



Sleep duration and sarcopenia risk: a systematic review and dose-response meta-analysis

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Abstract

Purpose Present systematic literature review and dose-response meta-analysis were carried out to evaluate the association between sleep duration and sarcopenia risk.

Methods Related studies were found by searching ISI Web of science databases, Scopus, and PubMed, up to May, 2019. Data were available from four studies. A total odds ratio of 17551 participants in these studies was pooled for the current study.

Results Pooled outcomes from random effects model demonstrated that lowest category of sleep duration (under 6 h) versus reference category (6–8 h) was significantly related with increased risk of sarcopenia (OR: 1.71 95% CI, 1.11, 2.64). Pooled OR also indicated that highest category (more than 8 h) of sleep duration versus reference category (6–8 h) was significantly associated with increased risk of sarcopenia (OR: 1.52 95% CI, 1.23, 1.88). Moreover, subgroup analysis by sex showed that women were affected by both short and long sleep while men were only affected by long sleep duration. The nonlinear dose-response meta-analysis revealed a U-shaped association between sleep duration and the risk of sarcopenia, with a nadir at 8 h per day. The linear dose-response meta-analysis illustrated that the risk of sarcopenia did not change significantly nor for a 0.5-h increment neither for 1-h increment in sleep duration per day.

Conclusion The outcomes from this meta-analysis indicate that the public should be made aware of the negative consequences of long and short sleep for sarcopenia especially among women. Further studies should now be undertaken to establish possible links between risk of sarcopenia and sleep duration.

Keywords Sleep duration · Sarcopenia · Systematic review · Meta-analysis

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Abbreviations

CI	Confidence interval
GH	Growth hormone
HPA	Hypothalamo-pituitary-adrenal
IGF-1	Insulin-like growth factor-1
OR	Odds ratio

Introduction

Aging has an important impact on changes in body composition including a loss of skeletal muscle mass [1]. The term *sarcopenia*, to define this condition with advancing age, was first proposed by Rosenberg et al. in 1989 [2]. Under normal conditions, after 30 years of age, muscle mass reduces by about 3 to 5% every 10 years [3]. At the tissue level, aging is accompanied with a decrease in the synthesis of muscle protein and an increase in fat mass [4, 5].

As the majority of the loss of power or physical function can also be caused by reduced skeletal muscle mass [6], the causes and consequences of sarcopenia need further investigation [7, 8]. Due to the multifactorial nature of the sarcopenic process, the importance of other risk factors should be considered. These include genetics [9, 10], physical activity [11–13], nutritional status (e.g., energy, protein, and vitamin D) [14–18], insulin resistance [19–24], hormonal changes (e.g., serum testosterone and growth hormone) [25, 26], atherosclerosis [20–22], and pro-inflammatory cytokines [27]. Moreover, sleep can affect the health of skeletal muscle through its effects on the function of endocrine factors [28, 29]. Sleep disorders are very common in older adults with up to 50% of the elderly complaining of regular sleep problems [30]. Reduced nocturnal sleep time, as a kind of sleep disturbance, can decrease quality of life and is related with depression, anxiety disorders, fatigue, and irritability as well as medical problems [31]. Furthermore, insomnia is associated with impaired social and occupational functions, to the extent that it can be considered a “24-h” disorder [31]. As such, insomnia has high social costs for both the individual and wider community [32]. Insomnia can be due to changes in hormones and pro-inflammatory factors [29], which have a negative effect on the musculoskeletal system [33, 34]. For example, sleep deprivation impacts testosterone and anabolic hormones such as insulin-like growth factor-1 (IGF-1) that regulate protein synthesis and thus maintain skeletal muscle mass [35]. Furthermore, insulin resistance associated with sleep deprivation can cause loss of muscle mass and function in the elderly [36].

A number of studies have indicated the relationship between sleep duration and risk of sarcopenia [37–39]. Chien et al. [38] showed that elder Taiwanese with both short sleep time or long sleep durations are more to develop sarcopenia. However, Kim et al. [40, 41] demonstrated that just higher

sleep duration was related with increased risk of sarcopenia among women. Although several studies exist in this field, considerable controversy has remained. The goal of current study was try to address these contradictory results, by evaluating the association between sleep duration and sarcopenia risk. It is expected that this will contribute to current knowledge and help to promote the health of all individuals at risk.

Methods

Literature search and selection

The present study was conducted according to the guidelines of the PRISMA. A systematic search was performed in the following electronic database: PubMed, Scopus, and ISI Web of science databases; search was performed from inception up to May, 2019. In order to perform current search strategy, medical subject heading (MeSHs) and keywords were used without any limitations for date or language. The following keywords and MeSHs were applied: (*sleep* OR *insomnia* OR *insomnias* OR “*sleep problems*” OR “*sleep quality*” OR “*sleep duration*” OR “*sleep deprivation*” OR “*sleep disturbance*” OR “*sleep disorders*” AND *sarcopenia* OR *sarcopenias* OR “*Muscular Atrophy*” OR “*Muscular Atrophies*” OR “*Muscle Atrophy*” OR “*lean mass*” OR “*lean body mass*” OR “*musculoskeletal aging*” OR “*muscle aging*” OR “*muscle mass*” OR “*muscle strength*” OR “*lean body mass*” OR “*dual-energy X-ray absorptiometry*” OR “*sarcopenias*” OR “*EWGSOP*” OR “*skeletal muscle mass*” OR “*appendicular skeletal muscle mass*.”

Eligibility criteria

Articles were included in present meta-analysis if they met following inclusion criteria: (1) observational articles that provided on the relationship between sleep duration and sarcopenia risk and (2) articles that reported odds ratios (OR) and 95% confidence intervals (CI) of sarcopenia risk. Articles were discarded in keeping with following exclusion criteria: (a) the data could not be used for definite outcomes; (b) specific articles without useful data (letters, conference reports, case reports, or reviews); and (c) inadequate data reporting.

Study selection

The headlines and abstracts of all obtained papers which retrieved initially were assessed separately by three investigators. An assessment form with specific above-mentioned inclusion criteria was used before any study could be entered in present study. Manual search also performed in related review articles and possible related references in the included studies.

Any disagreements between reviewers before inclusion of study were argued and solved by consensus.

Data collection

Data of interest were extracted independently by three reviewers (SM, AB, and HM) using predefined electronic form (Excel and Microsoft Office). Any disagreement was argued with fourth reviewer (E-GH). Following data were extracted: general characteristics of study (first author name, year of publication, country, study design, and number of participants), participants' features (age, gender, and setting), exposure (method), and the adjusted covariates for computing OR.

Quality assessment

Two independent reviewers (AB and SM) evaluated the quality of each included articles by the Newcastle-Ottawa scale (the scale also adapted for cross-sectional studies) [42]. This scale rewards a maximum of ten stars to each definite study. The method of quality assessment was mentioned in our previous study [43]. The quality assessment results for each article are showed in Table 1.

Data synthesis and statistical analysis

All of studies which reported OR for risk of sarcopenia and sleep duration entered in the last analysis using a restricted cubic spline regression model. A dose-dependent meta-analysis performed according to method recommended by Greenland and Longnecker [44] and Orsini et al. [45] for estimating the trend from the correlated log OR across different sleep duration categories. The midpoint of the sleep duration category assumed as the equivalent OR estimation. When changing the reference category needed, ORs and CIs were recalculated as involving to the referent for which data were necessitated [46]. All open-ended categories were considered as same width as the neighboring categories [44, 47]. Possible nonlinear relationship between risk of sarcopenia and sleep duration checked by two-stage random-effects dose-response meta-analysis. This meta-analysis was performed by sleep duration modeling and restricted cubic splines with four knots at fixed percentiles of 5%, 35%, 65%, and 95% of the distribution [48]. It must be noted that the restricted cubic spline model estimated by generalized least square regression considers the correlation within each set of available ORs [45]. In this method, first estimated study exact slope lines were then merged with studies in which the slopes were stated in order to acquire an general middling slope [47]. Then, the dose-response association, assuming within- and between-study variances, was assessed using spline transformations [45]. Restricted maximum likelihood method in a multivariate random-effects meta-analysis [49] performed to combine

study-specific estimates. A probability value for nonlinearity was calculated using the null hypothesis checking in which the coefficient of the second spline was assumed equal to 0. In linear dose-response, the ORs related to 0.5 and 1 h increases in sleep duration for each study was calculated [47] using the generalized least squares trend estimation [44, 47, 50]. Linearity in potential association by study-specific results was merged by a random-effects dose-response [51]. All reported data were converted to unit of interest (increasing 0.5 and 1 h of sleep duration) by specific commands in Stata. All published studies which reported the risk as any unit increase were also entered for the final analysis. The Cochran's Q test and I^2 statistic were applied to estimate statistical heterogeneity between the included studies [52]. Furthermore, publication bias was investigated using Egger regression [53]. In order to assess the impact of excluding one single study on overall RR estimation, sensitivity analysis was performed. All statistical analyses were conducted using STATA software, version 14 (Stata Corp LP, College Station, TX).

Results

Characteristics of the studies

At last, 2298 studies, without duplicates, were found from the different databases. Initially, 2275 papers were excluded according to eligibility criteria. Eventually, 23 articles remained (Fig. 1), of which only 5 met the full inclusion criteria [37–41]. However, another study [40] was excluded because it used the same population as another study [41]. Only 4 studies used a cross-sectional design and involved 17551 individuals from which the data was pooled for the current meta-analysis. These studies were published between 2015 and 2017 and performed in the China [39], Korea [41], Netherlands [37], and Taiwan [38]. The included studies assess sarcopenia risk by Dual-energy X-ray absorptiometry [40, 41] and calculation [37–39]. Table 1 describes the features of the included studies.

Main results of meta-analysis

Analysis between highest and lowest versus reference categories of sleep duration and sarcopenia risk was performed. Pooled effect size from random effects model showed that lowest category of sleep duration (under 6 h) versus reference category (6–8 h) was significantly associated with increased risk of sarcopenia (Pooled OR: 1.71 95% CI, 1.11–2.64, $p = 0.015$, $P_{heterogeneity} = 0.009$, $I^2 = 67.5%$; Fig. 2). We performed subgroup analysis according to sex to find out possible sources of heterogeneity among studies. For the stratified analysis by gender, the pooled RRs of sarcopenia were 1.10 (95% CI, 0.85–1.42, $p = 0.45$; $I^2 = 60.5%$) in studies carried

Table 1 Description of the studies included in the meta-analysis

Author (year; location)	Study design	Population (M/F)	Age (years)	Sleep category (hours/d)	Sleep reference (hours/d)	Outcome assessment	Sarcopenia risk assessment	Adjusted variables	Quality Score
Chien et al. (2015; Taiwan) [38]	Cross-sectional	488 (224/264)	≥ 65	< 6 h6–8 h ≥ 8 h	6–8 h	Questionnaire	Janssen et al. 200, (SM (kg) = (0.401 × [height ² /resistance] + [3.825 × gender] – [0.071 × age]) + 5.102)	Age, gender, study cohort, comorbidity index, regular exercise, depression, and physical disability.	+10/10
Hu et al. (2017; China) [39]	Cross-sectional	607 (251/356)	60–90	< 6 h6–8 h ≥ 8 h	6–8 h	Questionnaire	Wen et al. 2011, (ASM = 0.193 × body weight (kg) + 0.107 × height (cm) – 4.157 gender – 0.037 × age (year) – 2.631)	Age, gender, BMI, education level, marital status, smoking status, alcohol drinking status, physical activities, self-reported sleep quality, use of hypnotics, nutrition status, cognitive status, depression	+10/10
Kwon et al. (2017; Korea) [41]	Cross-sectional	16,148 (7,158 /8,990)	≥ 20	≤ 5 h6 h 7 h8 h ≥ 9 h	7 h	Questionnaire	Dual-energy X-ray absorptiometry	Age, sex, body mass index, household income, current smoking, regular exercise, alcohol drinking, hypertension, employment status, and work schedule	+10/10
Lucassen et al. (2017; Netherlands) [37]	Cross-sectional	915 (403/512)	≥ 45	Continuous	-	Questionnaire	Kim et al. 2010, (RASM below 29.9% and 25.1% for men and women, respectively)	Age, whole body fat mass ethnicity, education, alcohol intake, physical activity, vitamin D levels and season, usage of systemic corticosteroids, bisphosphonates, menopause	+10/10
Kim et al. (2018; Korea) [40], (a subset of Kwon 2017)	Cross-sectional	3,532 (1,542/1,990)	≥ 40	≤ 5 h6–8 h ≥ 9 h	6–8 h	Questionnaire	Dual-energy X-ray absorptiometry	Age, current smoking status, alcohol drinking status, regular exercise status, and household income quartile	+10/10

M, male; F, female; ASM, skeletal muscle mass; h, hour; SM, muscle mass; RASM, relative appendicular skeletal muscle mass

out in men, compared with 1.39 (95% CI, 1.06–1.82, $p = 0.016$; $I^2 = 77.3\%$) in studies carried out in women; therefore, subgroup analysis revealed that relationship remained significant only in women (Fig. 3).

Pooled OR from random effect model also indicated that highest category (more than 8 h) of sleep duration versus reference category (6–8 h) was significantly related with increase in the risk of sarcopenia (Pooled OR: 1.52 95% CI, 1.23–1.88, $p < 0.001$, $P_{heterogeneity} = 0.199$, $I^2 = 31.5\%$; Fig. 4). Subgroup analysis was also performed based on sex. Subgroup analysis showed that highest category of sleep duration was significantly related with higher risk of sarcopenia compared with reference category in women (Pooled OR: 1.48, 95% CI, 1.08–2.03, $p = 0.013$ $I^2 = 66.7\%$) and men (Pooled OR: 1.55, 95% CI, 1.17–2.07, $p = 0.002$, $I^2 = 0.0\%$; Fig. 5).

Sensitivity analysis and publication bias

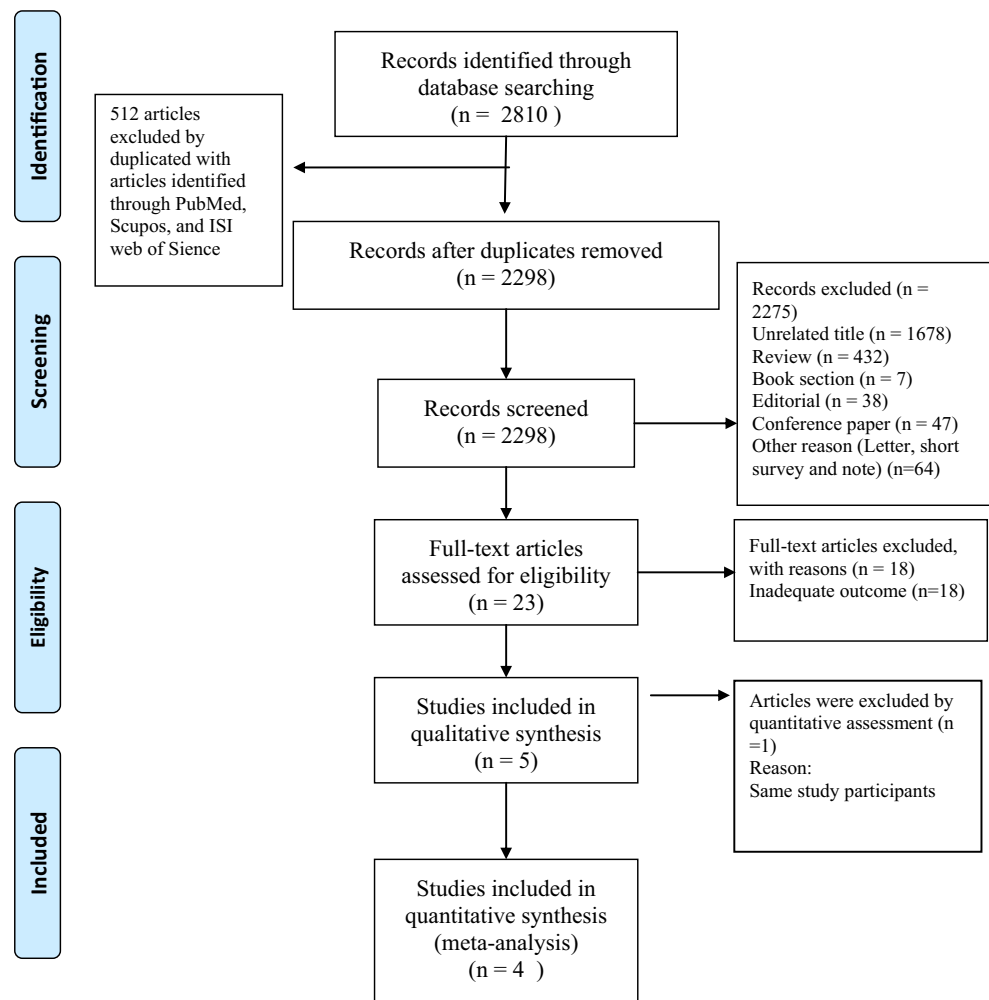
Sensitivity analysis also was conducted to evaluate the influence of each study using a pooled estimated effect size after

removing each individual study in turn. We found that a study by Kwon et al. could affect the pooled ORs when comparing lowest category versus reference category. When we removed that study, the pooled RRs changed to 3.14, (95 CI% 1.84, 5.34; $p < 0.0001$, $I^2 = 0.0\%$). Exclusion of each study did not impact the pooled OR. Analysis of highest category versus reference category showed no sensitivity analysis. In addition, there was no significant publication bias for highest (Egger test $p = 0.185$) and lowest (Egger test $p = 0.188$) analysis.

Dose-response association between sleep duration and risk of sarcopenia

After excluding studies included no data of interest, three studies were included in a nonlinear dose-response meta-analysis between sleep duration and risk of sarcopenia. Meta-analysis findings indicated a clear departure from linearity (P for nonlinearity < 0.001 , Fig. 6). The nonlinear dose-response meta-analysis revealed a U-shaped association between sleep duration and the risk of sarcopenia, with a nadir at 8 h per day. Sleep duration

Fig. 1 PRISMA flowchart describing the study’s systematic literature search and study selection



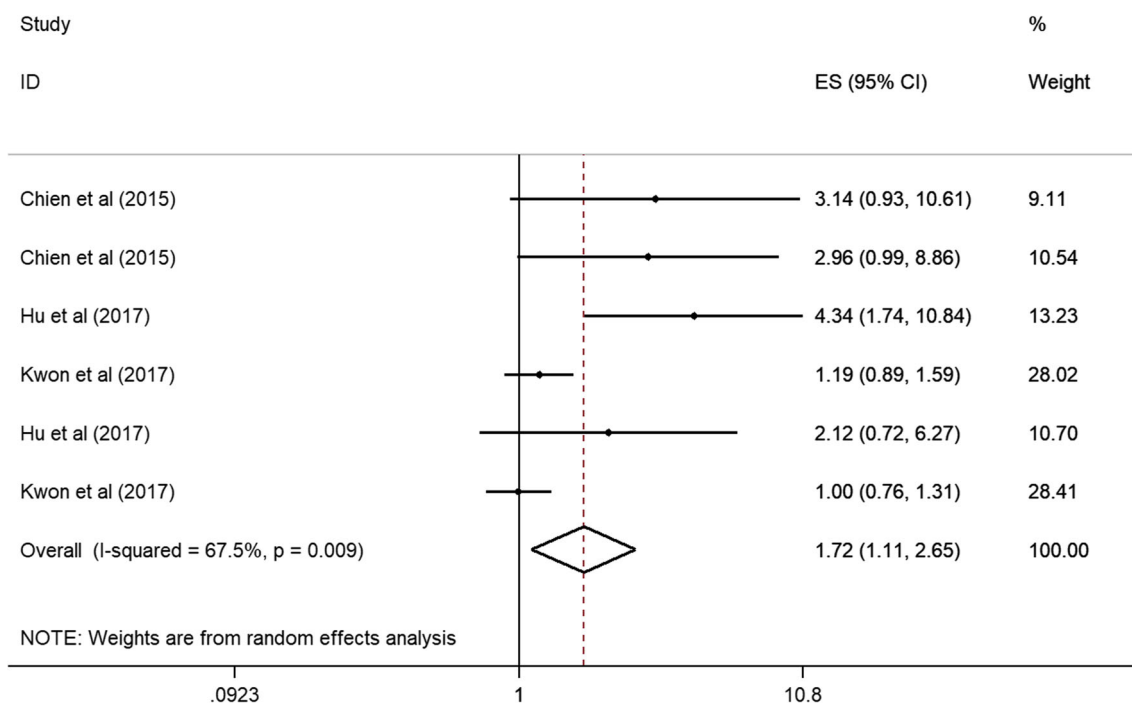


Fig. 2 Forest plots showing RR with 95% CI of pooled results from random effects model in lowest category of sleep duration (under 6 h) versus reference category (6–8 h)

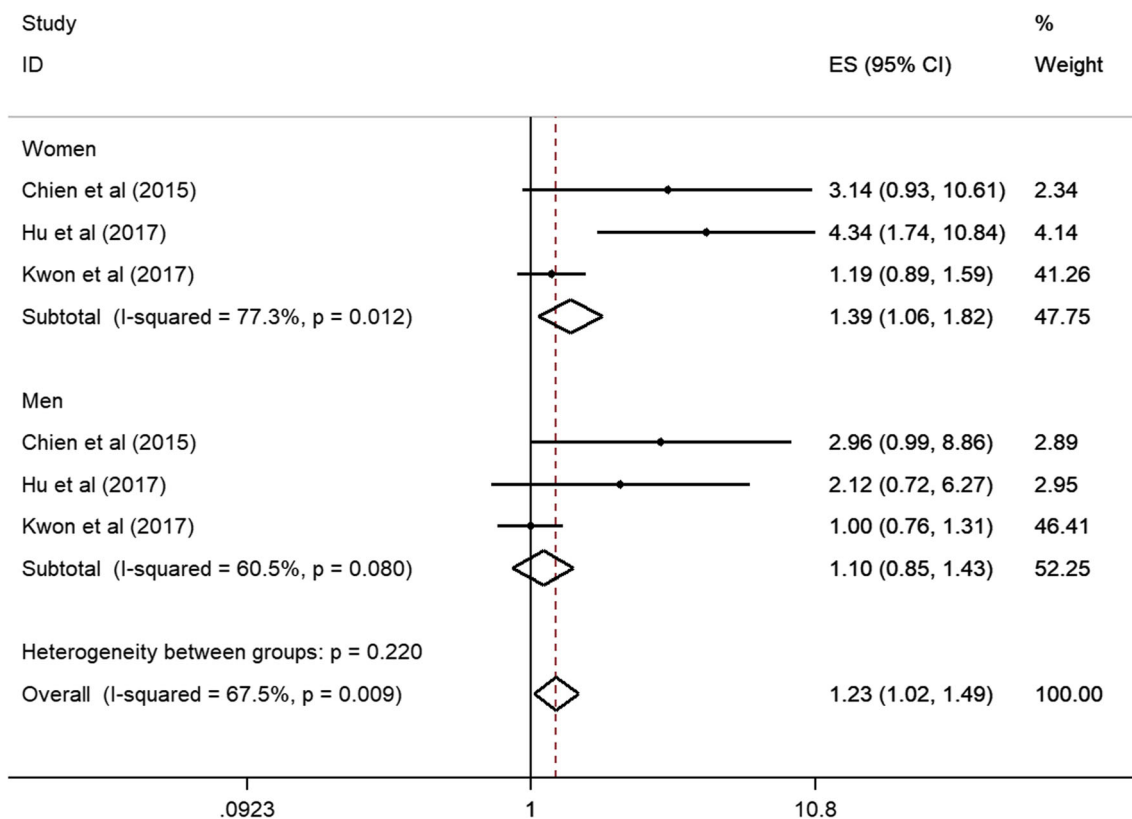


Fig. 3 Forest plots showing RR with 95% CI of pooled results from subgroup analysis based on sex in lowest category of sleep duration (under 6 h) versus reference category (6–8 h)

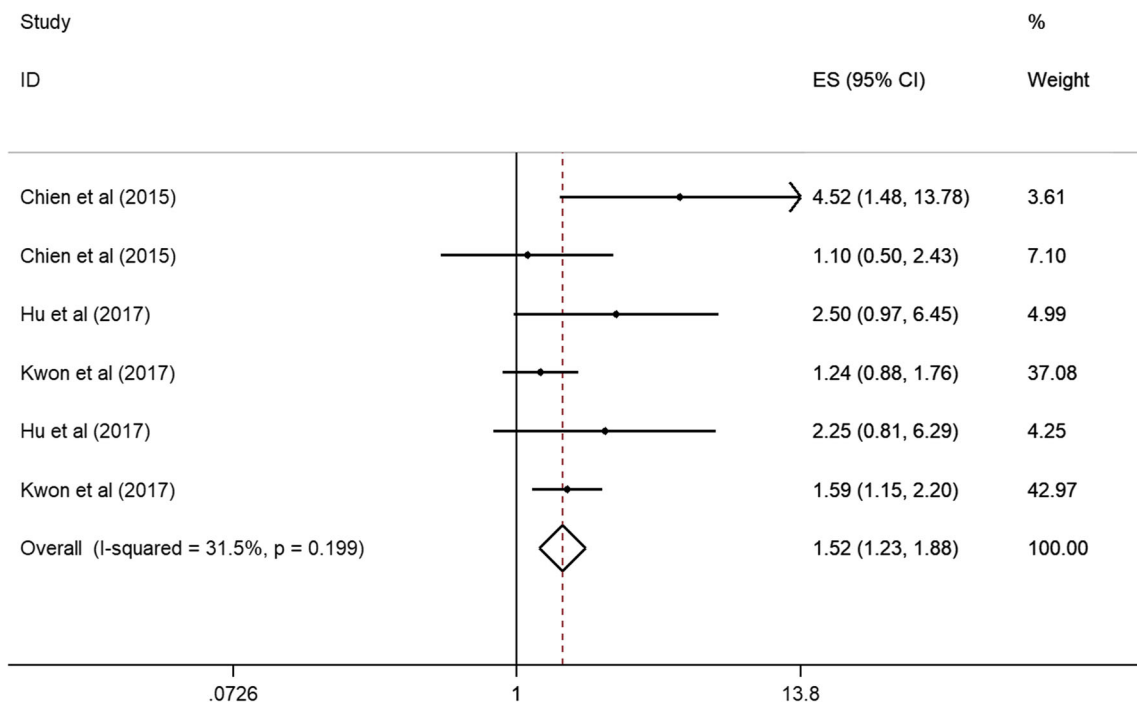


Fig. 4 Forest plots showing RR with 95% CI of pooled results from random effects model in highest category of sleep duration (upper 8 h) versus reference category (6–8 h)

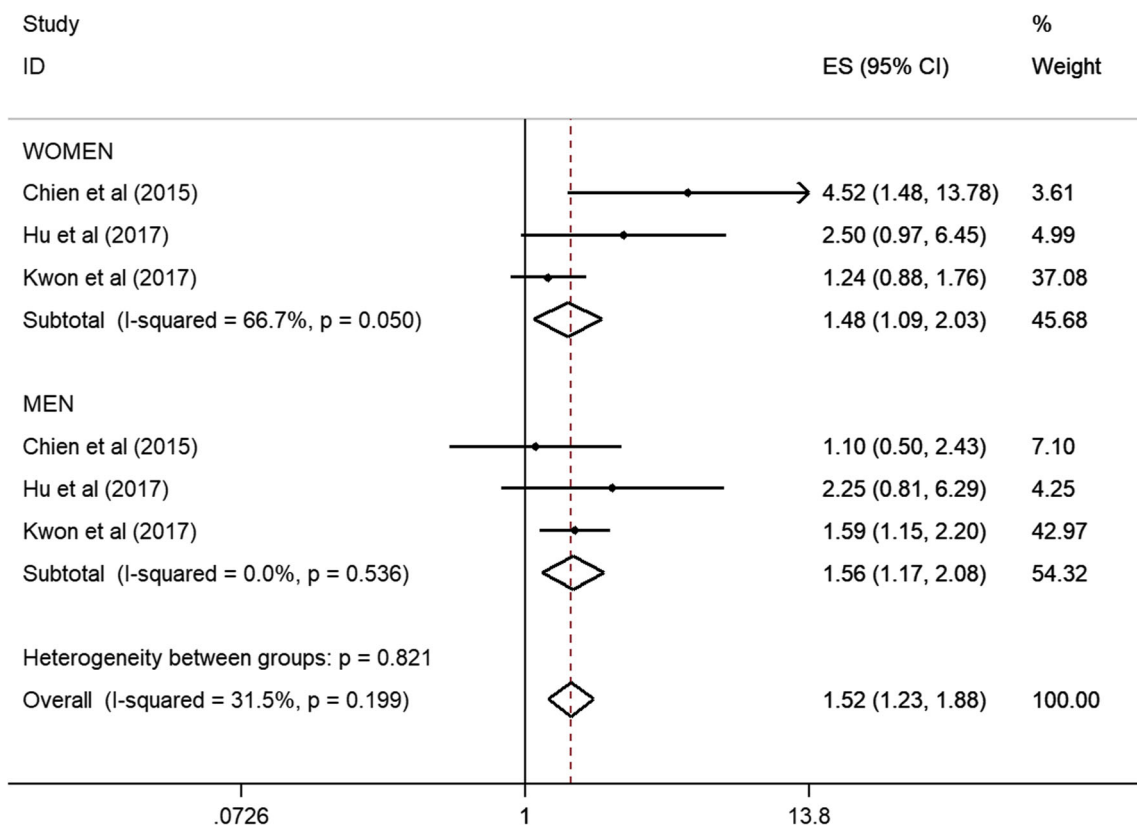


Fig. 5 Forest plots showing RR with 95% CI of pooled results from subgroup analysis based on sex in highest category of sleep duration (upper 8 h) versus reference category (6–8 h)

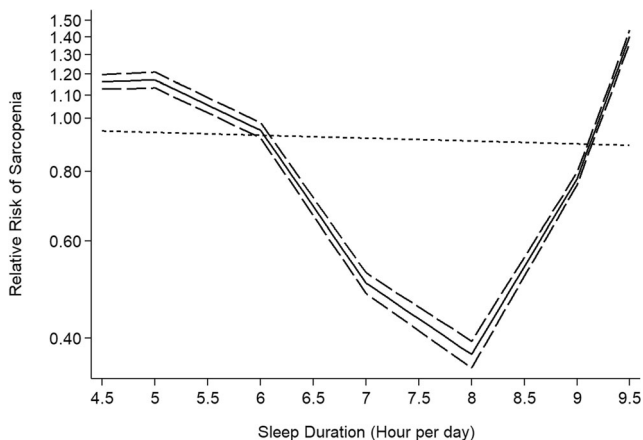


Fig. 6 Dose-response relationship between sleep duration (hour-horizontal axis) and relative risk of sarcopenia (RR-vertical axis); findings from 4 individual studies with 17 arms. The solid line represents point estimates of the association between sleep duration and sarcopenia risk, and the dotted lines are 95% CIs. The horizontal line is the reference line

increasing up to 8 h per day had a protective effect on the risk of sarcopenia, but sleep duration of more than 8 h per day leads to increase in the risk of sarcopenia risk with a steep slope (coefficient 6.07 compared with coefficient -1.86 for reducing slope).

Linear association

Four studies reporting the relationship between sleep duration and risk of sarcopenia were entered into the linear analysis.

The linear dose-response meta-analysis suggested that the risk of sarcopenia did not change significantly either for a 0.5-h (*Pooled OR: 0.994, 95% CI, 0.965, 1.024, P = 0.678; Fig. 7*) or 1-h increment (*Pooled OR: 0.987, 95% CI, 0.932, 1.045, P = 0.648; Fig. 8*) in sleep duration per day in the random effects model. This nonsignificant relationship highlighted that the association between sleep duration and risk of sarcopenia does not follow a linear association. However, a high heterogeneity was detected among studies in linear relationship ($I^2 = 99.7\%$, $P_{heterogeneity} < 0.001$) (Fig. 7).

Discussion

Sarcopenia is linked to the poor health-associated quality of life and a number of adverse social and occupational impacts. As such, it is a condition with large costs for both the society and individual [54]. Short nocturnal sleep, as a kind of insomnia disorder, or long sleep is clearly associated with strength, function, and losses of muscle mass, suggesting that normalizing duration of sleep might have protective effects [37–39]. Understanding the adverse effects of insufficient sleep is important for public health strategies to prevent sarcopenia. In this study, we describe a comprehensive literature review in which we assess the relationships between sleep duration and sarcopenia risk. Based on our knowledge, there is not any same study in this field. Together, our current review will help to highlight what is known about sarcopenia management related

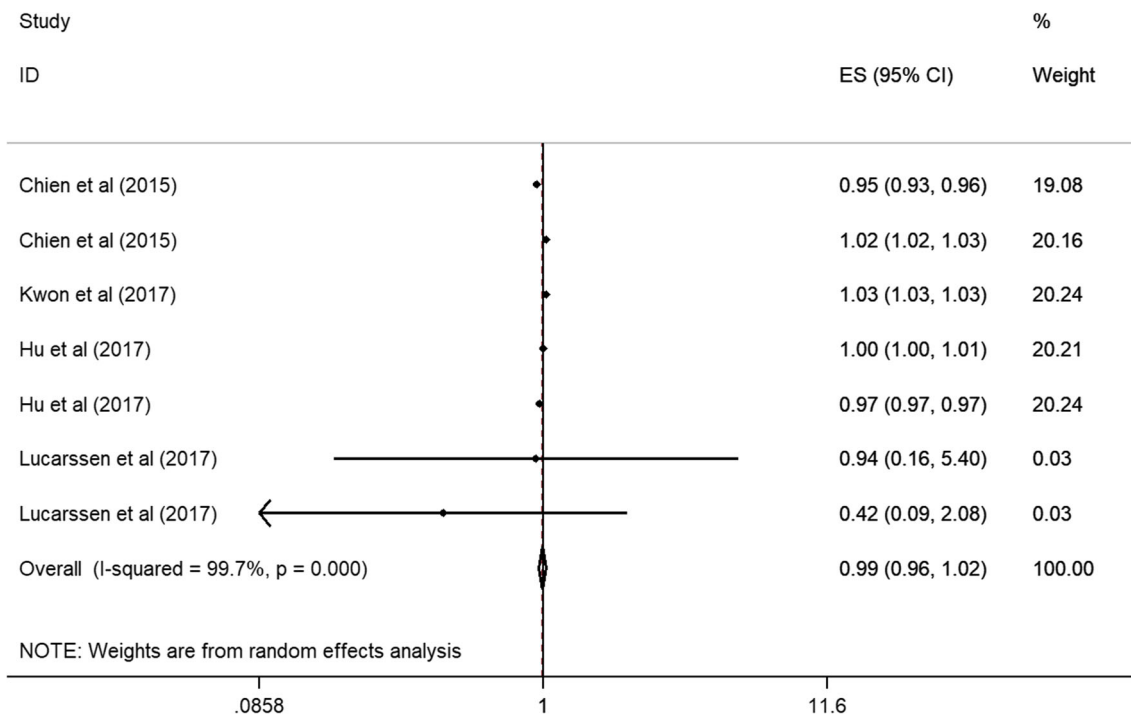


Fig. 7 Forest plots showing the linear dose-response meta-analysis of sarcopenia risk for 0.5-h change in sleep duration

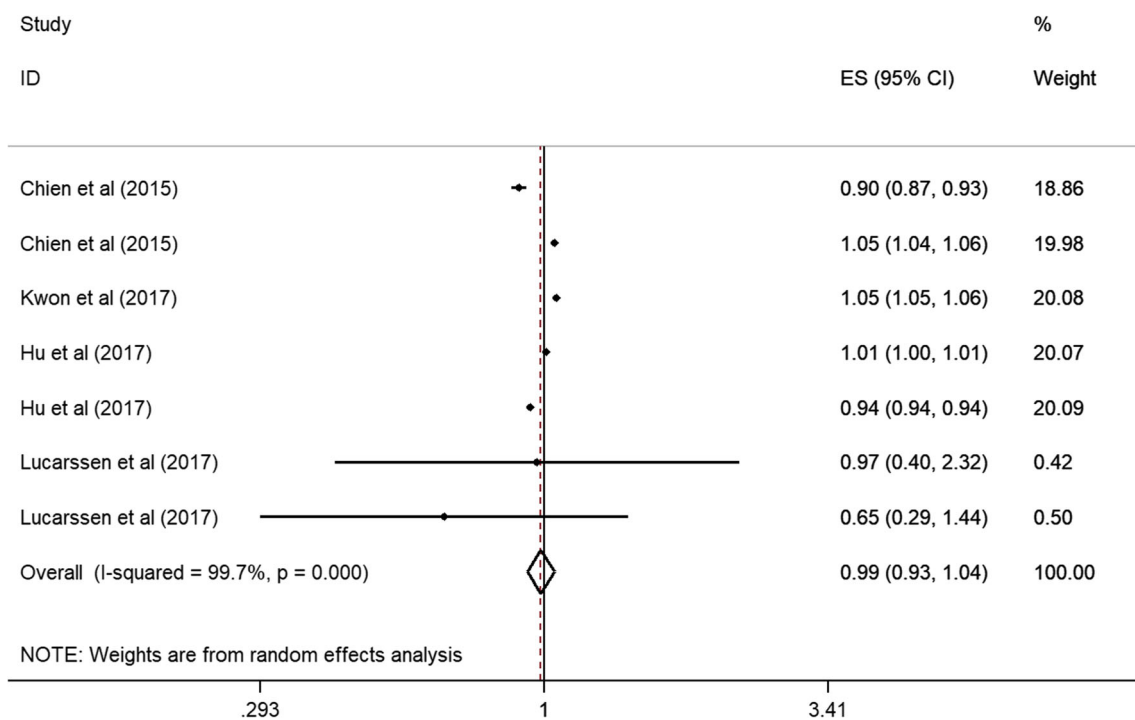


Fig. 8 Forest plots showing the linear dose-response meta-analysis of sarcopenia risk for 1-h change in sleep duration

to sleep duration and what more research is required to provide clinicians to manage this disorder.

Our meta-analysis of four studies demonstrated that short sleep duration was related with an increased risk of sarcopenia. Subgroup analysis revealed that women were affected by both less or higher than 6- to 8-h sleep duration while men were only affected by long sleep duration. Dose-response association analysis illustrated sleep duration increasing up to 8 h per day had a protective effect on sarcopenia risk, but sleep duration of higher than 8 h per day can increase the risk. We did find a linear association between sleep duration and sarcopenia risk.

Our results were in line with the previous study which supports the opinion of an association between sleep and muscle mass [55]. These results are also consistent with a cross-sectional study of 990 adolescents that showed sleep duration just in boys was associated positively with skeletal muscle index [56]. Shorter sleep duration has also been related with poorer muscle strength in males but not females [57]. A recent review in which data was pooled found inadequate sleep impairs maximal muscle strength in resistance exercise performance [58].

Interestingly, some related studies are conflicting regarding sex differences. This discrepancy maybe because of different daily sleep duration and/or requirements between sex due to their different hormone levels [56]. Sex hormones also regulate muscle mass and function and differ between sexes [59]. Females need higher sleep deprivation to be affected than males. Indeed, they are more resilient to environmental stressors [56]. Overall, metabolic differences among females

and males may have an effect on the sarcopenic phenotype and the onset of physical and metabolic dysfunction [60]. Conflicting factors, however, that influence sarcopenia include genetic factors [61] age, exercise, diet, cytokines, and endocrine-related changes [62].

Linear analysis further assessed the possible relationship between sleep duration and sarcopenia risk and compared 0.5 and 1 h [37–40] and confirmed our findings. In summary, increasing one unit (in our analysis 0.5 or 1 h of sleep duration) could not explain the link between sleep duration and risk of sarcopenia because, as we demonstrated, it has a U-shaped relationship and is not linear.

The impact of sleep duration on sarcopenia has been attributed to different mechanisms. The restorative theory indicates that sleep is critical for body repair as well as the rejuvenation processes. During sleep, many body functions for growth occur such as protein synthesis, tissue growth, muscle repair, and hormone release [63]. Intracellularly, reduced duration and quality of sleep potentially interfere by inhibiting protein synthesis pathways and inducing degradation in the skeletal muscles' pathways. Several anabolic and catabolic hormone cascades are mediated by growth hormone, IGF-1, cortisol, testosterone, and insulin, which have many functions on the cellular and molecular pathways to enhancement or re-establish muscle fiber, function, and strength [64, 65]. In addition, chronic insomnia might dysregulate the hypothalamo-pituitary-adrenal axis resulting in endocrine changes [66]. Sleep problems and increased prevalence of circadian rhythm disorders also induce proteolysis, change body composition,

and favor the risk of insulin resistance resulting in the sarcopenia [64, 65]. So, changes in sleep duration alter metabolic function, resulting in altered muscles mass, function, and metabolism.

Current meta-analysis had several limitations such as small number of included studies. The effects of confounding factors, namely, age, physical activity, diet, and genotype of individuals, remain unclear, and the outcomes should be interpreted with caution. Notwithstanding mentioned limitations, the strength of present study was the comprehensive dose-response, linear, and subgroup analysis which was according to PRISMA guidelines.

Implications for practice

The outcomes from current study indicate that the public should be made aware of the negative consequences of long and short sleep for sarcopenia.

Implications for research

Despite a good rationale for the relationship between sleep duration and sarcopenia, large-scale studies are required to be conducted with adequate follow-up to decrease heterogeneity and demonstrate the exact effect of sufficient sleep on sarcopenia. In addition, the confounding factors such as genetic background, dietary habits, age, and physical activity on this association should be assessed. Agreement on criteria to diagnose and classify sarcopenia also might provide a basis for studies to improve the assessment of association between sleep duration and this condition. Widely approved criteria for the diagnosis and staging of sarcopenia should be used in these patients in further studies.

Conclusion

The relationship between sleep duration and sarcopenia is well determined in the current meta-analysis although gender may effect this association. Women were affected by both less or higher than 6- to 8-h sleep duration, while men were only affected by long sleep duration.

Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflict of interest.

Informed consent Informed consent was obtained from all individual participants included in the study.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

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