Erratum

Taku K, Umegaki K, Sato Y, Taki Y, Endoh K, Watanabe S. Soy isoflavones lower serum total and LDL cholesterol in humans: a meta-analysis of 11 randomized controlled trials. Am J Clin Nutr 2007;85:1148–56.

On page 1151, the values listed in the "Sample size" column (the values for "Total population" for total cholesterol, LDL cholesterol, HDL cholesterol, and triacylglycerol for the comparison between ISP+ and ISP−) are incorrect. Instead of "185 vs 283," the values should read "285 vs 283."

Erratum

Vieth R, Bischoff-Ferrari H, Boucher BJ, et al. The urgent need to recommend an intake of vitamin D that is effective. Am J Clin Nutr 2007;85:649–50.

An incorrect e-mail address was listed for Reinhold Vieth. The correct address is rvieth@mtsinai.on.ca.

Erratum

Bischoff-Ferrari HA, Giovannucci E, Willett WC, Dietrich T, Dawson-Hughes B. Estimation of optimal serum concentrations of 25-hydroxyvitamin D for multiple health outcomes. Am J Clin Nutr 2006;84:18–28.

In the penultimate sentence in the abstract, the microgram value listed for vitamin D should be 25 μg instead of 40 μg. The sentence should read as follows: "An intake for all adults of $\geq$1000 IU (25 μg) vitamin D (cholecalciferol)/d is needed to bring vitamin D concentrations in no less than 50% of the population up to 75 nmol/L."

Erratum

Afaghi A, O'Connor H, Chow CM. High-glycemic-index carbohydrate meals shorten sleep onset. Am J Clin Nutr 2007;85:426–30.

On page 428, Table 1, the values listed in the "Low-GI meal, 4 h" column are incorrect for 6 of the sleep variables. The correct values (%) are as follows: 5.1 $\pm$ 2.0 (Sleep stage 1), 56.2 $\pm$ 6.2 (Sleep stage 2), 5.5 $\pm$ 2.1 (Sleep stage 3), 14.1 $\pm$ 4.2 (Sleep stage 4), 81.0 $\pm$ 4.8 (NREM sleep), and 19.1 $\pm$ 4.8 (REM sleep).

The American Journal of Clinical Nutrition

High-glycemic-index carbohydrate meals shorten sleep onset[1–3]

Ahmad Afaghi, Helen O'Connor, and Chin Moi Chow

ABSTRACT

Background: Dietary carbohydrate intake has been shown to increase the plasma concentration of tryptophan, a precursor of serotonin and sleep-inducing agent.

Objective: To investigate the role of carbohydrate in sleep induction, we explored the effect of glycemic index (GI) and meal time on sleep in healthy volunteers.

Design: We compared the effect of high- and low-GI carbohydrate–based meals ingested 4 h before bedtime on sleep quality. We also evaluated the effect of the timing of high-GI meals (4 h compared with 1 h) on sleep quality. Twelve healthy men (aged 18-35 y) were administered standard, isocaloric (3212 kJ; 8% of energy as protein, 1.6% of energy as fat, and 90.4% of energy as carbohydrate) meals of either Mahatma (low GI = 50) or Jasmine (high GI = 109) rice 4 h before their usual bedtime. On another occasion, the high-GI meal was given 1 h before bedtime. The participants underwent a familiarization night followed by 3 test nights in random order 1 wk apart.

Results: A significant ($P = 0.009$) reduction in the mean (±SD) sleep onset latency (SOL) was observed with a high-GI (9.0 ± 6.2 min) compared with a low-GI (17.5 ± 6.2 min) meal consumed 4 h before bedtime. The high-GI meal given 4 h before bedtime showed a significantly shortened SOL compared with the same meal given 1 h before bedtime (9.0 ± 6.2 min compared with 14.6 ± 9.9 min; $P = 0.01$). No effects on other sleep variables were observed.

Conclusions: We showed that a carbohydrate-based high-GI meal resulted in a significant shortening of SOL in healthy sleepers compared with a low-GI meal and was most effective when consumed 4 h before bedtime. The relevance of these findings to persons with sleep disturbance should be determined in future trials. *Am J Clin Nutr* 2007;85:426–30.

KEY WORDS Carbohydrates, glycemic index, sleep quality, sleep timing

INTRODUCTION

Common sleep difficulties include sleep initiation and maintenance. According to The Gallup Organization, 49% of adults in the United States do not sleep well for ≥5 nights/mo, 10–40% have intermittent insomnia, and 10–15% have long-term sleep difficulties (1). In Australia, a survey reported an insomnia prevalence of 17% in men and 25% in women in an urban community (2).

The current treatment options for insomnia are pharmacotherapy and cognitive behavioral therapy. Treatments are considered effective if they shorten sleep onset latency (SOL) or increase total sleep time by 30 min (3). Cognitive behavioral therapy is considered the best practice. Other popular remedies used to treat sleep difficulties include prescribed sedatives and tranquilizers, herbal extracts and complimentary medicines, massage and relaxation techniques, regular physical activity, and avoidance of stimulants such as caffeine before sleeping.

Both the timing (4, 5) and macronutrient content (6–9) of meals are known to influence sleep. A meal consumed close to bedtime is associated with sleep disturbance (4). A number of macronutrients influence sleep through tryptophan (Trp), which serves as a precursor for brain serotonin, a sleep-inducing agent (10, 11). A factor that promotes the entry of Trp into the brain is its plasma concentration relative to that of the other large neutral amino acids (LNAAs: tyrosine, phenylalanine, leucine, isoleucine, valine, and methionine) (12). It is now known that high-glycemic-index (GI) carbohydrates have the ability to increase the ratio of circulating Trp to LNAAs (Trp:LNAA) via a direct action of insulin, which promotes a selective muscle uptake of LNAAs (13). Thus, a high-GI meal would be expected to promote sleep via an increase in brain Trp and serotonin as the plasma Trp:LNAA increases (12). It would also be expected that a meal containing a high protein content, which contributes less Trp to the circulating blood compared with the other LNAAs (12) and thus a lower plasma Trp:LNAA, would reduce serotonin. Serotonin function may be measured indirectly through changes in melatonin concentrations, because serotonin is an intermediary product in the production of melatonin, a pineal hormone (14). Urinary 6-hydroxymelatonin sulfate, a stable end product of melatonin, is often used as a surrogate measure of melatonin, given their linear relation (14).

Therefore, the aim of the present study was to investigate the role of carbohydrate in inducing sleep, and specifically the effect of GI on sleep patterns in healthy sleepers. We hypothesized that carbohydrate-based high- compared with low-GI meals ingested 4 h before bedtime would improve sleep quality because of a greater insulin response and that the timing of the high-GI meal (4 h compared with 1 h) before bedtime would also influence sleep quality.

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[2] Supported by Sydney University's PhD student research budget. The rice was provided by Riviana Food Pty Ltd, Victoria, Australia.

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SUBJECTS AND METHODS

Subjects

Twelve healthy, young (aged 18–35 y), normal weight (body mass index: 18.5–25 kg/m^2) men were recruited from among 50 subjects screened. Subjects were excluded if they had a self-reported current or past history of significant medical, psychiatric, or sleep disorders (nocturnal eater inclusive), used prescribed medication (including sedatives or antidepressants) or recreational drugs, or regularly had an alcohol intake >20 g (2 drinks)/d on average, or exercised vigorously 24 h before a sleep study. The study was approved by the Human Research Ethics Committee of Sydney University.

Procedure

All subjects completed a medical evaluation questionnaire and a 2-wk sleep diary before undergoing a familiarization night of full sleep study (polysomnography). The subjects were given each of the following 3 standardized meals in a randomized order 1 wk apart: a high-GI meal administrated 4 h before their usual bedtime, a high-GI meal administrated 1 h before their usual bedtime, and a low-GI meal ingested 4 h before their usual bedtime. On the testing day, the subjects fasted for 5 h before the standardized meal. After voiding, the subjects ate their meals in 15 min. Finger prick blood samples for glucose analysis (with the use of a glucometer, Medisense Optium; Abott Laboratories, MediSense Products, Bedford, MA) were collected before the meal (baseline) and at 15, 30, 45, 60, 90, and 120 min after the high- and low-GI meals ingested 4 h before bedtime, and after high-GI meal ingested 1 h before bedtime (samples were collected at 15, 30, and 45 min before bedtime).

Urine for analysis of 6-sulfatoxymelatonin (6-SM) was collected in 2 parts: after the meal until bedtime and bedtime through to the next morning. The volume of collected urine and time period were recorded, and 2 aliquots of each volume were stored frozen (−80 °C) for 6-SM analysis with a commercially available enzyme-linked immunoassay kit (ImmunoBiological Laboratories, Hamburk, Germany). A 10-cm visual analogue scale (VAS) was checked on a 4-point scale, from extremely hungry to extremely full, before and after each meal to assess the subjects' hunger and fullness. Subjective feelings of sleepiness were assessed at 30 min and at 1, 2, 3, and 4 h after each meal by marking appropriately on a 10-cm 4-point sleepiness scale VAS from zero ("not at all sleepy") to +3 ("very sleepy"). The validity and reliability of this VAS sleepiness scale has been shown (15).

Meals

Standard isocaloric meals (3212 kJ; 8% of energy as protein, 1.6% of energy as fat, and 90.4% of energy as carbohydrate) included 600 g steamed rice (200 g raw) and 200 g steamed vegetables in tomato puree. The rice was either low (Mahatma long grain, Riviana Foods Inc, Sydney Australia; GI = 50) or high (Jasmine aromatic long grain; Riviana Foods Inc; GI = 109) GI (16). The glycemic load (GL) was calculated as (GI/100) × g available carbohydrate. A higher GL meal results in a greater elevation of blood glucose and insulin (16). The GL was 81.3 and 175 for the low- and high-GI meals, respectively.

Polysomnographic recording

A full polysomnography with an international 10–20 electrode placement (C3/A2, O2/A1) (17) and respiratory recordings (to exclude sleep breathing disorders) and leg electromyogram (to exclude limb movement disorders; Compumedics S-series Sleep system; Compumedics Ltd, Melbourne, Australia) were applied on the familiarization night. Subsequently on study nights, sleep electroencephalogram, electrooculogram, and electromyogram only were recorded. The sleep studies were scored by an expert sleep physiologist (CMC) who was blinded to the treatments. Sleep recordings were evaluated for variables of total sleep time (TST); sleep efficiency (SE); SOL; arousal index; non–rapid eye movement sleep stages 1, 2, 3, and 4; and rapid eye movement (REM) sleep.

Statistics

The sample size was calculated based on effect sizes obtained for sleep latency (0.82) and sleep efficiency (2.18) powered at 80% and an alpha of 5% in an intervention study of tryptophan-free drink on sleep (18). We estimated that an effect size of 1.6 and a calculated sample size of 12 were appropriate for our crossover design study. Data were inspected for normality of distribution before use of parametric statistics with SPSS version 13 (SPSS Inc, Cary, NC). Data are reported as means ± SDs. Sleep indexes were analyzed by repeated-measures analysis of variance (ANOVA) and test of within-subjects contrasts to determine the effect of the 3 treatments on sleep. Data obtained for sleepiness and hunger or fullness by VAS were analyzed by paired Student's *t* test. Urine 6-SM was analyzed by repeated-measures ANOVA to examine the effects of the high- and low-GI meals given 4 h before bedtime. The blood glucose response was analyzed by using the area under the curve (AUC) and paired Student's *t* test. The relation between blood glucose AUC and SOL was analyzed via Pearson's correlation coefficient for both the high- and low-GI (4 h) meals. The repeated-measures ANOVA was used to test for the effect of meal type, time post-meal, and their interaction on blood glucose measured at baseline and at 15, 30, 45, 60, 90, and 120-min intervals after eating.

RESULTS

Sleep variables

Sleep was ad libitum, with usual bedtime falling between 2145 to 0030 [$\bar{x}$ (±SD) time: 2330 h ± 52 min], and the timing was consistent for each subject for each study night. The mean values for all sleep variables are shown in **Table 1**. SOL was significantly different between the low- and high-GI meals given 4 h before bedtime and between the high-GI meal given 4 h and 1 h before bedtime. Mean SOL was reduced by 8.5 ± 9.3 min after the high-GI meal compared with the low-GI meal given 4 h before bedtime ($P = 0.009$) and by 5.6 ± 6.3 min when the high-GI meal was eaten 4 h before bedtime compared with 1 h ($P = 0.01$). Note that all subjects showed a reduction in SOL except for one (who showed an increase) (**Figure 1**). The other sleep indexes were not significantly different between meals of varying GI or with respect to the timing of the high-GI meal ingestion (Table 1).

Sleepiness scale

A rating of 2.0 corresponded to "sleepy" on the VAS scale. The greatest postprandial differences of sleepiness rating following the test meals occurred at bedtime. The subjects tended to feel sleepier and less awake after the high-GI meal ingested 4 h before

TABLE 1
Effect of the glycemic index (GI) and timing of meals on sleep[1]

Sleep variable	High-GI meal, 1 h	High-GI meal, 4 h	Low-GI meal, 4 h	P^2	P^3
SOL (min)	14.6 ± 9.9[4]	9.0 ± 6.2	17.5 ± 6.2	0.01	0.009
ROL (min)	84.1 ± 39.8	97.0 ± 33.2	82.6 ± 35.7	0.32	0.16
SE (%)	92.0 ± 2.2	92.4 ± 2.7	90.7 ± 2.7	0.63	0.06
Arousal index (no./h)					
REM	15.4 ± 10.4	15.4 ± 7.8	14.5 ± 7.1	0.99	0.70
NREM	12.1 ± 5.3	10.7 ± 4.5	10.6 ± 5.8	0.15	0.93
Total	12.6 ± 5.1	11.5 ± 4.2	11.4 ± 5.3	0.32	0.95
Sleep stage 1 (%)	5.7 ± 2.0	6.3 ± 2.8	17.5 ± 6.2	0.37	0.08
Sleep stage 2 (%)	56.2 ± 5.4	54.5 ± 4.8	82.6 ± 35.7	0.18	0.43
Sleep stage 3 (%)	5.3 ± 2.0	4.7 ± 2	90.7 ± 2.7	0.37	0.17
Sleep stage 4 (%)	14.7 ± 5.3	14.9 ± 7.2	17.5 ± 6.2	0.82	0.64
NREM sleep (%)	81.9 ± 4.3	80.6 ± 4.5	82.6 ± 35.7	0.15	0.77
REM sleep (%)	18.0 ± 4.3	19.4 ± 4.5	90.7 ± 2.7	0.14	0.75
Total sleep time (min)	478 ± 68.7	472.0 ± 66.4	464.1 ± 70.1	0.78	0.74
Total wake time (min)	26.0 ± 9.0	27.6 ± 7.55	29.3 ± 12.7	0.66	0.59

[1] $n = 12$. SOL, sleep onset latency; ROL, rapid eye movement latency; REM, rapid eye movement; SE, sleep efficiency (the ratio of total sleep time in bed); NREM, non–rapid eye movement. Sleep stages and proportion of NREM and REM sleep are presented as a percentage of total sleep time.
[2] Comparison of high-GI meal given 4 h and 1 h before bedtime.
[3] Comparison between high-GI meal ingested 4 h before bedtime with low-GI meal ingested 4 h before bedtime.
[4] $\bar{x}$ ± SD (all such values).

bedtime (2.3 ± 0.6) than they did after the low-GI meal ingested 4 h before bedtime (2.1 ± 0.3, $P = 0.1$). However, they were significantly sleepier after the high-GI meal ingested 4 h before bedtime than after that ingested 1 h before bedtime (1.9 ± 0.5, $P = 0.04$).

Hunger and fullness scale

The VAS rating of hunger or fullness confirmed that the large rice serving given was adequate so that subjects were not hungry after the meal. Respective ratings immediately after the meal and at bedtime were 3.21 and 2.17 for the low-GI meal ingested 4 h before bedtime; 3.17 and 2.13 for high-GI meal ingested 4 h before bedtime; and 3.17 and 2.25 for the high-GI meal ingested 1 h before bedtime. On a 4-point scale, a rating of 3 indicates that the subject feels "full" after the meal. These ratings were not significantly different between the high- and low-GI meals ingested 4 h before bedtime ($P = 0.67$ and $P = 0.72$ after the meal and at bedtime, respectively), between the high-GI meals ingested 4 h and 1 h before bedtime ($P = 1.0$ and $P = 0.28$ after the meal and at bedtime, respectively), and between the low-GI meal ingested 4 h before bedtime and the high-GI meal ingested 1 h before bedtime ($P = 0.72$ and $P = 0.17$ after the meal and at bedtime, respectively).

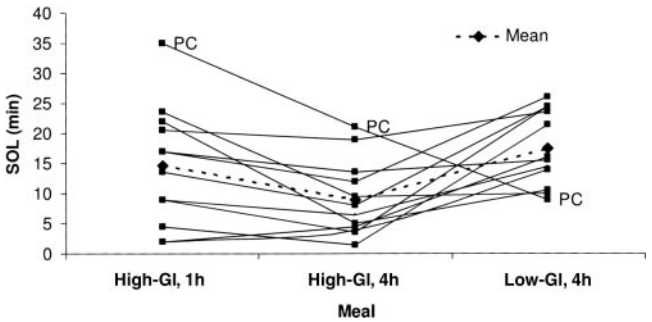

FIGURE 1. Comparison of sleep onset latency (SOL) between the high-glycemic-index (GI) meal ingested 1 h or 4 h before bedtime and the low-GI meal ingested 4 h before bedtime. PC, subject who showed an inappropriate trend in the SOL for the 3 meals. The mean (±SD) SOLs for the low-GI meal given 4 h before usual bedtime and the high-GI meals given 4 h and 1 h before the usual bedtime were 17.5 ± 6.2, 9.0 ± 6.2, and 14.6 ± 9.9 min, respectively ($n = 12$). The SOL was significantly shortened after the high-GI meal compared with the low-GI meal when given 4 h before usual bedtime ($P = 0.009$). When considering the timing of the high-GI meals, the SOL of the meal ingested 4 h before bedtime was significantly shortened compared to the meal ingested 1 h before bedtime, $P = 0.01$ (repeated-measures ANOVA).

6-SM analysis

The evening collection concentration of 6-SM, a metabolite of melatonin, showed no significant differences between the high-GI meal ingested 4 h before bedtime (661.8 ± 228.1 ng/h; CV = 34%), the high-GI meal ingested 1 h before bedtime (556.4 ± 209.8 ng/h; CV = 37%; $P = 0.3$), and the low-GI meal ingested 4 h before bedtime (602.4 ± 208.8 ng/h; CV = 34%; $P = 0.5$). Higher concentrations of 6-SM, as expected, were seen for the night collection (1783.6 ± 618.8 ng/h; CV = 34%; and 1718.5 ± 484.0 ng/h; CV = 28%; $P = 0.6$, and 1571.6 ± 482.6 ng/h, CV% = 30%; $P = 0.2$, respectively).

Blood glucose

The blood glucose response to both the high- and low-GI meals is shown in **Figure 2**. Blood glucose rose to a peak at ≈30 and 45 min after meal ingestion, followed by a steady decrease. The repeated-measures ANOVA confirmed that the blood glucose profiles over time differed between the 2 meal types ($P = 0.001$ for the group by time interaction). The AUC was significantly greater for the high-GI (336.2 ± 61.9) than for the low-GI (237.1 ± 69.3) meal (Student's t test, $P = 0.009$). The blood glucose response to ingestion of a high-GI meal 1 h before bedtime was similar to that observed after ingestion of a high-GI

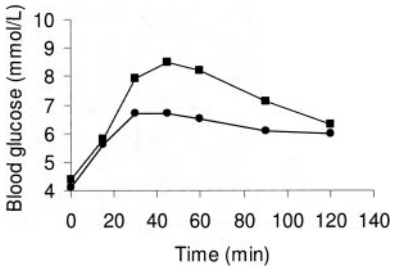

FIGURE 2. Blood glucose response to meals with a high (■) and low (●) glycemic index (GI) given 4 h before bedtime. The interaction of meal type and time after meal ingestion was significant ($P = 0.001$). Significant differences between the area under the curves for the glucose response to the high- and low-GI meals were observed: 336.2 ± 61.9 and 237.1 ± 69.3, respectively ($P = 0.009$; $n = 8$).

meal 4 h before bedtime for time points up to 45 min. No relation was observed between the subjects' glucose AUC and their SOL either for the high-GI meal ($r = 0.2$, $P = 0.6$) or the low-GI meal ($r = -0.3$, $P = 0.5$).

DISCUSSION

To our knowledge, this is the first study that showed a significant effect of glycemic index on SOL. We found that a high-GI meal given 4 h before bedtime significantly shortened SOL by 48.6% compared with a low-GI meal given 4 h before bedtime or by 38.3% compared with the same meal given 1 h before bedtime. If replicated in a cohort with sleep disturbance, such a nonpharmacologic approach to the management of insomnia may prove valuable, given the notorious side effects of medications used to achieve this outcome (19).

It is worth noting that one subject (PC) (Figure 1) showed an inappropriate trend in the SOL for the 3 meals. Further examination of his sleep data revealed a reduction in REM sleep, a delay in ROL, and an increase in stage 2 after both high-GI (4 h and 1 h) meals. These data contrasted those for the mean values obtained for the other 11 subjects. As an elite cyclist, PC claimed to have ceased all exercise for ≥24 h before each of the sleep studies, and thus fulfilled inclusion criteria for the present study. Although inappropriate to delete PC from the data, it is known that changes to sleep variables are associated with acute strenuous exercise (20). In the case of PC, 24 h cessation of exercise may have been insufficient to normalize sleep indexes.

The mechanism by which a high-GI carbohydrate meal shortens SOL is currently unknown, but it is possible that the high-GI meal works through an increased plasma concentration of insulin and Trp to large neutral amino acid ratio (Trp:LNAA) and its ability to compete for entry into the brain with other LNAAs. The entry of Trp into the brain is linked to its concentration relative to other LNAAs and the main determinant of brain serotonin concentration is a high plasma Trp:LNAA (21). It is now known that the plasma Trp:LNAA is affected by both dietary carbohydrates and dietary protein (12, 22).

Once in the brain, Trp is converted to serotonin, which is also spontaneously produced in the raphe system of the brainstem (10). Serotonin is necessary for normal sleep of mammals (10). Ingestion of L-Trp significantly reduces the SOL of insomniacs and that of healthy sleepers (10). Indeed, several studies and review articles of the effect of Trp on sleep (4, 11, 23) support the notion that Trp reduces SOL without affecting other sleep variables. Hartmann (11) revealed a 45% reduction in SOL after Trp loading compared with placebo. These findings are consistent with our data from the high-GI meal ingested 4 h before bedtime, which showed a SOL reduction of 48.6% compared with the low-GI meal ingested 4 h before bedtime. The VAS data also lent support for the SOL findings. The subjects were significantly sleepier after the high-GI meal ingested 4 h before bedtime than after that ingested 1 h before bedtime, and there was a trend toward being sleepy in a comparison between the high- and low-GI meals ingested 4 h before bedtime.

A high-GI carbohydrate meal hastens glucose entry into the blood and facilitates a greater insulin response in healthy subjects (24, 25), in noninsulin dependent diabetic subjects (26), and in obese insulin-resistant subjects (27). In response to an increasing plasma glucose concentration, insulin mediates the uptake of LNAAs into muscle, but not Trp that is largely bound to plasma albumin (25, 28), thus leaving a high Trp:LNAA. In our study, although we did not measure the insulin response, we provided evidence for a significantly higher blood glucose response after a high-GI meal compared with the low-GI meal (Figure 2). The GL of the high-GI meal of 175 in the present study was substantial compared with 81.3 for the low-GI meal. The higher the GL, the greater the insulinogenic effect (16). Meals with a GL >20 are considered to be high, and over a day a GL of 120 is rated as high (29). Hence, the GL of the high-GI meal was 1.5 times higher than the GI most persons would consume in an entire day. The test meals were almost entirely composed of rice and had low protein content. High protein content would have confounded the GI effects seen in the present study. Even the low-GI meal had a GL of 81.3, which is still high for a meal and around the cutoff (GL < 80/d is considered low) between high and low over a day. In the future, the effect of mixed meals with high and low GI or GL would be important, because regular consumption of meals with such a high GL would not be suitable for persons with diabetes or obesity (30–32).

The study conducted by Lyons and Truswell (25) showed that carbohydrates with a high GI induce a greater increase in the plasma Trp:LNAA than do carbohydrates with a low GI. Accordingly, Trp entry into the brain in response to a higher Trp:LNAA thus would predict a greater synthesis of serotonin. Indeed, it has been shown that a high Trp:LNAA when compared with a low ratio resulted in a significant increase of platelet-poor plasma serotonin in humans (33) and brain serotonin in rats (21). Although we did not measure serotonin directly, we did measure nocturnal urinary 6-SM, a byproduct of melatonin. 6-SM has been previously shown to be a valid measure of melatonin secreted under conditions of decreased serotonin function (14). The fact that serotonin acts as an intermediary product in pineal melatonin synthesis (34) means that melatonin metabolism directly reflects changes in serotonin function. A significant linear relation between plasma Trp and urinary 6-SM has been established (14). That study showed that after Trp depletion, the 6-SM concentration fell accordingly. In contrast, Trp supplementation led to a rise in 6-SM. In our study, the higher urinary 6-SM (per h) observed for the whole night collection than the evening collection is consistent with a peak melatonin secretion that occurs between 0200–0400, as would be expected of its metabolite. The highest concentration of 6-SM was observed after the high-GI meals (ingested 4 h and 1 h before bedtime), followed by the low-GI meal (ingested 4 h before bedtime). Although not statistically significant,

these results were consistent with our hypothesis. It is likely that our study was underpowered for this outcome.

The timing of the high-GI meal also affected sleep onset in our study. The high-GI meal ingested 4 h before bedtime was more effective in shortening the SOL than was the same meal ingested 1 h before bedtime. The effectiveness of the timing of meals on SOL relates to the plasma appearance of Trp and LNAAs after ingestion of a high-carbohydrate or -protein meal. Previous studies have shown that Trp:LNAA peaked around 2–4 h after ingestion of a high-carbohydrate meal, and the ratio reached a trough around 4 h after ingestion of a high-protein meal (12, 33, 25, 35). Thus, these findings support our observations that a high-GI carbohydrate meal with low protein content, consumed at 4 h before bedtime, had a greater effect on shortening the SOL than that consumed at 1 h before bedtime. The fact that a high-GI meal eaten 1 h before bedtime can also have an effect on SOL is important, because different work and lifestyle demands often render meals being ingested late at night close to bedtime.

In the present study, the determinant dietary factor of GI was simply the type of rice being eaten. The fact that a simple manipulation of food intake can significantly improve sleep onset lends itself to a possible convenient, inexpensive, and noninvasive therapy for treating difficulty with sleep initiation. Future research that explores the potential benefit of manipulating the GI or GL of meals for persons with sleep disturbance is warranted.

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AA, HOC, and CMC designed the study. AA and HOC planned the meal test. CMC, who was blinded to the treatments, scored all sleep studies. AA implemented the study protocol and did the statistical analysis of data. CMC and HOC oversaw the writing of the manuscript. No author had any financial interest in the organization supported this research. AA is a doctoral candidate at EXSS. HOC is a lecturer in Nutrition and CMC is a senior lecturer with a research focus on sleep at EXSS.

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