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Original Article

Associations of meal timing and sleep duration with incidence of obesity: a prospective cohort study

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ABSTRACT

Objectives: Late mealtime and short sleep are known to be associated with obesity risk due to a misaligned circadian rhythm. This study aimed to investigate the relationship between obesity and mealtime and sleep duration using the Korean Genome and Epidemiology Study (KoGES) data.
Design: Longitudinally prospective cohort study.
Setting: Population-based.
Participants: KoGES analysed data from 9,474 Korean adults with an average age of 54- years old at baseline.
Measurements: Meal timing was defined as the eating occasions of the day reported by the participant eating a 24-h dietary recall method. Sleep duration was categorized as <6, 6–7, 7–8, and ≥8 h. The Cox proportional hazard model was used to calculate hazard ratios (HRs) and 95% confidence intervals (CIs) for incident obesity according to meal timing, sleep duration, and nightly fasting duration.
Results: During a mean follow-up of 3.5 years, 826 participants developed obesity. In the multivariable-adjusted analysis, midnight snack eating (HR, 1.20; 95% CI, 1.02–1.41) and higher energy intake from midnight snacks (HR, 1.26; 95% CI, 1.06–1.49) were associated with a higher risk of obesity. Sleeping 8 h or more (HR, 0.67; 95% CI, 0.53–0.85) was associated with a lower risk of obesity.
Conclusions: Our findings highlight the importance of meal and sleep times and suggest that healthy eating habits related to the time of day.

1. Introduction

Obesity is a significant public health challenge in an increasing trend due to diet changes, decreased physical activity, and environmental changes. In Korea, the prevalence of obesity has been increasing in all age groups for the past 11 years, from 2009 to 2019 [1]. Moreover, obesity is related to higher rates of comorbidities, such as hypertension, type 2 diabetes mellitus, obstructive sleep apnea, fatty liver, and cancers [2]. Obesity is also thought to be a strong risk factor elevating the risk of disability and poor overall health among older adults, which are projected to be 21 billion worldwide by 2050 [3,4]. Thus, combating obesity through effective prevention and treatment strategies emerges as a paramount concern [5].

While diet and lifestyle modifications have long been recognized as cornerstone strategies for the management and prevention of obesity [6], emerging research over the past two decades has illuminated the role of additional factors such as inadequate sleep and circadian rhythm disruptions in the obesity epidemic [7,8]. The rapid adoption of Western dietary habits, combined with an increase in sedentary lifestyles and sleep deprivation, has notably contributed to the soaring obesity rates in Korea, spotlighting the urgent need for adaptive strategies in response to these lifestyle shifts [9,10].

The circadian rhythm, governing a myriad of biological processes including the sleep-wake cycle and metabolic regulation, plays a crucial role in maintaining health. Disruptions to this rhythm have been linked to metabolic diseases, underscoring the importance of aligning daily habits with our biological clocks to mitigate obesity risk [11–13]. Eating and sleeping are among certain repetitive human behaviors intrinsically linked to the daily 24-h rhythms of the neuroendocrine system [14]. Modern societal factors such as extended work hours, night shift work, and the pervasive use of electronic devices have facilitated late-night eating and sleep deprivation, thereby exacerbating circadian misalignment and its associated health risks [15,16].

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Significant research has pointed to the adverse effects of behaviors such as skipping breakfast, late-night eating, and inadequate sleep on both circadian rhythm integrity and obesity outcomes, as evidenced by both animal and epidemiological studies [17–28]. However, these investigations have predominantly explored sleep patterns and eating behaviors in isolation, without examining the interplay between the two, and have often relied on cross-sectional study designs.

In light of this gap, our study aims to delve into the intricate relationship between eating patterns, sleep duration, and obesity, leveraging data from the Korean Genome and Epidemiology Study (KoGES). By examining meal timing, energy intake distribution, and their joint associations with sleep duration, this research seeks to contribute valuable insights into the complex mechanisms underpinning obesity, thereby informing more targeted and effective public health interventions.

2. Methods

2.1. Study participants

This study used data from the Health Examinee (HEXA) study of the Korean Genome and Epidemiology Study (KoGES), which is a large-scale prospective study that is conducted by the Korea National Institute of Health (KNIH) [29]. In brief, the HEXA recruited and follow-up participants at 38 health examination centers and training hospitals located in the 8 regions of Korea. Data for each participant (socio-demographic characteristics, past medical history, family history, and dietary behaviors) were obtained by interviewing participants using structured questionnaires [29]. Anthropometric measures and blood tests were performed by trained staff and interviewers using standardized procedures and protocols.

For this analysis, anonymized data from 33,533 participants who completed the 24-h dietary recall survey between 2011 and 2013 were retrieved from the HEXA. Participants with the following conditions were excluded from the analyses: (1) missing anthropometric or biochemical measurements (n = 267), (2) implausible energy intake (<500 kcal/day or >5,000 kcal/day) (n = 262), (3) who did not attend the follow-up survey (n = 19,324), (4) those with obesity at baseline (n = 4,206). Thus, our analyses included 9,474 Korean adults (2,460 men and 7,014 women) (Fig. 1). Among the women, postmenopausal (n = 4,703) and premenopausal (n = 2,309) women were included.

2.2. Dietary assessment, meal frequency and meal timing

As mentioned above, the dietary intake of the study participants was assessed using a single-day, 24-h diet recall method. Trained staff surveyed participants and recorded all foods and beverages and portion size of those consumed within 24 h prior to the survey. Then, nutrients intakes consumed from each of all foods and beverages was converted into nutrients intakes based on Rural Development Administration database and daily intake of specific nutrients were calculated by summing those from all sources of foods and beverages. Meal frequency was determined by counting the number of times food or drink was consumed during the day, with each episode consisting of at least 1 kcal of intake [30]. Breakfast, dinner and midnight snack eating were evaluated as “yes” or “no” according to the energy intake during each occasion. Meal timing was defined as the eating occasions of day when the participants indicated which of the following ”snack before breakfast (05:00–07:00)”,

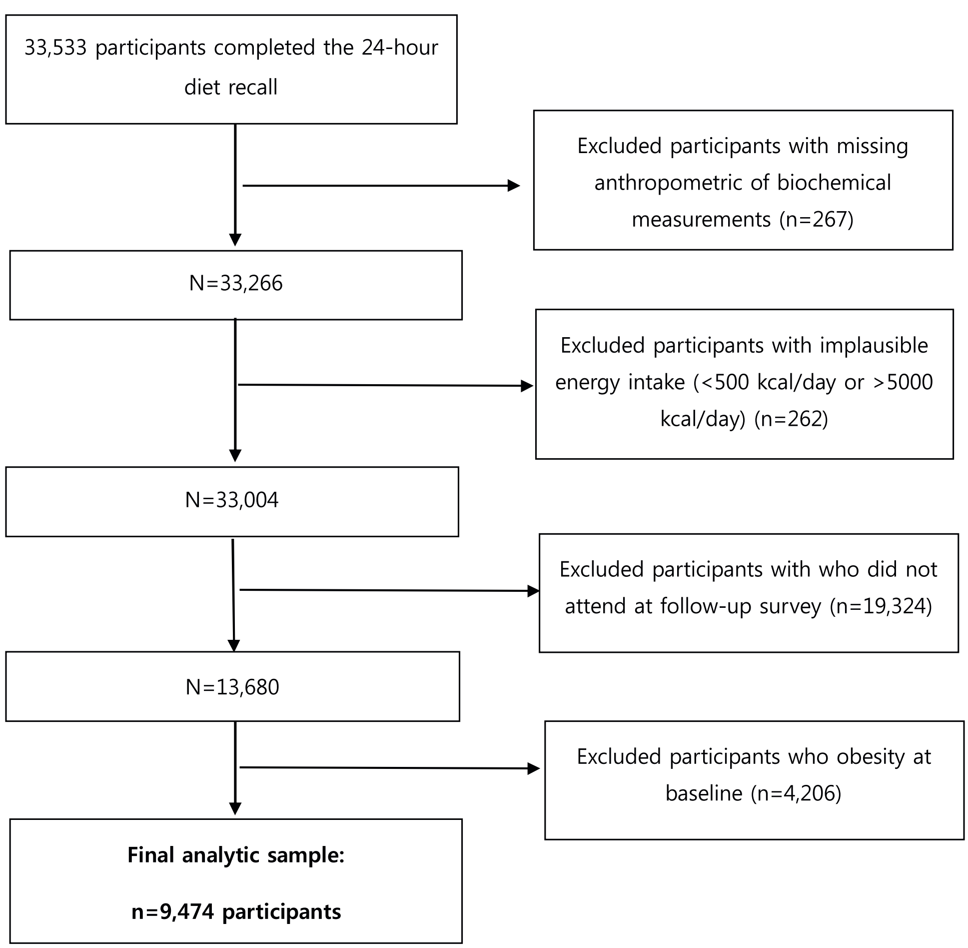

Fig. 1. Flow diagram of the study participants.

”breakfast (07:00–09:00),” ”mid-morning snack (09:00–12:00),” ”lunch (12:00–14:00),” ”afternoon snack (14:00–18:00),” ”dinner (18:00–21:00),” and ”midnight snack (21:00–23:00)” meals they ate in a 24-h period. Participants were classified into the top quartile for percentage of energy consumed in the ”breakfast,” ”lunch,” ”dinner” from the total energy. Nightly fasting duration was estimated by calculating the elapsed hours between the first and last eating episode for each day and subtracting this time form 24 h [31]. In our calculations, we considered snacks as distinct meal timings. Nightly fasting duration was categorized into 4 groups: 6−8 h, 8−10 h, 10−12 h, and more 12 h. These categories can be considered as prolonged nightly fasting [32].

2.3. Sleep duration assessment

sleep duration was measured using an interviewer-assisted questionnaire. Participants were asked to self-report the usual number of hours the slept per day, including naps, over the past year. In accordance with the American National Sleep Foundation's sleep time recommendations, a short sleep duration was defined as sleeping for ≤6 h [33]. Based on these criteria, the patients were divided into 4 groups according to sleep duration, of <6 h, 6−7 h, 7−8 h and ≥8 h.

2.4. Definitions of incident obesity and abdominal obesity

Anthropometric measures were obtained by trained staff with standardized protocols. Participants were measured while not wearing shoes and wearing lightweight clothing using digital stadiometer. Height and weight were measured to the nearest 1 cm and 0.1 kg, respectively. Waist circumference was measured at the middle area between the ribs and iliac crest [29]. Body Mass Index (BMI) (kg/m^2) was calculated as weight (kg) divided by height squared (m^2). Obesity was defined as BMI ≥ 25 kg/m^2 based on the criteria for the Asia-Pacific region of the World Health Organization (WHO) [34]. Abdominal obesity was defined as a waist circumference of mere then 90 cm in men and more than 85 cm in women in accordance with the definition of the Korean Society for the Study of Obesity [35].

2.5. Lifestyle variables

A structured questionnaire was used to obtain information on the lifestyle variables of the participants in the study [29]. Smoking status was grouped into “Never” for those who never smoked cigarettes or smoked <100 cigarettes over a lifetime, “Former” for those who smoked ≥100 cigarettes over a lifetime but not a current smoker, and “Current” for those who had smoked ≥100 cigarettes over a lifetime and a current smoker. Drinking status was “Never” for those who no consumption of any type of alcoholic beverage lifetime, “Former” for those who consumed alcoholic beverages a lifetime but not a current drinker, and “Current” for those who consumed alcoholic beverages more than once per month during the past year. The participants answered yes or no to the question ‘Do you exercise regularly enough to sweat?’. Further queries to subjects who participated in regular

Table 1
Baseline characteristics of the participants in KoGES_HEXA.

	Total	Men	Women	*P* value [1)]
N	9474 (100.0) [2)]	2460 (26.0)	7014 (74.0)	
Age (years)	54.0 ± 8.0	56.5 ± 8.2	53.2 ± 7.7	<0.001
Smoking status				
Non-smokers	7524 (79.4)	678 (27.6)	6846 (97.6)	<0.001
Former smokers	1228 (13.0)	1161 (47.2)	67 (1.0)	
Current smokers	722 (7.6)	621 (25.2)	101 (1.4)	
Drinking status				
Never-drinker	5406 (57.1)	561 (22.8)	4845 (69.1)	<0.001
Former-drinker	296 (3.1)	197 (8.0)	99 (1.4)	
Current-drinker	3772 (39.8)	1702 (69.2)	2070 (29.5)	
Regular Physical activity				
No	4041 (42.7)	963 (39.2)	3078 (43.9)	<0.001
Yes	5443 (57.3)	1497 (60.8)	3936 (56.1)	
Sleeping time (hour/day)	6.8 ± 1.2	6.8 ± 1.2	6.8 ± 1.2	0.0205
Body mass index (kg/m^2)	22.2 ± 1.8	22.6 ± 1.7	22.1 ± 1.8	<0.001
Waist circumference (cm)	76.5 ± 7.0	81.5 ± 6.1	74.7 ± 6.4	<0.001
Hip circumference (cm)	91.2 ± 4.8	92.8 ± 4.7	90.6 ± 4.8	<0.001
Systolic blood pressure (mmHg)	120.3 ± 14.4	123.4 ± 13.8	119.2 ± 14.5	<0.001
Diastolic blood pressure (mmHg)	73.7 ± 9.1	75.9 ± 8.8	72.8 ± 9.1	<0.001
Left grip strength (kg)	24.7 ± 8.7	34.8 ± 8.5	21.2 ± 5.4	<0.001
Right grip strength (kg)	26.1 ± 9.0	36.6 ± 8.7	22.5 ± 5.6	<0.001
Fasting blood glucose (mg/dL)	94.5 ± 17.3	99.2 ± 21.6	92.8 ± 15.1	<0.001
HDL-cholesterol (mg/dL)	57.1 ± 13.8	51.6 ± 12.8	59.0 ± 13.6	<0.001
Triglycerides (mg/dL)	113.1 ± 73.4	134.5 ± 91.2	105.6 ± 64.4	<0.001
Daily dietary intake				
Total daily energy intake (kcal/d)	2478.5 ± 928.6	2662.9 ± 987.8	2413.8 ± 898.0	<0.001
Carbohydrate (g)	415.6 ± 156.1	441.9 ± 162.3	406.4 ± 152.8	<0.001
Protein (g)	96.0 ± 40.9	103.9 ± 43.6	93.2 ± 39.5	<0.001
Fat (g)	52.1 ± 27.9	54.2 ± 29.2	51.4 ± 27.4	<0.001
Energy from carbohydrate (%)	67.6 ± 8.3	67.0 ± 8.3	67.7 ± 8.2	0.0002
Energy from protein (%)	15.4 ± 2.9	15.5 ± 2.8	15.4 ± 2.9	0.0132
Energy from fat (%)	18.6 ± 6.4	17.9 ± 6.2	18.8 ± 6.5	<0.001
Eating episodes (times/day)	4.3 ± 1.2	4.3 ± 1.2	4.3 ± 1.2	0.0419
Morning energy intake (kcal/day)	5.3 ± 40.1	5.5 ± 43.4	5.2 ± 38.8	0.7366
Morning energy intake (% of total energy)	0.2 ± 1.7	0.2 ± 1.5	0.2 ± 1.8	0.5546
Dinner energy intake (kcal/day)	782.1 ± 375.9	877.3 ± 401.2	748.7 ± 360.7	<0.001
Dinner energy intake (% of total energy)	31.6 ± 11.1	33.0 ± 10.4	31.1 ± 11.3	<0.001
Midnight snack energy intake (kcal/day)	48.5 ± 108.7	56.5 ± 132.9	45.7 ± 98.7	0.0002
Midnight snack energy intake (% of total energy)	1.6 ± 3.6	1.8 ± 4.0	1.6 ± 3.5	<0.0558

[1)] The *P* values were calculated using the chi-squared test or t-test procedure. [2)] Data are presented as means ± standard deviation (SD) or numbers (%).

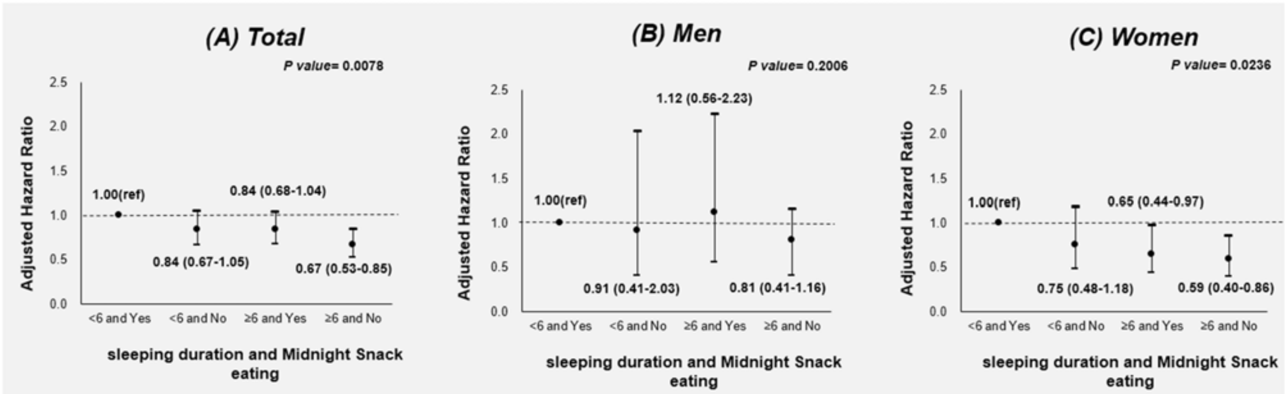

Fig. 2. Multivariable-adjusted hazard ratios according to sleep duration and midnight snack for risk of obesity in KoGES_HEXA. (A) Total, (B) Men (C) Women sleep duration (hours/day) and midnight snack eating categories (<6 and yes[ref], <6 and no, ≥6 and yes, ≥6 and no). HRs and 95% confidence intervals were calculated using Cox proportional hazards models with adjustment for age, sex (except for men, women), smoking status, alcohol consumption, physical activity, waist circumference and total energy intake.

physical activity asked about the average frequency per week and duration.

3. Statistical analysis

All data analyses were performed using SAS 9.4 (SAS Institute, Cary, NC, USA). Statistical significance was set at a *P*-value of <0.05. All continuous variables are presented as means ± standard deviation, and all categorical variables are presented as numbers (weighted percentage, %). The baseline characteristics of the study participants were compared using chi-square tests for the categorical variables and a *t*-test for continuous variables. Person-time was calculated for each participant from the date of attending an assessment center to the date of incident obesity or incident abdominal obesity or the date of last follow-up. Cox proportional hazards model was used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for incident obesity or incident abdominal obesity according to meal frequency, meal timing, percentage of energy at each eating occasion, sleep duration, and nightly fasting duration. The crude model was unadjusted; Model 1 was adjusted for age and sex (only for total); and Model 2 was additionally adjusted for smoking status, alcohol consumption, physical activity, sleep duration (except for the model having sleep duration as dependent variable), waist circumference (except for abdominal obesity), BMI (except for obesity), and total energy intake. We also conducted the analyses using defined cutoffs. Meal frequency was divided into quartiles of the number of eating episodes. Meal timing was evaluated as "yes" or "no" according to the energy intake at each eating occasion. Participants were categorized by the quartile of the percentage of total energy consumed in the morning, lunch, dinner and midnight snack. Sleep duration was categorized into <6, 6−7, 7−8, and ≥8 h. Nightly fasting duration was categorized into 6−8, 8−10, 10−12, and ≥12 h. To assess for a linear trend of the associations between the meal frequency, percentage of energy at each eating occasion, sleep duration, or nightly fasting, and the risk of obesity, participants were assigned a median value for each category. This variable was entered into the model as a continuous term. Stratified analyses were conducted to investigate the joint association of sleep duration and meal timing with the risk of obesity.

4. Results

Table 1 shows the general characteristics of the study participants. The mean age of men and women was 56.5 and 53.2 years, respectively. There were significant differences in smoking status, drinking status, and regular physical activity between men and women (*P* < 0.001 for all). The average number of daily eating episodes was 4.3 in men and women. Men consumed slightly more energy from dinner and midnight snacks than women.

During an average follow-up period of 3.5 years (33,338 person-years), 826 participants (273 men and 553 women) developed obesity, and during an average follow-up period of 3.5 years (37,703 person-years), 1,217 participants (354 men and 863 women) developed abdominal obesity. Meal frequency was not significantly associated with obesity (Table 2). However, there was significant association between meal frequency and abdominal obesity. A higher meal frequency significantly increased the risk for abdominal obesity (Q2: multivariable-adjusted HR, 1.23 95% CI, 1.04–1.45, Q3: multivariable-adjusted HR, 1.30 95% CI, 1.09–1.55, Q4: multivariable-adjusted HR, 1.31 95% CI, 1.07–1.60; P for trend = 0.0058) (Supplementary Table S1). In the fully adjusted multivariable models (Model 2), midnight snack eating significantly increased the risk for obesity, both in total (multivariable-adjusted HR, 1.20; 95% CI, 1.02–1.41; *P*-value = 0.0289) and men (multivariable-adjusted HR, 1.34; 95% CI, 1.01–1.77; *P*-value = 0.0437) (Table 2). In abdominal obesity, significant positive associations between midnight snack eating and abdominal obesity in women (Supplementary Table S1).

We found that participants with the highest energy vs. non-energy intake from midnight snacks had a higher risk of obesity both in total (multivariable-adjusted HR, 1.26; 95% CI, 1.06–1.49; *P* for trend = 0.0105) and men (multivariable-adjusted HR, 1.35; 95% CI, 1.00–1.83; *P* for trend = 0.0460) (Table 3). Similarly, a higher energy intake at midnight snack was significantly associated with an increased incidence of abdominal obesity (multivariable-adjusted HR, 1.18; 95% CI, 1.02–1.36; P for trend = 0.0304). In addition, men in the lower 2nd quartile energy intake at lunch had decreased incidence of abdominal obesity compared to men in the lowest quartile (multivariable-adjusted HR, 0.66; 95% CI, 0.48−0.91; P for trend = 0.0160) (Supplementary Table S2).

Table 4 shows the HRs of obesity according to sleep duration and nightly fasting duration. Individuals who slept ≥8 h had decreased HRs of obesity compared to those who slept <6 h (multivariable-adjusted HR, 0.67; 95% CI, 0.53–0.85; *P* for trend = 0.0078). When stratified by sex, there was no association between sleep duration and obesity in men. The association was significant only in women. Women who slept 7–8 h and ≥8 h had decreased HRs of obesity compared to those who slept <6 h (multivariable-adjusted HR, 0.76; 95% CI, 0.59–0.98 for sleep duration of 7–8 h; multivariable-adjusted HR, 0.66; 95% CI, 0.50–0.86 for sleep duration of ≥8 h). Further analyses focusing on women categorized by menopausal status did not indicate any significant changes in obesity risk (data not shown). In contrast to sleeping duration, nightly fasting duration was not associated with a high risk of obesity in Korean adults. In addition, sleep duration and nightly fasting duration had no significant association with abdominal obesity (Supplementary Table S2).

Fig. 2 shows the joint associations of sleep duration and midnight snack eating with obesity. Compared with those who slept <6 h and ate midnight snacks those who slept ≥6 h and did not eat midnight snacks, showed the lowest HR for obesity (multivariable-adjusted HR, 0.67; 95% CI, 0.53−0.85). Similarly, the HR for obesity was lowest among women who slept ≥6 h and did not eat midnight snacks (multivariable-adjusted HR, 0.59; 95% CI, 0.40−0.86), while men did not show significant difference between groups.

Sensitivity analyses showed that excluding individuals who intake of <50 kcal to define one occasion of meals did not change our results materially (data not shown) and different definitions of sleep duration categories did not change our results materially (data not shown).

5. Discussion

This prospective study found that midnight snack eating, higher energy intake from midnight snacks, and short sleep duration were associated with the risk of obesity in Korean adults after adjusting for various confounding factors based on data from the KoGES. In this study, midnight snack eating and higher energy intake from midnight snacks were positively associated with the HR of obesity. This was consistent with previous studies, which reported associations of midnight snack eating with obesity and abdominal obesity among adults in China and Japan [36–38]. Additionally, a study has shown that calorie intake after 8 pm correlates positively with BMI in healthy adults [21]. Furthermore, higher energy intake around midnight has been associated with a higher risk of obesity [24]. One possible explanation for these findings is circadian misalignment, which occurs when the body's natural internal clock is disrupted, often due to habits like waking up and eating late at night. This disruption can cause changes in hormone levels, including a decrease in plasma leptin, which regulates feelings of fullness [39]. Lower leptin levels may increase hunger and lead to overeating, potentially contributing to weight gain over time [40]. This suggests that the timing of food intake, especially late at night, could impact metabolic health and weight management. In order to clarify whether the association of sleep duration with obesity was linked to either sleeping time *per se* or nightly fasting duration, the potential associations of nightly fasting duration with obesity were also analyzed. However, no significant association between the nightly fasting duration and obesity was found. Studies in rodents show that intermittent fasting and restricting the availability of food to the normal nighttime feeding cycle improves metabolic profiles and reduces the risk of obesity [19,20]. However, data from related human studies are limited regarding the positive impacts of Time-restricted eating (TRE) on weight [31,40].

Sleep duration was a meaningful factor associated with obesity in Korean adults. When stratifying by sex, no association was observed between sleep duration and obesity among men. The risk of obesity was significantly lower for both the 7–8 and ≥8 h sleep durations among women, corresponding to 0.76 and 0.66 times lower HR, respectively, when compared to sleeping <6 h. Allied with our findings, Patel et al. found that a habitual sleep time of less than 7 h predicted increased future weight gain independent of baseline weight among middle-aged women [25]. Likewise, a cross-sectional analysis of the American population revealed that individuals with a sleep duration of less than 7 h exhibited an overweight and obesity rate nearly twice as high as those who slept for 7–9 h [27]. The association between sleep and weight status may be closely related to the circadian rhythm. Sleep deprivation has been shown to reduce leptin and increases ghrelin (a “hunger” hormone) [40]. Also, habitually short sleep duration could lead to insulin resistance by increasing sympathetic nervous system activity, raising evening cortisol levels, and decreasing cerebral glucose utilization, which over time could compromise pancreatic beta cell function and lead to diabetes. Consequently, prolonged wakefulness at night may precipitate late-night eating behaviors, which in turn may contribute to weight gain. Subsequently, stratified analyses were conducted for midnight snacking and short sleep duration, as these factors exhibited significant associations with the risk of obesity. The study results indicate that women who sleep for at least 6 h and abstain from midnight snacking have a lower risk of obesity.

The study found that midnight snacking and higher energy intake from midnight snacks were significantly positively associated with obesity risk in men, while short sleep duration was only significantly associated with obesity risk in women. This finding is consistent with a study of middle-aged women, which found no association between eating after 10 pm and BMI [41]. A previous study examining the association between sleep duration and overweight in the US adult population, using data from the 2015 to 2016 National Health and Nutrition Examination Survey (NHANES), found that among women, the short sleep group was more likely to be overweight than the normal sleep group when adjusted for age, race, marital status, and education. Conversely, the overweight incidence was statistically comparable between the short sleep and normal sleep groups among men [27].

The potential reasons behind our findings regarding the varying associations of sleep duration and midnight snack consumption with obesity risk according to sex remain unclear. Nevertheless, it is important to recognize that obesity is a complex condition influenced by numerous factors including age, gender, lifestyle choices, and physiological processes [42,43]. Both meal timing and sleep patterns may exert sex-specific effects on obesity [44–47]. One potential explanation could involve hormonal differences between sexes.

Alternatively, it's noteworthy that, on average, men in our study were three years older than women at the time of the baseline survey, and there were observed disparities in various lifestyle factors such as higher rates of alcohol consumption and smoking among men. These behavioral distinctions might have contributed to differences in obesity risk between men and women. Further investigations are warranted to explore the intricacies of sex-specific variations in obesity prevalence.

Epidemiological and metabolic evidence suggests that the negative metabolic consequence of excess fat is more closely related to the location of the fat than the amount of fat [48,49]. In this context, centralized accumulation of fat is a better predictor of increased risk of type 2 diabetes mellitus and cardiovascular disease than absolute fat mass [50]. Therefore, we further explored the effects of meal timing and sleep duration on abdominal obesity. The results of this study indicate that more eating episodes are associated with a higher risk of abdominal obesity.

This is consistent with findings in the US and UK that less frequent eating is associated with a reduced abdominal obesity risk and waist circumference [51,52]. However, opposite findings were reported in Swedish men, where more meal episodes were associated with reduced obesity and abdominal obesity [53], and in South Korea, where more meal episodes were associated with a lower prevalence of metabolic abnormalities, including abdominal obesity, elevated blood pressure, and elevated triglycerides in men [31]. Different definitions of frequency and dietary assessment. Some studies defined meal frequency as an intake of 50 kcal or more using a 24 -h recall or a 7-day weighed dietary record [51,52], while others defined it as an intake of 1 kcal or more of food or drink using a 24 -h recall [31]. The average daily food frequency was calculated using the FFQ [53].

Human meal timing and sleep duration are closely related not only to physiological factors but also to various environmental factors, such as longer working days, night shifts, and the use of electronics in modern society. As the risk of obesity increases in Korea, it is necessary to identify risk factors for obesity, abdominal obesity, and sex-specific obesity and to educate people about different dietary and lifestyle habits.

To the best of our knowledge, this is the first prospective study to investigate the association of both meal timing and sleep duration with obesity incidence as well as the modifying effect of various factors on this association in the Korean population. However, our study has some limitations. Firstly, the study subjects, drawn from health examination centers and training hospitals across 8 Korean regions, may exhibit a selection bias due to a tendency towards greater health consciousness. Consequently, these results may not fully extend to the broader Korean populace. Further research with a more representative demographic spectrum is warranted. Secondly, participant-reported sleep duration was utilized, which may introduce potential inaccuracies. Employing more objective measures, such as polysomnography [54], would yield more reliable results. Thirdly, meal timings are categorized and may exhibit slight biases as they are derived from converting 24-h time spans into fasting durations between the last and first meals. Implementing a validated questionnaire encompassing meal and fasting time assessments is imperative. Fourthly, while trained registered dietitians followed standardized protocols to mitigate bias by aiding participants in accurately reflecting on their daily dietary habits, relying solely on a 24-h recall might not adequately capture normal daily intake due to significant intra-individual variability in food and nutrient consumption. Lastly, several other factors that can contribute to obesity and circadian rhythms, such as shift work, other health status such as Obstructive sleep apnea (OSA) and Night eating disorder (NED), and medication that can affect body weight, were not collected and were not incorporated in the analysis. Finally, the short follow-up period of 3.5 years may bias the effect on obesity.

6. Conclusions

Midnight snack eating and higher energy intake from midnight snacks were associated with a higher risk of obesity. The association was greater in men than in women. Conversely, sleep duration was inversely associated with the risk of obesity only in women. Our findings underscore the importance of addressing nighttime eating habits and sleep duration in obesity prevention. Intervention strategies may include educational campaigns, behavioral interventions, nutritional guidance, and promoting healthy sleep hygiene practices within communities.

Author contributions

All authors contributed to the study conception and design; Material preparation, data collection and analysis were performed by J.L.; Supervision and funding acquisition was done by Y.J.P.; S.J. K.L critically reviewed, revised manuscript, and contributed to interpretation of data; All authors commented on previous versions of the manuscript and all authors read and approved the final manuscript.

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Ethics standards

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by Korea Disease Control and Prevention Agency Institutional Review Board (Date March 22. 2018/No 2018-03-05-P-A). Informed consent was confirmed by the IRB and obtained from all individual participants included in the study.

Conflict of interest

The authors declared no conflict of interest.

Acknowledgments

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jnha.2024.100220.

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