Good quality sleep and sufficient sleep duration are essential components for human health. Those who habitually sleep outside the normal range might have consequential effects on their health and well-being. Poor sleep is often associated with an increased risk for injuries (traffic- or occupation-related), reduced productivity, impaired mood, higher body mass, and development of noncommunicable diseases (NCDs). It has been suggested that there has been an increasing problem with sleep duration and quality in recent decades, especially among adult individuals who live in urban areas. This disturbance in sleep duration and quality has been observed in several countries, such as the United States, Europe, and Asia. Disturbances in sleep quality and quantity were associated with changes in human body homeostasis. These include changes in the hypothalamus-pituitary-adrenal system, sympathetic nervous system, insulin sensitivity, leptin, and ghrelin. These changes will induce reductions in energy expenditure and increases in energy intake, thereby contributing to weight gain and obesity.
in appetite, which, in turn, increase the risk for obesity and the development of NCDs.⁶⁻⁸ Investigating the role of sleep quality and duration of obesity is essential because obesity is responsible for an increasing economic and health burden worldwide.⁹,¹⁰ This study was aimed to evaluate the impact of sleep quality and duration on leptin, appetite, and obesity indices in Indonesian adults.

**METHODS**

This investigation was a cross-sectional study conducted among adult males and females in the urban area of Yogyakarta, Indonesia. The inclusion criteria were age between 18 and 56 years old, permanent resident of the area for at least 2 years, and agreement to become a subject of this study. Those diagnosed with having degenerative diseases by a medical doctor, were pregnant, and breastfeeding were excluded from this study. Subjects with a strict, prescribed diet or a problem performing physical activity in the last 6 months were also excluded from this study. This study has been approved by the Medical and Health Research Ethics Committee Faculty of Medicine, Universitas Gadjah Mada, Indonesia (KE/FK/791/EC/2015). All subjects provided informed consent before data collection.

Obesity indices included body mass index (BMI), waist-hip circumference, and body fat percentage. BMI was determined by dividing body weight (kg) by height (m) squared (m²). Body weight and percentage of body fat were measured using a digital body mass scale (Omron, Japan) with an accuracy of 0.1 kg. Height was measured using a wall-mounted tape measure (GEA Medical, Indonesia) with an accuracy of 0.1 cm. A non-elastic tape was used to measure the waist and hip circumferences with an accuracy of 0.1 cm.

Dietary intake, appetite, sleep quality, and sleep duration were measured by direct interviews. Dietary intake and food consumption were assessed using a semiquantitative food frequency questionnaire. The analysis was performed using the composition of nutrients from the Indonesian food database and the United States Department of Agriculture by the researchers (HFLM). The dietary intake was reported as the gram intake corrected for the total energy intake, gram/1,000 kcal. This was calculated by dividing grams of nutrient intake with total energy intake, and the result was multiplied by 1,000 kcal.

The sleep quality of subjects was determined by the Pittsburgh sleep quality index (PSQI).¹¹ The PSQI was chosen because of its reliability for evaluating sleep quality in a population.¹² An increase in the PSQI score shows a reduction in sleep quality. Sleep quality was determined as poor if the PSQI score >5 points. Sleep duration was the total hours spent sleeping in a previous 24-hour period.

Appetite was measured using the community nutrition appetite questionnaire.¹³ This questionnaire was developed to assess the appetite of individuals in a community setting. This questionnaire was initially designed for older adults but has been validated for younger individuals.

The measurement of leptin plasma was done under nonfasting conditions (DRG International Inc., USA). Ten ml of blood was collected in ethylenediaminetetraacetic acid-containing tubes. Within 2 hours after collection, the blood plasma anduffy coat were separated using a centrifuge. The plasma leptin was measured using an enzyme-linked immunosorbent assay (DRG International Inc.).

JASP software 0.8.3.1 was used for the statistical analysis (The University of Amsterdam, The Netherlands). Subjects were divided into two groups: those with poor sleep quality and those with good sleep quality. The difference in leptin, appetite, dietary intake, and obesity indices between good and poor sleep quality was analyzed using an independent t-test or Mann-Whitney test. The correlation between sleep quality, sleep duration, anthropometric measures, dietary, appetite, and leptin was evaluated by performing a Spearman analysis.

**RESULTS**

There were 244 adults who participated in this study. Their characteristics are shown in Table 1. The average BMI of the subjects was 25.1 kg/m² (14.5–46.4 kg/m²). In this study, 18.4% were obese, 25% were overweight, 50.4% were normal weight, and 6.1% were underweight. There were 59.8% subjects had poor sleep quality and 61.1% subjects had sleep duration <7 hours a day. Meanwhile, there were 47.9% who had both poor sleep quality and sleep duration <7 hours a day.

Subjects were equally distributed between males (47.5%) and females (52.4%). In comparison to females, males had a higher body weight (p = 0.020)
and height (p<0.001), but lower BMI (p<0.001), waist circumference (p = 0.001), hip circumference (p<0.001) and body fat (p<0.001). In addition, male subjects had better sleep quality (p = 0.032) but not sleep duration (p = 0.509).

The association between sleep quality, anthropometric measures, and leptin is presented in Table 1. Sleep quality was determined based on PSQI scores. Males were associated with better sleep quality than females (p = 0.028). We reported that poor sleep quality was related to higher BMI (p = 0.011), waist circumference (p = 0.008), hip circumference (p = 0.023), and body fat (p = 0.026).

In addition, poor sleep quality was associated with higher leptin concentration (p = 0.004). Appetite was not associated with sleep quality (p = 0.430). We also reported that dietary intake and typical food consumption were not associated with sleep quality (all p>0.05).

Correlation between sleep quality, sleep duration, anthropometric measures, leptin, appetite, and dietary intake were shown in Table 2. In this study, we showed that all anthropometric measures and leptin were positively correlated with the PSQI score. The correlation between sleep quality and appetite was not correlated.

### Table 1. Characteristics of subjects based on sleep quality

|                        | All subjects (N = 244) | Poor quality* (N = 146) | Good quality† (N = 98) | p     |
|------------------------|------------------------|--------------------------|-------------------------|-------|
| Age (years)†           | 41.4 (0.6)             | 42.0 (0.9)               | 40.6 (0.9)              | 0.276 |
| Sex (male/female)§     | 116/128                | 61/85                    | 55/43                   | 0.028 |
| Anthropometric measures‡ |                       |                           |                         |       |
| Body weight (kg)       | 62.6 (0.9)             | 63.8 (1.2)               | 60.8 (1.3)              | 0.081 |
| Height (cm)            | 158.2 (0.6)            | 157.5 (0.8)              | 159.1 (0.9)             | 0.196 |
| BMI (kg/m²)            | 25.1 (0.4)             | 25.8 (0.5)               | 24.0 (0.5)              | 0.011 |
| Waist circumference (cm)| 87.0 (0.9)             | 88.8 (1.2)               | 84.2 (1.2)              | 0.008 |
| Hip circumference (cm) | 94.2 (0.8)             | 95.6 (1.1)               | 92.0 (1.1)              | 0.023 |
| Body fat (%)           | 27.8 (0.6)             | 28.9 (0.8)               | 26.1 (0.9)              | 0.026 |
| Leptin (ng/ml)‡        | 7.8 (0.6)              | 9.3 (0.9)                | 5.7 (0.8)               | 0.004 |

SE=standard error; BMI=body mass index; PSQI=Pittsburgh sleep quality index

*Independent t-test; †based on PSQI; §Mann-Whitney test; §chi-square test

### Table 2. Correlation between sleep, anthropometric measures, leptin, appetite, and dietary intake

|                        | Sleep quality (PSQI score) | Sleep duration (hours) | r* | p     | r* | p     |
|------------------------|----------------------------|------------------------|----|-------|----|-------|
| Weight (kg)            | 0.129                      | 0.043                  | −0.228 | <0.001 |
| BMI (kg/m²)            | 0.176                      | 0.006                  | −0.202 | 0.001 |
| Waist circumference (cm)| 0.179                     | 0.005                  | −0.254 | <0.001 |
| Hip circumference (cm) | 0.185                      | 0.004                  | −0.191 | 0.003 |
| Body fat (%)           | 0.172                      | 0.007                  | −0.103 | 0.110 |
| Leptin (ng/ml)‡        | 0.186                      | 0.004                  | −0.095 | 0.141 |
| Appetite rating (CNAQ score) | 0.103                    | 0.109                  | −0.043 | 0.500 |
| Dietary intake (daily) |                           |                        |     |       |     |       |
| Energy (kcal)          | −0.052                     | 0.416                  | 0.067 | 0.295 |
| Protein (g/1,000 kcal) | −0.076                     | 0.239                  | 0.080 | 0.211 |
| Fat (g/1,000 kcal)     | −0.045                     | 0.489                  | 0.034 | 0.594 |
| Carbohydrate (g/1,000 kcal) | 0.075                    | 0.244                  | −0.050 | 0.437 |
| Fiber (g/1,000 kcal)   | 0.003                      | 0.962                  | 0.084 | 0.191 |
| Sugar (g/1,000 kcal)   | 0.038                      | 0.551                  | −0.032 | 0.622 |

PSQI=Pittsburgh sleep quality index; BMI=body mass index; CNAQ=community nutrition appetite questionnaire

*Spearman correlation test
DISCUSSION

We reported that poor sleep quality was associated with higher leptin and all obesity indices, but not appetite. In addition, this study showed a negative correlation between sleep duration and obesity indices. Our study indicated that sleep patterns might play a role in the development of obesity. Thus, achieving good sleep quality and sufficient sleep duration might be important lifestyle components to consider for obesity prevention.

Adults with poor sleep quality and duration were associated with higher adiposity indices. Findings from this study were similar to the findings of several studies that showed a correlation between sleep duration and body composition. It was previously reported that longer sleep duration was associated with lower BMI and body fat. This association was also reported in children and adolescents.

Several mechanisms have been investigated regarding the impact of sleep disturbances on the regulation of body weight. These mechanisms include altered metabolic parameters, appetite-related hormones, the sympathetic nervous system’s activity, appetite, and energy expenditure. Subjects with lower sleep duration, especially reduced rapid eye movement sleep and slow-wave sleep, were associated with a reduction in energy balance, leading to a higher chance of energy storage. In addition, shorter sleep duration has been associated with a reduction of leptin (satiety inducing hormone) and an incremental increase in ghrelin (hunger-inducing hormone), leading to an increase in appetite.

Sleep deprivation has been associated with a disruption in the activity of the appetitive evaluation region in the human brain. This condition, in addition to the up-regulation of orexin neurons and changes in appetite-regulating hormones, is followed by the increasing desire to eat, especially high-calorie foods. In this study, we showed that, sleep quality was not associated with appetite and dietary intake. It is argued that appetite is not the single driver of individual dietary intake. Some other factors might also play a role such as socioeconomic factors, age, and food availability.

There has been conflicting evidence regarding the association between sleep and plasma leptin concentration. In a cross-sectional study of individuals with obese and type 2 diabetes mellitus, Hirota et al reported that sleep quality was positively associated with leptin concentration. By contrast, Knutson et al reported that in obese males and females, leptin was not associated with sleep quality and duration. Several studies that supported our findings demonstrated that poor sleep quality was associated with higher leptin concentration. Van Leeuwen et al showed that prolonged sleep restriction was correlated with increased leptin concentration. Investigation in 443 police officers showed that sleep duration was associated with leptin concentration. This effect was seen in males and females. Leptin has an essential role in increased systemic inflammation. This also suggests that the increasing concentration of leptin because of poor sleep quality would potentially increase the risk for metabolic syndrome and cardiovascular diseases in the future.

In contrast to sleep quality, the relationship between sleep duration and leptin is unclear. A systemic review and meta-analysis recently reported the association between sleep restriction, leptin, obesity, hunger, and energy intake. It stated that sleep restriction was associated with weight gain, increased energy intake, and hunger. However, the meta-analysis concluded that there was no association between sleep restriction and leptin. It is argued that some factors, including circadian rhythm and the inert nature of leptin release, might influence its response to sleep restriction. It is still unclear how sleep quality and duration could affect leptin concentration. It has been suspected that shorter sleep duration could increase sympathetic activity and stress signals, which, in turn, could increase leptin concentration. The authors also suggested that this might be due to the reduction in leptin sensitivity because of poor sleep quality.

There were several limitations to this study. Subjects were in their free-living condition; thus, an objective sleep analysis cannot be used in this study. Instead, a subjective assessment of sleep quality and duration was used because of its reliability in a community setting. A causal-effect of both variables cannot be drawn in this study because of its cross-sectional design. Leptin was measured in a nonfasting condition because of the practicality for data collection in a community setting. This might influence the variability of leptin levels between subjects. To our knowledge, limited studies are investigating the relationship between sleep parameters and leptin in a free-living population-based study. Further studies are needed to evaluate
whether sleep improvement strategies could be used as an alternative to obesity prevention.

In conclusion, we reported that poor sleep quality and duration were associated with increased obesity indices among adults. Lower sleep quality also correlated with higher leptin. Lifestyle recommendations for obesity prevention should emphasize the importance of good sleep quality and sufficient sleep duration.

Conflict of Interest

The authors affirm no conflict of interest in this study.

Acknowledgment

We thank Mohammad Zaenal Sofro for his advice. His advice helped improve the design of this study. We also thank Cita Eri Ayuningtyas for her help during data collection.

Funding Sources

This study was funded by International Research Collaboration (IRec) Grant (IRec Grant No: 1002/PPSK/910408) (Universiti Sains Malaysia), Community Fund—Faculty of Medicine (Universitas Gadjah Mada), and Young Investigator Awards (Universitas Gadjah Mada).

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