Distinct Relevance of Nightly Sleep Duration to Metabolic, Anthropometric, and Lifestyle Factors in Patients with Type 2 Diabetes

Hitomi Nakayama$^{1,2}$, Yasushi Yamada$^3$, Kentaro Yamada$^4$, Shimpei Iwata$^1$, Nobuhiko Wada$^1$, Yuji Tajiri$^{1,5}$ and Masatoshi Nomura$^1$

Abstract:

Objective Although a number of studies have shown that both short and long sleep durations were associated with the risk of metabolic disorders related to obesity, the underlying mechanism is still not fully understood. In this study, we analyzed the association of sleep duration with metabolic, anthropometric, and lifestyle factors in patients with type 2 diabetes.

Methods The subjects were 279 patients with type 2 diabetes 63 (52-70) years old (median and interquartile range) with a body mass index of 25.0 (22.2-28.3) kg/m$^2$ and HbA1c levels of 8.7% (7.6-10.3%). Patients with advanced complications were excluded from the study. Diets were evaluated by registered dietitians using a software program. Body composition was assessed by the multifrequency bioelectrical impedance method.

Results The mean self-reported nightly sleep duration was 6.4 hours with no marked gender difference. Sleep duration was inversely correlated with the HbA1c levels, total energy intake, and intakes of carbohydrate, protein, and fat. The body fat ratio and skeletal muscle mass were correlated positively and negatively, respectively, with sleep duration. When the subjects were divided into three groups based on sleep duration, the intakes of total energy, carbohydrates, and fat tended to be high in those with <5.5 hours of sleep, and the percentage of patients who had habitual physical activities was lower in those with >7 hours of sleep.

Conclusion The observation that sleep duration is distinctly associated with excessive eating and a sedentary lifestyle may provide a basis for effective lifestyle management of patients with type 2 diabetes.

Key words: type 2 diabetes, obesity, sleep duration, dietary intake, habitual physical activity

(Intern Med 60: 681-688, 2021)
(DOI: 10.2169/internalmedicine.5078-20)

Introduction

Accumulating lines of evidence have shown that a short sleep duration is a risk factor for various health problems, including obesity/overweight (1-4), type 2 diabetes (1, 2), hypertension (3), and cardiovascular diseases (5, 6). In contrast, several epidemiological studies (7-10) and a meta-analysis (11) have shown that a long sleep duration is also associated with an increased risk of morbidity and mortality and that the correlation curve between sleep duration and obesity-related disorders, such as type 2 diabetes and cardiovascular diseases, is U-shaped (7, 9, 12-19). However, the mechanism by which both short and long sleep durations facilitate the onset of these obesity-related diseases is still not fully elucidated.

To assess the underlying mechanism of the U-shaped relationship, we analyzed the association of sleep duration with metabolic, anthropometric, and lifestyle factors in patients with type 2 diabetes.

1Division of Endocrinology and Metabolism, Department of Medicine, Kurume University School of Medicine, Japan, 2Division of Endocrinology and Metabolism, Chikugo Municipal Hospital, Japan, 3Department of Clinical Nutrition, Kurume University Hospital, Japan, 4Diabetes Center, Asakura Medical Association Hospital, Japan and 5Department of Endocrinology and Metabolism, Kurume University Medical Center, Japan

Received: April 13, 2020; Accepted: August 12, 2020; Advance Publication by J-STAGE: October 21, 2020
Correspondence to Dr. Hitomi Nakayama, nahitomi@med.kurume-u.ac.jp
**Materials and Methods**

**Subjects**

This was a retrospective analysis of electrical medical records in Kurume University Hospital. Subjects were patients >20 years old with type 2 diabetes who were admitted to the hospital for the treatment of dysglycemia and/or diabetes self-management education; 279 individuals, including 145 men and 134 women, who were 63 (52-70) years old (median and interquartile range) with a body mass index (BMI) 25.0 (22.2-28.3) kg/m², and HbA1c 8.7% (7.6-10.5%). Sleep duration was inversely correlated with HbA1c levels at admission. Sleep duration was negatively correlated with HbA1c levels at admission (Fig. 1). An anthropometric

**Table 1. Clinical Parameters of the Subjects.**

| Parameter | Value       |
|-----------|-------------|
| Number (male/female) | 279 (145/134) |
| Age (year) | 63 (52-70) |
| Diabetes duration (year) | 9.4 (3.0-16.8) |
| Body mass index (kg/m²) | 25.0 (22.2-28.3) |
| Body fat (%) | 32.6 (25.9-37.4) |
| Skeletal muscle mass (kg) | 24.2 (19.7-28.7) |
| HbA1c (%) | 8.7 (7.6-10.5) |
| Fasting plasma glucose (mg/dL) | 149 (123-194) |
| AST (U/L) | 21 (17-28) |
| ALT (U/L) | 23 (16-34) |
| ALP (U/L) | 216 (183-263) |
| γ-GTP (U/L) | 31 (20-54) |
| Albumin (g/dL) | 4.04 (3.83-4.27) |
| HDL cholesterol (mg/dL) | 45.8 (40.5-55.9) |
| LDL cholesterol (mg/dL) | 116.2 (95.0-136.1) |
| Triglyceride (mg/dL) | 140 (99-191) |
| eGFR (mL/min/1.73 m²) | 81.3 (66.5-100.5) |
| Diabetic retinopathy | 96/268 (35.8%) |
| Diabetic neuropathy | 107/279 (38.4%) |
| Diabetes medication | 32 (11.5%) |

**Figure 1. Correlation between nightly sleep duration and HbA1c levels.**

**The evaluation of nightly sleep duration, diet, body composition, and physical activities**

Usual nighttime sleep duration was self-reported at the admission interview. Daytime naps were not included. Diets were evaluated by registered dietitians using a software program (Shokuwa nari; Gleam, Kitakyushu, Japan). Alcohol ingestion twice a week or more was regarded as habitual drinking. Sweet snack intake on five days a week or more was regarded as habitual sweet snack consumption. Breakfast skipping was defined as eating breakfast <5 days per week. Body composition was assessed by the multifrequency (1, 5, 25, 50, 250, and 1,000 kHz) bioelectrical impedance method at each of 5 segments: arms, trunk, and legs (InBody720; InBody Japan, Tokyo, Japan). We defined habitual physical activity as ≥150 minutes of walking per week or equivalent, including occupational and household activities. The diagnosis of peripheral neuropathy was made based on the vibration time (128 Hz) at the lateral malleolus <10 seconds and negative Achilles tendon reflex in both legs.

The study was approved by the ethics committee of Kurume University.

**Statistical analyses**

Data are expressed as medians and interquartile ranges. The Kruskal-Wallis test, Mann-Whitney U test, and chi square test were used to compare the differences between groups. The statistical relationship between two continuous variables was evaluated by Pearson’s correlation coefficient. The Cochran-Armitage trend test was used to detect trends in binomial proportions. Results with p<0.05 were considered statistically significant.

**Results**

The self-reported nightly sleep duration was 6 (6-7) hours with a mean value of 6.4 hours and standard deviation (SD) of 1.4 hours. There were no marked age- or gender-related differences. Sleep duration was inversely correlated with HbA1c levels at admission (Fig. 1). An anthropometric
analysis showed that the BMI was not associated with sleep duration, but the body fat ratio and skeletal muscle mass were correlated positively and negatively, respectively, with sleep duration (Table 2). These correlations were significant, even after adjusting for age or both age and gender.

When subjects were divided into three groups based on sleep duration (group 1, <5.5 hours; group 2, 5.5-7 hours; group 3, >7 hours), the age and gender were not markedly different among the groups (Table 3). The HbA1c was highest in group 1 and lowest in group 3, whereas the body fat ratio was highest in group 3 and lowest in group 1. There were no marked differences in the diabetes duration, estimated glomerular filtration rate, diabetic neuropathy, diabetic retinopathy, diabetic medication, or hypnotic drug use among the groups.

To find an explanation for the relationships of nightly sleep duration with HbA1c levels and anthropometric parameters, we analyzed the dietary intake of nutrients and habitual physical activities. There were inverse correlations between sleep duration and the intakes of total calories, carbohydrates, protein, fat, and dietary fiber (Table 4). The fat intake was significantly different among the groups, with the lowest value being noted in group 3 (Fig. 2). A trend was also observed in the intake of total energy and carbohydrate, although the differences did not reach statistical significance.

Table 2. Correlation between Nightly Sleep Duration and Anthropometric Parameters. p values are Shown in Parentheses.

| BMI | %Body fat | Skeletal muscle mass |
|-----|-----------|----------------------|
| Pearson correlation coefficient | 0.0045 (0.939) | 0.1521 (0.008) | -0.1435 (0.013) |
| Age-adjusted | 0.0297 (0.607) | 0.1565 (0.009) | -0.1067 (0.048) |
| Age- and gender-adjusted | 0.0300 (0.605) | 0.1390 (0.009) | -0.0845 (0.037) |

Table 3. Clinical Parameters of the Subjects in Group 1 (Sleep Duration<5.5 Hours), Group 2 (5.5-7 Hours), and Group 3 (>5 Hours).

| Parameter | Group 1 | Group 2 | Group 3 | p value |
|-----------|---------|---------|---------|---------|
| Number (male/female) | 50 (29/21) | 162 (83/79) | 67 (33/34) | 0.621 |
| Age (year) | 61 (48.5-70.5) | 63 (51-69) | 66 (58-73) | 0.167 |
| Body mass index (kg/m²) | 24.2 (21.8-27.8) | 25.3 (22.7-28.2) | 25.2 (21.6-28.6) | 0.561 |
| Body fat (%) | 28.6 (22.7-35.5) | 32.2 (26.5-36.7) | 34.3 (25.9-40.8) | 0.046 |
| Skeletal muscle mass (kg) | 24.5 (20.4-29.7) | 24.3 (20.1-28.4) | 23.1 (18.2-28.5) | 0.294 |
| HbA1c (%) | 9.2 (8.0-10.7) | 8.9 (7.6-10.3) | 8.2 (7.3-9.3) | 0.007 |
| Fasting plasma glucose (mg/dL) | 149 (121-192) | 155 (129-199) | 140 (113-180) | 0.065 |
| AST (U/L) | 20 (16-25) | 22 (17-30) | 22 (18-29) | 0.345 |
| ALT (U/L) | 22 (15-33) | 24 (16-35) | 25 (17-38) | 0.296 |
| ALP (U/L) | 243 (195-261) | 216 (184-270) | 198 (170-253) | 0.081 |
| γ-GTP (U/L) | 32 (22-49) | 32 (20-55) | 28 (16-55) | 0.454 |
| Albumin (g/dL) | 3.98 (3.73-4.25) | 4.04 (3.86-4.30) | 4.04 (3.85-4.24) | 0.346 |
| HDL cholesterol (mg/dL) | 44.5 (39.2-56.9) | 46.4 (40.5-55.6) | 46.0 (41.3-55.3) | 0.854 |
| LDL cholesterol (mg/dL) | 119.1 (94.6-148.9) | 114.6 (95.0-133.2) | 116.5 (94.8-136.9) | 0.606 |
| Triglyceride (mg/dL) | 143 (98-185) | 134 (99-197) | 144 (100-194) | 0.674 |
| Diabetes duration (year) | 9.9 (3.3-17.2) | 8.8 (3.3-15.6) | 10.1 (2.6-18.0) | 0.837 |
| eGFR (mL/min/1.73 m²) | 85.4 (64.4-102.3) | 82.8 (66.9-101.5) | 76.5 (60.7-95.7) | 0.373 |
| Diabetic retinopathy | 17/46 (37.0%) | 53/159 (33.3%) | 26/63 (41.2%) | 0.531 |
| Diabetic neuropathy | 16/50 (32.0%) | 64/162 (39.5%) | 27/67 (40.3%) | 0.591 |

Medians and interquartile ranges. p values were assessed using the Kruskal-Wallis test or the chi square test.

Table 4. Correlation between Nightly Sleep Duration and Intakes of Total Energy and Nutrients.

| Energy | Carbohydrates | Protein | Fat | Dietary fiber |
|--------|---------------|---------|-----|--------------|
| p value | 0.0098 | 0.0108 | 0.0174 | 0.0024 |
| Correlation coefficient | -0.1544 | -0.1516 | -0.142 | -0.1801 |

| Hypnotic drugs | p value |
|---------------|---------|
| 7/50 (14%) | 0.614 |

To find an explanation for the relationships of nightly sleep duration with HbA1c levels and anthropometric parameters, we analyzed the dietary intake of nutrients and habitual physical activities. There were inverse correlations between sleep duration and the intakes of total calories, carbohydrates, protein, fat, and dietary fiber (Table 4). The fat intake was significantly different among the groups, with the lowest value being noted in group 3 (Fig. 2). A trend was also observed in the intake of total energy and carbohydrate, although the differences did not reach statistical significance.
In contrast, no significant difference was observed in habitual alcohol ingestion, habitual sweet snack consumption, or breakfast skipping based on the sleep duration (Fig. 3).

A significant negative trend was noted in the ratio of habitual physical activities of ≥150 minutes walking per week or equivalent among the 3 groups: 44.0%, 31.3%, and 25.4% in groups 1, 2, and 3, respectively (Fig. 4a). A post hoc analysis showed that the percentage of patients who had habitual physical activities was significantly lower in group 3 than in group 1. Patients with habitual physical activities tended to be older with a lower BMI and lower %body fat than those without such habits (Table 5). The associations were also observed when men and women were analyzed separately, although the difference in the BMI did not reach statistical significance (p=0.093) in women. In subjects without habitual physical activities, the nighttime sleep duration was positively and negatively correlated with the body fat percentage and skeletal muscle mass, respectively (Table 6). However, in subjects who had habitual physical activities, these associations were not observed. There was no significant difference in the prevalence of diagnosed clinical depression among the three groups (Fig. 4b).

Discussion

In the present study, we showed a significant inverse correlation between nighttime sleep duration and HbA1c levels in patients with type 2 diabetes, in accordance with previous studies reporting that sleep deprivation was associated with metabolic disorders, such as obesity (1-3), insulin resistance (2), and type 2 diabetes (1, 2). The postulated mechanisms include alterations in appetite-regulating hormones (20-22), activation of the food reward system (23, 24), and an increase in inflammatory responses (25, 26).

However, several studies have demonstrated that a long sleep duration was also associated with the incidence of obesity (8, 10, 22), type 2 diabetes (8-10), and cardiovascular disease (7, 16). In the present study, a longer sleep duration was associated with a high body fat ratio and low skeletal muscle mass. Although the body fat ratio and skeletal muscle mass are related to age and gender, these associa-
Figure 3. The prevalence of self-reported habitual alcohol drinking (a), habitual sweet snack consumption (b), and breakfast skipping (c) in group 1 (sleep duration<5.5 hours, n=50), group 2 (5.5-7 hours, n=162), and group 3 (>7 hours, n=67). Alcohol ingestion twice a week or more was regarded as habitual drinking. Sweet snack intake on five days a week or more was regarded as habitual sweet snack consumption. Breakfast skipping was defined as eating breakfast<5 days per week. NS: not significant by the chi square test.
Figure 4. The prevalence of habitual physical activity (a) and depression (b) in group 1 (sleep duration<5.5 hours, n=50), group 2 (5.5-7 hours, n=162), and group 3 (>7 hours, n=67). Habitual physical activity was defined as ≥150 minutes of walking per week or equivalent, including occupational and household activities. The dose-response association between sleep duration and the prevalence of habitual physical activities was assessed by the Cochran-Armitage trend test. The p value in parenthesis is the result of a post-hoc analysis using the chi square test. NS: not significant

Table 5. Clinical Parameters of the Subjects with and without Habitual Physical Activities.

| Parameter                              | Habitual physical activities | p value |
|----------------------------------------|------------------------------|---------|
| Number (male/female)                   | 190 (94/96)                  | 89 (51/38) | 0.222 |
| Age (year)                             | 61 (51-69)                   | 66 (53.5-72) | 0.037 |
| Diabetes duration (year)               | 8.45 (2.6-16.6)              | 10.1 (3.8-17.1) | 0.351 |
| Body mass index (kg/m²)                | 25.8 (22.5-28.8)             | 24.1 (21.7-26.2) | 0.012 |
| Male                                   | 26.0 (22.6-28.7)             | 24.4 (22.4-26.1) | 0.047 |
| Female                                 | 25.2 (22.4-28.9)             | 23.6 (21.5-26.5) | 0.093 |
| Body fat (%)                           | 33.4 (26.6-39.4)             | 29.7 (23.2-34.1) | 0.001 |
| Male                                   | 28.4 (24.1-35.2)             | 26.1 (21.7-29.8) | 0.039 |
| Female                                 | 36.2 (32.8-42.2)             | 33.9 (31.7-37.4) | 0.041 |
| Skeletal muscle mass (kg)              | 23.9 (19.3-29.1)             | 24.4 (20.0-28.4) | 0.987 |
| HbA1c (%)                              | 8.9 (7.5-10.3)               | 8.6 (7.7-10.2) | 0.969 |
| Fasting plasma glucose (mg/dL)         | 149 (123-195)                | 153 (125-194) | 0.658 |
| AST (U/L)                              | 21 (16-28)                   | 22 (18.5-31) | 0.335 |
| ALT (U/L)                              | 22 (16-35)                   | 24 (17-33) | 0.269 |
| ALP (U/L)                              | 214 (185-268)                | 218 (174-258) | 0.664 |
| γ-GTP (U/L)                            | 31 (19-54)                   | 31 (21-60) | 0.622 |
| Albumin (g/dL)                         | 4.01 (3.81-4.21)             | 4.07 (3.87-4.33) | 0.048 |
| HDL cholesterol (mg/dL)                | 46.2 (40.1-55.9)             | 45.3 (41.3-55.3) | 0.836 |
| LDL cholesterol (mg/dL)                | 115.4 (94.0-135.0)           | 117 (95.7-140.1) | 0.532 |
| Triglyceride (mg/dL)                   | 140 (103-188)                | 140 (91-198) | 0.874 |
| eGFR (mL/min/1.73 m²)                  | 84.5 (68.7-104.1)            | 76.3 (63.7-90.4) | 0.047 |
| Diabetic retinopathy                   | 64/182 (35.2%)               | 32/86 (37.2%) | 0.961 |
| Diabetic neuropathy                    | 77/190 (40.5%)               | 30/89 (33.7%) | 0.275 |
| Diabetes medication                    |                            |          | 0.116 |
| Insulin alone or in combination        | 34 (18.0%)                   | 25 (28.1%) |        |
| Drugs except insulin                   | 99 (52.0%)                   | 44 (49.4%) |        |
| No drug                                | 57 (30.0%)                   | 20 (22.5%) |        |
| Hypnotic drugs                         | 20 (10.5%)                   | 12 (13.5%) | 0.470 |

Medians and interquartile ranges. p values were assessed using the Mann-Whitney U test or the chi square test.
not get enough sleep because of longer work hours. Low physical activity might have been associated with a low energy intake in group 3.

When the subjects were divided into two groups based on habitual physical activities, those without habitual physical activities had a greater BMI and higher body fat ratio than those with such activities. The association between sleep duration and body composition was observed in subjects without habitual physical activities but not in those who had habitual physical activities. These observations suggest that a long sleep duration is not deleterious when physical activities are sufficient and that the adipose body composition in group 3 was caused by both the low prevalence of habitual physical activities and low nonexercise thermogenesis due to a sedentary lifestyle.

Another possible explanation is that some patients in group 3 may have psychological problems that cause a physically inactive lifestyle, as hypersomnolence and physical inactivity are common features of major depressive disorders (32, 33). Depression increases the risk of cardiovascular morbidity and mortality, probably through an unfavorable lifestyle (34). However, while the prevalence of depression tended to be higher in groups 1 and 3 than in group 2 in our study, the difference was not statistically significant. Further studies will be required to elucidate the influence of depression on the association between sleep duration and metabolic disorders. Finally, the use of hypnotic drugs was not associated with clinical parameters in this study, indicating that the associations were unlikely attributable to the effects of hypnotics.

Several limitations associated with the present study warrant mention. This was a cross-sectional study of a single facility, and the number of subjects was not large. The nightly sleep duration was not evaluated by electroencephalography but by self-report. Habitual physical activities were also self-reported and not measured using accelerometers or pedometers. We did not use a standardized assessment tool to screen for depression; therefore, there may have been some overlooked cases. Finally, although some studies have defined long sleepers as subjects with a sleep duration of ≥10 hours, in the present study, only 8 subjects had a sleep duration of ≥10 hours. This may be because daytime naps were not included in the sleep duration. Further studies will be required to elucidate the influence of habitual physical activities on the association between nightly sleep duration and body composition in long sleepers.

In conclusion, although a U-shaped relationship was not observed between sleep duration and either HbA1c or body fat percentage, sleep duration was negatively and positively associated with HbA1c and body fat ratio, respectively, indicating that both sleep deprivation and oversleeping are deleterious to health. Thus, nightly sleep durations have distinct relevance to metabolic and anthropometric factors in patients with type 2 diabetes. These observations may provide a basis for effective lifestyle management of patients with type 2 diabetes.

The authors state that they have no Conflict of Interest (COI).

References

1. Schmid SM, Hallischmid M, Schultes B. The metabolic burden of sleep loss. Lancet Diabetes Endocrinol 3: 52-62, 2015.
2. McNeil J, Doucet E, Chaput JP. Inadequate sleep as a contributor to obesity and type 2 diabetes. Can J Diabetes 37: 103-108, 2013.
3. Bjorvatn B, Sagen IM, Øyane N, et al. The association between sleep duration, body mass index and metabolic measures in the Hordaland Health Study. J Sleep Res 16: 66-76, 2007.
4. Xiao Q, Aren H, Moore SC, Hollenbeck AR, Matthews CE. A large prospective investigation of sleep duration, weight change, and obesity in the NIH-AARP Diet and Health Study cohort. Am J Epidemiol 178: 1600-1610, 2013.
5. Eguchi K, Pickering TG, Schwartz JE, et al. Short sleep duration as an independent predictor of cardiovascular events in Japanese patients with hypertension. Arch Intern Med 168: 2225-2231, 2008.
6. Bertisch SM, Pollock BD, Mittleman MA, et al. Insomnia with objective short sleep duration and risk of incident cardiovascular disease and all-cause mortality: Sleep Heart Health Study. Sleep 41: zsy047, 2018.
7. Reis C, Dias S, Rodrigues AM, et al. Sleep duration, lifestyles and chronic diseases: a cross-sectional population-based study. Sleep Sci 11: 217-230, 2018.
8. Tan X, Chapman CD, Cedernaes J, Benedict C. Association between long sleep duration and increased risk of obesity and type 2 diabetes: a review of possible mechanisms. Sleep Med Rev 40: 127-134, 2018.
9. Shan Z, Ma H, Xie M, et al. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. Diabetes Care 38: 529-537, 2015.
10. Cespedes EM, Bhupathiraju SN, Li Y, Rosner B, Redline S, Hu HB. Long-term changes in sleep duration, energy balance and risk of type 2 diabetes. Diabetologia 59: 101-109, 2016.
11. Jike M, Itani O, Watanabe N, Buyssse DJ, Kaneita Y. Long sleep duration and health outcomes: a systematic review, meta-analysis and meta-regression. Sleep Med Rev 39: 25-36, 2018.
12. van den Berg JF, Neven AK, Tulen JH, et al. Actigraphic sleep duration and fragmentation are related to obesity in the elderly: the Rotterdam Study. Int J Obes (Lond) 32: 1083-1090, 2008.
13. Chaput JP, Després JP, Bouchard C, Tremblay A. The association

Table 6. Correlation between Nightly Sleep Duration and Anthropometric Parameters in Subjects with and without Habitual Physical Activities. p values are Shown in Parentheses.

| Habitual physical activities (+) | BMI | %Body fat | Skeletal muscle mass |
|---------------------------------|-----|-----------|---------------------|
|                                 | 0.0298 (0.792) | 0.0857 (0.424) | -0.0040 (0.971) |
| Habitual physical activities (-) | 0.0292 (0.689) | 0.1557 (0.032) | -0.2068 (0.004) |
between sleep duration and weight gain in adults: a 6-year prospective study from the Quebec Family Study. Sleep 31: 517-523, 2008.
14. Ayas NT, White DP, Al-Delaimy WK, et al. A prospective study of self-reported sleep duration and incident diabetes in women. Diabetes Care 26: 380-384, 2003.
15. Yaggi HK, Araujo AB, McKinlay JB. Sleep duration as a risk factor for the development of type 2 diabetes. Diabetes Care 29: 657-661, 2006.
16. Cappuccio FP, D’Elia L, Strazzullo P, Miller MA. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. Diabetes Care 33: 414-420, 2010.
17. Krittanawong C, Tunhasiriwet A, Wang Z, et al. Association between short and long sleep durations and cardiovascular outcomes: a systematic review and meta-analysis. Eur Heart J Acute Cardiovasc Care 8: 762-770, 2019.
18. Cappuccio FP, Cooper D, D’Elia L, Strazzullo P, Miller MA. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. Eur Heart J 32: 1484-1492, 2011.
19. Ohkuma T, Fujii H, Iwase M, et al. Impact of sleep duration on obesity and the glycemic level in patients with type 2 diabetes: the Fukuoka Diabetes Registry. Diabetes Care 36: 611-617, 2013.
20. Spiegel K, Tasali E, Penev P, Van Cauter E. Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann Intern Med 141: 846-850, 2004.
21. Cooper CB, Neufeld EV, Dolezal BA, Martin JL. Sleep deprivation and obesity in adults: a brief narrative review. BMJ Open Sport Exerc Med 4: e000392, 2018.
22. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med 1: e62, 2004.
23. St-Onge MP, McReynolds A, Trivedi VB, Roberts AL, Sy M, Hirsch J. Sleep restriction leads to increased activation of brain regions sensitive to food stimuli. Am J Clin Nutr 95: 818-824, 2012.
24. Benedict C, Brooks SJ, O’Daly OG, et al. Acute sleep deprivation enhances the brain’s response to hedonic food stimuli: an fMRI study. J Clin Endocrinol Metab 97: E443-E447, 2012.
25. Irwin MR, Olmstead R, Carroll JE. Sleep disturbance, sleep duration, and inflammation: a systematic review and meta-analysis of cohort studies and experimental sleep deprivation. Biol Psychiatry 80: 40-52, 2016.
26. Möller-Levet CS, Archer SN, Bucca G, et al. Effects of insufficient sleep on circadian rhythmicity and expression amplitude of the human blood transcriptome. Proc Natl Acad Sci U S A 110: E1132-E1141, 2013.
27. Grandner MA, Jackson N, Gerstner JR, Knutson KL. Dietary nutrients associated with short and long sleep duration. Data from a nationally representative sample. Appetite 64: 71-80, 2013.
28. Kant AK, Graubard BI. Association of self-reported sleep duration with eating behaviors of American adults: NHANES 2005-2010. Am J Clin Nutr 100: 938-947, 2014.
29. Ma X, Shen Q, Pu Y, et al. Skipping breakfast is associated with overweight and obesity: a systematic review and meta-analysis. Obes Res Clin Pract 14: 1-8, 2020.
30. Yasuda J, Asako M, Arimitsu T, Fujita S. Skipping breakfast is associated with lower fat-free mass in healthy young subjects: a cross-sectional study. Nutr Res 60: 26-32, 2018.
31. Kredlow MA, Capozzoli MC, Heaton BA, Calkins AW, Otto MW. The effects of physical activity on sleep: a meta-analytic review. J Behav Med 38: 427-449, 2015.
32. Kaplan KA, Harvey AG. Hypersomnia across mood disorders: a review and synthesis. Sleep Med Rev 13: 275-285, 2009.
33. Plante DT, Cook JD, Goldstein MR. Objective measures of sleep duration and continuity in major depressive disorder with comorbid hypersomnolence: a primary investigation with contiguous systematic review and meta-analysis. J Sleep Res 26: 255-265, 2017.
34. Penninx BW. Depression and cardiovascular disease: epidemiological evidence on their linking mechanisms. Neurosci Biobehav Rev 74 (Pt B): 277-286, 2017.

The Internal Medicine is an Open Access journal distributed under the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License. To view the details of this license, please visit (https://creativecommons.org/licenses/by-nc-nd/4.0/).