env Health. 2005 Oct;31(5):329-35. https://doi. org/10.5271/sjweh.915.
27. Lombardi DA, Folkard S, Willetts JL, Smith GS. Daily sleep, weekly working hours, and risk of work-related injury: US National Health Interview Survey (2004-2008). Chronobiol Int. 2010 Jul;27(5):1013-30. https://doi.org/10.3109/074205 28.2010.489466.
28. Vegso S, Cantley L, Slade M, Taiwo O, Sircar K, Rabinowitz P, et al. Extended work hours and risk of acute occupational injury: A case-crossover study of workers in manufacturing. Am J Ind Med. 2007 Aug;50(8):597-603. https://doi. org/10.1002/ajim.20486.
29. Folkard S, Lombardi DA. Modeling the impact of the components of long work hours on injuries and "accidents." Am J Ind Med. 2006 Nov;49(11):953-63. https://doi. org/10.1002/ajim.20307.
30. Van Orden KA, Witte TK, Cukrowicz KC, Braithwaite SR, Selby EA, Joiner TE. The interpersonal theory of suicide. Psychol Rev. 2010 Apr;117(2):575-600. https://doi. org/10.1037/a0018697.
31. Eurofound. Sixth European Working Conditions Survey - Overview report (2017 update) [Internet]. Publications Office of the European Union; 2017. Available from: https://www. eurofound.europa.eu/sites/default/files/ef_publication/ field_ef_document/ef1634en.pdf.
32. Karhula K, Puttonen S, Ropponen A, Koskinen A, Ojajärvi A, Kivimäki M, et al. Objective working hour characteristics and work-life conflict among hospital employees in the Finnish public sector study. Chronobiol Int. 2017;34(7):876-85. https://doi.org/10.1080/07420528.2017.1329206.
33. Korean psychological autopsy center. 2018 psychological autopsy report [Internet]. Seoul; 2019 Aug [cited 2019 Oct 23]. Available from: http://www.psyauto.or.kr/sub/notice_view. asp?mode=&page=1&direction=&idx=&bbsid=biNotice& editIdx=730&SearchKey=ALL&SearchStr=.
34. Lee S-U, Oh I-H, Jeon HJ, Roh S. Suicide rates across income levels: Retrospective cohort data on 1 million participants collected between 2003 and 2013 in South Korea. J Epidemiol. 2017 Jun;27(6):258-64. https://doi.org/10.1016/j. je.2016.06.008.
Received for publication: 30 October 2019

DETAILS

Subject:	Population; Mental health; Accidents; Mortality; Mental disorders; Nutrition; Statistical analysis; Risk; Workers; Fatalities; Working hours; Suicide; Confidence intervals; Cardiovascular disease; Occupational safety; Family income; Households
Business indexing term:	Subject: Workers Working hours
Location:	Japan
Company / organization:	Name: Organization for Economic Cooperation &Development; NAICS: 928120
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46

Issue:	5
Pages:	480-487,480A
Publication year:	2020
Publication date:	2020
Section:	Original article
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal
Language of publication:	English
Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3890
ProQuest document ID:	2444685932
Document URL:	https://www.proquest.com/scholarly-journals/association-long-working-hours-with-accidents/docview/2444685932/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2020-09-22
Database:	Public Health Database

Document 11 of 11

Psychosocial work exposures of the job strain model and cardiovascular mortality in France: results from the STRESSJEM prospective study

Niedhammer, Isabelle, PhD; Milner, Allison, PhD; Geoffroy-Perez, Béatrice, MD; Coutrot, Thomas, PhD; Lamontagne ScD, Anthony D; Chastang, Jean-François, PhD

ABSTRACT (ENGLISH)

Objectives The study aims to explore the prospective associations of the psychosocial work exposures of the job strain model with cardiovascular mortality, including mortality for ischemic heart diseases (IHD) and stroke, using various time-varying exposure measures in the French working population of employees. Methods The study was based on a cohort of 798 547 men and 697 785 women for which job history data from 1976 to 2002 were linked to mortality data and causes of death from the national death registry. Psychosocial work exposures from the validated job strain model questionnaire were assessed using a job-exposure matrix (JEM). Three time-varying measures of exposure were studied: current, cumulative, and recency-weighted cumulative exposure. Cox proportional hazards models were used to examine the associations between psychosocial work exposures and cardiovascular mortality. Results Within the 1976-2002 period, there were 19 264 cardiovascular deaths among men and 6181 among women. Low decision latitude, low social support, job strain, iso-strain, passive job, and high strain were associated with cardiovascular mortality. Most of these associations were also observed for IHD and stroke mortality. The comparison between the different exposure measures suggested that current exposure may be more important than cumulative (or past) exposure. The population fractions of cardiovascular mortality attributable to job strain were 5.64% for men and 6.44% for women. Conclusions Psychosocial work exposures of the job strain model may play a role in cardiovascular mortality. The estimated burden of cardiovascular mortality associated with these exposures underlines the need for preventive policies oriented toward the psychosocial work environment.

FULL TEXT

Headnote

Objectives The study aims to explore the prospective associations of the psychosocial work exposures of the job strain model with cardiovascular mortality, including mortality for ischemic heart diseases (IHD) and stroke, using various time-varying exposure measures in the French working population of employees.

Methods The study was based on a cohort of 798 547 men and 697 785 women for which job history data from 1976 to 2002 were linked to mortality data and causes of death from the national death registry. Psychosocial work exposures from the validated job strain model questionnaire were assessed using a job-exposure matrix (JEM). Three time-varying measures of exposure were studied: current, cumulative, and recency-weighted cumulative exposure. Cox proportional hazards models were used to examine the associations between psychosocial work exposures and cardiovascular mortality.

Results Within the 1976-2002 period, there were 19 264 cardiovascular deaths among men and 6181 among women. Low decision latitude, low social support, job strain, iso-strain, passive job, and high strain were associated with cardiovascular mortality. Most of these associations were also observed for IHD and stroke mortality. The comparison between the different exposure measures suggested that current exposure may be more important than cumulative (or past) exposure. The population fractions of cardiovascular mortality attributable to job strain were 5.64% for men and 6.44% for women.

Conclusions Psychosocial work exposures of the job strain model may play a role in cardiovascular mortality. The estimated burden of cardiovascular mortality associated with these exposures underlines the need for preventive policies oriented toward the psychosocial work environment.

Key terms cardiovascular disease; cumulative exposure; job-exposure matrix; JEM; ischemic heart disease; job stress; stroke.

Psychosocial work factors have been at the heart of occupational health issues in working populations of developed countries for the last decades. They have been found to influence cardiovascular diseases (1-4). While the literature has been extensive on cardiovascular morbidity, there is a lack of research on the associations between psychosocial work factors and cardiovascular mortality.

Among the existing theoretical models, the job strain model has been widely used for the measurement of psychosocial work factors through Karasek's Job Content Questionnaire (JCQ) (5). This model relies on three factors: psychological demands (intense and complex workload), decision latitude (skill discretion and decision authority), and social support from supervisor and colleagues. These three factors may have adverse effects on health outcomes, and the model also assumes that the combination of these factors may be still more detrimental for health, in particular job strain (combi- nation of high demands and low latitude) and isostrain (combination of job strain and low support).

A recent literature review summarized the results on the associations between psychosocial work factors and coronary heart disease mortality (6). Twelve studies explored the associations between the job strain model factors and cardiovascular mortality (7-18), three of them were based on the same study sample and hence can be considered as one study only (8, 14, 15). Among eight studies exploring decision latitude, five highlighted the influence of low decision latitude (7, 12, 14, 16, 17). Only one study (13) of five found psychological demands as a risk factor. Three studies explored job strain, and two (13, 14) found that job strain was associated with cardiovascular mortality. Of two studies, only one (11) found an association between iso-strain and cardiovascular mortality. Finally, two studies observed unexpected protective effects of high demands (16), low decision authority and low support from coworkers on cardiovascular mortality (9). These mixed findings make it difficult to draw conclusions about the associations between job strain model factors and cardiovascular mortality.

Three of the previous studies used a job-exposure matrix (JEM) for exposure assessment (7, 12, 17). Such a JEM has at least two major advantages in the topic of psychosocial work exposures: it provides exposure assessment in datasets that do not include any measure of exposure (except job title), and it reduces the bias related to self-reported measures, ie, reporting bias. The use of JEM is not a novel approach in the literature. Indeed, the pioneer studies by Alfredsson et al (19) and Johnson et al (20) already constructed and studied JEM for the job strain model exposures in the 1980-1990s. However, the study of cumulative exposure in association with cardiovascular mortality has been very rare for the studies using JEM and in the literature in general.

Consequently, there is a lack of prospective studies based on large national representative samples of the working population of men and women followed up over a long period of time, exposures from the validated JCQ and derived from JEM, and repeated measures of exposure over time. The present study thus attempted to overcome shortcomings of previous studies by using a very large nationally representative sample of the working population of men and women, evaluating exposures via the validated JCQ and a JEM over a long period of time, constructing various time-varying measures of exposures, and conducting long-term follow-up for exposure and outcome.

The objective of the study was to explore the prospective associations between the psychosocial work exposures of the job strain model and cardiovascular mortality. An additional objective was to examine various time-varying measures of exposure as well as subtypes of cardiovascular mortality.

Methods

The protocol of the STRESSJEM study has been presented in a previous publication (21).

Study sample

Briefly, the study was based on a nationally representative prospective cohort of the working population of employees that combined the national SUMER survey data from DARES (French Ministry of Labor) and the COSMOP program data from Santé publique France. For the studied sample of 1 511 456 individuals, two sources of data were linked: job history from 1976-2002 (INSEE DADS panel data, a random population-based sample, 1724th, of the French national working population of employees, excluding self-employed workers, agricultural workers/employees, employees of some public sectors and of household activities and extraterritorial organizations) and mortality data and causes of death coded according to the 10th, 9th, or 8th revisions of the ICD over the period

1976-2005 (French national death registry, INSERM-CépiDc, which is the official statistical organization in charge of mortality and causes of death in France). Data included information about job history over the period 1976-2002, in particular for all jobs held: dates of start and end of job, occupation, economic activity of the company, and company size. Exposure estimates for the validated job strain model questionnaire (JCQ) (22, 23) were assessed through a JEM that was constructed and validated for men and women separately using the national SUMER data. The JEM construction was based on a segmentation method with cross-validation that used the three job title variables: occupation, economic activity of the company, and company size. Validity was assessed in two main steps: comparison between self-reported individual versus JEM exposures, and study of the predictive validity of JEM exposures in association with self-reported health. The JEM estimates were then imputed using the same three job title variables for each job held during 1976-2002 in the COSMOP dataset. One of our previous publications described the construction and study of validity of this JEM (24).

Outcomes

Three outcomes were studied, namely, deaths for all cardiovascular diseases (I00-I99 in ICD-10 or the corresponding ICD-9 and ICD-8 codes); deaths for ischemic heart diseases (IHD) (I20-I25 or the corresponding ICD-9 and ICD-8 codes); and deaths for stroke (I60, I61, I63, I64 in ICD-10 or the corresponding ICD-9 and ICD-8 codes).

Measures of exposure

We constructed three time-varying exposure measures, using all jobs held during 1976-2002: (i) current exposure at time i; (ii) cumulative exposure until time i, calculated from an average measure at time i using the estimates of exposure and the time spent in all jobs up to and including time i; and (iii) recency-weighted cumulative exposure at time i, calculated from both past and current exposures and the time elapsed since exposure with higher weights assigned to more recent exposures (25). Psychosocial work exposure effects were assumed to persist for <5 years after the end of exposure (26) and decrease linearly over a 5-year period to be null after 5 years. We chose this 5-year period in accordance with the study by Amick et al (26) and also because this was a balanced choice between current exposure at time i only and cumulative exposure that assessed exposure over the whole 26-year follow-up period.

The current exposure measures were: (i) the binary exposures for psychological demands, decision latitude, and social support, derived from the JEM scores dichotomized at the median of the distribution for the first job among the total sample of men and women; (ii) the binary variables for job strain (combination of high demands and low latitude) and isostrain (combination of job strain and low support), constructed from the binary variables of demands, latitude, and support (11, 13); (iii) the 4-category variable related to the four quadrants by Karasek (13), from the combination of demands and latitude, that were: high strain (ie, high demands and low latitude), low strain (ie, low demands and high latitude), passive job (ie, low demands and low latitude), and active job (ie, high demands and high latitude). Two alternative reference groups were used: active job and low strain.

The two cumulative exposure measures (cumulative and recency-weighted cumulative exposure) were based on time-weighted scores for demands, latitude, and support as previously defined and dichotomized. Job strain, isostrain, and the 4-quadrant variable were constructed as mentioned earlier.

Statistical methods

The hazard ratio (HR) of cardiovascular mortality was estimated according to the studied exposures using Cox proportional hazards models. The proportional hazard assumption checked by graphical analysis was not violated (supplementary material, www.sjweh.fi/ show_abstract.php?abstract_id=3902, figures S1-3). It can be noticed that the survival curves at older ages, especially for women, were based on a very low number of persons and had large confidence intervals (CI). The studied exposures were time-dependent variables. Age was used as the time scale. Calendar time and four occupational variables related to biomechanical, physical, chemical, and biological exposures were included as adjustment variables. These four occupational variables were assessed by occupational physicians in the SUMER survey (27) and imputed through a JEM whose construction followed the same methodology as the JEM for the job strain model factors (24). They were used as a marker of social position as they displayed strong social gradients (28). We used a model with delayed entry. Individuals entered the cohort on the 1 January 1976 if they already had a job or when they started a first job within the 1976-2002 period.

As we had full information about exposures over the study period, both mortality during time intervals with job and mortality during the total follow-up (ie, including mortality after the end of last job/exposure) were studied.

For the three exposure measures, we used cardiovascular mortality until the end of last job to study mortality during time intervals with job (called "on-thejob" cardiovascular mortality); thus in this analysis, the follow-up ended at the time of death or at the end date of the last job within the 1976-2002 period, or at the end of follow-up (31 December 2002) if still working at this time, whichever came first.

For the two cumulative exposure measures, as delayed effects may be expected, a second analysis was performed in which the follow-up ended at the time of death or 31 December 2002, whichever came first. Compared to on-the-job cardiovascular mortality, the study of cardiovascular mortality until 2002 included cardiovascular deaths that occurred after the end of last job.

Death for causes other than cardiovascular diseases were censored at that time.

Three types of models were performed which included: (i) each exposure separately, (ii) demands, latitude and support simultaneously, (iii) job strain, isostrain, and the 4-quadrant variable separately. In the model that included demands, latitude, and support together, we tested the interaction term between high demands and low latitude, following Karasek's hypothesis.

Akaike's Information Criterion (AIC) was used to compare the three models with the three exposure measures - current, cumulative, and recency-weighted cumulative exposure - in association with on-the-job cardiovascular mortality to identify the highest relative quality model.

Finally, the population fractions of cardiovascular mortality attributable (PAF) to job strain and isostrain were calculated for France, with Pe being the exposure prevalence, and HR, the hazard ratio for mortality associated with exposure:

$$PAF = Pe(HR-1)/[1+Pe(HR-1)]$$

Pe was assessed by the weighted prevalence of job strain (19.94% among men, 28.70% among women) and iso-strain (12.72% among men, 17.44% among women) using the SUMER survey data. HR was provided by the present study. We used simulation-modelling techniques for the CI of PAF (29).

As interaction terms were found to be statistically significant between psychosocial work exposures and gender in the total sample, all analyses were performed for men and women separately and using SAS (SAS Institute, Cary, NC, USA) and R statistical software. Gender-related interactions were presented.

Sensitivity analyses

To check the robustness of the results, we performed the sensitivity analyses: (i) using scores for the measure of exposure instead of binary variables; (ii) adjusting for occupation in addition to the occupational exposures of biomechanical, physical, chemical and biological nature; (iii) imputing the lowest level of exposure instead of the highest level of exposure in case of multiple job-holder (only 3% of the sample had more than one job at the same time); (iv) studying mortality until 2005 instead of 2002; (v) excluding the first years of follow-up (1976-1978).

Results

Description of the study sample

The studied sample included 1 496 332 individuals, 798 547 men and 697 785 women (1%, N=15 214, had missing values for job history). Men and women had a mean age of 28 and 27 years, respectively, at entrance in the cohort. There were 13.6 million and 11.6 million person-years in the follow-up period for men and women, respectively. The mean duration of follow-up was 17 years for both genders (21). Over 1976-2002, there were 19 264 cardiovascular deaths (14.19 cases per 10 000 person-years) among men and 6181 (5.35 per 10 000 person-years) among women, including 2988 cardiovascular deaths (2.89 per 10 000 person-years) among men and 474 (0.56 per 10 000 person-years) among women during time intervals with a job (on-the-job cardiovascular mortality). All details about person-years, number of cases, and cases per 10 000 person-years for cardiovascular, IHD and stroke mortality are provided in supplementary table S1. The description of exposures was presented in our study protocol (21). Women had a higher prevalence of exposure to high psychological demands, low decision latitude, low social support, job strain, and iso-strain than men. Low correlations for demands were found with latitude (0.28) and support (-0.18) and a moderate correlation was observed between latitude and support (0.57).

Associations between psychosocial work factors and cardiovascular mortality

The results are presented in table 1 for current exposure, table 2 for cumulative exposure, and table 3 for recency-weighted cumulative exposure. The results of these tables were very close and can be summarized as follows. Low latitude and low support were found to be risk factors in almost all models. High demands displayed either no effects or protective effects on cardiovascular mortality. Job strain, iso-strain, passive job, and high strain were found to be risk factors in almost all models. No interaction term between high demands and low latitude was found to be statistically significant in the models exploring current exposure to demands, latitude, and support together. Information about personyears, number of cases, and cases per 10 000 personyears for cardiovascular mortality according to the studied exposures of job strain, isostrain, quadrants is provided in supplementary tables S2-5.

Gender-related interactions

Statistically significant gender-related interactions were observed suggesting differences in exposure-outcome associations by gender. The effect of low support on on-the-job cardiovascular mortality was stronger among women than among men. The association of job strain and iso-strain with cardiovascular mortality until 2002 was stronger among men than women.

Study of subtypes of cardiovascular mortality

Information about person-years, number of cases, and cases per 10 000 person-years for IHD and stroke tality to the studied exposures of job strain, strain, isostrain, quadrants is provided in supplementary tables S2-5. Most of the associations observed in tables 1-3 for cardiovascular mortality remained statistically significant for IHD and stroke mortality among men whereas only a smaller number of the associations remained significant among women, probably due to a low number of cases (supplementary tables S6-11).

Comparison between models

Tables 2-3 showed that, although the statistical power was higher, the study of cardiovascular mortality after the end of the last job (until 31/12/2002) reduced the effect size of most HR (dilution of the effects over time) compared to the study of on-the-job cardiovascular mortality (during time intervals with job). AIC calculation showed that the model with the lowest value of AIC (ie, the highest relative quality) was the model with current exposure. However, although the AIC value was lower for current exposure, it was not significantly different from the AIC values from the two models with cumulative exposure or recency-weighted cumulative exposure.

Sensitivity analyses

The sensitivity analyses found similar results compared to tables 1-3, except including occupation as additional adjustment variable, which reduced the statistical significance of the associations (supplementary tables S12-14).

Population attributable fractions

Following the AIC results, the population fractions of cardiovascular mortality attributable to current exposure to job strain and iso-strain were calculated. For job strain, the PAF were 5.64% (95% CI 3.09-8.42) among men and 6.44% (95% CI 0-14.3) among women. For iso-strain, the PAF were 3.20% (95% CI 1.38-5.09) among men and 4.02% (95% CI 0-9.20) among women.

Discussion

Summary of the results

Low decision latitude, low social support, job strain, iso-strain, passive job, and high strain were found to be associated with cardiovascular mortality for both genders. These results were observed for current, cumulative and recency-weighted cumulative exposure. Similar results were found for IHD and stroke mortality, especially among men. The population fractions of cardiovascular mortality attributable to current exposure to job strain were 5.64% and 6.44% for men and women respectively.

Comparison with the literature

Our study underlined the effect of low decision latitude on cardiovascular mortality. This result is in agreement with the findings of five studies among men mainly (7, 12, 14, 16, 17) and with a recent review and meta-analysis (6), whose results showed that the only statistically significant psychosocial work factor was low latitude in association with coronary heart disease mortality. In our study, high psychological demands were found to be a non-significant or protective factor. These findings echo some rare previous studies that reported no effect (7, 12, 14) or a protective effect of high demands (16). Only one study reported an association between high demands and cardiovascular mortality (13). Low social support was found to be a risk factor in our study. Three previous studies either did not find any effect of low support (12, 16) or found a protective effect of low support from coworkers (9). Our study is thus the first one to show low social support as a risk factor for cardiovascular mortality.

Job strain was associated with cardiovascular mortality in our study, confirming two previous studies (13, 14). We found iso-strain to be a risk factor, which is in agreement with Johnson et al's study (11) but not that of Padyab et al (16) who reported no effect of isostrain. Regarding the study of the four quadrants, our study showed that passive job and high strain were both associated with cardiovascular mortality. Two previous studies explored the four quadrants but did not find any associations (16, 18). Our study is thus the first one to report an association between passive job and cardiovascular mortality.

In addition, our HR estimates were very close in effect size to previous estimates published in the literature for both cardiovascular mortality and morbidity. Indeed, the recent review and meta-analysis on job strain in association with cardiovascular mortality (6) provided a summary HR of 1.3 (95% CI 0.9-1.9), though nonsignificant, and the last update of a meta-analysis (30) on the association between job strain and cardiovascular morbidity reported a summary relative risk of 1.33 (95% CI 1.19-1.49).

All previous studies explored a measure of exposure at baseline in association with cardiovascular mortality, except Johnson et al's (12), which used a JEM and explored a cumulative exposure based on job history but limited to the past five occupations held by the subjects for >2 years, meaning a potential incomplete job history. In addition, Johnson et al's study examined the association between retrospective cumulative exposure (before 1977) and mortality over the period 1977-1990, leading to an absence of information about exposure during follow-up. The results of this study underlined the effects of low work control on cardiovascular mortality, but no other association was observed except for the combination of low control and low support. The two other studies using JEM also reported an association between low decision latitude and cardiovascular mortality (7, 17).

A small number of studies calculated population attributable fractions, although most of them did so for cardiovascular morbidity and not mortality (3, 29, 31, 32). Two studies (33, 34) estimated the fractions of cardiovascular mortality attributable to occupational exposures, including job strain, shift work, noise, exhaust gases, combustion products, and/or environmental tobacco smoke together. Nurminen et al (34) provided estimates of the fractions of cardiovascular fatalities attributable to job strain in Finland that were 16% for men and 19% for women. We may consider that these estimates were high given that Nurminen et al (34) as well as Järvholm et al (33) provided estimates of the fractions of cardiovascular mortality attributable to all occupational exposures together of 23.0% (women) and 20.1% (men) for acute myocardial infarction (33) and of 18.9% (men) and 9.1% (women) for IHD, and of 14.4% (men) and 6.7% (women) for all diseases of the circulatory system (34). In that sense, the attributable fractions of our study are more in line with the fractions of cardiovascular morbidity attributable to job strain, provided by the studies of Kivimaki et al (3), who found an estimate of 3.4% (95% CI 1.5-5.4), and Niedhammer et al (29), who reported an estimate of 4.46% (95% CI 1.26-7.65). The present study adds to the literature by providing estimates for mortality among men and women separately. The PAF estimated among women (6.44% for job strain and 4.02% for isostrain) were found to be non-significant (as 0 was included in the CI). This was explained by the non-significant HR from table 1 used for the calculations. Given the differences between HR (both in statistical significance and effect size) associated with job strain and iso-strain among women between table 1 and tables 2-3 for onthe-job cardiovascular mortality, these PAF estimates may be considered conservative among women. In addition, these PAF estimates may underestimate the global burden of cardiovascular mortality attributable to psychosocial work exposures as only job strain or iso-strain were considered and not other exposures such as, for example, job insecurity or long working hours.

Limitations and strengths of the study

The strengths of this study deserve to be mentioned. The studied cohort was a very large and national representative sample of the working population of men and women followed up for a long period of time for both exposure and outcome. As the data were routine, participation, response or selection bias were unlikely. However, a healthy worker effect may not be excluded for the association of current exposure and on-the-job cardiovascular mortality, but this would bias the exposure-outcome associations towards the null hypothesis. As we had complete follow-up, there was no attrition bias. As the data were provided by sources that were not the individuals themselves, no reporting bias for both exposure (assessed using a JEM) and outcome (derived from national registry data) could be suspected. We adjusted for physico-biomechanical-chemical-biological exposures that were used as proxies of social position. These occupational exposures are known to display strong social differences, the lower the social groups, the higher the prevalence of exposure (28). Indeed, the results without adjustment for these exposures showed stronger and more significant results than those presented in tables 1-3, underlying the appropriateness of taking them into account. Exposure was assessed by the validated JCQ, and we explored both main factors and combinations of factors. Thus, job strain, isostrain, and the four quadrants were found to be risk factors, which may be explained by the main effects of low decision latitude and low social support on cardiovascular mortality. This point was confirmed by the absence of interaction between high demands and low latitude. Exposure was defined using various time-varying measures. The comparison between the three exposure measures showed that the most recent (current) exposure may be more important than past (cumulative) exposure, although the results were not significant. This may also suggest that repeated measures of exposure (what we called current exposure at each time i) may be most powerful to capture the effects of exposure on cardiovascular mortality. Mortality was provided by the national death registry. We studied two outcomes (on-the-job and until 31/12/2002 cardiovascular mortality) and the results suggested that the effects of exposure on cardiovascular mortality may decrease after the end of exposure (potential reversibility of the effects). Two subtypes of cardiovascular mortality (IHD and stroke mortality) were studied, which has not been done previously. Finally, sensitivity analyses confirmed the robustness of the findings.

However, the study included limitations. Few adjustment variables were available, thus residual confounding bias was possible. We had no data on baseline cardiovascular morbidity. This may have led to a potential selection bias, as people with cardiovascular morbidity at baseline may have changed to less exposed jobs or left the labor market, but this would bias towards the null hypothesis. This may also have led to the reserve bias (bias away from the null) if people with cardio- vascular morbidity at baseline changed to jobs with low decision latitude for example. We may think that our study of cumulative and recency-weighted cumulative exposure might have reduced this bias as these exposure measures took past exposure into account. More information would have been useful to control for major cardiovascular risk factors. Nevertheless, controlling for cardiovascular risk factors in the association between psychosocial work factors and cardiovascular mortality may be considered as an over adjustment as these cardiovascular risk factors may be on the causal pathways between exposures and outcome. Although we controlled for other occupational exposures used as proxies of social position, this adjustment may not be completely satisfactory. Our sensitivity analysis additionally adjusted for occupation, a way to control further for social position. However, as occupation was among the job title variables used to construct the JEM, this may have led to over adjustment. Indeed, as expected, although most of the results were found to be similar, the associations were weaker and less significant. The true measure of the exposure-outcome associations may be in between. Although using a JEM may have the advantage of reducing reporting bias, imprecision in the exposure assessment, non-differential misclassification and bias towards the null hypothesis may be present and may have led to an underestimation of the observed associations. Furthermore, the validity of the JEM was lower for psychological demands and social support, which may contribute to explain discrepancies in the results for psychological demands (24). There was rare missing information about some jobs that was treated using midcensoring. As we only had data for 1976-2002, we were unable to assess complete working life-course exposure. We studied two subtypes of cardiovascular mortality (IHD and stroke), but the study of ischemic and hemorrhagic stroke separately provided non-significant results and did not confirm previous results on morbidity reporting an association of job strain with ischemic stroke but not with hemorrhagic stroke (1). Low statistical power was related to both a substantial proportion of stroke cases not specified as hemorrhage or infarction and consequently a low number of cases for each subtype of stroke. The total effect of all these limitations on our results may be difficult to evaluate as they included both over- and underestimations of the exposure-outcome associations.

Concluding remarks

Our findings suggested that the psychosocial work exposures of low decision latitude, low social support, job strain, iso-strain, high strain, and passive job showed concerning patterns of association with cardiovascular mortality. The same associations were also found for IHD and stroke especially among men. The PAF observed in our study were substantial and reaffirm the need for preventive policies at the workplace to improve cardiovascular health.

Acknowledgements

We wish to dedicate this paper to the memory of our highly valued colleague, Allison Milner, who died tragically and prematurely in August 2019.

The authors thank the members of the DARES (French Ministry of Labour), all the occupational physicians and médecins inspecteurs régionaux du travail, and all the employees who participated in the SUMER survey. The authors thank Catherine Buisson, Laurence Chérié-Challine, and Pascal Empereur-Bissonnet at Santé publique France (Direction Santé Travail) for the access to the COSMOP data. The authors also thank all people and public institutions who contributed directly or indirectly to the final COSMOP database, especially INSEE and INSERM-CépiDc.

Conflict of interest

The authors declare no conflicts of interest.

Funding

The French National Research Program for Environmental and Occupational Health of ANSES, France, supported this study (grant number: EST-2016/1/49).

Ethics approval

Ethical permissions were granted by French ethics committees: Commission Nationale de l'Informatique et des Libertés (no 762430V1 and no 04-1274) and Conseil National de l'Information Statistique (no 2009X705TV).

Sidebar

This study is one of the first large-scale prospective studies on the associations between psychosocial work exposures and cardiovascular mortality. It underlined the effects of these exposures on cardiovascular, including ischemic heart diseases and stroke, mortality. It also provided first estimates of the fraction of cardiovascular mortality attributable to these exposures.

Niedhammer I, Milner A, Geoffroy-Perez B, Coutrot T, LaMontagne AD, Chastang J-F. Psychosocial work exposures of the job strain model and cardiovascular mortality in France: results from the STRESSJEM prospective study. Scand J Work Environ Health. 2020;46(5):542-551. doi:10.5271/sjweh.3902

Correspondence to: Isabelle Niedhammer (ORCID: 0000-0002-8042-8925), INSERM U1085 - IRSET - Equipe ESTER, Faculté de Médecine - Université d'Angers, 28 rue Roger Amsler, CS 74521, 49045 ANGERS Cedex 01, France. [E-mail: isabelle.niedhammer@inserm.fr]

Footnote

1 INSERM, Univ Angers, Univ Rennes, EHESP, Irset (Institut de recherche en santé, environnement et travail) - UMR_S 1085, Epidemiology in Occupational Health and Ergonomics (ESTER) Team, Angers, France.

2 Centre for Health Equity, Melbourne School of Population and Global Health, University of Melbourne, Melbourne, Victoria 3010, Australia

3 Santé publique France, Saint-Maurice, France.

4 DARES, Ministere du Travail, Paris, France.

5 Institute for Health Transformation, School of Health &Social Development, Deakin University, Geelong, Victoria 3125, Australia.

6 Sadly, Professor Allison Milner died tragically on 12 August 2019.

References

References

1. Fransson EI, Nyberg ST, Heikkilä K, Alfredsson L, Bjorner JB, Borritz M et al. Job strain and the risk of stroke: an individual-participant data meta-analysis. Stroke 2015 Feb;46(2):557-9. https://doi.org/10.1161/ STROKEAHA.114.008019.

2. Huang Y, Xu S, Hua J, Zhu D, Liu C, Hu Y et al. Association between job strain and risk of incident stroke: A metaanalysis. Neurology 2015 Nov;85(19):1648-54. https://doi. org/10.1212/WNL.0000000000002098.

3. Kivimäki M, Nyberg ST, Batty GD, Fransson EI, Heikkilä K, Alfredsson L et al.; IPD-Work Consortium. Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis of individual participant data. Lancet 2012 Oct;380(9852):1491-7. https://doi.org/10.1016/S01406736(12)60994-5.

4. Xu S, Huang Y, Xiao J, Zhu W, Wang L, Tang H et al. The association between job strain and coronary heart disease: a meta-analysis of prospective cohort studies. Ann Med 2015;47(6):512-8. https://doi.org/10.3109/07853890.2015. 1075658.

5. Karasek R, Brisson C, Kawakami N, Houtman I, Bongers P, Amick B. The Job Content Questionnaire (JCQ): an instrument for internationally comparative assessments of psychosocial job characteristics. J Occup Health Psychol 1998 Oct;3(4):322-55. https://doi.org/10.1037/10768998.3.4.322.

6. Taouk Y, Spittal MJ, LaMontagne AD, Milner AJ. Psychosocial work stressors and risk of all-cause and coronary heart disease mortality: A systematic review and meta-analysis. Scand J Work Environ Health 2020 Jan 1;46(1):19-31. https://doi.org/10.5271/sjweh.3854.

7. Alterman T, Shekelle RB, Vernon SW, Burau KD. Decision latitude, psychologic demand, job strain, and coronary heart disease in the Western Electric Study. Am J Epidemiol 1994 Mar;139(6):620-7. https://doi.org/10.1093/oxfordjournals. aje.a117051.

8. Brunner EJ, Kivimäki M, Siegrist J, Theorell T, Luukkonen R, Riihimäki H et al. Is the effect of work stress on cardiovascular mortality confounded by socioeconomic factors in the Valmet study? J Epidemiol Community Health 2004 Dec;58(12):1019-20. https://doi.org/10.1136/ jech.2003.016881.

9. Joensuu M, Kivimäki M, Koskinen A, Kouvonen A, PulkkiRåback L, Vahtera J et al. Differential associations of job control components with mortality: a cohort study, 19862005. Am J Epidemiol 2012 Apr;175(7):609-19. https://doi. org/10.1093/aje/kws028.

10. Joensuu M, Kivimäki M, Pentti J, Virtanen M, Väänänen A, Vahtera J. Components of job control and mortality: the Finnish Public Sector Study. Occup Environ Med 2014 Aug;71(8):536-42. https://doi.org/10.1136/ oemed-2014-102111.

11. Johnson JV, Hall EM, Theorell T. Combined effects of job strain and social isolation on cardiovascular disease morbidity and mortality in a random sample of the Swedish male working population. Scand J Work Environ Health 1989 Aug;15(4):271-9. https://doi.org/10.5271/sjweh.1852.

12. Johnson JV, Stewart W, Hall EM, Fredlund P, Theorell T. Long-term psychosocial work environment and cardiovascular mortality among Swedish men [see comments]. Am J Public Health 1996 Mar;86(3):324-31. https://doi.org/10.2105/AJPH.86.3 324.

13. Karasek R, Baker D, Marxer F, Ahlbom A, Theorell T. Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. Am J Public Health 1981 Jul;71 (7):694-705. https://doi.org/10.2105/AJPH.7E7.694.

14. Kivimäki M, Leino-Arjas P, Luukkonen R, Riihimäki H, Vahtera J, Kirjonen J. Work stress and risk of cardiovascular mortality: prospective cohort study of industrial employees. BMJ 2002 Oct;325(7369):857. https://doi.org/10.1136/ bmj.325.7369.857.

15. Kivimäki M, Leino-Arjas P, Kaila-Kangas L, Luukkonen R, Vahtera J, Elovainio M et al. Is incomplete recovery from work a risk marker of cardiovascular death? Prospective evidence from industrial employees. Psychosom Med 2006 May-Jun;68(3):402-7. https://doi.org/10.1097/01. psy.0000221285.50314.d3.

16. Padyab M, Blomstedt Y, Norberg M. No association found between cardiovascular mortality, and job demands and decision latitude: experience from the Västerbotten Intervention Programme in Sweden. Soc Sci Med 2014 Sep;117:58-66. https://doi.org/10.1016/j. socscimed.2014.07.033.
17. Toivanen S, Hemström O. Income differences in cardiovascular disease: is the contribution from work similar in prevalence versus mortality outcomes? Int J Behav Med 2006;13(1):89-100. https://doi.org/10.1207/ s15327558ijbm1301_11.
18. Tsutsumi A, Kayaba K, Hirokawa K, Ishikawa S; Jichi Medical School Cohort Study Group. Psychosocial job characteristics and risk of mortality in a Japanese community-based working population: the Jichi Medical School Cohort Study. Soc Sci Med 2006 Sep;63(5): 1276-88. https://doi.org/10.1016/j.socscimed.2006.03.028.
19. Alfredsson L, Spetz CL, Theorell T. Type of occupation and near-future hospitalization for myocardial infarction and some other diagnoses. Int J Epidemiol 1985 Sep;14(3):37888. https://doi.org/10.1093/ije/14.3.378.
20. Johnson JV, Stewart WF. Measuring work organization exposure over the life course with a job-exposure matrix. Scand J Work Environ Health 1993 Feb;19(1):21-8. https:// doi.org/10.5271/sjweh.1508.
21. Niedhammer I, Milner A, Geoffroy-Perez B, Coutrot T, LaMontagne AD, Chastang JF. Prospective associations of psychosocial work exposures with mortality in France: STRESSJEM study protocol. BMJ Open 2019 Oct;9(10):e031352. https://doi.org/10.1136/ bmjopen-2019-031352.
22. Niedhammer I, Chastang JF, Gendrey L, David S, Degioanni S. Propriétés psychométriques de la version française des echelles de la demande psychologique, de la latitude décisionnelle et du soutien social du 4 Job Content Questionnaire,) de Karasek : résultats de l'enquete nationale SUMER. [Job Content Questionnaire" and its scales measuring psychological pressures, decisional latitude and social support: the results of the SUMER] Sante Publique 2006 Sep;18(3):413-27. https://doi.org/10.3917/ spub.063.0413.
23. Niedhammer I. Psychometric properties of the French version of the Karasek Job Content Questionnaire: a study of the scales of decision latitude, psychological demands, social support, and physical demands in the GAZEL cohort. Int Arch Occup Environ Health 2002 Mar;75(3):129-44. https://doi.org/10.1007/s004200100270.
24. Niedhammer I, Milner A, LaMontagne AD, Chastang JF. Study of the validity of a job-exposure matrix for the job strain model factors: an update and a study of changes over time. Int Arch Occup Environ Health 2018 Jul;91(5):523-36. https://doi.org/10.1007/s00420-018-1299-2.
25. Sylvestre MP, Abrahamowicz M. Flexible modeling of the cumulative effects of time-dependent exposures on the hazard. Stat Med 2009 Nov;28(27):3437-53. https://doi. org/10.1002/sim.3701.
26. Amick BC 3rd, McDonough P, Chang H, Rogers WH, Pieper CF, Duncan G. Relationship between all-cause mortality and cumulative working life course psychosocial and physical exposures in the United States labor market from 1968 to 1992. Psychosom Med 2002 May-Jun;64(3):370-81. https:// doi.org/10.1097/00006842-200205000-00002.
27. Niedhammer I, Chastang JF, David S. Importance of psychosocial work factors on general health outcomes in the national French SUMER survey. Occup Med (Lond) 2008 Jan;58(1):15-24. https://doi.org/10.1093/occmed/kqm115.
28. Niedhammer I, Chastang JF, David S, Kelleher C. The contribution of occupational factors to social inequalities in health: findings from the national French SUMER survey. Soc Sci Med 2008 Dec;67(11):1870-81. PubMed https://doi. org/10.1016/j.socscimed.2008.09.007
29. Niedhammer I, Sultan-Taieb H, Chastang JF, Vermeylen G, Parent-Thirion A. Fractions of cardiovascular diseases and mental disorders attributable to psychosocial work factors in 31 countries in Europe. Int Arch Occup Environ Health 2014 May;87(4):403-11. https://doi.org/10.1007/s00420013-0879-4.
30. Kivimäki M, Kawachi I. Work Stress as a Risk Factor for Cardiovascular Disease. Curr Cardiol Rep 2015 Sep;17(9):630. PubMed https://doi.org/10.1007/s11886-0150630-8
31. Olsen O, Kristensen TS. Impact of work environment on cardiovascular diseases in Denmark. J Epidemiol Community Health 1991 Mar;45(1):4-9. https://doi. org/10.1136/jech.45.1.4.

32. Rosengren A, Hawken S, Ounpuu S, Sliwa K, Zubaid M, Almahmeed WA et al.; INTERHEART investigators. Association of psychosocial risk factors with risk of acute myocardial infarction in 11119 cases and 13648 controls from 52 countries (the INTERHEART study): case-control study. Lancet 2004 Sep;364(9438):953-62. https://doi.org/10.1016/S0140-6736(04)17019-0.
33. Järvholm B, Reuterwall C, Bystedt J. Mortality attributable to occupational exposure in Sweden. Scand J Work Environ Health 2013 Jan;39(1):106-11. https://doi.org/10.5271/ sjweh.3284.
34. Nurminen M, Karjalainen A. Epidemiologic estimate of the proportion of fatalities related to occupational factors in Finland. Scand J Work Environ Health 2001 Jun;27(3):161213. https://doi.org/10.5271/sjweh.605.
Received for publication: 14 February 2020

DETAILS

Subject:	Occupational stress; Womens health; Working conditions; Mortality; Heart diseases; Exposure; Cardiovascular diseases; Ischemia; Cardiovascular disease; Occupational health; Social interactions; Stroke; Statistical models; Time measurement; Coronary artery disease
Business indexing term:	Subject: Occupational stress
Location:	France
Publication title:	Scandinavian Journal of Work, Environment &Health; Stockholm
Volume:	46
Issue:	5
Pages:	542-551,542A
Publication year:	2020
Publication date:	2020
Section:	Original article
Publisher:	Scandinavian Journal of Work, Environment &Health
Place of publication:	Stockholm
Country of publication:	Finland, Stockholm
Publication subject:	Occupational Health And Safety
ISSN:	03553140
e-ISSN:	1795990X
Source type:	Scholarly Journal

Language of publication:	English
Document type:	Journal Article
DOI:	https://doi.org/10.5271/sjweh.3902
ProQuest document ID:	2444685639
Document URL:	https://www.proquest.com/scholarly-journals/psychosocial-work-exposures-job-strain-model/docview/2444685639/se-2?accountid=211160
Copyright:	Copyright Scandinavian Journal of Work, Environment &Health 2020
Last updated:	2020-09-22
Database:	Public Health Database

Bibliography

Citation style: APA 6th - Annotated with Abstracts - American Psychological Association, 6th Edition

Åkerstedt, T., PhD, Narusyte, J., PhD., & Svedberg, P., PhD. (2020). Night work, mortality, and the link to occupational group and sex. Scandinavian Journal of Work, Environment & Health, 46(5), 508-515,508A. doi:https://doi.org/10.5271/sjweh.3892

Objective Night shifts are associated with several major diseases. Mortality has been studied only to a limited extent, and the association with night shifts remains unclear. The purpose of the present study was to investigate the association between duration of night shift exposure and mortality in a large sample from the Swedish Twin Registry (the SALT cohort). Results The hazard ratio (HR) for "ever" night shifts for total mortality was 1.07 95% confidence interval (CI) 1.01-1.15] but 1.15 (95% CI 1.07-1.25) for longer exposure (>5 years). Also, HR for cause-specific mortality due to cardiovascular disease was significant, with higher HR for longer night shift exposure. Mortality due to cancer was significant for longer exposure only. White-collar workers showed significant HR for longer exposure. In particular, male white-collar workers showed a significant HR, with a highest value for longer exposure HR 1.28 (95% CI 1.09-1.49)]. Heredity did not influence the results significantly. Conclusions Long duration of exposure to night shift work is associated with increased mortality, particularly in male white-collar workers. The lack of effects of accumulated exposure suggests that the results should be interpreted with caution.

Fadel, M., Evanoff, B. A., Andersen, J. H., d'Errico, A., Dale, A. M., Leclerc, A., & Descatha, A. (2020). Not just a research method: If used with caution, can job-exposure matrices be a useful tool in the practice of occupational medicine and public health? Scandinavian Journal of Work, Environment & Health, 46(5), 552-553,552A. doi:https://doi.org/10.5271/sjweh.3900

The potential usefulness of a JEM as a decision tool for compensation of workrelated musculoskeletal disorders has been examined (3). Because occupational diseases are often under-recognized, another practical application is using a JEM to screen for occupational exposures as part of health surveillance. ...]these JEM developed for research might also be used as a public health tool, provided that their limitations are properly taken into account. Refers to the following texts of the Journal: 2020;46(3):259-267 2020;46(3):231-234 Key terms: JEM; job exposure matrix; job-exposure matrix; method; methodology; occupational health; occupational medicine; public health; tool This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32367143 Marc Fadel, MSc, MD resident,1, Bradley A Evanoff, MD, MPH,2 Johan HAndersen, MD, PhD,3 Angelo d'Errico, MD,4 Ann Marie Dale, PhD,2 Annette Leclerc, PhD,5Alexis Descatha, MD, PhD 1 1 UNIV Angers, CHU Angers, Univ Rennes, Inserm, EHESP, Irset (Institut de recherche en santé, environnement et travail) - UMR_S1085, CAPTV CDC, Angers, France 2 Division of General Medical Sciences, Washington University School of Medicine, St. Louis, Missouri, USA 3 Occupational Medicine, University Research Clinic, Herning, Denmark 4 Unit of Epidemiology, Regional Health Service ASL T03, Grugliasco (Turin), Italy. 5 Inserm, UMS 011 Population-based Epidemiologic Cohorts Unit, Villejuif, France.

Rugulies, R., & Burdorf, A. (2020). Scandinavian journal of work, environment and health goes full open access. Scandinavian Journal of Work, Environment & Health, 46(5), 455-456,455A. doi:https://doi.org/10.5271/sjweh.3916

Unlike other major journals in occupational health and safety, SJWEH is published not by a large commercial publishing house but by a not-for-profit organization: the Nordic Association of Occupational Safety and Health (NOROSH) (4). ...]the journal is not published to make a financial profit, and we operate on a relatively small budget with the constant challenge to balance income and expenses. The five most often cited articles in 2019 among those published in SJWEH in 2018 were all open access articles (11-15). ...]OA also benefits authors. Key terms: editorial; open access; open science This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/32852043 Reiner Rugulies, PhD, Editor-in-Chief National Research Centre for the Working Environment, Copenhagen, Denmark e-mail: rer@nfa.dk] Alex Burdorf, PhD, Editor-in-Chief Department of Public Health, Erasmus Medical Centre, Rotterdam, The Netherlands e-mail: a.burdorf@erasmuscmc.nl] References 1.

Kuijer, P. P., & Burdorf, A., PhD. (2020). Prevention at work needed to curb the worldwide strong increase in knee replacement surgery for working-age osteoarthritis patients. Scandinavian Journal of Work, Environment & Health,

46(5), 457-460,457A. doi:https://doi.org/10.5271/sjweh.3915

In the upcoming decades, hospitals and clinics around the world face a steep rise in demand from patients seeking knee replacement surgery. An absolute increase in knee replacement surgery of 297%-to 57,893 procedures-is forecasted in The Netherlands between 2005 and 2030. The situation is similar in many countries: Sweden, 163% to 21,700 (2013-2030); Italy, 45% to about 100,000 (2017-2050), the UK, 916% to about 1.2 million (2015-2035); Australia, 276% to 65,569 (2013-2030); and the USA, 673% to 3.48 million (2005-2030). No projections are available for Asia, however, similar growth percentages have already been seen in Japan of 373% (2007-2014) and Korea (407%, 2001-2010). Here, Kuijer and Burdorf discuss how work might play a significant role in reducing the steep rise in replacement surgery for knee osteoarthritis across the world.

Loef, B., M.Sc, Van der beek, Allard J, PhD, Hulsegge, G., PhD., van Baarle, D., PhD, & Proper, K. I., PhD. (2020). The mediating role of sleep, physical activity, and diet in the association between shift work and respiratory infections. Scandinavian Journal of Work, Environment & Health, 46(5), 516-524,516A. doi:https://doi.org/10.5271/sjweh.3896

Objectives Shift work may be associated with an increased incidence of respiratory infections. However, underlying mechanisms are unclear. Therefore, our aim was to examine the mediating role of sleep, physical activity, and diet in the association between shift work and respiratory infections. Methods This prospective cohort study included 396 shift and non-shift workers employed in hospitals. At baseline, sleep duration and physical activity were measured using actigraphy and sleep/activity diaries, sleep quality was reported, and frequency of meal and snack consumption was measured using food diaries. In the following six months, participants used a smartphone application to report their influenza-like illness/acute respiratory infection (ILI/ARI) symptoms daily. Mediation analysis of sleep, physical activity, and diet as potential mediators of the effect of shift work on ILI/ARI incidence rate was performed using structural equation modeling with negative binomial and logistic regression. Results Shift workers had a 23% incidence rate ratio (IRR) 1.23, 95% CI 1.01-1.49] higher incidence rate of ILI/ARI than non-shift workers. After adding the potential mediators to the model, this reduced to 15% (IRR 1.15, 95% CI 0.94-1.40). The largest mediating (ie, indirect) effect was found for poor sleep quality, with shift workers having 29% more ILI/ARI episodes via the pathway of poorer sleep quality (IRR 1.29, 95% CI 1.02-1.95). Conclusions Compared to non-shift workers, shift workers had a higher incidence rate of ILI/ARI that was partly mediated by poorer sleep quality. Therefore, it may be relevant for future research to focus on perceived sleep quality as an underlying mechanism in the relation between shift work and increased infection susceptibility.

Costello, S., PhD., Chen, K., M.P.H., Picciotto, S., PhD., Lutzker, L., M.P.H., & Eisen, E., ScD. (2020). Metalworking fluids and cancer mortality in a US autoworker cohort (1941–2015). Scandinavian Journal of Work, Environment & Health, 46(5), 525-532,525A. doi:https://doi.org/10.5271/sjweh.3898
