Association between Objectively Sleep Pattern and Obesity in the Elderly

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Abstract

Background: Previous studies on the relationship between sleep patterns and obesity in the elderly are limited and have conflicting results. Moreover, few studies have measured sleep patterns objectively. In this study, we investigated objective sleep patterns and their relationship with obesity in the elderly in Tehran, Iran.

Methods: In this cross-sectional study, 88 elderly (≥60 years old) who were members of health homes of zone 5 in Tehran, Iran, were included by simple random sampling method in 2014. Sleep patterns were objectively assessed using actigraphy for a mean of 43 ± 1.7 days. Height, weight, and waist circumference (WC) were measured by standard methods and body mass index (BMI) was calculated. Data entry and statistical analyses were performed using SPSS version 21.

Results: Mean actigraphy-assessed sleep duration, sleep efficiency (percentage of time in bed spent sleeping), and sleep latency (time required to fall asleep) were 427 ± 62 min, 71.3 ± 18%, and 14.2 ± 3.8 min, respectively. A negative relationship was found between BMI and sleep duration (r = −0.2, p = 0.03), BMI and sleep efficiency (r = −0.3, p=0.01), and WC and sleep efficiency (r = −0.2, p = 0.04). Also, a positive association was observed between BMI and sleep latency (r = 0.4, p = 0.006).

Conclusions: In the elderly, actigraphy-assessed sleep duration was associated with obesity and the sleep efficiency was poor in obese participants. It seems that sleep patterns and BMI are correlated with each other. However, there is a need for prospective studies to affirm causal relationships between these constructs.

Keywords: Sleep, Obesity, Aged, Actigraphy.

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Introduction

Aging is associated with many physiological changes, including changes associated with increased adiposity and obesity.1,2 Additionally, the results of some cross-sectional studies have shown that changes in circadian rhythms, as well as the use of multi-drug therapy, can cause sleep disorders during aging.3,4 It has been reported that more than 50% of the elderly have trouble falling asleep or staying asleep.5 In the elderly, insomnia is more often related to increasingly frequent awakening during sleep rather than longer durations of awakening.6 Adequate length and quality of sleep is important for normal daily metabolic and hormonal functioning, as well as for appetite regulation.7 Previous studies have shown that the amount and quality of sleep is associated with obesity.8-11 Kim et al. showed that poor sleep quality is an independent risk factor for obesity.12 Knuston et al. mentioned that sleep deprivation is associated with increased risk of obesity, impaired glucose metabolism, and risk of type 2 diabetes.13 Although some studies have shown negative linear associations between sleep time and body mass index (BMI),14-16 others have proposed that sleep time and BMI have a U-shaped relationship.17,18,19 While it seems that objective measurements have shown sleep patterns to be associated with obesity in the elderly,20-24 some studies have failed to show this correlation.25

According to our review of the literature, only a limited number of studies have assessed the relationship between sleep patterns and obesity in the elderly, as most of the previous studies have instead focused on children or adolescents.26-28 Also, most epidemiological studies have assessed sleep duration by self-reporting methods that show high rates of false estimation. However, the American Sleep Disorders Association has presented the actigraphy method as a more reliable way for assessing sleep-wake patterns, sleep efficiency (SE), and sleep onset latency in adults based on the person’s movement.19-21

The present study sought to objectively evaluate sleep patterns using actigraphy and to investigate the relationship between sleep patterns and obesity in the elderly.

Materials and Methods

This cross-sectional study involved elderly participants who were residents of health homes located in zone 5 of Tehran, Iran. A total of 90 elderly (≥60 years old) participants were selected by simple random sampling based on the proportional populations of each facility. Participants recruitment started in February 2014 and was completed in August 2014. After obtaining informed consent from all the participants, they were enrolled in the study. Individuals with any health condition that made them unable to use the Actiwatch devices, such as physical or mental disorders, mental retardation, Alzheimer’s disease, Cognitive impairment, or the use of sedative drugs, and people unwilling to participate in the study were excluded.

Anthropometric parameters for each participant were measured by the same researcher between 8–10 am.
Participants were measured without shoes and wearing minimal clothing. Body weight was measured using a digital scale with an accuracy of 100 g. Height was measured in a relaxed position and freely hanging arms using a wall-mounted height chart with an accuracy of 0.5 cm. Waist circumference (WC) was measured using a non-elastic tape from the middle of the last rib to the iliac crest. BMI was calculated by dividing weight (in kg) by height (in m) squared. Obesity was defined as a BMI of 30 kg/m² or higher.

Sleep patterns were objectively evaluated using the GT3X Actiwatch device (Pensacola, Florida, USA). The validity and reliability of this method has been demonstrated in previous studies. The Actiwatch is a small device worn on the wrist or waist. Due to the possible hand vibrations in the elderly, we asked participants to wear the device on their waist. The accelerometer sensors in the device measured the number of bodily accelerations based on the person’s movement for each minute and information can be stored for a long time. Participants were asked to wear the Actiwatch continuously over a period of 3 to 7 consecutive nights. During the actigraphy period, participants were asked to record information about what time they went to bed, what time they woke up in the morning, and what time they removed the Actiwatch. This information was used to ensure the accuracy of the stored data. The stored data on each Actiwatch was then transferred to the computer and the row data was analyzed using ActiLife software, version 4.4.0 (Pensacola, Florida, USA). Collected data from participants with less than two nights were excluded from analysis. Nocturnal sleep duration was categorized into groups of <5 h, 5 h to 8 h, and >8 h.

The actigraphy data was transferred to Excel 2013 from the ActiLife software. The Kolmogorov-Smirnov test was used to examine the normal distribution of data. Continuous data were expressed as means and standard deviations, and categorical variables were presented as numbers and percentages. Independent sample t-tests were used to compare continuous parametric data. The Pearson correlation coefficient test was used to examine the relationship between the normal variables of sleep pattern and obesity. Participants were categorized into two groups based on BMI: obese (BMI ≥30 kg/m²) and non-obese (BMI <30 kg/m²). Actigraphically measured nocturnal sleep duration was divided into three categories. The Chi-square test was used to compare categorical data between the obese and non-obese groups. Analyses of variance (ANOVA) followed by Tukey post-hoc tests were conducted to examine between-group differences for BMI and WC values. The alpha value was set at 0.05 to indicate statistical significance for all comparisons. All data analyses were conducted using SPSS (version 21; SPSS Inc., Chicago, Illinois, USA).

**Results**

In total, 88 participants (56 women and 32 men) completed the study, and the final data for 2 participants were excluded because of gaps in their sleep duration data. The mean age of the participants was 71.3 ± 2.6 years (with a range of 60–93 years). The mean BMI and WC were 28.6 ± 4.0 kg/m² and 95.5 ± 8.5 cm, respectively. A total of 25 participants (28.4%) had a BMI equal to or higher than 30 kg/m² and were classified as obese. The characteristics of the participants are presented in Table 1.

For the 59 participants (67%), the study period included 3 continuous nights, and all other participants had data for 7 nights. The mean time of actigraphy recording was 4.3 ± 1.7 nights.

There were no significant differences between men and women in their actigraphy data, except for sleep latency, which was higher in women (P = 0.04). Table 2 shows the correlation between anthropometric measurements and sleep parameters. WC was not associated with sleep duration or sleep latency in the studied population. A significant negative correlation was observed between sleep duration and BMI (r = 0.03), between sleep efficiency and BMI (P = 0.01), and between sleep efficiency and WC (P = 0.04). Also, there was a significant positive correlation between sleep latency and BMI (r = 0.006). Table 3 shows the relationship between the categories of actigraphic sleep duration and obesity. We found that short sleepers (<5 h), were significantly more obese than longer sleepers (P = 0.002). The percentage of people who slept 5–8 hours per night in the obese group was higher than for non-obese participants, but the observed difference was not significant. Obese participants had lower sleep efficiency (P = 0.04) and greater sleep latency than non-obese participants.

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**Table 1. Characteristics of the study population by gender, N = 88**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Men mean±SD (N=23)</th>
<th>Women mean±SD (N=56)</th>
<th>Total mean±SD (N=88)</th>
<th>P.V*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>70.5±3.3</td>
<td>72.0±1.9</td>
<td>71.3±2.6</td>
<td>0.2*</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.2±3.8</td>
<td>29.1±4.1</td>
<td>28.6±4.0</td>
<td>0.5*</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>95.3±7.5</td>
<td>96.3±11.5</td>
<td>95.5±8.5</td>
<td>0.2*</td>
</tr>
<tr>
<td>Actigraphic sleep measure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep duration (min)</td>
<td>43±17</td>
<td>42±71</td>
<td>42±16</td>
<td>0.4*</td>
</tr>
<tr>
<td>SE (%)</td>
<td>73.2±19.3</td>
<td>69.4±11.4</td>
<td>71.3±18</td>
<td>0.09*</td>
</tr>
<tr>
<td>SL (min)</td>
<td>11.2±4.6</td>
<td>17.3±6.1</td>
<td>14.2±3.8</td>
<td>0.04*</td>
</tr>
</tbody>
</table>

*Independent t-Test

Abbreviations: SD, standard deviation; BMI, body mass index; WC, waist circumference; min, minutes; SE, sleep efficiency; SL, sleep latency.
secretion by affecting the hypothalamic–pituitary–adrenal axis that has a secondary role in increasing the accumulation of abdominal fat and systemic obesity. Inversely, obesity can lead to respiratory problems and obstructive sleep apnea, which are associated with shorter sleep duration and poorer sleep quality in obese individuals. The relationship between sleep duration and obesity may therefore be bidirectional. Bradley et al. has suggested that sleep problems in patients with obstructive sleep apnea may be due to an overactive sympathetic nervous system, which in turn stimulates the beta receptors in adipose cells, prevents leptin production, and subsequently alters appetite.

The mechanisms linking longer sleep duration to obesity has not been studied as thoroughly, but it is likely that the mechanisms involved such a relationship would differ from those involved in the relationship between shorter sleep duration and obesity.

Few past studies have measured SE. Among the participants in our study, SE was inversely associated with BMI and WC. Moreover, obese participants had lower SE. A number of studies support our findings. Among those involved in the relationship between shorter sleep duration and obesity, it is likely that the relationship between SE and obesity, like that of sleep duration and obesity, is bidirectional. Polysomnographic findings have indicated that obese people, even in the absence of sleep apnea, have lower SE and higher SL. Similar results have been seen in a study by Vgontzas. One explanation for this may be that obese people have circadian abnormalities related to endocrine and metabolic processes that contribute to sleep disorders. In addition, laboratory studies have found that plasma levels of cytokines are elevated in obese participants, which might be responsible for sleep disturbances.

Our study has some key strengths, including the use of an objective assessment of sleep patterns using actigraphy, and a focus on an older population that has been neglected in most previous studies. The main limitation of our study was the rather small sample size which did not allow us to divide the sample into more numerous groups based on sleep duration. Because of the cross-sectional design, we were unable to investigate causality. Also, our data did not account for participant dietary habits and physical activity that may mediate the relationship between sleep pattern and obesity. In addition, while many studies agree on the validity of actigraphy in normal participants, some recent studies have raised new concerns about it. Bland and Altman showed that actigraphy overestimated total sleep time, sleep latency, and sleep efficiency while underestimating the number of awakenings.

One explanation for their findings could be that, with actigraphy, lack of motion is interpreted as sleep, so errors will occur when participants wake up but remain motionless.

The present study found a link between obesity and both the amount and quality of sleep. Because sleep disorders in the

Table 2. Association between obesity indices and sleep characteristics, N= 88

<table>
<thead>
<tr>
<th>Sleep parameters</th>
<th>Obesity indices</th>
<th>t’</th>
<th>P.V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep duration (min)</td>
<td>BMI (kg/m²)</td>
<td>-0.2</td>
<td>0.03</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>-0.1</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td>SE (%)</td>
<td>-0.3</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>WC (cm)</td>
<td>-0.2</td>
<td>0.04</td>
<td></td>
</tr>
<tr>
<td>SL (min)</td>
<td>-0.4</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td>WC (cm)</td>
<td>0.02</td>
<td>0.3</td>
<td></td>
</tr>
</tbody>
</table>

*Pearson correlation
Abbreviations: BMI, body mass index; WC, waist circumference; cm, centimeters; min, minutes; SE, sleep efficiency; SL, sleep latency.

Table 3. Sleep pattern of the participants by BMI, N= 88

<table>
<thead>
<tr>
<th>Sleep parameters</th>
<th>Obese (BMI≥30)</th>
<th>Nonobese (BMI&lt;30)</th>
<th>Total</th>
<th>P.V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actigraphic sleep duration</td>
<td>≤5 h, n (%)</td>
<td>8 (32)</td>
<td>26 (41.2)</td>
<td>34 (38.6)</td>
</tr>
<tr>
<td>5-8 h, n (%)</td>
<td>7 (28)</td>
<td>15 (23.8)</td>
<td>22 (25)</td>
<td>0.09</td>
</tr>
<tr>
<td>28 h, n (%)</td>
<td>10 (40)</td>
<td>22 (35)</td>
<td>32 (36.3)</td>
<td>0.06’</td>
</tr>
<tr>
<td>SE (%), means SD</td>
<td>63.2 ±16.1</td>
<td>77.1 ±22.1</td>
<td>71.3 ±18</td>
<td>0.04’</td>
</tr>
<tr>
<td>SL (min), means SD</td>
<td>16.6 ± 9.1</td>
<td>11.3 ± 7.4</td>
<td>14.2 ± 8.8</td>
<td>0.08’</td>
</tr>
</tbody>
</table>

*Chi Square Test,*’Independent t-Test
Abbreviations: SE, sleep efficiency; SL, sleep latency.

Discussion

In the present study, reduced sleep duration was associated with greater BMI. An association between reduced sleep duration and obesity has been supported by previous studies on younger participants. Although some studies did not find evidence for an association between sleep duration and obesity, studies conducted by van den Berg et al. and Patel et al. among elderly participants using actigraphy showed a negative relationship between sleep duration and obesity, which supports our findings. In the study by VenDen Berg et al., long sleepers (>8 hrs) had higher BMI and we found a marginally significant correlation between these variables. The same relationship has been reported by other studies. In contrast to our findings, a number of studies have shown a U-shaped relationship between sleep duration and obesity, indicating that longer and shorter sleep durations alike are associated with higher BMI. Magee et al., in a self-report sleep duration study, found that shorter and longer sleep durations were associated with an increased risk of obesity in middle-aged adults (55–64 years old), but not in older adults. Similarly, Stranges et al. found an association between sleep duration and BMI in a cross-sectional study.

There are several possible explanations for the relationship between shorter sleep duration and obesity. Sleep deprivation can cause changes in hormone levels, such as decreases in leptin and increases in ghrelin, as well as altering glucose metabolism. Leptin has an appetite reducing effects while ghrelin can increases appetite and any changes in serum levels of mentioned hormones can alter human appetite and facilitate the weight gain and obesity over time. Moreover, it seems that people with short sleep durations have greater opportunities to consume more calories, which could result in obesity. In addition, short sleep can elevate cortisol secretion by affecting the hypothalamic–pituitary–adrenal axis that has a secondary role in increasing the accumulation of abdominal fat and systemic obesity. Inversely, obesity can lead to respiratory problems and obstructive sleep apnea, which are associated with shorter sleep duration and poorer sleep quality in obese individuals. The relationship between sleep duration and obesity may therefore be bidirectional. Bradley et al. has suggested that sleep problems in patients with obstructive sleep apnea may be due to an overactive sympathetic nervous system, which in turn stimulates the beta receptors in adipose cells, prevents leptin production, and subsequently alters appetite.

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The present study found a link between obesity and both the amount and quality of sleep. Because sleep disorders in the
elderly are a common phenomenon and researchers agree about the adverse effects of obesity, future studies should focus on larger elderly populations to assess the relationship between sleep patterns and obesity prospectively and to determine the extent to which sleep duration can alter metabolic processes, or what is the normal weight for prevention of sleep disorders.

Acknowledgement

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Conflict of Interest

The authors declared that they have no conflict of interest.

References

6. Dijk DJ, Duffy JF, Rechtschaffen A. Age-related increase in awakenings: impaired consolidation of nonREM sleep at all circadian phases. Sleep 2001;24:565-77.
44. Kim S, DeRoo LA, Sandler DP. Eating patterns and nutritional characteristics associated with sleep duration. Public Health Nutr 2011;14:889-95. doi:10.1017/S1368946511000296X.