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Original Research

Leisure Time Physical Activity and Sleep Predict Mortality in Men Irrespective of Background in Competitive Sports

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ABSTRACT

Introduction: Physical activity and sleep are closely related behaviors with suggested synergistic influence on cardiovascular health. Physical activity potentially modifies associations between sleep and mortality. Our aim was to study the interrelationships between sleep, leisure-time physical activity (LTPA), a history of sports, and mortality.

Methods: A prospective cohort of former elite male athletes (n = 1,028), and age- and region-matched nonathlete men (n = 610) completed a health questionnaire in 1985. Their mortality was followed up until December 31, 2011. Analyses included Cox proportional hazards models with sleep duration and sleep quality as main predictors of all-cause and cardiovascular disease (CVD) mortality.

Results: Sleep duration or sleep quality were not independently associated with mortality after controlling for socioeconomic and lifestyle factors. The interaction between sleep duration and LTPA was significant, with higher risk of all-cause and CVD mortality if sleeping 6 hours or less and not achieving 450 metabolic equivalent minutes LTPA weekly, as compared with sleeping 6.5–8.5 hours and achieving 450 metabolic equivalent minutes of LTPA. Also, the relative excess risk due to interaction between short sleep and low LTPA was significant for CVD mortality.

Discussion: Significant interactions between sleep duration and LTPA with regard to mortality were observed. In particular, short sleep and low LTPA jointly predicted all-cause and CVD mortality irrespective of a history of sports. Findings suggest important synergistic associations of short sleep and low LTPA with CVD mortality risk.

Keywords: Sleep, Physical activity, Mortality, Former athlete, Cardiovascular disease

Introduction

Both short and long sleep duration are suggested to associate with higher all-cause mortality while the relationships with cause-specific mortality have not conclusively been agreed on.[1–3] The role of confounding behaviors and health parameters in the sleep mortality relationship need more evaluation.[4] Short sleep associates with impaired glucose metabolism and obesity, low-grade inflammation, and hypertension,[5,6] all possible mechanisms for higher mortality.[1,2,4,5] Fewer biological explanations for the increased mortality risk of long sleepers have been presented.[1,7,8] Underlying disease and poor health or disruptions in the circadian rhythm may particularly be behind the risk of excessive mortality for long sleep.[9,10] Where the mortality risk in short and long sleepers may be related to and modified by different factors, physical activity and more precisely the lack thereof, is one suggested confounder in both long and short sleepers.[7,10,11] Physical activity can be situated on either side of sleep on a causal pathway to health and mortality[4,12,13] and physical activity and sleep are suggested to have a synergistic effect upon cardio-metabolic risk factors.[14]

Despite important interrelationships between physical activity and sleep, there are only few studies that have focused on the interaction between sleep and physical activity in relation to mortality. One such study was performed among U.S. men and women (aged 50–72 years), and both the multiplicative and additive interaction between sleep duration and moderate-to-vigorous intensity physical activity with mortality was examined. However, in this study, physical activity was not found to interact with sleep in relation to mortality.[15] In another study consisting of Swedish adult men and women aged 45–83 years, long sleep (> 8 hours) predicted higher all-cause mortality only in those with low physical activity.[11] Short sleep (< 6 hours) was found to predict mortality at all levels of physical activity.[11]

Preliminary results of the study have been presented at the American Heart Association Scientific Sessions, November 2015, Orlando, Fla.

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While the modifying effect of physical activity on the sleep-mortality association needs clarifying, physical activity as such reduces the risk of mortality.[16–18] Physical inactivity, that is, not meeting the current guidelines for physical activity accounts for about 9% of premature deaths worldwide.[17] Regular physical activity improves cardiorespiratory fitness level and also a good cardiorespiratory fitness reduces the risk of mortality.[19,20] Most competitive sports require training for a high cardiorespiratory fitness level, and lower mortality in former top-level athletes is partly due the good cardiorespiratory fitness they have achieved through a history of vigorous physical activity.[21,22] However, attaining sufficient levels of physical activity throughout life plays an important role for health and mortality risk with increasing age.[21,23,24] Remaining physically active or taking up physical activity at older ages (> 65 years) improves the odds of healthy ageing and survival.[25]

Sleep and physical activity are closely related behaviors both from energy expenditure as well as a time use point of view.[26,27] The relationship between physical activity and sleep is likely bidirectional where both acute and regular physical activity can improve sleep,[12,28] and sleep duration and quality may likewise influence physical activity behavior.[29] Physical activity is thought to affect sleep through mechanisms including fluctuations in body temperature and glucose metabolism, autonomic nervous system activity, and changes in body composition, mood and cardiorespiratory fitness.[28–30] However, among highly active and well-trained athletes, poor or insufficient sleep is surprisingly common and sleep patterns in this population are rather varying.[31,32] Athletes seem to spend rather long time in bed, but their actual sleep time often no more than 7 hours, with both sleep duration and sleep quality varying in relation to sports discipline, training, and competition schedules.[33,34] Among active Masters athletes Shephard et al.[35] observed better sleep quality among the athletes than the general population.

Our aim was to study the interrelationships between a history of sports, sleep, leisure time physical activity (LTPA), and all-cause and cardiovascular disease (CVD) mortality, in a population cohort enriched with individuals with a high physical activity level. Our cohort comprises male former elite level athletes and age- and residential area–matched nonathletic males. Concerning this cohort, we have learned that having a background in competitive sports increased the likelihood of attaining a healthy life style including physical activity.[22,36,37] Previous reports in this cohort also suggest lower mortality and morbidity in former athletes than nonathletes,[22,36,38] but sleep or the associations between sleep and mortality have so far not been studied.

Methods

The Finnish former elite athletes' cohort

A detailed description of study subjects has been published previously.[22] In brief, the prospective cohort is based on former elite male athletes (N = 2,448), that is, those who represented Finland at least once in international or inter-country sports competitions between the years 1920 and 1965. Athletes represented endurance sports such as cross-country skiing or long-distance running, power sports such as boxing, wrestling, weight lifting, and mixed sports including, for example, track and field jumping, short distance running, soccer, ice hockey, and shooting.[22] The nonathletes (N = 1712) were selected among Finnish men classified as completely healthy (military class A1, fully fit for ordinary military service, which is obligatory in Finland) at 20 years of age at the medical examination preceding their conscription.[22] The nonathletes were identified from public archives of the register of men liable for military service and matched on birth cohort and area of residence with the athletes.[22] In this study, a history of sports refers to the former athlete and nonathlete division.

In 1985, a health questionnaire was simultaneously mailed to all athletes and nonathletes alive at that time point (n = 2,528, 60.8% of all the identified athletes and nonathletes). In 1985, the response rates varied between 77% and 90% for nonathletes and athletes by sport, respectively. The questionnaire assessed demographic background, anthropometrics, symptoms and diseases, health-related factors, and health behaviors such as sleep and physical activity.

Ethical approval for this study (N° 173/13/03/2008) was provided by the HUCH Ethics Committee, Biomedicum Helsinki 2 C P.O. Box 705, FI-00029 Helsinki, Finland on 20th May, 2008. Informed consent was obtained from all individual participants included in the study.

Main exposure variables

Sleep duration was assessed on the health questionnaire by asking "How many hours do you usually sleep per 24 hours?" with 9 response alternatives (≤ 6 hours, 6.5, 7, 7.5, 8, 8.5, 9, 9.5, and ≥ 10 hours). Sleep duration was categorized into ≤ 6 hours/d (short sleep), 6.5–8.5 hours/d (mid-range sleep), and ≥ 9 hours/d (long sleep). This categorization was based upon previous literature[10] and a univariate Cox model where those sleep duration categories that were related with an increased risk of mortality as compared with 7 hours sleep were recognized. Sleep quality was reported on a question about usual sleep quality with responses "Well," "Fairly well," "Fairly poorly," "Poorly," and "Cannot say," further dichotomized into "Good sleep" (including "fairly well" and "fairly poorly") and "Poor sleep." Those reporting "cannot say" were considered missing data. The questions about sleep duration and quality have previously been used in the 1981 survey of the Finnish Twin cohort.[39–41]

Three structured questions about LTPA were used to calculate a physical activity index, representing cumulative leisure metabolic equivalent (MET) hours per week.[37] A multiple of the resting metabolic rate (1 MET referring to resting metabolic rate) was assigned to the type of LTPA reported and then was the product of intensity, duration, and frequency calculated for a total weekly LTPA. The method has been validated against a detailed physical activity interview.[42] Following current physical activity guidelines,[18] subjects were grouped according to their weekly amount of LTPA into those with sufficient (i.e., achieving 450 MET minutes per week) and insufficient LTPA (not-achieving 450 MET minutes per week).

Covariates

Occupational data were collected partly from the Central Population Registry of Finland and partly from the 1985 questionnaire. Occupational groups were classified into the following main categories: executives, white collar, blue collar, unskilled workers, and farmers. Each person was classified according to the occupation he had practiced for the longest period.[22] Previous studies in this cohort have reported on significant differences between athletes and nonathletes in occupational status and significant associations of occupational status, as a marker of socioeconomic status, with mortality.[22]

Marital status was assessed on the questionnaire, and answers were grouped into the categories married (including married, cohabiting, remarried) and not married (including single, divorced, and widowed), as previously in this cohort.[43] In one

question, typical working hours of the participant were assessed, and the information was used to group subjects as day-workers and night- or shift-workers (including all night-work, 2 shift- and 3 shift-work).

Subjects' smoking status was determined based on a detailed smoking history[44] and further classified into: never, occasional, former, and current smokers. Alcohol consumption based on quantity-frequency measures of beverage use was converted into grams of pure alcohol per month as previously reported.[45] The respondents were categorized as abstainers, light-to-moderate users (no more than 14 standard drinks a week, with 1 standard drink containing 12 g of alcohol) and heavy users (on average more than 2 drinks a day).[46]

Self-reported data on height (m) and weight (kg) were used to calculate the body mass index in kg/m^2, further categorized into normal (< 25 kg/m^2), overweight (25–29.9 kg/m^2), and obese (≥ 30 kg/m^2).

Life satisfaction was measured by a score including the items "interestingness of life," "life happiness," "life easiness," and "loneliness," with a high total score indicating high dissatisfaction.[47] Life satisfaction has previously been shown to correlate with depression in this cohort.[48]

Diurnal type or chronotype was assessed by one self-rated question, used in several Finnish data sets[49] originating in the Morningness-Eveningness questionnaire by Horne and Ostberg[50] as follows: "One hears about morning and evening type of people. Which one of these do you consider yourself to be?" Response categories were "Definitely morning," "More morning-than-evening," "More evening-than-morning," and "Definitely evening." Sleep medication use was assessed as days of use within the past 12 months. Those who reported no use or having used during less than 10 days were categorized as "No sleep medication," whereas more frequent use was categorized as "Sleep medication use." Most (14.5% points) of the 18.6% missing data on sleep medication use were reclassified as nonusers as they reported to sleep well and to become alert in less than 20 minutes in the morning. The rest with missing information were excluded as later explained.

Self-reported and physician-diagnosed chronic diseases in 1985, including coronary heart disease (coronary heart disease, angina pectoris), pulmonary disease (chronic bronchitis, emphysema, asthma), diabetes, arthritis (rheumatoid arthritis, osteoarthritis), Parkinson's disease, and cancer were dichotomized into a health status variable as having a history of disease (with some of the mentioned diseases) or not. Data on cancer diagnoses (yes/no) were obtained from the Finnish Cancer Registry and from the hospital discharge reports as International Classification of Diseases codes (ICD-8 from 1970 to 1985) to neoplasms of all organs (ICD 140–239). Discharge reports are held in a nationwide register by the national board of health and include both private hospitals and covered records (discharge dates) from the beginning of 1970 to the end of 1985.

Mortality follow-up data

All residents of Finland have a unique personal identification number, which was used for record linkages. Personal identifiers and possible dates of emigration or death for the cohort were obtained from the Population Register of Finland. Information on death was obtained using the national cause-of death register at Statistics Finland until December 31, 2011. Causes of death were classified according to the International Classification of Diseases (the eighth version in 1969–1986; the ninth version in 1987–1995; the 10th version in 1996 and onwards).

Inclusion criteria

In the current study, criteria for inclusion were as follows: (1) those alive at the start of the follow-up on January 1, 1985 and participating in the survey (n = 2,141), (2) availability of data on sleep duration, sleep quality, chronotype, LTPA, occupational status, marital status, shiftwork, BMI, smoking, alcohol consumption, sleep medication, life satisfaction, history of chronic diseases, and mortality until 31st December 2011 (n = 1,782), (3) not deceased due to cancer in 1985 or 1986 (n = 1,726). Furthermore were those responding "cannot say" to the sleep quality question (n = 12) and the shooters (n = 76) among the former athletes excluded. Shooters have a different age distribution and shooting as a sport has a different nature compared with the other sport disciplines included.[22]

Statistical analysis

The descriptive data are presented as frequencies and percentages by history of sports (ie, former athletes and nonathletes; Table 1). Cox proportional hazards model with age as the time variable and age at baseline in 1985 as the entry was used to estimate crude and adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) for total (all causes) and cardiovascular (CVD) mortality. Sleep duration and sleep quality were regarded as the main explanatory variables. Full adjustments of the models included history of sports (in full sample), occupational status, marital status, shift work, smoking, alcohol use, BMI, chronotype, sleep medication, and history of chronic disease. In models for CVD mortality, death of any other reason was treated as censoring.

The proportional hazards assumption for each exposure was tested for by including an interaction with time in crude, univariate models and visually by Kaplan–Meier plots. All included variables, except occupational status satisfied the assumption. For this reason, extended Cox proportional hazards models including an interaction between occupational status and time were calculated. Testing of the difference in -2 LogLikelihood between the normal and the extended Cox regression models supported that the extended model performs better for all-cause mortality but not for CVD mortality. Therefore, the results for all-cause mortality are based on the extended Cox regression model, whereas results for CVD mortality are based on normal Cox regression models.

First, we tested crude as well as adjusted Cox proportional hazards models with sleep duration and sleep quality for all-cause and CVD mortality stratified by the history of sports. Then, we tested the association between sleep duration and sleep quality with all-cause and CVD mortality in the full sample, and history of sports was added among the covariates. In the full sample, we tested interactions between a history of sports, a history of chronic disease, and LTPA with sleep duration and sleep quality, respectively, regarding both all-cause and CVD mortality. No statistically significant interaction was found between a history of sports and sleep duration and sleep quality, or between a history of chronic disease and sleep duration and sleep quality, respectively. There was a statistically significant interaction between sleep duration and LTPA, and sleep quality and LTPA regarding all-cause mortality (P = 0.03 and P = 0.03, respectively). For CVD mortality, the interaction between sleep duration and LTPA was significant (P = 0.01), but the interaction between sleep quality and LTPA was not (P = 0.06).

According to the aims of the study and the significant interaction between LTPA and sleep, subjects were grouped by their combination of sleep duration (3 categories) and LTPA level (2 categories) in the following 6 groups: short sleep + insufficient LTPA; short sleep + sufficient LTPA; long sleep + insufficient LTPA; long sleep + sufficient LTPA; mid-range sleep + insufficient LTPA; and

TABLE 1.
Characteristics of the Study Sample (N = 1638) by History of Sports

	Athletes, 1,028 (63%)	Nonathletes, 610 (37%)	*P* value for Chi-square test
Occupational status			
Executives	272 (26)	71 (12)	<0.0001
Clerical workers	419 (41)	164 (27)	
Skilled workers	283 (28)	273 (45)	
Unskilled workers	16 (2)	23 (4)	
Farmers	38 (4)	79 (13)	
Marital status			
Married or co-habiting	883 (86)	508 (83)	0.15
Living alone	145 (14)	102 (17)	
Type of work			
Day-work	856 (83)	524 (86)	0.16
Night- or shift work	172 (17)	86 (14)	
Leisure time physical activity			
Less than 450 MET minutes/wk	262 (25)	326 (53)	<0.0001
At least 450 MET minutes/wk	766 (75)	284 (47)	
Sleep duration short sleep	73 (7)	63 (10)	0.07
Mid-range sleep	835 (81)	480 (79)	
Long sleep	120 (12)	67 (11)	
Sleep quality			
Good sleep	940 (91)	525 (86)	0.001
Poor sleep	88 (9)	85 (14)	
Chronotype			
Morning type	398 (39)	227 (37)	0.01
More morning type	271 (26)	202 (33)	
More evening type	276 (27)	129 (21)	
Evening type	83 (8)	52 (9)	
Body mass index			
Normal < 25	419 (41)	216 (35)	0.10
Overweight 25–29.9	490 (48)	315 (52)	
Obese ≥ 30	119 (12)	79 (13)	
Alcohol consumption			
Abstainers	75 (7)	69 (11)	0.02
Light or moderate users	793 (77)	458 (75)	
Heavy users	160 (16)	83 (14)	
Smoking status			
Never	515 (50)	164 (27)	<0.0001
Occasional	49 (5)	16 (3)	
Former	297 (29)	250 (41)	
Current	167 (16)	180 (30)	
Use of sleep medications			
No	967 (94)	581 (95)	0.31
Yes	61 (6)	29 (5)	
History of chronic disease			
No chronic disease	670 (65)	393 (64)	0.76
Chronic disease	358 (35)	217 (36)	
Life satisfaction (median, range)	7 (4–20)	8 (4–20)	

mid-range sleep + sufficient LTPA. Also, 4 groups by combination of sleep quality and LTPA level was formed as follows: poor sleep + insufficient LTPA; poor sleep + sufficient LTPA; good sleep + insufficient LTPA; and good sleep + sufficient LTPA.

All-cause and CVD mortality risk in the combinations of sleep and LTPA were tested, adjusting as previously for a history of sports, occupational status, marital status, shift work, smoking, alcohol use, BMI, and chronotype, sleep medication, and history of chronic disease. The group of mid-range sleep and sufficient LTPA, or good sleep and sufficient LTPA, respectively, was considered as the reference category. Finally, additive interaction was estimated by the relative excess risk due to interaction (RERI) with corresponding 95% CIs using an excel spreadsheet created by Knol and VanderWeele[51] available as a supplement to their article. For the purpose of the additive interaction analyses were short sleepers with insufficient LTPA, short sleepers with sufficient LTPA, and mid-range and long sleepers with insufficient LTPA compared with mid-range and long sleepers with sufficient LTPA, adjusting for all covariates as in previous models. Statistical analyses were performed using SAS version 9.3. (SAS Institute Inc., Cary, N.C.), with significance at $P < 0.05$.

Results

Our analyses included 1,638 (77.5%) of the initial 2141 former athletes and nonathletes who participated in the baseline survey in 1985. Compared with the included, the excluded were older (mean age, 62 versus 55 years; $P < 0.0001$) and more likely had a history of chronic disease [odds ratio (OR), 2.11; $P < 0.0001$], slept either ≤ 6 hours (OR, 1.5; $P = 0.02$) or ≥ 9 hours (OR, 1.38; $P = 0.03$) than 6.5–8.5 hours, reported poor sleep (OR, 2.65; $P < 0.0001$), and did not achieve the LTPA recommendations (OR, 1.58; $P < 0.0001$).

Table 1 presents the distribution in background characteristics by history of sports. Athletes were on average older than the nonathletes with a mean age of 56 years, (SD, ±10 years) versus nonathletes mean 53 years (SD, ±9 years). Of the former athletes included in this study, 13% (n = 132) were categorized as endurance athletes, 34% (n = 348) as power athletes, and 53% (n = 548) as mixed sports athletes. Mixed sports athletes had the lowest prevalence of long sleep with 9% as compared with 17% in endurance athletes and 14% in power sport athletes. The prevalence of short sleep was 7% for all different sports. Regarding sleep opinion, 7% of the endurance athletes reported poor sleep as compared with 9% in mixed and power athletes, respectively. Seventy percentage of the power athletes and 77% of endurance and mixed sports athletes had sufficient LTPA.

Results for mortality in relation to sleep duration, sleep quality, and LTPA in the full sample are presented in Table 2. There was a crude association for short sleep duration (HR, 1.33), poor sleep quality (HR, 1.29), and insufficient LTPA (HR, 1.31), with all-cause mortality. Regarding CVD mortality, only insufficient LTPA showed a statistically significant crude association (HR, 1.40). Adjusting models for history of sports and demographic and health-related covariates, all independent associations of sleep and LTPA were attenuated and no longer significant (Table 2).

Table 3 presents the results for the combinations of sleep duration and sleep quality with LTPA regarding all-cause mortality, respectively. A combination of short sleep duration and insufficient LTPA, as compared with mid-range sleep and sufficient LTPA, was associated with higher all-cause mortality, also after full adjustment for covariates. A combination of poor sleep quality and insufficient LTPA compared with good sleep and sufficient LTPA was also significantly associated with all-cause mortality in crude model, but no longer in the fully adjusted model. The analysis of RERI showed a positive, but nonsignificant additive interaction between short sleep and insufficient LTPA for all-cause mortality ($RERI_{\text{short sleep + insufficient LTPA}} = 0.36$; 95% CI, -0.23 to 0.96) and a positive, but nonsignificant interaction between sleep quality and LTPA for all-cause mortality ($RERI_{\text{poor sleep + insufficient LTPA}} = 0.27$; 95% CI, -0.20 to 0.74).

Table 4 presents the results for the combinations of sleep duration and sleep quality with LTPA regarding CVD mortality, respectively. Short sleep and insufficient LTPA significantly associated with CVD mortality throughout the models. Also mid-range sleep

TABLE 2.

Hazard Ratios and 95% CIs for Sleep Duration, Sleep Quality, and Leisure Time Physical Activity Predicting All-Cause and Cardiovascular Mortality

	Crude	Model 1	Model 2	Model 3
All-cause mortality (824 deaths/1,638)				
Short sleep	**1.33 (1.05–1.68)**	1.23 (0.95–1.59)	1.21 (0.93–1.57)	1.16 (0.89–1.50)
Long sleep	0.92 (0.75–1.13)	0.89 (0.73–1.10)	0.90 (0.73–1.10)	0.85 (0.69–1.05)
Poor sleep	**1.29 (1.05–1.59)**	1.16 (0.92–1.46)	1.14 (0.91–1.44)	0.99 (0.78–1.26)
Insufficient LTPA	**1.31 (1.14–1.50)**	**1.28 (1.10–1.48)**	**1.27 (1.09–1.48)**	1.12 (0.96–1.31)
CVD mortality (391 CVD cases/1,638)				
Short sleep	1.33 (0.95–1.86)	1.22 (0.84–1.78)	1.15 (0.79–1.68)	1.13 (0.78–1.65)
Long sleep	0.91 (0.68–1.23)	0.88 (0.65–1.18)	0.86 (0.64–1.16)	0.79 (0.59–1.07)
Poor sleep	1.31 (0.97–1.77)	1.19 (0.85–1.65)	1.17 (0.84–1.63)	1.00 (0.71–1.41)
Insufficient LTPA	**1.44 (1.17–1.76)**	**1.42 (1.15–1.76)**	**1.42 (1.14–1.77)**	1.21 (0.97–1.51)

Model 1: sleep variables, history of sports and physical activity; Model 2: Model 1 + socioeconomic variables; Model 3: Model 2 + other lifestyles, sleep medication, and chronic disease. Models 2 and 3 for all-cause mortality are extended Cox models including an interaction between occupational status and time. Reference categories for sleep and LTPA were following: mid-range sleep, good sleep, sufficient LTPA. Statistically significant (p<0.05) hazard ratios and CIs are bolded.

and insufficient LTPA was significantly associated with CVD mortality until fully adjusted model. The combination of poor sleep and insufficient LTPA, as well as good sleep and insufficient LTPA showed a significant association with CVD mortality in the first steps of modeling, but not in the fully adjusted model. According to the RERI, there was a significant positive additive interaction between short sleep and insufficient LTPA for CVD mortality ($RERI_{\text{short sleep + insufficient LTPA}}$ = 1.12; 95% CI, 0.19–2.06). The additive interaction between sleep quality and insufficient LTPA for CVD mortality was nonsignificant ($RERI_{\text{poor sleep + insufficient LTPA}}$ = 0.45; 95% CI, -0.26 to 1.15).

Sensitivity analyses

Further adjusting the Cox proportional hazards models for life satisfaction as a proxy for depression had a minor impact on the HRs or the interpretational outcome of the models. A small percentage of persons in the mid-range sleep and sufficient LTPA group, as well as in the good sleep and sufficient LTPA group reported sleep medication use (4.8% and 3.4%, respectively). Excluding those reporting use of sleep medication from the reference groups, respectively, resulted in virtually no changes in HRs or interpretational outcome of the models. Finally, no significant crude associations were found for sleep duration or sleep quality or the combinations of sleep duration and insufficient LTPA and sleep quality and insufficient LTPA with cancer mortality (results not shown).

Discussion

A few significant multiplicative and additive interactions between sleep duration and insufficient LTPA regarding all-cause and CVD mortality were found. The main finding was that short sleepers (6 hours or less) with insufficient LTPA (less than 450 MET minutes per week) had an increased all-cause and CVD mortality compared with those with mid-range sleep and sufficient LTPA. No other significant associations were found.

The interaction between sleep and physical activity for health has been recognized,[52–54] but the few results regarding interrelationships between sleep, physical activity, and mortality are not coherent and leave some unanswered questions. First of all, the classification of physical activity in the studies has differed. Bellavia et al.[11] who reported on a significant interaction between physical activity and sleep in relation to both all-cause and CVD mortality classified daily physical activity of the participants into thirds, where physical activity represented the usual total amount of occupational, domestic, leisure time, and walking or biking during the previous year. The association between sleep duration and mortality was U-shaped in the two lowest thirds, but not in the highest.

TABLE 3.

Hazard Ratios and 95% Confidence Intervals for All-Cause Mortality by Sleep Duration and Leisure Time Physical Activity Groups and Sleep Quality and Leisure Time Physical Activity Groups, Respectively

	Crude	Model 1	Model 2	Fully Adjusted Model
Sleep duration and LTPA, reference: mid-range sleep and sufficient LTPA (387/861)				
Short sleep and insufficient LTPA (38/56)	**2.23 (1.59–3.11)**	**2.12 (1.51–2.98)**	**1.93 (1.37–2.71)**	**1.49 (1.05–2.11)**
Short sleep and sufficient LTPA (42/80)	1.12 (0.81–1.55)	1.10 (0.80–1.52)	1.13 (0.82–1.56)	1.04 (0.75–1.45)
Long sleep and insufficient LTPA (47/78)	0.96 (0.71–1.30)	0.93 (0.68–1.26)	0.95 (0.70–1.30)	0.80 (0.58–1.11)
Long sleep and sufficient LTPA (68/109)	1.05 (0.81–1.36)	1.05 (0.81–1.36)	1.03 (0.80–1.34)	0.99 (0.76–1.29)
Mid-range sleep and insufficient LTPA (242/454)	**1.34 (1.14–1.58)**	**1.30 (1.10–1.53)**	**1.29 (1.09–1.53)**	1.15 (0.97–1.37)
Sleep quality and LTPA, reference: good sleep and sufficient LTPA (446/950)				
Poor sleep and insufficient LTPA (51/73)	**2.05 (1.53–2.74)**	**1.94 (1.44–2.61)**	**1.84 (1.36–2.49)**	1.29 (0.94–1.77)
Poor sleep and sufficient LTPA (51/100)	1.05 (0.79–1.41)	1.03 (0.77–1.38)	1.04 (0.77–1.39)	0.94 (0.69–1.27)
Good sleep and insufficient LTPA (276/515)	**1.23 (1.06–1.43)**	**1.19 (1.02–1.39)**	**1.20 (1.03–1.41)**	1.08 (0.92–1.27)

Model 1: history of sports; model 2: model 1 + socioeconomic variables; fully adjusted model: model 2 + other lifestyles, sleep medication, and chronic disease. Models 2 and the fully adjusted model for all-cause mortality are extended Cox models including an interaction between occupational status and time. Number of cases and number of men in that category at baseline are given in parentheses. Statistically significant (p<0.05) hazard ratios and CIs are bolded.

TABLE 4.

Hazard Ratios and 95% Confidence Intervals for Cardiovascular Mortality by Sleep Duration and Leisure Time Physical Activity Groups and Sleep Quality and Leisure Time Physical Activity Groups, Respectively

	Crude	Model 1	Model 2	Fully adjusted model
Sleep duration and LTPA, reference: mid-range sleep and sufficient LTPA (180/861)				
Short sleep and insufficient LTPA (23/56)	**2.90 (1.87–4.48)**	**2.79 (1.79–4.36)**	**2.57 (1.64–4.02)**	**1.98 (1.25–3.12)**
Short sleep and sufficient LTPA (15/80)	0.85 (0.50–1.45)	0.84 (0.50–1.43)	0.81 (0.47–1.38)	0.75 (0.44–1.30)
Long sleep and insufficient LTPA (23/78)	0.99 (0.64–1.54)	0.97 (0.62–1.51)	0.95 (0.60–1.49)	0.75 (0.47–1.18)
Long sleep and sufficient LTPA (31/109)	1.04 (0.71–1.53)	1.04 (0.71–1.52)	1.03 (0.70–1.51)	0.96 (0.65–1.42)
Mid-range sleep and insufficient LTPA (119/454)	**1.41 (1.18–1.78)**	**1.37 (1.08–1.75)**	**1.38 (1.08–1.76)**	1.18 (0.92–1.52)
Sleep quality and LTPA, reference: good sleep and sufficient LTPA (204/950)				
Poor sleep and insufficient LTPA (27/73)	**2.37 (1.58–3.54)**	**2.28 (1.50–3.46)**	**2.17 (1.42–3.31)**	1.46 (0.94–2.27)
Poor sleep and sufficient LTPA (22/100)	0.99 (0.64–1.54)	0.98 (0.63–1.52)	0.97 (0.62–1.51)	0.87 (0.55–1.39)
Good sleep and insufficient LTPA (138/515)	**1.33 (1.07–1.65)**	**1.30 (1.04–1.63)**	**1.31 (1.04–1.66)**	1.14 (0.90–1.44)

Model 1: history of sports; model 2: Model 1 + socioeconomic variables; fully adjusted model: Model 2 + other lifestyles, sleep medication, and chronic disease. Number of cases and number of men in that category at baseline are given in parentheses. Statistically significant ($p<0.05$) hazard ratios and CIs are bolded.

Xiao et al.[15] who did not find any significant interaction assessed physical activity as the typical frequency of moderate-to-vigorous intensity physical activity during the past 10 years. A cutoff for high physical activity was set at 1 hour of moderate-to-vigorous intensity physical activity per week. This is less than the recommended weekly physical activity dose for adults that were used as a criterion for sufficient LTPA in the current study. However, it must be kept in mind that our measure of physical activity did not include occupational or commuting physical activity, which can lead to lower proportion of those meeting the sufficient physical activity level.

Not only sleep duration, but also sleep quality in relation to mortality warrant consideration. We also studied sleep quality but did not find any statistically significant interaction between poor sleep quality and insufficient LTPA with all-cause or CVD mortality. Neither the study of Bellavia et al.[11] or Xiao et al.[15] examined sleep quality. Sleep duration and sleep quality may be overlapping, yet different characteristics of sleep[5,10] and the solution to poor sleep is not always a change in sleep duration.[10,55] Epidemiological studies have shown significant associations between measures of sleep quality and cardiovascular health.[56] However, in adult Finnish twin pairs,[40] and among American men and women,[57] sleep duration rather than sleep quality showed a significant association with mortality. Poor sleep can be initiated by and related to several factors,[58] one being chronotype.[59] In this current cohort of former athletes, Broms et al.[49] previously observed evening types to have a higher mortality than morning types. We included chronotype as a covariate in the Cox models, but our result did not show any independent effect for chronotype in the models.

Our findings add to the sparse literature on the topic and suggest that LTPA modifies the association particularly between short sleep and cardiovascular mortality where the coexistence of short sleep and insufficient LTPA contribute significantly to the excess CVD mortality risk. Short sleep associates with impaired glucose metabolism and obesity, low-grade inflammation, and hypertension,[6,56] all that make up possible mechanisms for the relationship between short sleep and CVD mortality.[1,2,5] Also, physical activity modifies cardiometabolic risk factors, and low physical activity is an established risk factor for CVD mortality.[17,20] Thus, it is not surprising to find the combination of short sleep and insufficient LTPA to predict a higher CVD mortality. Similar to us, Xiao et al.[15] also studied the additive interaction between sleep and physical activity, but they did not find any significant additive interaction. Where we chose to include the long sleepers in the reference group for our additive interaction analyses, Xiao et al. excluded the long sleepers from those analyses.

Contrary to previous literature, we did not see any clear U-shaped association between sleep duration and mortality in our models. The two ends of the U-shaped association between sleep duration and mortality are proposed to be explained by different mechanisms.[1,8,10] It has, for example, been suggested that health status modifies the associations between sleep and mortality, particularly long sleep and mortality.[4,9] In the current study, long sleep was not related to increased mortality neither in crude or adjusted models and there were no statistically significant interactions between history of disease and sleep with regard to mortality. The survival of the healthiest until baseline measurements may have affected the result in the current study, especially regarding long sleep. Bellavia et al.[11] who observed that long sleep associate with mortality only among subjects with low physical activity, could not control for depression. We assessed life satisfaction, a correlate of depression,[48] but did not find it to impact the nonsignificant associations between long sleep and mortality.

Some differences in the distribution of sleep duration and sleep quality and level of LTPA between former athletes and nonathletes were observed in this study. The former athlete men more often had mid-range than short sleep as compared with the nonathletes, and sufficient LTPA was as common in short as in long sleepers among the former athletes. As a comparison, in the cohort of Bellavia et al.,[11] high physical activity was reported for a substantially higher percentage of short than long sleepers (53% and 16%, respectively). In their review, Driver and Taylor[28] suggested that athletic individuals sleep better than nonathletic counterparts whether they are training or not. Cardiorespiratory fitness that develops through regular physical activity and exercise is suggested as one mechanism linking physical activity with good sleep.[12,28,29] Many former athletes sustain good fitness levels after their active career mainly by remaining physically active.[22] Nevertheless, in the current study, the association between sleep duration or sleep quality and mortality was not different between athletes and nonathletes, and sleep was not an independent predictor of mortality in either group.

The occurrence of diseases and health issues differ between the former athletes and nonathletes, and also to some extent between athletes from different sports.[23,60] Genetic factors that might underlie the selection to sports and different training regimens, but also the background of vigorous physical activity included in training for high-level competitive sports may explain some of the health differences between athletes and nonathletes and also between different athletes, respectively.[60] The longer life

expectancy in former athletes is to a high degree explained by a decreased CVD mortality and lower cancer risk in this population, as reported earlier.[22] We did not observe any interactions between sleep, LTPA, and cancer mortality (results not shown), and nor did Bellavia et al.[11] in their study. Frequent snoring was reported by 45% in the short sleep and insufficient LTPA group, but it is unlikely though that mortality risk is due to undiagnosed sleep apnoea. Snoring was not independently related to mortality and excluding 5% of the total sample with frequent snoring and a BMI ≥ 30 kg/m^2 did not change the outcome of the final analyses.

Limitations of the study

One weakness of our study is the use of self-reported measures of sleep and physical activity that include the possibility of reporting bias. However, at the time of the baseline questionnaire, availability and use of objective accelerometers was limited. Sleep duration was measured with one categorical question, and it is therefore not possible to analyze a true continuous measure of sleep duration. Furthermore, we do not know how the participants, especially the athletes slept when they were active. Thus, it remains unknown whether or not there have been changes in their sleep duration after their active career, and if so, to what extent. This kind of transition has also not, to our knowledge, been studied elsewhere. Where almost half of those with short sleep and insufficient LTPA were former athletes, these subjects can have had insufficient sleep already in their active days, causing them to eventually stop being physically active. Another scenario is that they have become short sleepers only later in life, as a consequence of the athletic retirement. Findings supporting the latter theory come from the Aerobics Centre cohort and suggest that a decline in cardiorespiratory fitness that mimics a reduced physical activity level predisposes to shorter sleep and poorer quality of sleep.[61] It has also been observed that decreasing the amount of daily physical activity in active athletes has a negative impact on sleep.[62]

Other factors such as health status or socioeconomic status are also prone to change over time. In addition, there are many important bidirectional associations between health and behaviors and a change in weight can, for example, have major effects on sleep quality and quantity as well as numerous health indices. We are limited by the fact that we do not know what changes in the athletes' and nonathletes' life have led to the reported sleep in 1985. The role of health status was controlled for by excluding early deaths during the follow-up, and models were adjusted for by the known history of disease. The interaction between history of disease and sleep with mortality was also examined but found nonsignificant. Regarding socioeconomic status, each person was classified according to the occupation he had practiced for the longest period until 1985,[22] thus partly controlling for the change in occupational status taken place before the baseline measurement. Furthermore, marital status at baseline was also taken into account.

It also needs to be acknowledged that the sample comprised only men and thus the study leaves a lack of knowledge regarding women athletes' sleep and mortality. There may also be some selection bias related to the forming of the cohort. As stated, our cohort includes former top-level athletes who are a selected group with good health at baseline.[22,60] Furthermore, the nonathletes also represent the most healthy and fit population as determined in the medical examination preceding their conscription at age 20. It is not easy to find matching subjects to world class athletes, but in this cohort, the comparability of the nonathletes to the athletes can be considered good.[22] Even if the results are not directly generalizable to the average population, they highlight the importance of joint behaviors such as physical activity and sleep for CVD risk, even in such a selected population.

Conclusions

In this unique cohort including former elite athletes and nonathlete men, we observed a significant interaction between sleep and LTPA with mortality, particularly CVD mortality risk. Higher risk was observed for men having short sleep and insufficient LTPA even after adjusting for a history of sports, and a variety of other demographic, behavioral, and health-related factors. The results also suggest that on an additive scale, the combination of short sleep and insufficient LTPA is associated with a larger risk than the sum of the estimated risk of short sleep or LTPA alone. It can be concluded that a former sports career does not fully protect with regard to mortality risk, and the negative impact of short sleep rather varies according to LTPA level. Future studies are still needed to replicate the findings of an interaction between sleep and physical activity for mortality, yet in different populations. The CVD risk profile including both behavioral and health indices of short sleepers with low LTPA, need to be determined further to provide more tools for health promotion and preventive work. It is especially important also to investigate how changes in sleep duration or physical activity levels and how different types of physical activity affect the risk profile.

Disclosure

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