# The influence of oral water load on energy expenditure and sympatho-vagal balance in obese and normal weight women

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## Abstract

**Introduction:** Oral water load may increase the energy expenditure (EE) by stimulation of sympathetic dependent thermogenesis. Thus, drinking of water may be helpful in weight reduction. The aim of the study is to assess the influence of water load on energy expenditure and sympathetic activity in obese and normal weight women.

**Material and methods:** Forty-five women were included. Energy expenditure was measured twice, in the morning and after oral water load, by the indirect calorimetric method. The heart rate variability parameters low frequency (LF), high frequency (HF), LF/HF index, standard deviation of normal RR intervals (SDNN) and root mean square difference among successive RR normal intervals (rMSSD) were used for the indirect assessment of the sympatho-vagal balance.

**Results:** Resting energy expenditure (REE) was significantly higher in obese than in normal weight women (1529 ±396 kcal/day vs. 1198 ±373 kcal/day; $p = 0.02$). In both study groups after water load EE increased significantly (by 20% and by 12%, corresponding to 8.6 kcal/h and 5.2 kcal/h respectively), while, LF/HF index increased simultaneously. The increase of energy expenditure (EE) did not exceed the energetic cost of water heating, from room to body temperature – 15 kcal/1000 ml. There was no correlation between changes of energy expenditure (EE) and heart rate variability (HRV) parameters.

**Conclusions:** The increase of EE induced by water load is mostly related to the heating of the consumed water to body temperature. The assessment of autonomic balance by means of standard HRV indices had been found insufficient for detection of actually operating mechanisms.

**Key words:** energy expenditure, sympatho-vagal balance, oral water load, obesity.

## Introduction

The prevalence of obesity and its co-morbidities is increasing worldwide, inspiring a search for methods that increase energy expenditure (EE), other than physical activity. Stimulation of thermogenesis by enhancing sympathetic activity is one of these methods [1].

Thermogenesis includes heat production during enzymatic reactions taking part in all body cells, digestion and absorption of food (diet-induced thermogenesis) and homeostatic reaction of the body exposed to low temperature and exercise (adaptive thermogenesis) [2, 3]. Diet-induced thermogenesis is modified by daily intake of macronutrients and a number of meals [4, 5]. A diet rich in saturated fatty acids, preferred by the obese, decreases heat production during digestion and absorption of food [5]. The main regulator of adaptive thermogenesis is sympathetic activity, partially related to enhanced metabolism of brown adipose tissue [6, 7]. It was shown that obesity is associated with impaired adaptive thermogenesis [8].

Pharmacological methods of sympathetic activity stimulation (enhancing thermogenesis) are associated with adverse drug reactions including hypertension, cardiac arrhythmias and exacerbation of coronary artery disease. Therefore these agents have been withdrawn from the therapy of obesity [9, 10]. Recently, sibutramine, an anorexigenic drug, centrally acting serotonin-norepinephrine reuptake inhibitor, and peripheral stimulator of sympathetic induced thermogenesis, was also withdrawn for similar reasons [11].

One of the possible non-pharmacological methods of sympathetic induced thermogenesis is water load [12-16]. It has been confirmed by observation that the use of a β-adrenolytic agent prior to water load may prevent increased thermogenesis [16].

The aim of the study is to assess the influence of oral water load on energy expenditure and sympatho-vagal balance in obese and normal weight women.

## Material and methods

The study group consisted of 45 females, including 24 obese (BMI (body mass index) ≥ 30 kg/m$^2$) and 21 normal weight (BMI 18.5-24.9 kg/m$^2$), without concomitant diseases. The characteristics of study groups are presented in Table I.

The exclusion criteria included the presence of morbid obesity (BMI ≥ 40 kg/m$^2$), acute and chronic diseases, any drug use, including contraceptive agents, changes of body mass exceeding 3 kg during the last 6 months, abnormal resting electrocardiogram, smoking, drinking more than 3 drinks per week, endocrine disorders, such as hyper- and hypothyroidism, Cushing's syndrome, and polycystic ovary syndrome. The study was approved by the Local Bioethics Committee. All the subjects gave their informed consent for participation in the study.

Study procedures were conducted between 8.00 and 9.00 in the morning, after 16-hour overnight fast. Anthropometric measurements (body weight and height) were performed, and body mass index (BMI) was calculated according to the standard formula. Body composition was assessed by the bioimpedance method using a Bodystat 1500 analyser (Douglas, Isle of Man) and standardised resting electrocardiograph (Aspel A-100, Zabierzow, Poland) [17] was performed. Resting energy expenditure (REE) measurement (during 30 min, after 60 min of rest) was performed by the indirect calorimetric method (MedGraphics, Minnesota, USA) [18], on the basis of oxygen and carbon dioxide concentrations analysed in exhaled air. The calibration was performed before each examination. The second energy expenditure (EE) measurement was performed for 1 h after the ingestion of 1000 ml of low mineralized non-sparkling water (Nałęczowianka) stored at room temperature (22°C) in 5 min.

Heart rate variability (HRV) analysis for the indirect assessment of autonomic cardiac control was performed based on 12-lead electrocardiographic monitoring (Suprima 12, Oxford, United Kingdom) [19] during the whole individual examination. Two time-domain measures, i.e. standard deviation of all

**Table I.** Characteristics of study groups (median values and inter-quartile range in brackets)

| Parameter | Obese ($n$ = 24) | Normal weight ($n$ = 21) | Value of $p$ |
|---|---|---|---|
| Age [years] | 27 (23-32) | 23 (22-25) | < 0.01 |
| Body weight [kg] | 94.0 (80.1-96.1) | 57.0 (55.0-60.0) | < 0.001 |
| BMI [kg/m$^2$] | 31.6 (30.0-35.6) | 21.6 (20.6-22.9) | < 0.001 |
| Free fat mass [%] | 55.3 (53.5-57.0) | 71.1 (66.8-73.0) | < 0.001 |
| Free fat mass [kg] | 50.7 (46.0-53.8) | 40.0 (38.6-42.5) | < 0.001 |
| Fat mass [%] | 44.7 (43.0-46.5) | 28.9 (27-33.2) | < 0.001 |
| Fat mass [kg] | 41.9 (34.1-46.4) | 17.4 (14.6-20.1) | < 0.001 |
| Respiratory quotient (RQ) | 0.78 (0.73-0.84) | 0.76 (0.75-0.79) | NS |

normal RR intervals (SDNN, ms) and root-mean-square difference between successive RR normal intervals (rMSSD, ms) and four frequency-domain measures, including total spectral power (TP, 0.04-0.4 Hz), low frequency (LF, 0.04-0.15 Hz, $ms^2$), high frequency (HF, 0.15-0.4 Hz, $ms^2$), as well as the LF/HF index. The SDNN and TP were considered as reflecting the level of overall cardiac autonomic control, RMSSD and HF as indices of parasympathetic control, and LF and LF/HF as indices of sympathetic activity [19]. All measurements were performed in 5-minute epochs. Only 5-minute periods of uninterrupted sinus rhythm were accepted. In the final analysis averaged values of baseline HRV parameters from three consecutive 5-minute periods that did not change by more than 15% were considered as the reference [20].

### Statistical analysis

Statistical analyses were performed using Statistica 8.0 PL (StatSoft Polska, Krakow, Poland). The results are presented as median values with inter-quartile ranges or mean values with 95% confidence interval (for serial measurements).

Mann-Whitney *U* test was used for comparison of quantitative variables between study groups and Wilcoxon test was used to show changes of parameters after water load. The univariate correlation coefficients were calculated according to Spearman. The results were considered as significant with a *p* value of less than 0.05.

## Results

As expected, the REE was significantly higher in obese than in normal weight women (Table II). However, REE calculated per body surface area and respiratory quotient (RQ) were similar in both groups.

In both study groups, water load was followed by a significant increase in EE, slightly higher in the obese (Table II). The increase of EE in an hour per body surface area in the obese (8.6 ±14.1 kcal/1.73 $m^2$) and in the normal weight group (5.2 ±8.7 kcal/1.73 $m^2$) did not differ significantly (Figure 1). The RQ value did not change in the obese (0.78 (95% CI: 0.76-0.80) vs. 0.79 (95% CI: 0.75-0.84), $p < 0.05$), but diminished in the normal weight group (0.79 (95% CI: 0.75-0.83) vs. 0.76 (95% CI: 0.71-0.81), $p < 0.05$).

The initial values of HRV parameters of cardiac autonomic control were similar in both study groups (Table III). After water load no change of heart rate was observed (Table III). There was a moderate increase of LF/HF index values in both study groups. The TP and LF increased significantly, exclusively in the obese (Table III).

The HRV response to water load was heterogeneous. Significant changes of HRV parameters, defined as at least 30% increase or decrease of the initial values of total power (TP) and SDNN [20], were observed only in a few subjects. A TP increase was found in 7 obese and 5 normal weight, and a decrease in 2 obese and 3 normal weight women. An SDNN increase was observed in 2 obese and 1 normal weight and decrease in a single obese and 2 normal weight women. The subgroups of subjects with clinically significant changes were not sufficient for further statistical comparisons.

There was no correlation between changes of EE and studied HRV parameters induced by water load in both study groups and in the combined analysis.

## Discussion

The results presented in this study demonstrate that the increase of the energy expenditure induced by oral water load is relatively small, sufficient for ingested water warming to body temperature, but not as a supportive method of weight reduction.

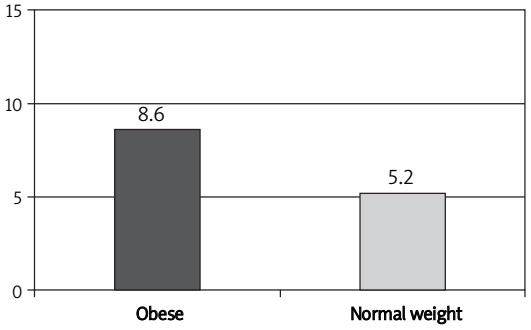


**Figure 1.** The increase of energy expenditure (EE) after water load in normal weight ($n = 21$) and obese ($n = 24$) women

**Table II.** The influence of water load on metabolic parameters (means and 95% confidence interval)

| | Obese ($n = 24$) | | Normal weight ($n = 21$) | |
|---|---|---|---|---|
| | REE | EE | REE | EE |
| kcal/day | 1529^ (1344-1715) | 1755* (1610-1900) | 1198 (1028-1368) | 1315** (1140-1491) |
| kcal/$m^2$/h | 31.3 (28.2-34.4) | 36.3 (33.3-39.3) | 30.8 (26.4-35.2) | 33.8*** (29.1-38.6) |
| kJ/min | 3.7^^^ (3.3-4.1) | 4.3*& (3.9-4.7) | 1.6 (1.4-1.9) | 1.8*** (1.5-2.0) |

*EE – energy expenditure, REE – resting energy expenditure; *p < 0.05, **p < 0.01, ***p < 0.001 – before vs. after water load; ^p < 0.05, ^^^p < 0.001 – before water load obese vs. normal weight; &p < 0.001 – after water load obese vs. normal weight*

**Table III.** Heart rate variability parameters in studied groups before and after water load (means and 95% confidence interval)

| Water load | Obese ($n$ = 24) | | Normal weight ($n$ = 21) | |
|---|---|---|---|---|
| | Before | After | Before | After |
| RR [ms] | 877 (833-921) | 901 (856-947) | 863 (800-926) | 875 (816-934) |
| Total power [ms$^2$] | 4067 (2977-5158) | 5447* (3829-7065) | 4684 (3502-5866) | 5227 (3528-6927) |
| LF [ms$^2$] | 1003 (709-1297) | 1248** (958-1538) | 1317 (966-1668) | 1493 (970-2016) |
| HF [ms$^2$] | 898 (573-1224) | 888 (615-1160) | 1081 (723-1438) | 858 (622-1095) |
| LF/HF | 1.32 (1.04-1.60) | 1.73* (1.33-2.10) | 1.47 (1.10-1.80) | 1.79* (1.40-2.10) |
| SDNN [ms] | 70.8 (59.3-82.4) | 74.4 (62.3-86.5) | 75.3 (64.4-86.1) | 72.2 (58.7-85.6) |
| rMSSD [ms] | 62.5 (45.6-79.5) | 63.2 (51.5-75.0) | 65.2 (53.0-77.4) | 58.2 (45.1-71.3) |

*RR – duration between two consecutive R waves of the ECG, LF – low frequency (0.04-0.15 Hz), HF – high frequency (0.15-0.4 Hz), LF/HF – low frequency/high frequency index, SDNN – standard deviation of all normal RR intervals, rMSSD – root-mean-square difference between successive RR normal intervals; *p < 0.05, **p < 0.01 – after vs. before water load*

The increase of energy expenditure after water load observed in the present study reached 20% on average in the obese and 12% in normal weight women. It was lower than previously reported (30%) [16], despite a higher water load (1000 ml vs. 500 ml).

As mentioned, the increase in energy expenditure after water load of room temperature (22°C) is mostly the consequence of the thermic effect of the ingested water (water heating) [16]. Heating of 1000 ml of ingested water from room temperature (22°C) is estimated at 15 kcal (1000 ml × 15 cal/g × K). The energy for this process derives from blood redistribution (constriction of peripheral vessels) due to sympathetic activation induced by water load [13]. Therefore, the increase of energy expenditure after water load may be lower than its thermic effect. In both studied groups of females during the first hour after water load the enhancement of energy expenditure was lower: 8.6 kcal and 5.2 kcal over 60 min in obese and normal weight women, respectively. Thus we suggest that blood redistribution reduced heat loss though the skin due to vessel constriction. The results of our study are in line with those obtained by Brown *et al.* [21], who reported that lowering of the temperature of drinking water by 3°C increased energy expenditure by 5%.

The redistribution of circulating blood from the skin to the gastrointestinal tract after water load is the effect of sympathetic and parasympathetic activity changes. Distension of the stomach, especially up to the volume of 125 ml, is one of the mechanisms of sympathetic activation [22, 23]. Moreover, ingestion of hypo-osmotic fluid results in sodium-dependent stimulation of sympathetic receptors [24-26]. We could not exclude that other mechanisms of sympathetic/parasympathetic modulation by room temperature water load, such as stimulation of the sensory receptor of the trigeminal nerve in the oral cavity, may participate in it. Cold oral water load may potentially stimulate the trigemino-cardiac reflex (TCR) [27-29].

It should be stressed that only some [12-16], and not all [30-36] studies revealed that the increase in EE induced by water load is mediated by sympathetic activation. The conducted studies have varied in methodology of EE determination and study subject characteristics. Some of them included only normal weight subjects [30, 31, 34], others both normal weight and overweight subjects [32, 35, 36], or males and females with different nutritional status [35]. Moreover, the number of studied subjects usually did not exceed 20.

After water load, we have observed an increase of the LF/HF index, independent from body mass. The index according to some investigators reflects the balance between sympathetic and parasympathetic components of the autonomic nervous system [37], while to others it reflects increased sympathetic modulation [19, 38]. In studies assessing HRV parameters and additionally gastric electric activity by electrogastrography after water load, vagus nerve activation was shown [39, 40]. In addition, the increased thermogenesis may be partially related to the ingestion of hypo-osmotic fluids [41], enhanced anabolic processes in swollen cells [42] and in consequence increased EE [16, 21].

We failed to find a correlation between changes of HRV and of EE parameters. However, it is a weak piece of evidence to deny the important role of sympathetic activation in the thermic effect of water ingestion. As we used the constant volume of water load, the expected sympathetic activation should be quite similar in all study subjects. Unfortunately, the assessment of sympathetic activity by spectral analysis of HRV is a low specific method [43].

In line with a previous study [16], we observed a decline of RQ after water load in normal weight subjects exclusively, which may reflect the increased utilisation of carbohydrates. The lack of RQ

changes in obese women proves the "metabolic inflexibility" phenomenon referring to the impaired switch between carbohydrate and lipid oxidation in the obese [41].

It should be stressed that regardless of the mechanisms increasing EE after oral water load, the results of the present study show that drinking water does not lead to a clinically significant increase of EE that might be helpful in the management of obesity. However, this observation does not exclude that drinking of water before food ingestion may accelerate the appearance of postprandial satiety.

The main limitation of our study is the assessment of sympatho-vagal balance by spectral analysis of HRV only. The assessment of autonomic balance by means of standard heart rate variability indices had been found insufficient for detection of actually operating mechanisms. Moreover, we did not test the various volumes and temperatures of water load.

In conclusion, the increase of EE induced by water load is mostly related to the heating of the consumed water to body temperature. The assessment of autonomic balance by means of standard HRV indices was found insufficient for detection of actually operating mechanisms.

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