Short-term sleep loss decreases physical activity under free-living conditions but does not increase food intake under time-deprived laboratory conditions in healthy men1–4

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ABSTRACT
Background: Short sleep duration is correlated with an increased risk of developing obesity and cardiovascular disease, but the mechanisms behind this relation are largely unknown.
Objective: We aimed to test the hypothesis that acute sleep loss decreases physical activity while increasing food intake, thereby shifting 2 crucial behavioral components of energy homeostasis toward weight gain.
Design: In 15 healthy, normal-weight men, spontaneous physical activity was registered by accelerometry during the entire experiment, and food intake as well as relevant hormones were assessed during a 15-h daytime period after 2 nights of regular sleep (bedtime: 2245–0700) and after 2 nights of restricted sleep (bedtime: 0245–0700). Experiments were performed in a crossover design.
Results: Sleep restriction significantly decreased physical activity during the daytime spent under free-living conditions after the first night of sleep manipulation (P = 0.008). Also, intensities of physical activity were shifted toward lower levels, with less time spent with intense activities (P = 0.046). Total energy intake, feelings of hunger, and appetite as well as ghrelin and leptin concentrations during day 2 remained unaffected by acute sleep restriction.
Conclusions: In contrast to our expectation, short-term sleep loss neither increased food intake nor affected concentrations of the hunger-regulating hormones leptin and ghrelin. However, the observed decrease in daytime physical activity may point to another potentially important behavioral mechanism for the health-impairing influence of sleep loss. Am J Clin Nutr 2009;90:1476–82.

INTRODUCTION

The decrease in average sleep duration over the past century (1) has been paralleled by an increase in the prevalence of obesity (2, 3). Epidemiologic studies indicate an inverse relation between sleep duration and body mass index (BMI) in adults (4, 5) as well as in children and adolescents (6, 7). Although the relation between sleep loss and disturbances of energy homeostasis is the subject of extensive debate (8–10), only a few experimental studies have addressed the basis of the connection between sleep loss and risk factors for obesity. In a seminal study, Spiegel et al (11) showed that 2 consecutive nights of sleep restriction to 4 h instead of 10 h of sleep induced an 18% reduction in circulating concentrations of the hunger-suppressing hormone leptin in conjunction with a 24% elevation in concentrations of the appetite-stimulating hormone ghrelin. These hormonal changes were paralleled by markedly increased feelings of hunger and appetite. However, most recently, subchronic sleep restriction to 5 h/night for 14 d has been shown to not affect total energy intake and orexigenic/anorexigenic hormone balance, yielding only a relative increase in snack intake and raising the question of whether other critical factors of energy homeostasis may be sensitive to decreases in sleep duration (12). We investigated the effects of short-term sleep loss on spontaneous physical activity. We hypothesized that sleep restriction decreases physical activity, thus favoring a positive energy balance and, in the long run, weight gain. Surprisingly, to our knowledge, this hypothesis, although very plausible, has not been examined in humans so far. We also expected an orexigenic effect of sleep restriction on spontaneous food intake and circulating concentrations of leptin and ghrelin that were also assessed in our experiments.

SUBJECTS AND METHODS

Subjects

The study was carried out in a crossover design in 15 healthy, normal-weight men [mean (±SEM) BMI (in kg/m2): 22.9 ± 0.3] aged 20–40 y (27.1 ± 1.3 y) with a regular sleep-wake cycle during the 4 wk before the experiments. They were recruited via ads and flyers stating the inclusion criteria. A standardized interview on sleep habits revealed a habitual sleep duration of 459 ± 7 min (range: 450–540 min) with bedtime starting between 2200 and 0000 and wake-up time from 0600 to 0800. Exclusion

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2 The Deutsche Forschungsgemeinschaft had no influence on the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript.
3 Supported by the Deutsche Forschungsgemeinschaft, KFO 126 (“Selfish Brain”).
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Received April 26, 2009. Accepted for publication September 20, 2009.
criteria were chronic or acute illness, current medication of any kind, smoking, alcohol or drug abuse, obesity, and diabetes in first-degree relatives. Also, subjects who displayed a high level of cognitive control in the Three-Factor Eating Questionnaire (13) were excluded to ensure unrestrained eating behavior. Subjects were not informed about the assessment of eating behavior but were told that the study would focus on the effects of sleep restriction on cognitive functions that were repeatedly assessed throughout the experiments with a battery of cognitive tests (the results of these tests are not reported here). Thus, subjects were unaware that their food intake was monitored during the study. The study protocol was approved by the Ethics Committee on Research Involving Humans at the University of Luebeck, and all participants gave written informed consent before participation. The initial recruitment date was 1 April 2006.

Study design and procedure
After an adaptation night in the laboratory that included standard polysomnographical recordings, participants were tested 6 wk apart in a counterbalanced crossover design on 2 conditions. The experiments included 2 consecutive nights of 4 h of sleep (“4-h sleep”) and 2 consecutive nights of 8 h of sleep (“8-h sleep”). Experimental day 1 followed the first 4-h-sleep/8-h-sleep night and was spent under free-living conditions outside the laboratory with physical activity measured by accelerometry throughout the day. Thereafter, the second night of sleep manipulation was performed. After the second 4-h-sleep/8-h-sleep night, the assessment of physical activity, spontaneous food intake, blood variables, and symptom ratings was performed in a 15-h time-deprivation laboratory setting (day 2; see below).

Before each experimental night, subjects arrived at the research unit at 2000. They had been instructed to eat a light dinner before arrival. Thereafter, only water was allowed to be drunk until the next morning. After the installment of polysomnographic and accelerometric recordings, subjects went to bed and lights were turned off at 2245 in the 8-h-sleep condition. In the 4-h-sleep condition, subjects remained awake in a sitting position until 0245. They were allowed to read and to watch nonstimulating movies. Brisk physical activities were avoided, and subjects were constantly monitored by the experimenters. After each experimental night in both conditions, subjects were awakened at 0700.

After the first of the 2 consecutive experimental nights (night 1), subjects in both conditions received a standard breakfast and were allowed to leave the laboratory (day 1). They were instructed to not deviate from their usual eating habits and to avoid intense physical activities (eg, working out) and naps during the day. Subjects reported back to the laboratory at 2000 to be prepared for experimental night 2, the procedure and setting of which were identical to night 1.

After waking up the subjects at 0700 at the end of night 2, the laboratory assessment day (day 2) started. An intravenous catheter was inserted into a vein of the subject’s nondominant distal forearm to allow the drawing of blood samples at 0740 and thereafter at 1-h intervals between 0800 and the end of the experiment at 2300. Immediately before each blood drawing, on a semi-quantitative questionnaire subjects rated from 0 (none) to 9 (severe) each of the following 6 symptoms: hunger, appetite, satiety, activity, weakness, and tiredness. During the 15-h day-time assessment period (0800–2300), subjects were deprived of all time cues by removing all clocks, radios, and other time indicators from the sound-attenuated laboratory room (with adjacent bathroom) that had no natural light. Subjects were allowed to read and to play video games.

Sleep recordings
Recordings were performed with a Nihon Kohden amplifier (EEG 4400 series; Nihon Kohden GmbH, Rosbach, Germany) and were scored offline according to standard criteria (14). The following sleep variables were determined: total sleep time, time spent in sleep stage 1, 2, 3, 4, and slow-wave sleep (ie, sleep stage 3 + 4) and in REM sleep (all in min and % of total sleep time), time spent awake after sleep onset, and movement time (in % of total sleep time).

Physical activity
Physical activity was assessed by standard accelerometric recordings of wrist activity (Acti-Watch; Cambridge Neurotechnology, Cambridge, United Kingdom). Recordings of 3 subjects had to be excluded from analyses for technical reasons, ie, because of insufficient recording quality in at least one condition. Analyses of daytime physical activity included the time interval from 0800 to 2000 during day 1 (ie, under free-living conditions after night 1) and during day 2 (ie, when subjects stayed in the laboratory). The sampling interval was 1 min with a subsequent reduction of activity counts (AC) to 5-min intervals. Total activity counts comprise the sum of activity counts registered on the respective day. The intensity of physical activity was grouped into low (below mean: AC/5 min – 1 SD), middle (within mean: AC/5 min ± 1 SD), and high (above mean: AC/5 min + 1 SD) activity with reference to the recordings under the 8-h-sleep condition on day 1.

Food intake
At 0800 on day 2, subjects were presented with a large standardized breakfast buffet (5060 kcal) from which they were allowed to eat ad libitum. At 1100, the breakfast buffet was replaced by a snack buffet (5010 kcal at first serving), which remained in the experimental room until the end of the day (at 2300). Buffet components were refilled whenever necessary. In addition to the snack buffet, subjects could select main meals from a menu (1200 kcal at first serving) whenever they wanted to from 1100 on (for details on buffets and main meals, see Table 1). Food intake was measured outside the experimental room by weighing buffet components before serving and after clearing the table. Analyses of food intake were performed for the whole experimental day and separately for the standardized breakfast period (0800–1100) as well as for the snack buffet/main meals period (1100–2300). Nutritional analyses were performed with the use of a software program for macronutrient analyses (DGE-PC professional 3.3; Stuttgart, Germany), and buffet components and main meals were also analyzed with regard to different food categories according to recommendations of the German Nutrition Society (15). On the basis of the power calculations inferred from previous experiments on food intake (16), the sample size of 15 was sufficient to detect a significant
difference in food intake, assuming a study power of 0.95 at a significance level of 0.05.

Assays
Concentrations of serum leptin (Human Leptin RIA kit; Linco Research, St Charles, MO) and plasma ghrelin [ghrelin (total) RIA kit; Linco Research] were determined from stored (−80°C) samples by radioimmunoassays.

Statistical analyses
All values are expressed as means ± SEMs. Analyses of sleep data were based on an analysis of variance (ANOVA) for repeated measures, including the factors “condition” (for 4 h compared with 8 h of sleep) and “night” (for night 1 compared with night 2). Analyses of physical activity data were based on ANOVA for repeated measures, including the factors “condition” and “day” (for day 1 compared with day 2) and “activity level” (low-, middle-, and high-intensity physical activity). Analyses of hormonal data as well as symptom ratings were based on ANOVA for repeated measures, including the factors condition and time (for repeated measurements during day 2). Pairwise comparisons of single timepoint values were performed by using the Student’s t test. Analyses of food intake were based on ANOVA for repeated measures, including the factors condition and macronutrients (for the respective macronutrient composition of ingested food). Pairwise comparisons of single macronutrients as well as food categories were performed by using the Student’s t test. A P value <0.05 was considered significant. Analyses were run with SPSS 12.0 for Windows (SPSS Inc, Chicago, IL).

RESULTS

Sleep
During both nights of the 4-h-sleep condition, subjects slept on average ~229 min less than in the 8-h-sleep condition [236 ± 2 compared with 465 ± 4 min (first night) and 238 ± 1 compared with 467 ± 3 min (second night) for the 4-h and 8-h conditions, respectively; P < 0.001 for the condition main effect]. The longer sleep duration in the 8-h-sleep condition was primarily due to more pronounced shallow sleep—ie, S1 and S2—as well as REM sleep.

Physical activity and related self-reports
Overall analyses of accelerometric activity data obtained on days 1 and 2 revealed distinctly lower cumulative daytime AC in the 4-h-sleep than in the 8-h-sleep condition (P = 0.021 for the condition main effect). In general, subjects were markedly more active under the free-living conditions of day 1 than under the laboratory conditions of day 2 (P < 0.001 for the day main effect) with this difference being independent of the sleep condition (P = 0.12 for the condition × day interaction effect; Figure 1A). Separate analyses of day 1 and day 2 revealed distinctly lower cumulative AC after 4 h than after 8 h of sleep during day 1 (43,622 ± 4713 compared with 50,190 ± 4554; P = 0.008), whereas on day 2, against the background of overall greatly decreased activity, this difference was not significant (14,688 ± 1814 compared with 16,177 ± 1799; P = 0.52). Including the factor activity level in the ANOVA models revealed a significant condition × activity level interaction on day 1 (P = 0.12 for the condition × day interaction effect; Figure 1A). Separate analyses of day 1 and day 2 revealed distinctly lower cumulative AC after 4 h than after 8 h of sleep during day 1 (43,622 ± 4713 compared with 50,190 ± 4554; P = 0.008), whereas on day 2, against the background of overall greatly decreased activity, this difference was not significant (14,688 ± 1814 compared with 16,177 ± 1799; P = 0.52). Including the factor activity level in the ANOVA models revealed a significant condition × activity level interaction on day 1 (P = 0.12 for the condition × day interaction effect; Figure 1A).
activities (22.6 ± 3.5% compared with 25.4 ± 3.4%; P = 0.044) than after 8 h of sleep (Figure 1B). Throughout day 2, the self-reported level of activity was lower after the 2 nights of 4-h sleep than after the 2 nights of 8-h sleep (P = 0.002 for the condition main effect; Figure 1C). A roughly corresponding pattern was revealed for feelings of weakness (P = 0.019) and tiredness (P = 0.001; Figure 1D).

Food intake, ratings of appetite, hunger, and satiety and related hormones

Total energy intake during the entire experimental day 2 did not differ between the 4-h-sleep and the 8-h-sleep condition (Table 2). Although subjects consumed relatively more fat in the 4-h-sleep than in the 8-h-sleep condition and analyses of food categories revealed a higher intake of food belonging to the category “fat” in the 4-h-sleep condition (394 ± 43 compared with 305 ± 46 kcal; P = 0.029), ANOVA did not reveal a significant condition × macronutrient interaction (P = 0.31). Note that consumption of sweet and salty snacks did not differ between the 4-h-sleep and the 8-h-sleep condition (1169 ± 151 compared with 1246 ± 184 kcal and 461 ± 101 compared with 520 ± 98 kcal, respectively; P > 0.54). Separate analyses of the breakfast buffet and postbreakfast energy intake again did not reveal any differences in energy intake or macronutrient composition (all P > 0.64), except for a trend toward increased fat intake after breakfast in the sleep loss condition (P = 0.06; Table 2).

Ratings before breakfast did not show any differences in appetite (5.4 ± 0.5 compared with 4.7 ± 0.5; P = 0.17), hunger (4.7 ± 0.5 compared with 4.4 ± 0.6; P = 0.68), and satiety (1.3 ± 0.3 compared with 1.0 ± 0.5; P = 0.62) between the 4-h-sleep and 8-h-sleep condition. During breakfast, ratings of appetite (Figure 2A) and hunger decreased rapidly and remained at rather low levels for the rest of the experiment (P < 0.001 for the time main effects and P > 0.29 for the condition × time interaction of both variables). Satiety ratings mirrored those of appetite and hunger and likewise

### TABLE 2

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
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<th>Snacks and main meals</th>
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<tr>
<td><strong>Total energy intake (kcal)</strong></td>
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<tr>
<td>4-h sleep</td>
<td>3969 ± 258</td>
<td>1471 ± 121</td>
<td>2496 ± 220</td>
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<tr>
<td>8-h sleep</td>
<td>4070 ± 285</td>
<td>1498 ± 127</td>
<td>2572 ± 237</td>
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<td>P</td>
<td>0.70</td>
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<td><strong>Fat (%)</strong></td>
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<td></td>
<td>34.0 ± 1.3</td>
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<td>P</td>
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<td>0.68</td>
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<tr>
<td><strong>Carbohydrate (%)</strong></td>
<td>50.5 ± 1.1</td>
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<td>55.8 ± 1.2</td>
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<td></td>
<td>51.7 ± 1.9</td>
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<tr>
<td>P</td>
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<td>0.90</td>
<td>0.44</td>
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<tr>
<td><strong>Protein (%)</strong></td>
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<td>14.7 ± 0.6</td>
<td>13.3 ± 1.0</td>
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<tr>
<td></td>
<td>14.3 ± 0.8</td>
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<td>13.9 ± 1.3</td>
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<tr>
<td>P</td>
<td>0.61</td>
<td>0.58</td>
<td>0.72</td>
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</table>

1 All values are means ± SEMs. n = 15. P values are derived from Student’s t tests. ANOVA did not show significant condition × macronutrient interactions.
did not display differences between conditions ($P < 0.001$ for time, $P = 0.44$ condition $\times$ time, and $P > 0.11$ for pairwise single-timepoint comparison of respective symptom ratings during day 2). As shown in Figure 2, B and C, sleep restriction had no effect on serum and plasma concentrations, respectively, of leptin and ghrelin (for statistical comparisons, see also Figure 2, B and C).

**DISCUSSION**

Our data show that short-term sleep loss reduces daytime overall spontaneous physical activity and shifts the intensity of physical activity toward lower levels under free-living conditions. In contrast to previous results (9) and to our expectations, short-term sleep loss affected neither food intake and self-rated hunger and appetite nor circulating concentrations of leptin and ghrelin. Collectively, our results significantly add to the still limited knowledge of the health-impairing influences of sleep loss and probably point to more diverse underlying mechanisms than previously suggested.

Although intuition and everyday experience may label this finding as obvious, the reduction of physical activity and the shift toward less intense activities as measured by wrist accelerometry is a novel and highly interesting result. A reduction of time periods spent at high physical activity levels can be assumed to, in the long run, reduce physical fitness (17), thereby increasing the risk of metabolic diseases such as obesity (18) and diabetes (19) as well as cardiovascular events (20, 21). Although to our knowledge similar results have not been obtained in humans before, canines display markedly reduced motor activity after 1 d of total sleep deprivation (22). It should be noted that wrist accelerometry as measured in our study, although highly correlated with whole-body trunk movements (23), does not represent total-body physical activity and in particular does not allow any reliable conclusion on daily energy expenditure (24). Thus, our finding cannot necessarily be taken as an indicator of a reduction in energy expenditure. Also, the reduction in self-reported physical activity has to be cautiously interpreted because sleep-loss-induced fatigue might have biased the subjective perception of activity. However, these obvious limitations should not distract from the important and dependable finding of markedly reduced physical activity that certainly can be expected to adversely affect health.

Circulating leptin concentrations were previously shown to be reduced and ghrelin concentrations to be elevated after a roughly comparable sleep restriction regimen of 4 h in 2 consecutive nights (11). This apparent contrast to our result of unchanged circulating concentrations of leptin and ghrelin may be due to subtle, but probably essential, differences in the designs of both studies. Although sleep duration in the respective sleep loss conditions was comparable ($\approx 237$ compared with $\approx 233$ min; reference 11), sleep duration in our 8-h-sleep control condition was on average $\approx 80$ min shorter than in the previous study in which subjects were actually tested in an extended sleep condition ($\approx 466$ compared with $\approx 543$ min). There is some evidence that the effects of sleep loss on leptin and ghrelin concentrations follow a dose-dependent relation (4, 25, 26). For example, ghrelin concentrations are distinctly elevated in the morning after 1 night of total sleep deprivation as compared with regular sleep, whereas concentrations after 4.5 h of sleep

**FIGURE 2.** Mean ($\pm$SEM) subjective ratings of (A) appetite and concentrations of (B) serum leptin and (C) plasma ghrelin during an experimental day after 2 nights of 8 h of sleep/night (open circles) and 2 nights each containing 4 h of sleep (solid circles), respectively. Fasting prebreakfast concentrations of leptin (4-h sleep, 2.9 $\pm$ 0.5 ng/mL, compared with 8-h sleep, 3.0 $\pm$ 0.7 ng/mL; $P = 0.79$, Student’s t test) and ghrelin (4-h sleep, 603 $\pm$ 39 pg/mL, compared with 8-h sleep, 615 $\pm$ 47 pg/mL; $P = 0.57$) were not altered by preceding sleep restriction. ANOVA showed that during the experimental day leptin concentrations increased ($P < 0.001$ for time) but did not display differences between conditions ($P = 0.21$ for condition, and $P = 0.18$ for condition $\times$ time). Concentrations of ghrelin rapidly decreased during breakfast and remained at low concentrations during the rest of the experimental day ($P < 0.001$ for time). Again, there was no effect of sleep restriction ($P = 0.22$ for condition, and $P = 0.86$ for condition $\times$ time), $n = 15$. 

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are intermediate but not significantly different from regular sleep (26). Thus, in the present study the difference in sleep duration between conditions may have been too small to elicit detectable effects on the concentrations of ghrelin and leptin. Furthermore, in our study the time point of awakening was identical in both conditions (0700), whereas in the foregoing studies, sleep restriction was associated with a somewhat early awakening at 0500 in contrast to ~0800 in the sleep-extension condition. Therefore, subjects in the sleep loss condition had been awake ~3 h longer than in the control condition before the first hormone measurements were obtained at 0900. Such an additional fasting period spent awake may be an important factor in the sleep-dependent regulation of leptin and ghrelin.

In accordance with unchanged leptin and ghrelin concentrations, self-rated hunger and appetite were not affected by sleep shortage. Moreover, we were unable to detect differences in total energy intake. The proportion of ingested fat was slightly greater after sleep loss, but this finding must be interpreted with caution because it is not buttressed by a significant statistical interaction between treatment and macronutrient composition. Our subjects overall ingested a rather high amount of energy that on average exceeded their estimated daily energy demand by ~60% (27). A recent study has shown that food intake critically depends on the amount of food provided to a subject (28). Presented with a great variety of highly palatable foods, our subjects likely displayed a ceiling effect that might have masked more subtle effects of sleep loss on spontaneous energy intake. However, sleep restriction already failed to affect food intake during breakfast, ie, at a time when potential ceiling effects were presumably less pronounced. This suggests that, provided that the time of awakening is comparable to normal sleep, sleep loss does not acutely increase food intake in the morning.

In conclusion, our study indicates a profound deteriorating influence of sleep deprivation on physical activity that in the long-run possibly adversely affects metabolic and cardiovascular health. It should be noted that our observations have been obtained in an acute setting and cannot be directly extrapolated to the effects of long-term sleep loss. Nevertheless, in conjunction with a previous series of cogent experimental studies (25, 29, 30) and a growing number of epidemiologic reports (4, 31, 32), our results point to reduced physical activity as another potentially important behavioral mechanism linking sleep loss to the development of obesity, type 2 diabetes, and cardiovascular disease.

We are grateful to Mareike Kück, Elisa Gustke, Claudia Frenzel, Jutta Schwanbohm, and Kathleen Kurwahn for their expert and invaluable laboratory assistance.

The authors’ responsibilities were as follows—SMS, MH, KJ-C, JB, and BS: designed the study; SMS, MH, BW, JB, and BS: analyzed the data; SMS, MH, BW, KJ-C, CB, HL, JB, and BS: contributed to writing the manuscript; and SMS: collected data and performed experiments for the study. All authors had full access to all data in the study and take responsibility for the integrity and accuracy of the data analysis. None of the authors had a conflict of interest.

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