Psychoneuroendocrinology (2013) 38, 1668—1674

Available online at www.sciencedirect.com

SciVerse ScienceDirect

journal homepage: www.elsevier.com/locate/psyneuen

Acute sleep deprivation increases portion size and affects food choice in young men☆

Pleunie S. Hogenkamp[a,*], Emil Nilsson[a], Victor C. Nilsson[a], Colin D. Chapman[a], Heike Vogel[b], Lina S. Lundberg[a], Sanaz Zarei[a], Jonathan Cedernaes[a], Frida H. Rångtell[a], Jan-Erik Broman[a], Suzanne L. Dickson[b], Jeffrey M. Brunstrom[c], Christian Benedict[a,1], Helgi B. Schiöth[a,1]

[a] Department of Neuroscience, Uppsala University, 751 24 Uppsala, Sweden
[b] Department of Physiology/Endocrinology, The Sahlgrenska Academy at the University of Gothenburg, 405 30 Gothenburg, Sweden
[c] Nutrition and Behaviour Unit, School of Experimental Psychology, University of Bristol, Bristol BS8 1TU, UK

Received 3 December 2012; received in revised form 25 January 2013; accepted 25 January 2013

KEYWORDS
Sleep loss;
Sleep;
Satiety;
Food intake;
Ghrelin;

Summary Acute sleep loss increases food intake in adults. However, little is known about the influence of acute sleep loss on portion size choice, and whether this depends on both hunger state and the type of food (snack or meal item) offered to an individual. The aim of the current study was to compare portion size choice after a night of sleep and a period of nocturnal wakefulness (a condition experienced by night-shift workers, e.g. physicians and nurses). Sixteen men (age: 23 ± 0.9 years, BMI: 23.6 ± 0.6 kg/m^2) participated in a randomized within-subject [illegible] orning using levels of ghrelin were measured. In the morning after TSD, subjects had increased plasma ghrelin levels (13%, $p = 0.04$), and chose larger portions (14%, $p = 0.02$), irrespective of the type of food, as compared to the sleep condition. Self-reported hunger was also enhanced ($p < 0.01$). Following breakfast, sleep-deprived subjects chose larger portions of snacks (16%, $p = 0.02$), whereas the selection of meal items did not differ between the sleep interventions (6%, $p = 0.13$). Our results suggest that overeating in the morning after sleep loss is driven by both homeostatic and hedonic factors. Further, they show that portion size choice after sleep loss depend on both an individual's hunger status, and the type of food offered.

☆ Supported by the Swedish Research Council, Åhlens stiftelse, Swedish Brain Research Foundation, and Forskning och Utvecklingsarbete/Avtal om Läkarutbildning och Forskning Göteborg. The study is registered with www.ClinicalTrials.gov (NCT01730742).

* Corresponding author at: Department of Neuroscience, Uppsala University, Box 593, SE-751 24 Uppsala, Sweden. Tel.: +47 18 47 14 123.
E-mail addresses: pleunie.hogenkamp@neuro.uu.se (P.S. Hogenkamp), christian.benedict@neuro.uu.se (C. Benedict).

[1] Contributed equally.

0306-4530
http://dx.doi.org/10.1016/j.psyneuen.2013.01.012

1. Introduction

Sleep loss has been linked to increased appetite (Taheri et al., 2004), enhanced food intake (Bosy-Westphal et al., 2008; Nedeltcheva et al., 2009; Brondel et al., 2010; St-Onge et al., 2011), higher circulating levels of the orexigenic hormone ghrelin (Spiegel et al., 2004; Taheri et al., 2004; Schmid et al., 2008; Nedeltcheva et al., 2010; Benedict et al., 2011), greater brain reward activation in response to food images (Benedict et al., 2012; St-Onge et al., 2012a), decreased energy expenditure (Benedict et al., 2011), and reduced physical activity (Schmid et al., 2009). In combination, these findings could explain why people who experience irregular or poor quality sleep such as night-shift workers are more prone to gain weight than those with normal sleep/wake patterns (Patel et al., 2006; Schiöth et al., 2012). However, not all laboratory studies have demonstrated increases in food intake following sleep loss (Schmid et al., 2009), and a recent meta-analysis indicated only small to moderate enhancing effects of sleep deprivation on acute energy intake (Chapman et al., 2012). One reason for this wide range in effect sizes might be that the level of food intake under experimental condition increases with the amount of food provided (Rolls et al., 2002; Steenhuis and Vermeer, 2009). Thus, offering a large amount of foods to sleep deprived participants may stimulate eating beyond satiety, thereby potentially masking modest effects of sleep deprivation on food intake.

Recently, it has been argued that the control of meal size is more likely to be expressed in decisions about portion size, before a meal begins (Fay et al., 2011). Accordingly, rather than focusing on ad libitum intake, we sought to explore the effects of sleep deprivation using a computer-based task (Brunstrom et al., 2008) as a measure of self-selected portion size. The task was administered to 16 healthy, normal-weight men following either a night of 8-h sleep or total sleep deprivation (TSD), both before and after breakfast. Previous studies have shown that TSD affects ghrelin concentrations (Spiegel et al., 2004; Schmid et al., 2008; Benedict et al., 2011). Thus, additionally, plasma levels of total ghrelin were measured. It was hypothesized that the selected portion size would be larger after TSD than after one night of sleep (Bosy-Westphal et al., 2008; Nedeltcheva et al., 2009; Brondel et al., 2010; St-Onge et al., 2011), and that this difference in portion size would be greater for snack items than for meal items (Nedeltcheva et al., 2009). Further, we expected that these effects would remain after breakfast.

2. Methods

2.1. Participants

Based on power calculations and previous experiments on sleep loss and food intake, 16 healthy normal-weight subjects (body mass index 23.6 $\pm$ 0.6 kg/m^2, age 23 $\pm$ 0.9 years) who were nonsmokers were included in the study. In an interview prior to the experiment, all subjects reported that they had a regular sleep–wake rhythm (i.e. 7–8 h per night), were not on shift work, ate breakfast regularly ($\geq$5 times per week), and were not on medication. Exclusion criteria included a history of endocrine or psychiatric disorders, irregular bedtimes, or sleep complaints. The study was approved by the Regional Ethical Review Board in Uppsala (EPN), and the procedures followed were in accordance with the Helsinki Declaration. The study is registered with www.ClinicalTrials.gov (NCT01730742). All participants gave written informed consent, and were paid for participation in the study.

2.2. Study design and procedure

In a randomized and balanced within-subject design, all subjects participated in two experimental conditions that were at least spaced apart by 28 days: sleep (i.e. 8-h sleep opportunity) and total sleep deprivation (TSD).

Each experimental session started with a 28.5-h baseline period. Upon arrival at 1800 (day 0), subjects received a standardized dinner at 2000 h (ready-to-eat meal with minced meat, baked potatoes, and vegetable; 945 kcal). Following a baseline night of 8-h sleep opportunity (i.e. 2230–0630 h), on day 1, participants received breakfast at 0800 h (pancakes, yogurt, and juice; 494 kcal), a warm lunch at 1300 h (ready-to-eat meal with chicken, rice, and vegetable; 462 kcal), a snack at 1530 h (2 cinnamon buns; 119 kcal/piece), and a warm dinner at 2000 h (ready-to-eat meal with minced meat, baked potatoes, and vegetable; 945 kcal) – all in fixed amounts, and two 30-min walks (at 1000 h and 1500 h). The baseline period in each experimental session was used to ensure that participants had a normal sleep–wake cycle before sleep interventions (i.e., sleep and TSD respectively), to control for physical activity and food intake, and to habituate the subjects to the experimental setting.

The baseline period was followed by a nighttime intervention period (2230–0630 h) in which subjects slept or stayed awake. In the morning after both sleep and TSD (i.e. day 2), subjects were requested to rate their perceived hunger on a 100-mm visual analog scale (VAS) at 0700 h and blood was sampled at 0730 h. Following the blood sampling procedure, participants conducted the portion size task (description below) in a fasted state (i.e. $\sim$0750 h), and again rated their hunger. At 0800 h, they received a breakfast comprising a yogurt drink (Gainomax Recovery Vanilj; Norrmejerier, Umeå) poured into 2 glasses (500 ml in total; 100 kcal/100 g) and 2 bars of crisp bread (Wasa Sandwich Cream Cheese Naturell; Barilla Sverige AB, Stockholm) (75 kcal/bar), providing 650 kcal in total. This meal was consumed in its entirety within 10 min, and subjective hunger feelings were again assessed after breakfast at 0830 h. Following this, participants once more conducted the second portion size task, i.e. a second time but now in sated state.

2.3. Portion size task

The ideal portion size was measured using the 'method of adjustment' (based on Brunstrom et al., 2008), in which pictures of 13 commonly consumed foods were shown on a laptop: 7 meal items including spaghetti Bolognese, penne and tomato sauce, rice curry (chicken tikka masala), boiled potatoes, oven fries, baguettes with garlic and herb butter, and cheese and tomato pizza; and 6 snack items including cashew nuts, pretzels, 'Pringles' (savory snack; Proctor and Gamble), banana, 'KitKat' (chocolate confectionary;

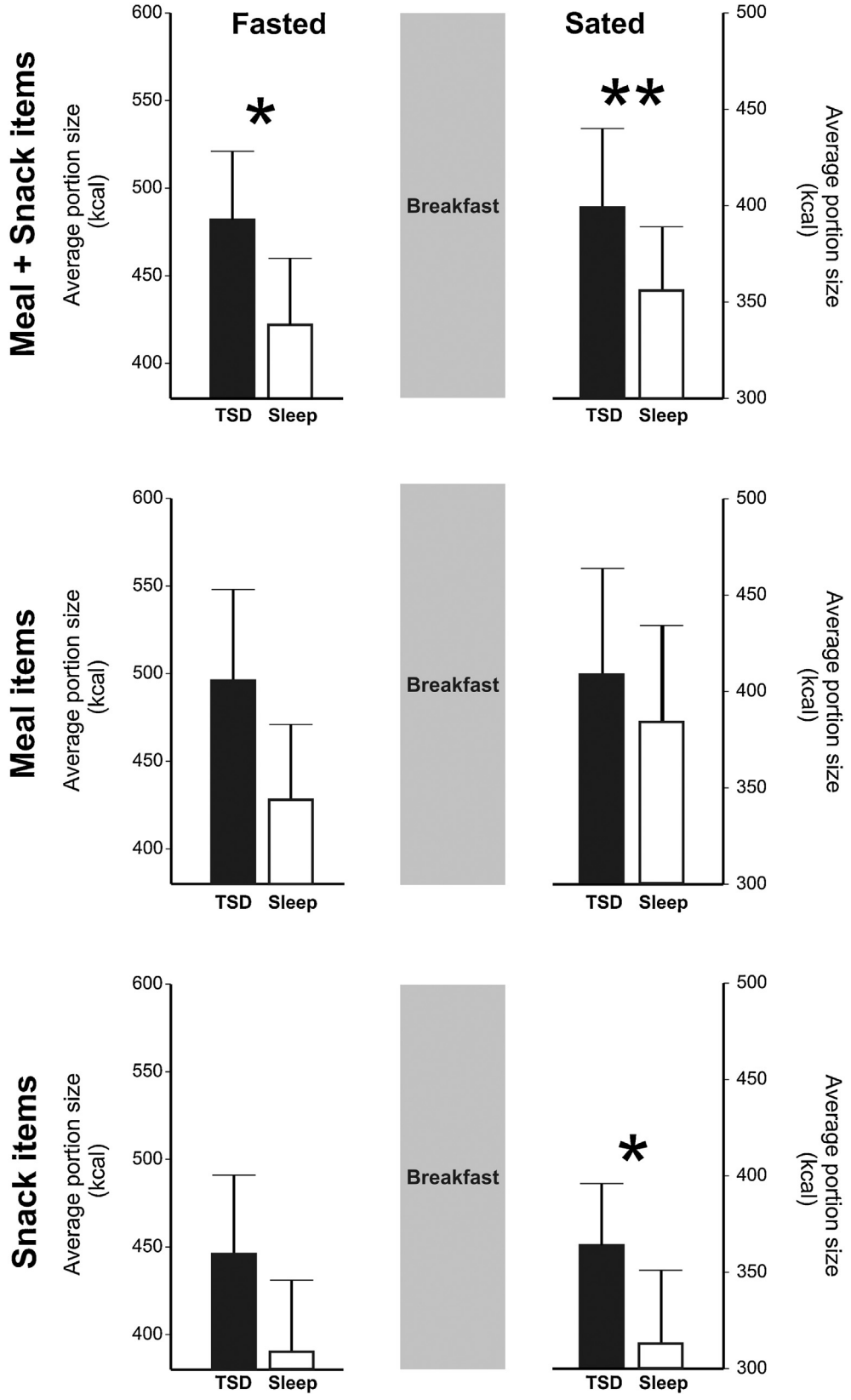

Figure 1 Selected portion size (kcal; mean ± SEM) of the most-liked food items following total sleep deprivation (TSD; black bars) and a night of sleep (white bars). The individual's most-liked food items with the highest pleasantness ratings were selected based on a median split for all food categories, i.e. 7 of all the food items ('meal + snack items'), 4 of the meal items ('meal items') and 3 of the snack items ('snack items'). The portion size task was administered both before ('fasted') and after ('sated') a breakfast of 650 kcal ($n = 16$). TSD vs. sleep (*$p < 0.05$; **$p < 0.01$).

Nestlé), and peanut M&Ms (chocolate confectionary; Mars). Participants were familiar with all of these foods. Participants were asked to 'imagine that they would have the food on the screen right now', and to 'select their ideal portion size' for 13 food items, comprising 7 meal items and 6 snack items. Ideal portion size for each food item was selected by using the arrow keys on the keyboard, i.e. depression of the left and right arrow keys caused a decrease and increase in the portion shown, respectively. The pictures were loaded with sufficient speed that the change in portion size appeared animated. The amount that could be displayed ranged from 83 kcal to 750 kcal. A total of 51 pictures were used to display these amounts. Picture number 25 displayed a 250 kcal portion. Picture number 0 displayed 0.3 times and picture number 50 displayed 3 times this amount. The order of appearance of these 13 food items was randomized across participants, and each trial (i.e. each food item) started with a different and randomly selected amount for each food. Importantly, participants were informed that this task was a mock test, and that they would not be given the food to eat after the test. For each participant, a mean portion size (kcal) was calculated based on the response to all food items interest (i.e. a mean of all 13 food items; a mean of meal items; and a mean of snack items). A previous study has shown that the computer-based task used here to assess portion size has been validated against selection and consumption of real foods (Wilkinson et al., 2012). Moreover, it has been demonstrated that portion size is a reliable predictor of food intake (Rolls et al., 2002; Wansink et al., 2005).

In order to assess individual differences in food preferences, participants rated pleasantness ('how much do you like this food in general?') of the 13 test food items (showing the 250-kcal portion) on the baseline day (after breakfast) of their first experimental session on 100-mm VAS, anchored 'not at all' to 'extremely'.

2.4. Sleep interventions

In the nights with sleep opportunity, lights were turned off at 2230 h, and switched on the next morning at 0630 h. Polysomnography was performed by use of Embla A10 recorders (Flaga hf, Reykjavik, Iceland) and comprised electroencephalography (EEG), electrooculography (EOG), and electromyography (EMG). Sleep stages were determined according to standard criteria (Rechtschafen and Kales, 1968) by an experienced scorer blinded to the study hypothesis. To keep subjects awake during the nocturnal vigil, they were provided with a selection of movies, games, and books, given access to bottled water, and the lights were kept on. Participants were continuously monitored by the experimenters to ensure wakefulness.

2.5. Assessments

For the assessment of plasma concentrations of total ghrelin, blood was sampled at 0730 h in the morning following the sleep intervention. Blood samples were immediately centrifuged and frozen at $-80\,^{\circ}C$ until analysis. A commercially available ELISA was used for assaying total ghrelin (EZGRT-89K; Millipore, Billerica, MA, USA).

2.6. Data analysis

In order to account for inter-individual differences in food preferences, as well as the amount of food an individual might be satisfied by, our primary statistical analysis testing the influence of acute total sleep deprivation (TSD) on portion size choice was run for the most-liked items. To this aim, we selected the individual's most-liked food items with the highest pleasantness ratings, based on a median split, i.e. 7 of all the food items, 4 of the meal-items and 3 of the snack items. In a secondary step, all food items (7 meal and 6 snack items, respectively) were entered into the statistical analyses. Overall, ANOVA was used to explore the effects of sleep interventions (sleep vs. TSD) and/or the breakfast provided (fasted vs. sated) on the selected portion size, perceived hunger ratings, and hormonal data. A recent meta-analysis has shown that sleep loss increases food intake in humans (Chapman et al., 2012). Further, following total sleep deprivation, increased morning fasting ghrelin concentrations have been observed in healthy young men (Schmid et al., 2008; Benedict et al., 2011). With this in mind, we predicted that both selected portion size and fasting ghrelin would be increased after TSD as compared with values obtained after one night of sleep. Thus, weighted contrasts were used for pairwise comparisons between the sleep and TSD conditions. All data are presented as means $\pm$ SEM. $P < 0.05$ was considered significant. Data were analyzed using SAS (version 9.5; SAS Institute Inc.).

3. Results

3.1. Sleep recordings

In the sleep intervention night of the sleep condition, sleep characteristics were as follows: total sleep time: 442 ± 6 min; wake: 30 ± 6 min; sleep stage 1: 5 ± 1 min; stage 2: 219 ± 11 min; slow-wave sleep: 115 ± 6 min; rapid eye movement (REM) sleep: 103 ± 8 min. Sleep-onset latency was 8 ± 4 min, and sleep efficiency was $92 \pm 1\%$. Sleep recordings in the baseline period showed similar results, and did not differ between conditions (data not shown).

3.2. Effects of total sleep deprivation on selected portion size of most-liked food items

Fig. 1 shows the mean selected portion size (in kcal) of the most-liked food items. Portion size was larger after TSD compared to sleep (main effect TSD/sleep $p < 0.01$), and larger in fasted compared to the sated state (main effect breakfast $p < 0.001$). In the fasted state (TSD vs. sleep, all food items: 482 ± 39 vs. 422 ± 38 kcal, $p = 0.02$), the effect of TSD was not specific for the food item category (TSD vs. sleep, meal items: 496 ± 52 vs. 428 ± 43 kcal, $p = 0.06$; snack items: 446 ± 45 vs. 390 ± 41 kcal, $p = 0.08$). Following breakfast, the portion size was again larger after TSD compared to sleep in the sated state (TSD vs. sleep, all food items: 399 ± 41 vs. 356 ± 33 kcal, $p < 0.01$). However, in contrast to the portion size choice pattern before the breakfast, subjects selected larger portions of snack but not meal items in their TSD condition (TSD vs. sleep, snack items:

364 ± 32 vs. 313 ± 38 kcal, $p = 0.02$; meal items: 408 ± 55 vs. 384 ± 50 kcal, $p = 0.13$).

3.3. Effects of total sleep deprivation on selected portion size of all food items

Descriptive results can be found in Table 1. Repeating the analysis including all 13 food items revealed a similar results pattern. The portion size (in kcal) was larger after TSD compared to sleep (main effect TSD/sleep $p = 0.01$). While this effect was significant after breakfast (TSD vs. sleep, all foods 322 ± 34 vs. 303 ± 33 kcal, $p < 0.01$), it was not in the fasted state (TSD vs. sleep, 400 ± 36 vs. 359 ± 31 kcal, $p = 0.07$). In line with the results obtained from the analysis including the most-liked food items, subjects again selected larger portions of snack but not meal items in their TSD condition when sated (TSD vs. sleep, snack items: 277 ± 20 vs. 242 ± 29 kcal, $p = 0.02$; meal items: 361 ± 51 vs. 351 ± 45 kcal, $p = 0.13$).

3.4. Effects of total sleep deprivation on hunger sensations

Participants reported higher hunger ratings after TSD than after sleep. Hunger ratings (mm) for wakefulness vs. sleep were 76 ± 7 vs. 61 ± 6 mm at 0700 h ($p < 0.01$), and 80 ± 4 vs. 69 ± 6 mm at 0800 h ($p = 0.04$). Hunger reductions following the breakfast were similar for both TSD (37 ± 6 mm) and sleep (36 ± 5 mm; $p = 0.45$), and reported hunger at 0830 h remained higher following TSD (43 ± 7 mm) as compared to sleep (33 ± 7 mm; $p = 0.02$).

3.5. Effects of total sleep deprivation on plasma levels of ghrelin

Plasma levels of total ghrelin were higher after TSD (442 ± 61 pg/ml) than after a night of sleep (390 ± 44 pg/ml; $p = 0.04$).

4. Discussion

These results demonstrate that overnight wakefulness, a condition that is typically observed in night-shift workers, increases both feelings of hunger and plasma ghrelin levels the following morning. Furthermore, participants selected larger portions following sleep deprivation as compared to normal sleep, and importantly, this effect was not specific to the type of food offered. A breakfast (650 kcal) reduced subjective hunger sensations equally in both conditions. However, post-consumption, sleep-deprived subjects selected larger portions only of snack items. These results demonstrate that both an individual's hunger state, as well as the specific type of food that is available, are relevant for portion size decisions after sleep deprivation. Thus, when determining the effect of sleep deprivation on food intake in humans, it is important to take into account experimental conditions, such as test meal composition, food liking, and hunger state.

The fact that sleep deprivation increased portion size in the fasted state irrespective of the type of food offered suggests that overeating following sleep loss may represent a homeostatic compensatory response. This homeostatic response is likely produced to compensate for the energy deficit that results from sleep deprivation (Jung et al., 2010): it has been observed that nocturnal wakefulness, as compared to a day including a nocturnal 8-h sleep episode, produces a ~7% increase in 24-h energy expenditure. Moreover, when subjects stay awake overnight, the physiologic decline in core body temperature (highly correlated with the basal metabolic rate; Silva, 2006) that typically emerges after sleep onset is dampened (Benedict et al., 2011), and concomitantly, the central nervous system's energy needs are increased (Boyle et al., 1994).

However, the added effect of sleep loss on portion size when sated was observed only for snack items, suggesting the existence of a secondary, non-homeostatic mechanism for sleep-loss induced consumption. In previous studies, sleep deprivation has been shown to increase the overall brain responses to palatable food images (Benedict et al., 2012; St-Onge et al., 2012a). These effects were particularly pronounced in areas involving reward processing, including the nucleus accumbens, thalamus, insula, and anterior cingulate cortex. Additionally, and in line with our results, in one of these functional magnetic resonance imaging (fMRI) studies, these effects were obtained following a caloric load (Benedict et al., 2012). Bearing in mind this consistency of evidence, the overeating associated with sleep loss may be the result of two independent mechanisms: a homeostatic drive to compensate for nocturnal energy deficits, and an increase in reward system susceptibility to calorically dense foods. Importantly, whereas the homeostatic drive thus can be reset by a caloric load, e.g. a breakfast, this does not appear to be the case for the portion size choices driven by the reward system.

Our results are in line with studies that observed an increase in ad libitum intake after loss of sleep

Table 1 Selected portion size (kcal; mean ± SEM) of all food items, meal items, and snack items after total sleep deprivation (TSD) and after a night of sleep ($n = 16$).

	All food items		Meal items		Snack items	
	TSD[a]	Sleep[b]	TSD[a]	Sleep[b]	TSD[a]	Sleep[b]
Fasted	400 ± 36[c]	359 ± 31[d]	455 ± 48[c]	400 ± 36[d]	337 ± 30	310 ± 29
Sated	322 ± 34[e]	303 ± 33[f]	361 ± 51	351 ± 45	277 ± 20[e]	242 ± 29[f]

[a,b] Portion size was larger after TSD as compared to a night of sleep (main effect sleep: $p < 0.05$ for all categories).
[c,d] Portion size was larger after TSD as compared to a night of sleep in the fasted state ($p = 0.07$ for all foods, $p = 0.06$ for meal items).
[e,f] Portion size was larger after TSD as compared to a night of sleep in the sated state for all food items and snack items ($p < 0.01$).

(Bosy-Westphal et al., 2008; Nedeltcheva et al., 2009; Brondel et al., 2010; St-Onge et al., 2011), and support the observation that sleep loss may increase energy intake of snacks in particular (Nedeltcheva et al., 2009). This latter study, however, reported differences in snack intake particularly in the hours after an evening dinner (i.e. between 1900 h and 0700 h), which included most of the extra waking hours in which no food other than snack items was available. In the current study, we did not measure actual food intake and food items were not readily available in the current study. However, screen-based measures of ideal portion size were significantly related to intake (Wilkinson et al., 2012), and earlier studies showed that portion size is a good predictor of food intake (Rolls et al., 2002; Wansink et al., 2005). One of the strengths of using the portion size task in the current study is that this avoided giving the participants the opportunity to overeat from a buffet, or from only one available food type. Also, the sleep interventions were preceded by a baseline period to minimize potential noise in the data produced by spontaneous food intake and/or exercise, which may affect subsequent eating behavior (Laan et al., 2010).

Our results are also in agreement with previous studies showing consistently that sleep-deprivation induces increases in plasma ghrelin concentrations (Taheri et al., 2004; Schmid et al., 2008). Ghrelin plays an important role in the short-term control of food intake: ghrelin administration stimulates a quick and robust increase in consumption (Druce et al., 2005). Moreover, intravenous infusions of ghrelin increase neural activation in response to food in brain areas involved in hedonic appetitive control, such as the striatum (Malik et al., 2008), suggesting that ghrelin is likely to promote an increase in the brain reward response to food. This neuroendocrine mechanism may thus underly both the homeostatic and hedonic control of food intake following sleep deprivation. However, we did not determine ghrelin concentrations in the sated state, and therefore the role of ghrelin in the selection of a larger portion size, as observed in the fasted compared to sated states, requires further investigation.

Several limitations apply: people who usually do not have breakfast might react differently to TSD than breakfast eaters. Second, we studied normal-weight healthy men, and generalization to women may not be appropriate (St-Onge et al., 2012b). Third, the aspect of different chronotypes may be relevant. Fourth, participants were informed that portion size task was a mock test. Thus, they were not given the food to eat after the test. There are some additional issues related to the portion size task that warrants further discussion. Participants could have considered some meal items as snack items (e.g. pizza), and vice versa (e.g. banana). Another issue relates to a person's like for a particular food versus the person's actual consumption of that food. One might crave chocolate bars but not be willing to eat them because of potential negative health outcomes. Finally, the most liked food among those presented could be less appealing than each individual's unique favorite food, which may have resulted in even larger differences in portion size between the TSD and sleep conditions. Inter-individual differences in food preferences cannot be ignored. This latter view is also supported by our study findings. Inter-individual differences in food preferences cannot be ignored. This latter view is also supported by our study findings. When taking individual food preferences into account, the effects of TSD on portion size choices reached significance. In contrast, when including all food items into analysis, i.e. when individual preferences were ignored, the effects of TSD on portion size choices were less pronounced. This suggests that the type of test food provided should be taken into account when exploring the influence of sleep loss on food intake in humans.

In conclusion, our results suggest that two independent mechanisms — one homeostatic, and one hedonic — may combine to explain the effect of sleep deprivation on hunger and consummatory behavior. This experiment further suggests that variables such as hunger state and the type of test food provided should be taken into account when considering the influence of sleep loss on food intake in humans. However, future research is needed to confirm our findings under conditions of partial sleep deprivation.

Role of the funding sources

Work from P.S.H., C.B. and H.B.S. was supported by the Swedish Research Council, Åhlensstiftelse, the Swedish Brain Research Foundation, Tore Nilsons Foundation, Ingrid Thurings Foundation, Brain Foundation, Åke Wiberg Foundation, Novo Nordisk,Olle Engkvist Bygmästare, and the Swedish Research Council. Work from C.B. was also supported by the German research Foundation (DFG, SFB 654, Project B7). Work from S.L.D. and H.V. was supported by the Swedish Research Council (2009-S266), the Forskning och Utvecklingsarbete/Avtal om Läkarutbildning och Forskning Göteborg (ALFGBG-138741), and DFG.

Conflict of interest

None of the authors had a conflict of interest.

Acknowledgements

We thank all participants.

References

Benedict, C., Brooks, S.J., O'Daly, O.G., Almèn, M.S., Morell, A., Åberg, K., Gingnell, M., Schultes, B., Hallschmid, M., Broman, J.-E., Larsson, E.-M., Schiöth, H.B., 2012. Acute sleep deprivation enhances the brain's response to hedonic food stimuli: an fMRI study. J. Clin. Endocrinol. Metab. 97, E443—E447.

Benedict, C., Hallschmid, M., Lassen, A., Mahnke, C., Schultes, B., Schiöth, H.B., Born, J., Lange, T., 2011. Acute sleep deprivation reduces energy expenditure in healthy men. Am. J. Clin. Nutr. 93, 1229—1236.

Bosy-Westphal, A., Hinrichs, S., Jauch-Chara, K., Hitze, B., Later, W., Wilms, B., Settler, U., Peters, A., Kiosz, D., Müller, M.J., 2008. Influence of partial sleep deprivation on energy balance and insulin sensitivity in healthy women. Obes. Facts 1, 266—273.

Boyle, P.J., Scott, J.C., Krentz, A.J., Nagy, R.J., Comstock, E., Hoffman, C., 1994. Diminished brain glucose metabolism is a significant determinant for falling rates of systemic glucose utilization during sleep in normal humans. J. Clin. Invest. 93, 529—535.

Brondel, L., Romer, M.A., Nougues, P.M., Touyarou, P., Davenne, D., 2010. Acute partial sleep deprivation increases food intake in healthy men. Am. J. Clin. Nutr. 91, 1550—1559.

Brunstrom, Shakeshaft, N.G., Scott-Samuel, N.E., 2008. Measuring 'expected satiety' in a range of common foods using a method of constant stimuli. Appetite 51, 604—614.

Chapman, C.D., Benedict, C., Brooks, S.J., Schiöth, H.B., 2012. Lifestyle determinants of the drive to eat: a meta-analysis. Am. J. Clin. Nutr. 96, 492—497.

Druce, M., Wren, A., Park, A., Milton, E., Patterson, M., Frost, G., Ghatei, M., Small, C., Bloom, S., 2005. Ghrelin increases food intake in obese as well as lean subjects. Int. J. Obes. 29, 1130—1136.

Fay, S.H., Ferriday, D., Hinton, E.C., Shakeshaft, N.G., Rogers, P.J., Brunstrom, J.M., 2011. What determines real-world meal size? Evidence for pre-meal planning. Appetite 56, 284—289.

Jung, C.M., Melanson, E.L.L., Frydendall, E.J., Perreault, L., Eckel, R.H., Wright, K., 2010. Energy expenditure during sleep, sleep deprivation and sleep following sleep deprivation in adult humans. J. Physiol. 589, 235—244.

Laan, D.J., Leidy, H.J., Lim, E., Campbell, W.W., 2010. Effects and reproducibility of aerobic and resistance exercise on appetite and energy intake in young, physically active adults. Appl. Physiol. Nutr. Metab. 35, 842—847.

Malik, S., McGlone, F., Bedrossian, D., Dagher, A., 2008. Ghrelin modulates brain activity in areas that control appetitive behavior. Cell Metab. 7, 400—409.

Nedeltcheva, A.V., Kilkus, J.M., Imperial, J., Kasza, K., Schoeller, D.A., Penev, P.D., 2009. Sleep curtailment is accompanied by increased intake of calories from snacks. Am. J. Clin. Nutr. 89, 126—133.

Nedeltcheva, A.V., Kilkus, J.M., Imperial, J., Schoeller, D.A., Penev, P.D., 2010. Insufficient sleep undermines dietary efforts to reduce adiposity. Ann. Int. Med. 153, 435—441.

Patel, S.R., Malhotra, A., White, D.P., Gottlieb, D.J., Hu, F.B., 2006. Association between reduced sleep and weight gain in women. Am. J. Epidemiol. 164, 947—954.

Rechtschafen, A., Kales, A., 1968. A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects. BIS/BRI, UCLA, Los Angeles.

Rolls, B.J., Morris, E.L., Roe, L.S., 2002. Portion size of food affects energy intake in normal-weight and overweight men and women. Am. J. Clin. Nutr. 76, 1207—1213.

Schiöth, H.B., Brooks, S.J., Benedict, C., 2012. Healthcare systems never sleep: are medical residents today the patients of tomorrow? Sleep Med. 13, 965.

Schmid, S.M., Hallschmid, M., Jauch-Chara, K., Born, J.A.N., Schultes, B., 2008. A single night of sleep deprivation increases ghrelin levels and feelings of hunger in normal-weight healthy men. J. Sleep Res. 17, 331—334.

Schmid, S.M., Hallschmid, M., Jauch-Chara, K., Wilms, B., Benedict, C., Lehnert, H., Born, J., Schultes, B., 2009. Short-term sleep loss decreases physical activity under free-living conditions but does not increase food intake under time-deprived laboratory conditions in healthy men. Am. J. Clin. Nutr. 90, 1476—1482.

Silva, J.E., 2006. Thermogenic mechanisms and their hormonal regulation. Physiol. Rev. 86, 435—464.

Spiegel, K., Tasali, E., Penev, P., Cauter, E.V., 2004. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann. Int. Med. 141, 846—850.

St-Onge, M.-P., Roberts, A.L., Chen, J., Kelleman, M., O'Keeffe, M., RoyChoudhury, A., Jones, P.J., 2011. Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals. Am. J. Clin. Nutr. 94, 410—416.

St-Onge, M.-P., McReynolds, A., Trivedi, Z.B., Roberts, A.L., Sy, M., Hirsch, J., 2012a. Sleep restriction leads to increased activation of brain regions sensitive to food stimuli. Am. J. Clin. Nutr. 95, 818—824.

St-Onge, M., O'Keeffe, M., Roberts, A., RoyChoudhury, A., Laferrè, B., 2012b. Short sleep duration, glucose dysregulation and hormonal regulation of appetite in men and women. Sleep 35, 1503—1510.

Steenhuis, I.H., Vermeer, W.M., 2009. Portion size: review and framework for interventions. Int. J. Behav. Nutr. Phys. Act. 6, 58.

Taheri, S., Lin, L., Austin, D., Young, T., Mignot, E., 2004. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med. 1, e62.

Wansink, B., Painter, J.E., North, J., 2005. Bottomless bowls: why visual cues of portion size may influence intake. Obes. Res. 13, 93—100.

Wilkinson, L.L., Hinton, E.C., Fay, S.H., Ferriday, D., Rogers, P.J., Brunstrom, J.M., 2012. Computer-based assessments of expected satiety predict behavioural measures of portion-size selection and food intake. Appetite 59, 933—938.