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Association between habitual sleep duration/quality and appetite markers in individuals with obesity

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ABSTRACT

Study Objectives: To assess if habitual sleep duration/quality was associated with appetite in individuals with obesity, and if the association was modulated by sex.
Methods: Sleep duration/quality was measured with Pittsburgh Sleep Quality Index score in 95 healthy adults with obesity (BMI: 36.6 ± 4.2 kg/m^2). Subjective feelings of appetite were assessed using visual analogue scales, and plasma concentrations of active ghrelin, total peptide YY, active glucagon-like peptide 1, cholecystokinin (CCK) and insulin were measured in fasting and every 30 min up to 2.5 h after a meal.
Results: No significant associations were found between sleep duration, or overall quality, and appetite in all participants. However, a worse sleep efficiency was associated with lower postprandial CCK, a shorter habitual sleep was associated with lower postprandial desire to eat and a lower daytime dysfunction was associated with higher prospective food consumption in fasting ($P<0.05$, for all). In males, a shorter habitual sleep duration and a worse subjective sleep quality were associated with increased basal and postprandial active ghrelin ($P<0.05$, $P<0.01$, $P<0.01$ and $P<0.05$, respectively). Also, a shorter habitual sleep was associated with lower basal and postprandial insulin ($P<0.05$ for both) and a worse overall sleep quality with lower postprandial insulin ($P<0.05$). In females, a worse overall sleep quality was associated with lower postprandial active ghrelin ($P<0.05$), and short habitual sleep with higher postprandial insulin ($P<0.05$).
Conclusion: A worse habitual sleep efficiency is associated with blunted postprandial CCK secretion in individuals with obesity. The association between habitual sleep duration/quality and insulin and active ghrelin seems to be modulated by sex, but more studies are needed to confirm these findings.

1. Introduction

The prevalence of obesity has increased rapidly over the last couple of decades [1]. This has happened almost in parallel to a decrease in sleep duration [2-4]. There is good epidemiological evidence for an association between sleep deprivation and obesity [4-9]. Moreover, several longitudinal studies have found sleep deprivation [10-14], as well as sleep fragmentation and bad sleep quality [15-19], to be associated with increased risk of weight gain prospectively. The mechanisms behind this association are not completely understood, but increased drive to eat, coupled with more available time to eat and increased preference for energy dense foods seem to be especially important [20-22].

A large number of laboratory studies have been conducted on the association between acute sleep restriction and/or fragmentation and appetite, and the majority show deregulated appetite, with increased hunger and reduced fullness feelings, paralleled by a concomitant increase in the plasma concentration of the hunger hormone ghrelin and/

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or decreased concentration of satiety peptides [23-35]. However, most of these findings are from studies in young males with normal-weight and are limited by their small sample size. Moreover, as highlighted in the review articles by St-Onge MP (2013) [36] and Bayon et al. (2014) [37], there is great variation among studies regarding the impact of acute sleep deprivation on ghrelin plasma concentrations, with some reporting an increase [24,29,37-39], others a reduction [26] and still others no change [35,40,41].

In contrast, very few studies have investigated how habitual short sleep impacts appetite [16,39,42]. Taheri et al. (2004) found that habitual short sleep was associated with reduced leptin and increased ghrelin plasma concentrations in the fasting state in people with overweight or obesity [39]. McNeil et al. (2013), reported that habitual short sleepers had a lower total satiety quotient, which indicates a lower satiety response to a meal, in males with obesity [16]. St-Onge MP et al. (2012) found a sex difference in the association between sleep duration and appetite, with an increase in total ghrelin secretion for males with short sleep both for fasting and postprandially. In addition, GLP-1 concentrations were lower with short sleep compared to habitual sleep in females postprandially [43].

More studies are needed in this area to investigate if an association between habitual sleep duration/quality and appetite exists and if sex modulates that association.

The primary aim of this study is, therefore, to assess the potential association between habitual sleep duration and appetite in individuals with obesity. Secondary aims are to assess the potential association between sleep quality and appetite and to investigate if sex differences exist in the association between habitual sleep duration/quality and appetite.

2. Materials and methods

2.1. Participants

This analysis used baseline data from a weight loss intervention study run at the Obesity Research Group at the Norwegian University of Science and Technology (NTNU) [44]. Participants were recruited by newspaper advertising and St. Olavs Hospital and NTNU intranet. Ninety-five healthy adults (56% females) with obesity (30< BMI <50 kg/m^2), stable weight (<2 kg variation the last 3 months), who were not currently dieting to lose weight and who had an inactive lifestyle (<150 min of physical activity, of at least moderate intensity/week) [45] were recruited for this study. Exclusion criteria included pregnancy, breast feeding and clinically significant illness, including diabetes, previous weight loss surgery and/or medication known to affect appetite/metabolism or induce weight loss. This study was conducted according to the guidelines laid down in the Declaration of Helsinki and was approved by the regional ethics committee (Ref., 2012/1901), registered in ClinicalTrial.gov (NCT01834859). All participants provided written informed consent.

2.2. Procedure and measurements

Participants arrived at the laboratory at 8 am following a 12 h overnight fast. They were instructed not to consume alcohol, or caffeine containing products, or engage in structured physical activity for at least 24 h before the test day and to follow their usual sleeping habits the previous night. Upon arrival, height and weight were measured following standardized procedures and body composition assessed by air-displacement plethysmography (BodPod, COSMED, Italy).

This was followed by a standardized breakfast meal, ingested during 20 min. The meal contained bread, orange juice, milk (alternatively yoghurt), cheese, jam and butter. The nutritional composition was 600 kcal (2512 kJ) (14% protein, 38% fat and 47% carbohydrate). Subjective feelings of hunger, fullness, desire to eat and prospective food consumption were measured with a 10 cm Visual Analogue Scale (VAS) [46], in the fasting state, immediately after breakfast and every 30 min for up to 2.5 h post-breakfast (fasting state, and immediately after breakfast, 30, 60, 90, 120 and 150 min, post breakfast).

Blood samples for the analysis of appetite related hormones were also taken in fasting and every 30 min for 2.5 h (fasting state, and 30, 60, 90, 120 and 150 min, post breakfast). Plasma samples were analysed in duplicates for active ghrelin, active glucagon-like peptide 1 (GLP-1), total peptide YY (PYY) and insulin using a Human Metabolic Hormone Magnetic Bead Panel (LINCOplex Kit, Millipore, Germany), and cholecystokinin (CCK) using an "in-house" RIA method [47]. Intra-and inter-assay CV were <10% and <20% for active ghrelin, active GLP-1 and total PYY; <10% and <15% for insulin and <5% and <15% for CCK, respectively.

Subjective sleep duration and quality were measured retrospectively for the last month before the laboratory assessments, using a Norwegian validated version of the Pittsburgh Sleep Quality Index (PSQI) [48]. The PSQI has strong reliability and validity as a sleep screening tool in both clinical and non-clinical settings in a variety of populations [49]. The PSQI is made of 7 components: subjective sleep quality (C1), sleep latency (C2), sleep duration (C3), sleep efficiency (C4), sleep disturbances (C5), use of sleeping medication (C6) and daytime dysfunction (C7). A total score of >5 indicates poor sleep quality, and the higher the score the worse the sleep quality [48].

2.3. Statistical analysis

Statistical analysis was performed with SPSS version 23.0 (SPSS IBM, New York, USA). Data are presented as median (first and third quartiles) or as mean±SD if otherwise not mentioned. Statistical significance was assumed at $P<0.05$, unless otherwise stated. Total area under the curve (AUC) for subjective appetite sensations and appetite hormones was calculated from 0 to 150 min using the trapezoid rule.

Some variables were not normal-distributed and were either log-transformed or square-root transformed for normalization purposes. The potential association between habitual sleep (duration/quality) and appetite was assessed by multiple linear regression analyses, with appetite markers (appetite sensations and hormone plasma concentrations) used as dependent variables and with sleep variables (duration/quality) as predictors, after adjusting for age, sex, moderate intensity physical activity (min/day) and BMI. Separate regression models were run for each dependent variable and for each predictor. A sex interaction was also included in the model. All the assumptions for multiple linear regression were checked and met.

3. Results

Table 1 presents the general characteristics of all 95 participants included in this analysis.

Participants had a median age of 43 years, an average BMI of 36.6 ± 4.0 kg/m^2, and 56% were females. Males were younger ($P<0.01$), heavier and had a lower fat mass (%) ($P<0.001$ for both), and a longer duration of moderate-to-vigorous intensity physical activity (MVPA) ($P<0.01$), compared with females. Most of the participants presented with glucose plasma concentration in fasting within the normal range, even though 65% of the participants ($n = 57$) with available data were classified as having significant insulin resistance derived from HOMA-index (a score of >2.9 was an indicator for significant insulin resistance). The mean global score from the PSQI (overall sleep quality) was 5.5 ± 3.0 and sleep duration was 7 h/night (6.0–7.3 h/night).

Detailed information regarding sleep duration and quality can be seen in Supplementary Table I. The majority of the participants (68%, 65 participants and 60%, 57 participants, respectively) reported an overall short sleep duration (< 7 h/night) and poor sleep quality (PSQI total score > 5).

A significant negative association was found between minutes of MVPA and overall sleep quality, but not duration, in all participants

Table 1
Characteristics of the study participants.

		All(n = 95)	Females(n = 53)	Males(n = 42)
General	Age (years)	43 (36, 48)	44 (40, 51)	40 (32, 45)
	Weight (kg)	109.6 ± 9.6[a]	102.5 ± 13.0	118.5 ± 18.0
	BMI (kg/m^2)	36.6 ± 4.0	36.7 ± 3.6	36.3 ± 4.6
	Fat mass (kg)	48.0 ± 10.3	49.4 ± 9.1	46.2 ± 11.6
	Fat mass (%)	44.1 ± 6.4[a]	48.0 ± 3.9	39.0 ± 5.4
	MVPA (min/day)	60.7 ± 49.8 [a]	44.4 ± 31.9	83.0 ± 60.7
	Steps/day	6557±2737	6857±3019	6148±2278
Sleep	Sleep duration (h/night)	7.0 (6.0, 7.3)	7.0 (6.0, 7.3)	6.8 (6.0, 7.5)
	Sleep quality (total PSQI score)	5.5 ± 3.0	5.6 ± 3.0	5.2 ± 3.0
Appetite - Hormones - Subjective feelings	Basal AG (pg/ml)	79 (55, 117)[a]	89 (66, 136)	63 (39, 93)
	Basal Insulin (pg/ml)	929 (599, 1368)[a]	720 (499, 1205)	1137 (836, 1627)
	AUC AG (pg/ml*min)	8254 (5882,11,560) [a]	9217 (6731, 12,376)	6419 (4976, 10,043)
	AUC Insulin (pg/µl*min)	531 (390, 720) [a]	488 (368, 693)	584 (472, 752)
	AUC total PYY (pg/ml*min)[b]	6497 (3743, 12,679)	5898 (2296, 9849)	7636 (5233, 14,451)
	AUC active GLP-1 (pg/ml*min)	1421 (809, 2068)	1453 (977, 2034)	1395 (697, 2248)
	AUC CCK (pmol/L*min)	355 (279, 480)	387 (294, 509)	332 (270, 440)
	Fasting Hunger (cm)	3.8 ± 2.1	3.9 ± 2.1	3.7 ± 2.2
	Fasting DTE (cm)	4.5 ± 2.1	4.5 ± 1.9	4.5 ± 2.2
	Fasting PFC (cm)	5.8 ± 2.2[a]	5.2 ± 1.9	6.5 ± 2.3
	AUC Fullness (cm*min)	887.0 ± 270.0[a]	947.0 ± 253.0	810.0 ± 273.0

Data presented as median (first, third quartile) or as mean±SD. [a]Significant sex differences. [b]Data available in only n = 61 (35 females). Abbreviations: MVPA, moderate-to-vigorous physical activity; PSQI, Pittsburgh Sleep Quality Index; AG, active ghrelin; PYY, peptide YY; GLP-1, glucagon-like-peptide-1; CCK, cholecystokinin. DTE: desire to eat; PFC: prospective food consumption.

(β=−0.018, P = 0.019, n = 77).

The association between habitual sleep duration/quality and the plasma concentration of appetite hormones, in all participants, males and females can be seen in Table 2.

No significant association was found between habitual sleep duration or overall quality, derived from the PSQI, and the plasma concentration of any of the appetite hormones measured, either in fasting or after a meal. However, sleep efficiency (PSQI; component 4) was negatively associated with postprandial CCK (β =−60.090, P = 0.028, n = 84).

A significant sex interaction was found in the association between both basal and postprandial active ghrelin plasma concentrations and overall sleep quality (PSQI total score/global score) (P = 0.040 and P = 0.012, respectively) and subjective sleep quality (PSQI; C1) (P = 0.013 and P = 0.010, respectively), also between both basal and postprandial insulin plasma concentrations and sleep duration (P = 0.023 and P = 0.002, respectively) and overall sleep quality (PSQI total score) (P = 0.037 and P = 0.001, respectively) and, finally, between postprandial insulin plasma concentrations and sleep efficiency (PSQI, C4) (P = 0.002).

In males only, sleep duration was negatively associated with basal and postprandial active ghrelin concentrations ((β=−0.287, P = 0.016, n = 36) and (β=−0.302 P = 0.002, n = 36), respectively). A significant positive association was found between subjective sleep quality (PSQI; C1) and both basal and postprandial active ghrelin concentrations (β=0.584, P = 0.008, n = 36 and β=0.420, P = 0.028, n = 36, respectively). There was also a significant positive association between sleep duration and both basal and postprandial concentrations of insulin ((β=0.275, P = 0.024, n = 36) and (β=0.154, P = 0.043, n = 36), respectively) and overall sleep quality (PSQI total score) was negatively associated with postprandial insulin concentrations (β=−0.064 P = 0.028, n = 36) . Finally, sleep efficiency (PSQI, C4) was negatively associated with postprandial insulin concentrations (β=−0.509, P = 0.006, n = 36).

In females only, a significant negative association was found between sleep duration and postprandial levels of insulin (β=−0.205, P = 0.030, n = 47), and between overall sleep quality (PSQI total score) and postprandial concentrations of active ghrelin (β=−0.091, P = 0.027, n = 47). Also, overall sleep quality (PSQI total score) was positively associated with postprandial insulin concentrations (β=0.082, P = 0.018, n = 47).

The association between habitual sleep duration/quality and subjective feelings of appetite in all participants, males and females can be seen in Table 3.

A significant positive association between sleep duration and postprandial desire to eat (β=1.666, P = 0.029, n = 91) and a significant negative association was found between daytime dysfunction (PSQI, C7) and prospective food consumption in fasting (β=−0.721, P = 0.040, n = 91).

In males only, a significant positive association was found between sleep duration and fasting prospective food consumption (β=0.73, P = 0.03, n = 42) and postprandial desire to eat (β=2.24, P = 0.04, n = 42).

4. Discussion

This is the first study to investigate a potential association between habitual sleep duration/quality, and both objective and subjective appetite measures, in individuals with obesity. A worse sleep efficiency was found to be associated with decreased postprandial CCK. The worst sleep quality (short habitual sleep and worse daytime dysfunction) was associated with decreased subjective feelings of desire to eat after a meal and prospective food consumption in fasting, respectively.

Moreover, some important associations emerged when males and females were analyzed separately. In males, worse overall sleep quality and shorter habitual sleep were associated with lower insulin plasma concentrations. Moreover, the worst sleep quality (habitual short sleep duration and a worse subjective sleep quality) was associated with increased active ghrelin concentrations in males only, both in the fasting state and after a meal. In females, habitual short sleep and worse overall sleep quality were associated with increased postprandial insulin but decreased postprandial active ghrelin concentrations.

This study's findings are not in line with Taheri et al. (2004) [39], who found that habitual short sleep duration was associated with increased levels of ghrelin in the fasting state, in a mixed sample of males and females with overweight or obesity. Moreover, they reported no association between sleep duration or quality and basal insulin plasma concentrations. This study reports habitual short sleep duration to be associated with increased basal and postprandial active ghrelin and reduced basal insulin concentrations in males only. Methodological differences in the measurement of both habitual sleep duration and ghrelin may play a role in the inconsistency of the findings. First, the association was only found when sleep was measured with

Table 2
Association between habitual sleep duration/quality assessed by PSQI and the plasma concentration of appetite hormones in all participants, males, and females.

	Sleep duration (h/night)		Sleep quality (global score)		Subjective sleep quality (C1)		Sleep efficiency (C4)		Daytime dysfunction (C7)	
Basal(pg/ml)	β Coeff	p	β Coeff	p	β Coeff	p	β Coeff	p	β Coeff	p
AG										
- All[a]	−0.139	0.075	0.017*	0.577	0.222*	0.087	−0.171	0.212	0.071	0.535
- Males[a]	−0.287	*0.016*	0.076	0.104	0.584	*0.008*	−0.147	0.643	0.158	0.418
- Females[a]	−0.048	0.664	−0.045	0.275	−0.001	0.993	−0.174	0.222	0.028	0.846
Insulin										
- All[b]	0.069*	0.495	0.013*	0.729	−0.210	0.206	0.149	0.393	0.083	0.570
- Males[b]	0.275	*0.024*	−0.069	0.150	−0.517	*0.023*	−0.140	0.665	−0.105	0.599
- Females[b]	−0.239	0.170	0.123	0.052	−0.005	0.984	0.324	0.149	0.275	0.222
AUC (pg/ml*min^{-1})										
AG										
- All[a]	−0.135	0.062	−0.008*	0.776	0.068*	0.577	−0.134	0.290	−0.050	0.641
- Males[a]	−0.302[b]	*0.002*	0.058	0.151	0.420	*0.028*	−0.048	0.859	0.105	0.531
- Females[a]	0.006	0.957	−0.091	*0.027*	−0.186	0.249	−0.175	0.232	−0.201	0.169
Active GLP-1										
- All[a,c]	−0.036	0.734	0.029	0.473	−0.046	0.791	−0.078	0.672	0.167	0.277
Total PYY										
- All[a]	−0.048	0.660	0.007	0.872	−0.158	0.461	−0.114	0.537	0.163	0.326
CCK										
- All	−1.912	0.905	−7.201	0.238	−32.239	0.222	−60.090	*0.028*	0.999	0.966
Insulin										
- All[a]	0.001*	0.984	0.003*	0.906	−0.103	0.285	0.043*	0.674	0.045	0.599
- Males[a]	0.154	*0.043*	−0.064	*0.028*	−0.302	*0.034*	−0.509	*0.006*	−0.115	0.350
- Females[a]	−0.205	*0.030*	0.082	*0.018*	0.033	0.810	0.228	0.065	0.170	0.172

Adjusted for age, sex, BMI, and minutes of moderate-to-vigorous intensity physical activity (MVPA) in all participants, and for age, BMI, and minutes of MPA for males and females. Each coefficient is from a separate regression model. *Significant sex interaction. [a]Log transformation used in these models. [b]Square rot transformation used in these models. [c]Even after log and square rot transformation, the standardized residuals were not normally distributed. CCK AUC is in pmol/L*min. AG: active ghrelin, AUC: area under the curve, GLP-1: glucagon like peptide-1, PYY: peptide YY, CCK: cholecystokinin. Results for males and females separately are presented only when there was a sex interaction.

Table 3
Association between habitual sleep duration/quality assessed by PSQI and subjective feelings of appetite in all participants, males, and females.

	Sleep duration (h/night)		Sleep quality (global score)		Sleep efficiency (C4)		Sleep medication (C6)		Daytime dysfunction (C7)	
	β Coeff	p	β Coeff	p	β Coeff	p	β Coeff	p	β Coeff	p
Fasting										
Hunger										
- All[a]	0.068	0.302	−0.018	0.453	−0.175	0.093	−0.055	0.614	−0.012	0.896
DTE										
- All	0.139	0.573	−0.008	0.928	−0.240	0.537	0.233	0.562	−0.246	0.480
PFC										
- All	0.361	0.148	−0.084	0.346	−0.374	0.345	0.281	0.492	−0.721	*0.040*
AUC										
Hunger										
- All[a,b]	1.369	0.075	−0.492	0.071	−2.026	0.096	−0.921	0.467	−1.134	0.302
Fullness										
- All	−29.209	0.360	8.412	0.458	79.179	0.114	40.733	0.434	−26.347	0.560
DTE										
- All[a]	1.666	*0.029*	−0.314	0.251	−1.416	0.245	−0.302	0.812	−0.756	0.490
PFC										
- All	74.853	0.058	−17.712	0.209	−75.342	0.230	−11.306	0.862	−47.386	0.401

Appetite feelings in fasting and AUC are reported in their relevant state. Adjusted for age, sex, BMI, and minutes of moderate-to-vigorous intensity physical activity (MVPA) in all participants, and for age, BMI, and minutes of MPA for males and females. Each coefficient is from a separate regression model. [a]Square rot transformation used in these models. [b]Even after log and square rot transformation, the standardized residuals were not normally distributed. AUC: area under the curve, DTE: desire to eat, PFC: prospective food consumption. No significant sleep*sex interaction was noted, therefore the results for males and females are not presented.

polysomnography, performed on the night before blood sampling or when sleep duration was assessed from a 6-day sleep diary (which included naps). No association was found when sleep duration was derived from the 6-day sleep diary (without naps) or a sleep questionnaire, which is in line with this study's findings. Unfortunately, the paper from Taheri et al. does not mention the timeframe over which participants were asked about usual sleep in the questionnaire. Even though polysomnography is the gold standard method for measuring sleep, it may be that the artificial environment in which the polysomnograph is performed (sleep in a laboratory) does not reflect habitual sleep and that subjective methods, such as PSQI, may be a better option, particularly in individuals with obesity [50]. Second, in the study from Taheri et al., blood samples were taken after awakening in the laboratory, while in the present study, participants had to drive to the research center to have their blood samples taken, and it is possible that they were awake for several hours before blood collection, which could potentially have affected the appetite data. Third, in the study from Taheri and colleagues, total ghrelin was measured, while in the present study, active ghrelin was quantified.

The present study is the first to investigate the potential association between habitual sleep duration/quality and subjective feelings of appetite in a mixed sample of males and females. This study is unable to find any significant association either in all participants, or when each sex was analyzed separately. This is in line with McNeil and colleagues

(2013) [16], who reported no differences in subjective feelings of appetite between males with short and normal sleep duration, and good and poor sleep quality.

In the present study, a shorter habitual sleep and worse subjective sleep quality (PSQI, C1) were associated with increased basal and postprandial active ghrelin in males, but not in females. This was in line with the evidence derived from laboratorial studies. Indeed, most studies including males only, have found acute short sleep/no sleep to be associated with increased levels of ghrelin the day after [28-32]. However, studies including mixed samples of males and females, or females only, have not found any association between acute sleep deprivation and circulating concentrations of ghrelin and, regarding the other appetite hormones, the results are inconsistence [27,40,41,51-53]. In fact, St-Onge et al. (2012) [27], reported no differences in the plasma concentration of appetite-related hormones between acute sleep restriction and normal sleep in females, while in males, acute sleep restriction was associated with increased total ghrelin plasma concentrations, in line with the findings from the present study. The same study also found that in females, acute sleep deprivation was associated with lower total GLP-1 postprandial concentrations. This study finds no significant association regarding GLP-1, but measures active, not total GLP-1. However, and overall, laboratorial studies in females report an association between sleep deprivation and increased calorie intake, especially calories from carbohydrates and palatable foods [40,41,52]. It is therefore likely that there are separate mechanisms behind the association between sleep deprivation and increased food intake in males and females. In males it is probably driven by upregulation of ghrelin secretion and potential downregulation of satiety signals, while in females, and as already suggested by St-Onge et al. [27] it may be a result of upregulated hedonic signals.

Regarding subjective feelings of appetite, studies on acute sleep deprivation have mainly been performed in males with normal-weight and the majority report that short sleep is associated with increased hunger [23,25,28,31] and reduced postprandial fullness [25,33]. However, Schmid et al. (2009) [35], found, similarly to this study, no difference in hunger or other subjective appetite feelings between acute sleep deprivation and normal sleep in males who are healthy. Moreover, Hart et al. (2015), in the only study on subjective appetite feelings and acute sleep in females, reported no differences in hunger feelings between sleep restriction and sleep elongation in females with overweight and obesity [53]. This is not in line with this study's findings where no association was found between subjective feelings of appetite and sleep duration/quality either when all participants were included or when each sex was analyzed separately. Discrepancies in the results could be due to comparison between sleep manipulation studies to induce sleep deprivation vs. habitual sleep measurements. A short sleeper who has been sleeping less than 7 h/night for many years may not experience as large of an effect of sleep deprivation on changes in appetite, whereas someone who is a normal sleeper who is sleep deprived would likely experience more drastic changes in appetite in response to this acute "sleep deprivation stressor".

This study has both strengths and limitations. Appetite was assessed in fasting and for 2.5 h after a meal. Another strength is that this study included both males and females. However, this study also suffers from some limitations. First, sleep duration/quality the night before blood sampling was not assessed and this might have distorted findings. Second, appetite sensations and hormones in the fasting state were measured sometime after awakening (wake-time and early wake-time was not assessed or standardized). Third, this study does not take into consideration the phase of the menstrual cycle, which has been shown to modulate appetite [54]. Fourth, the multiplex assay for the measurements of appetite hormones (except for CCK) is likely to result in less accurate and precise measurements compared with optimized assays for each individual hormone. This was a large study and it was inevitable that different plates were run, which could affect the variability of the results. Fifth, this is a cross-sectional study and a cause-effect relationship cannot be established. Finally, no data was collected on food intake, and this study was underpowered to look at sex differences.

5. Conclusions

In conclusion, a worse habitual sleep efficiency seems to be associated with a blunted postprandial secretion of CCK in a mixed sample of males and females with obesity. Sex seems to modulate the association between habitual sleep duration/quality and insulin and active ghrelin secretion, however more studies are needed to confirm these findings.

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Supplementary materials

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