general obesity, but also with increased subcutaneous fat mass, supporting a role of chronic sleep restriction in obesity pathogenesis.

Reviews of several cross-sectional and prospective studies among child and adult populations around the world have found fairly uniform results that short sleep duration is associated with obesity⁷ and weight gain. However, in their most recent review of prospective studies, Nielsen *et al.* concluded that short sleep duration is consistently associated with development of obesity in children and young adults, but the findings were less consistent in older adults. Furthermore, sleep duration was not associated with BMI in a population-based cohort study among Japanese aged 40 to 69 years, well as in a prospective multicenter cohort study among early-middle-aged adults (age range of 38 to 50 years) in the United States. In a German study, the significant association between short sleep duration and BMI did not persist after controlling for physical health and emotional status. In

This study is the first in the field to formally assess physical and psychiatric illnesses and to assess the relationship between sleep duration and obesity independent of these factors. These confounders may lead to a relationship in the opposite direction; obesity predisposes to physical or psychiatric illnesses, which in turn cause reduced sleep duration. Previous studies have ignored this explanatory pathway or attempted to address it by using selfreported data obtained from a single question on the overall physical and mental health of the participants.²¹ Such a measure is not sensitive and does not capture severity of the illnesses. In our analyses, we were not able to show a significant attenuation of the association between short sleep duration and obesity after excluding subjects with psychiatric illnesses and controlling for physical illnesses. These findings suggest that the association among our study population may not be explained by this pathway. Further studies are needed to investigate the possible confounding effects of physical and mental disorders on the relationship between sleep duration and obesity.

It is worth noting that short sleep duration did not show any significant association with general obesity and central abdominal fat areas among women in this study. Similar findings were also found in a study among a large Japanese working population in which no prospective association between sleep duration and obesity or weight gain was detected in women.¹⁹ This finding is also consistent with results obtained from other studies in western populations.²⁸ In contrast, a study in Spain showed that the significant association between sleep duration and weight gain was observed in women, but not in men.²⁰ However, direct comparison with men might be made with caution as the mean age of women in our study was roughly 5 years older than that in men. In the Zurich Cohort Study, the relationship between sleep duration and weight weakened as participants aged.²⁹ Furthermore, our bivariate results show that sleep duration was not related to any obesity-related characteristics in women.

Based on experimental studies of sleep deprivation, a number of causal pathways linking short sleep duration with obesity have been suggested. One mechanism by which sleep deprivation might predispose to weight gain is by increasing caloric intake. In short-term trials, sleep restriction leads to reduction in circulating leptin, elevations in ghrelin, subjective hunger and preferences for calorie-dense, refined-carbohydrate foods, ³⁰ which contribute to the development of obesity. Alternatively, some have argued that, in an environment where food is readily available, curtailed sleep may simply represent an increased opportunity to eat, especially if most of the wake-time is spent in sedentary activities such as watching television where snacking is common.³¹ Chronic sleep deprivation clearly leads to feeling fatigue that may in turn lead to obesity-related behavior including decreased energy expenditure, irregular eating habit and low consumption of fruits and vegetables.³² In addition, activation of inflammatory pathways by sleep restriction may also be implicated in the development of obesity.³³ The strengths of this study include the large sample size of men, the use of CT scanner to measure central abdominal fat areas and the comprehensive assessments of important covariates. The relationship between sleep duration and obesity may vary in association with underlying risk factors such as insomnia and psychological disorders that are potential comorbidities of sleep deprivation and other severe medical conditions that might affect body composition. With a broad variety of data obtained from a standardized collection, we were able to exclude subjects with a history of psychiatric illnesses, insomnia, stroke, myocardial infarction, cancer and diabetes mellitus. In this way, we extended previous findings by systematically assessing the association between sleep duration and obesity independent from the effects of these potential confounding factors.

Several limitations should also be recognized. First, because of the cross-sectional design, a causal relationship cannot be definitively established. However, experimental studies have confirmed that sleep restriction can have metabolic effects that may be relevant to weight homeostasis.³¹ Future studies should evaluate how changes in sleep duration are related to changes in weight and body fat composition over time. Second, daily sleep duration was self-reported, which is a continued limitation in sleep epidemiological studies. However, the Nurses' Health Study has shown a good validity for sleep duration measured by using a similar question against 1-week sleep diaries.34 Third, long sleepers (>8h) were not specifically separated from normal sleepers (7 to 8h). As a result, we were unable to examine the relation between long sleep and obesity, as many studies have reported a U-shaped association. 12,35 Furthermore, information regarding sleep duration did not allow us to distinguish the real 'sleep duration' and 'time in bed'. Finally, although we excluded subjects with history of insomnia, no adjustment was made for other important sleep disorders such as obstructive sleep apnea, which is presumed to play an important role in both sleep disruption and obesity.³⁶ Future research should examine whether obstructive sleep apnea accounts for the gender differences in the association between sleep duration and adiposity as previous studies found that Asian men appear to have an increased risk of obstructive sleep apnea at lower BMI levels than observed in Caucasian men.³⁷

In conclusion, our findings suggest that short sleep duration is associated not only with general obesity, but also with subcutaneous fat mass in Japanese working men. Further research is needed to further explicate the biological mechanisms behind this relationship and to see whether interventions addressing inadequate sleep or poor sleep quality could treat or prevent obesity by taking gender differences into consideration.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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