

Overnight weight loss: relationship with sleep structure and heart rate variability

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Abstract

Background: Weight loss can be caused by a loss of body mass due to metabolism and by water loss as unsensible water loss, sweating, or excretion in feces and urine. Although weight loss during sleep is a well-known phenomenon, it has not yet been studied in relation to sleep structure or autonomic tonus during sleep. Our study is proposed to be a first step in assessing the relationship between overnight weight loss, sleep structure, and HRV (heart rate variability) parameters.

Methods: Twenty-five healthy volunteers received a 487 kcal meal and 200 ml water before experiment. Volunteers were weighed before and after polysomnography. Absolute and relative weight indices were calculated. Time and frequency domain analysis of heart rate variability was assessed during stages 2, 4, and REM. Nonparametric linear regression analysis was performed between night weight loss parameters, polysomnographic, and HRV ariables.

Results: High Frequency domain correlated positively with weight loss during stage 4. Slow wave sleep duration correlated positively with weight loss and weight loss rate. The duration of Stage 2 correlated negatively with absolute and relative weight loss.

Conclusions: Weight loss during sleep is dependent upon sleep stage duration and sleep autonomic tonus. Slow-wave sleep and sleep parasympathetic tonus may be important for weight homeostasis.

Key words: weight loss, sleep stages, sleep structure, HRV, polysomnography

1. Background

Weight loss can be caused by loss of body mass due to metabolism and by water loss as insensible water loss, sweating, or excretion in feces and urine.¹ Eighty-three percent of the total weight loss is due to insensible water loss from airways and skin.¹ Water loss rate varies according to changes in activity and ambient temperature and humidity.¹ Although weight loss during sleep is a well-known phenomenon, there are no studies relating it to sleep structure or autonomic tonus during sleep. Studies on the variation of body composition over 24 hours using bioimpedance methods showed an increase in weight and a reduction in height during daytime.^{3,4} In these studies, bioimpedance was influenced by food and drink ingestion.^{3,4,5} Many studies assessed the effects of sleep debt on metabolism and weight gain for long periods of time, suggesting that it may cause obesity and metabolic syndrome but there are no studies for the effects during short periods.^{6,7,8} There is evidence suggesting a homeostatic mechanism for weight control in animal models and humans.^{9,10} This mechanism is thought to be dependent on energy intake, energy expenditure, and environmental conditions. In this context, sleep homeostasis could also influence this process.^{9,10} Autonomic tonus varies according to different sleep stages and influences overnight fluid loss, blood pressure, and heart rate variability.^{12,13,14} HRV (heart rate variability) is an easy-to-access non-invasive marker of the autonomic tonus during sleep.^{12,13,14} Our study is proposed as a first step in assessing the relationship between overnight weight loss, sleep structure, and HRV parameters.

2. Methods

2.1 Experimental subjects

Experiments were performed in the sleep laboratory of the Psychobiology Department of the Universidade Federal de São Paulo, São Paulo – Brazil. Twenty-eight healthy young adult volunteers who were regular sleepers and free of medications were selected for the experiment. There were three drop-outs during the experiment due to data loss. The final sample consisted of 25 subjects, 7 males and 18 females aged 18-29 years. Female subjects were not in their menstruation period. Eleven were in estrogenic and 7 were in progestagenic phases.

2.2. Ethics

Written informed consent forms were signed by all subjects. The protocol was approved by the Ethics Committee of the Universidade Federal de São Paulo.

2.3. Procedure

The laboratory procedure is summarized in Figure 1. Subjects arrived in the sleep laboratory at 8 pm after 4 hours of fasting. They underwent a clinical interview and completed a standard questionnaire about sleep and physical status. All subjects were eutrophic and normo-hydrated. Subjects were not allowed to perform physical activity or to be sleep deprived for at least 48 hrs before the experiment. They received a standard 487 kcal diet and 200 ml water at 9 pm. All

the following measurements were carried out by a single investigator. A pre-sleep weighing was performed (initial night weight W1) in a 1 g precision Bod Pod® digital scale with computer interface. After that, their height was measured and sleep clothes were weighed in order to be subtracted from body weight. After body measuring, subjects were asked not to ingest liquids or solids, not to use the restroom, and to wear standard clothing during the entire night of sleep. Body temperature was measured twice (pre and post-sleep). All subjects slept in a standard room with controlled temperature and humidity (humidity 40-50%, temperature 23-24°C). Subjects were also asked not to wash any part of their bodies until the end of the experiment. Polysomnography recording finished at 7 am and weight and height were measured again (post-sleep weight, W2). The weight variables analyzed were absolute weight loss in grams (AWL), absolute weight loss rate in grams per hour (AWLR), relative weight loss (RWL = absolute weight loss/initial weight x 100), relative weight loss rate (RWLR = relative weight loss per hour), and height variation (HV). Age, gender, menstrual cycle phase, body temperature, and room temperature were also analyzed. The present study was limited to the analysis of body-weight variation during the night. We did not measure lean and fat mass due to technical limitations.

2.3. Polysomnographic recording and scoring

Polysomnography was performed in equipment Meditron Sonolab® sampling at 256 Hz for 4 EEG, 2 electro-oculogram, 1 chin electromyogram, 1 leg electromyogram, and 1 tracheal microphone; sampling at 500 Hz for 1 electrocardiogram (EKG); sampling at 16Hz for 1 oronasal thermistor, 1 nasal

pressure transducer, 2 chest and abdominal effort sensors, and 1 oximeter (Nellcor™ oximeter). Polysomnograms lasted at least 7 hours. Recordings were scored following Rechtschaffen and Kales and American Academy of Sleep Medicine (AASM) criteria.^{15,16,17} The polysomnographic variables analyzed were total sleep time, sleep efficiency (sleep time/record time x 100), sleep latency, REM sleep latency, REM and non-REM sleep percentage, respiratory disturbance index (RDI), microarousals/hour (MAI), periodic leg movements/hour (PLMI), and oxygen saturation.

2.4. Heart rate variability (HRV)

Analyses were performed on one polysomnography EKG-channel (sampling rate 500Hz). A careful manual review was performed in order to exclude artifacts or arrhythmias.

2.4.1 Time domain analysis

In a continuous EKG recording, each QRS complex was detected and the normal-to-normal (NN) intervals were determined. Five time domain indexes were derived: the standard deviation of all NN intervals (SDNN); the square root of the mean squared differences of successive NN intervals (RMS); the standard deviation of successive differences between adjoining normal cycles (SDSD); and the proportion of adjacent normal NN intervals differing by >50 msec (NN50 and pNN50).

2.4.2 Frequency Domain analysis

Artifact-free stable sleep stages 2, 4, and REM, in sleep epochs of 5-minute duration were selected for analysis. We chose the central 5-minute period of the longest above-mentioned sleep stages. The power densities in the Very Low Frequency (VLF, 0.0033-0.04Hz), Low Frequency (LF, 0.04-0.15Hz), and High Frequency (HF, 0.15-0.4Hz) were calculated by integrating the power spectral density in the respective frequency bands. Normalized power spectra for LF and HF and LF/HF ratio were also calculated.

2.5. Statistical analysis

For comparison of repeated measurements, the interval data were transformed into ordinal data because the precondition of normal distribution was not fulfilled for all variables. Therefore, comparisons of night weight loss, polysomnographic, and HRV variables were performed by use of nonparametric Spearman correlation coefficients. Significance was set at the 0.05 level of probability (two-tailed). Mann-Whitney U-test was utilized to assess differences between genders and menstrual cycle phases.

3. Results

3.1. Sleep results

Mean RDI was higher in male subjects (4.1 ± 6.2 vs. 0.4 ± 0.8 ; $U=29$, $p=0.04$). No snoring was detected for any subject during the night of the experiment. No significant differences were found according to menstrual cycle phase for any sleep variable. All individual polysomnography variables are outlined in Table 1.

3.2. Body-measurement results

All individual body measurements are outlined in Table 2. Body temperature (before and after the sleep period) ranged from 35.9 to 36.7°C. As seen in Table 2, overall weight loss (300 ± 68 g) during sleep for both genders was a common phenomenon in our population of healthy and young subjects. There were no differences in weight loss indices between genders and menstrual cycle phases. There was an overnight increase in height of 1.0 ± 0.7 cm. Height variation did not correlate with any sleep parameters.

3.3. HRV results

The mean and standard deviations of HRV parameters for all volunteers during sleep stages 2 and 4 non-REM sleep and REM sleep are provided in Table 3. SDNN (118.7 ± 48.3 vs. 58.5 ± 29.5 ; $U=15$, $p=0.003$), RMSSD (145.1 ± 81.3 vs. 52.2 ± 34.6 ; $U=18$, $p=0.06$), and SDSD (114.9 ± 58.5 vs. 38.9 ± 27.9 ; $U=14$,

$p=0.003$) were higher in men during REM sleep. No significant differences were found according to menstrual cycle phase for any HRV variable.

3.4. Weight, HRV and polysomnography correlations

There were significant positive correlations between SWS (more specifically stage 4) and relative weight loss indices. However we also found a negative correlation between stage 2 and night weight loss.

The HF (parasympathetic component) correlated positively with all absolute and relative weight loss indices. Correlation R values are outlined in Table 4.

4. Discussion

To our knowledge, this is the first study to report that overnight weight loss is dependent upon sleep structure. Although we found that weight loss during sleep was a universal phenomenon, its magnitude was affected by sleep stages and autonomic tonus. The literature suggests that sleep is important for weight homeostasis on a long term since sleep shortage is associated with overweight status, but overnight weight variation had not yet been studied.^{5,6,7,8} In short periods of time, body-weight is a cyclic phenomenon in which the lowest values are registered after the sleep period.^{3,4} Sleep is not a uniform state and there are documented differences in cardiovascular function, autonomic tonus, transepidermal water loss, and many other functions during the distinct sleep stages, particularly SWS and REM sleep.^{18,19,20,21} Considering these facts, we hypothesized that the overnight weight loss rate is not uniform throughout the sleep period.

In our study, we found a positive correlation between weight loss and SWS length. Indeed, SWS is a stage when transepidermal water loss is higher due to differences in autonomous system activity favoring weight loss.^{20,21,22} In support of this hypothesis, we found that higher SWS length and parasympathetic tonus (HF component of HRV) were directly related to higher overnight weight loss rates.

The present study was not designed to evaluate hormone secretion, but we can speculate that the endocrine system is also involved in this phenomenon. A

previous study found that ghrelin promotes SWS in humans.²³ Since ghrelin levels tend to be higher during longer fasting periods, higher amounts of SWS in faster weight losing subjects could be explained by higher ghrelin levels.^{23,24} Furthermore, reduced GH secretion due to shorter SWS periods could also be related to reduced overnight weight loss since most human GH is secreted during SWS.²⁵ GH promotes overnight weight loss through increased lipolysis.^{25,26}

As far as we are aware, this is the first study relating HRV to overnight weight changes. In our population, men presented higher sympathetic activity during REM sleep, a state in which surges of sympathetic tone are found. Gender differences in HRV have also been reported in previous studies.²⁷ However, this increase in the sympathetic component in male subjects did not correlate or seem to affect the weight loss rate during sleep. Other variables that increased the sympathetic component are arousals and apnea/hypopnea, which did not affect weight changes in our sample.¹⁸ Our results suggest that the main autonomic parameter linked to overnight weight loss is the vagal tonus. Autonomic tonus is proven to be related weight homeostasis in long term studies.²⁸

In summary, we have confirmed our initial hypothesis that weight loss during sleep seems to be dependent on sleep-stage duration and autonomic tonus. SWS and sleep parasympathetic tonus may be important for weight homeostasis. Consequently, sleep debt and sleep deprivation may negatively affect weight homeostasis through SWS debt. More precise methods need to be developed in order to determine the relationship between sleep structure, variations in body composition, weight loss, autonomic tonus, and hormone secretion.

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Table 1: Polysomnographic results

Subject	Sleep latency (min)	REM sleep latency (min)	Total sleep time (min)	Sleep efficiency (%)	S1%	S2%	S3%	S4%	REM%	RDI (/h)	MAI (h)	PLMI (/h)	Mean oxygen saturation (%)
1	21.5	80.0	383.0	90.0	4.0	49.1	13.4	10.4	23.0	0.0	9.7	0.0	96.5
2	5.5	138.5	421.0	88.9	13.2	55.1	8.2	12.1	11.4	0.0	10.8	16.1	97.9
3	8.0	155.0	356.0	84.4	6.7	58.7	10.8	5.9	17.8	0.2	20.4	0.0	96.3
4	5.0	153.0	437.5	96.5	3.1	54.2	4.1	22.7	15.9	0.0	17.7	3.2	97.9
5	4.0	177.0	392.5	94.4	6.6	57.3	4.1	15.3	16.7	2.1	21.2	0.0	97.7
6	3.5	117.0	405.5	96.6	11.0	52.4	7.2	19.7	9.7	7.6	18.6	0.0	95.4
7	5.0	77.0	364.5	94.8	4.3	37.6	7.0	17.6	33.0	0.0	7.9	0.0	95.8
8	5.5	178.5	375.5	96.0	4.4	52.3	10.5	16.4	16.4	2.2	4.3	0.0	93.4
9	14.5	102.5	327.0	85.4	6.1	38.1	14.2	19.4	22.2	4.0	4.8	0.0	98.4
10	11.0	64.5	409.5	95.1	4.4	38.7	13.2	18.4	25.3	1.2	3.4	0.0	96.2
11	5.0	70.5	320.5	93.6	5.9	46.3	9.7	15.1	22.9	0.0	11.8	0.0	96.1
12	13.0	74.5	382.0	93.5	6.2	45.4	6.9	15.2	26.3	2.0	6.4	0.0	96.9
13	8.0	146.5	326.5	80.9	8.7	45.6	9.8	26.3	9.5	4.2	18.0	0.0	96.0
14	9.0	302.0	305.5	79.7	12.1	49.3	7.5	25.0	6.1	1.8	13.2	0.0	95.0
15	13.0	129.5	360.5	83.4	11.0	33.6	8.9	31.3	15.3	0.0	7.7	0.0	95.9
16	21.5	147.5	404.5	89.6	6.9	56.5	5.4	22.7	8.4	0.0	4.7	0.0	98.1
17	32.0	129.0	264.0	76.3	13.6	46.2	8.9	24.8	6.4	0.7	7.3	0.0	98.3
18	8.0	108.0	346.5	94.7	4.6	47.9	4.3	31.5	11.7	0.0	18.9	0.0	96.2
19	16.5	13.5	358.5	87.0	6.0	40.6	8.4	29.8	15.2	0.0	10.9	0.0	96.0
20	28.5	178.5	368.5	85.7	7.7	54.8	3.5	18.6	15.3	0.0	15.1	0.0	92.2
21	5.5	187.5	402.5	97.5	1.1	37.6	12.3	29.8	19.1	0.0	8.8	0.0	97.9
22	41.5	126.5	298.0	75.3	6.9	48.5	4.5	25.3	14.8	0.0	7.7	0.0	96.0
23	2.0	179.5	404.0	98.2	1.4	32.7	17.1	35.5	13.4	0.0	2.8	0.0	97.1
24	46.5	155.0	296.5	71.2	16.4	42.8	6.7	8.8	25.3	0.0	14.2	0.0	97.5
25	11.5	201.5	340	85.9	8.5	49.0	10.7	17.6	14.1	0.0	3.9	0.0	95.9

min= minutes /h= per hour

Table 2: Individual body measurement data

Subject	Age (years)	Gender	Height (cm)	Absolute initial weight (g)	AWL (g)	AWLR (g/h)	RWL (%)	RWLR (%/h)	HV (cm)
1	22	f	158	46,800	220	23.0	0.47	0.049	1.5
2	28	m	164	54,573	244	26.6	0.45	0.049	1.5
3	24	f	163	53,139	261	27.6	0.49	0.052	1.5
4	24	f	163	54,756	294	31.0	0.54	0.057	2.0
5	22	f	164	59,235	303	33.4	0.51	0.056	2.5
6	20	m	169	56,937	255	27.6	0.45	0.048	2.0
7	28	f	170	54,841	269	28.5	0.49	0.052	2.0
8	18	f	160	58,293	262	30.5	0.45	0.052	0.5
0	21	m	179	68,909	316	36.7	0.46	0.053	2.0
10	20	m	174	64,725	307	33.7	0.47	0.052	0.5
11	24	f	165	54,062	196	21.8	0.36	0.040	1.0
12	25	f	180	56,674	263	29.0	0.46	0.051	0.5
13	21	m	167	57,646	341	37.9	0.59	0.066	1.5
14	23	m	181	78,728	304	33.6	0.39	0.043	0.0
15	22	f	161	51,634	469	49.5	0.91	0.096	1.5
16	24	f	162	53,145	289	31.3	0.54	0.059	1.0
17	23	f	157	57,175	236	27.1	0.41	0.047	1.0
18	21	f	153	47,196	419	47.5	0.89	0.101	1.0
19	20	f	158	50,934	327	33.8	0.64	0.066	1.5
20	29	f	162	72,307	262	29.1	0.36	0.040	1.0
21	18	f	166	53,862	323	33.9	0.60	0.063	1.0
22	19	f	163	73,779	265	28.3	0.36	0.038	0.3
23	20	f	152	47,755	318	30.5	0.67	0.064	0.8
24	21	m	181	74,716	480	46.4	0.64	0.062	0.1
25	24	f	165	55,711	303	30.1	0.54	0.054	0.3

g= grams cm= centimeters h= hours f= female m= male

Table 3: Mean Heart Rate Variability data

	S2	S4	REM
Mean NN interval	807.3±243.4	884.3±187.7	853.6±249.8
SDNN	58.6±38.8	62.5±42.7	75.3±44.3
RMSSD	61.2±43.8	72.6±57.2	78.2±65.6
SDSD	45.2±35.8	54.0±52.2	60.2±51.1
NN50	18.0±14.1	19.7±14.7	14.5±11.3
PNN50	27.9±23.2	32.4±24.1	25.4±22.1
VLF	1770.5±984.9	1628.3±1318.5	4242.5±3399.1
LF	2870.2±1948.8	2542.2±1798.8	3265.7±1652.7
HF	2478.9±1612.4	2826.8±1623.8	2101.3±1013.1
Total power	7326.6±3877.0	6873.1±3404.7	9858.7±4998.8
LF/HF	1.4±0.8	1.1±0.7	1.7±1.0

Mean ± standard deviation

Table 4: Correlations

	AWL	AWLR	RWL	RWLR
S2 %	-0.42*	-0.38	-0.40*	-0.34
S2D (min)	-0.38	-0.36	-0.24	-0.21
S4 %	0.34	0.38	0.49*	0.50*
S4D (min)	0.32	0.33	0.52*	0.51*
SWS %	0.27	0.27	0.44*	0.42*
SWSD (min)	0.23	0.21	0.46*	0.43*
HF S4	0.60*	0.57*	0.47*	0.45*

*Significant for $p < 0.01$ min=minutes

Figure 1: Study procedure

