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Water-Induced Thermogenesis Reconsidered: The Effects of Osmolality and Water Temperature on Energy Expenditure after Drinking

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Context: A recent study reported that drinking 500 ml of water causes a 30% increase in metabolic rate. If verified, this previously unrecognized thermogenic property of water would have important implications for weight-loss programs. However, the concept of a thermogenic effect of water is controversial because other studies have found that water drinking does not increase energy expenditure.

Objective: The objective of the study was to test whether water drinking has a thermogenic effect in humans and, furthermore, determine whether the response is influenced by osmolality or by water temperature.

Design: This was a randomized, crossover design.

Setting: The study was conducted at a university physiology laboratory.

Participants: Participants included healthy young volunteer subjects.

Intervention: Intervention included drinking 7.5 ml/kg body weight (~518 ml) of distilled water or 0.9% saline or 7% sucrose solution

(positive control) on different days. In a subgroup of subjects, responses to cold water (3 C) were tested.

Main Outcome Measure: Resting energy expenditure, assessed by indirect calorimetry for 30 min before and 90 min after the drinks, was measured.

Results: Energy expenditure did not increase after drinking either distilled water ($P = 0.34$) or 0.9% saline ($P = 0.33$). Drinking the 7% sucrose solution significantly increased energy expenditure ($P < 0.0001$). Drinking water that had been cooled to 3 C caused a small increase in energy expenditure of 4.5% over 60 min ($P < 0.01$).

Conclusions: Drinking distilled water at room temperature did not increase energy expenditure. Cooling the water before drinking only stimulated a small thermogenic response, well below the theoretical energy cost of warming the water to body temperature. These results cast doubt on water as a thermogenic agent for the management of obesity. (*J Clin Endocrinol Metab* **91**: 3598–3602, 2006)

THE HIGH PREVALENCE of overweight and obesity has led to the search for compounds that can increase energy expenditure and fat oxidation, thereby promoting weight loss. Because thermogenesis is partly regulated by sympathetic activity, substances that interact with the sympathetic nervous system can be considered as potential agents for weight reduction (1). Sympathomimetic compounds such as ephedrine are effective at increasing thermogenesis (2) but can have undesirable side effects (3). Safe, preferably nonpharmacological substances that can stimulate thermogenesis without causing side effects are therefore sought. A surprising candidate for a thermogenic agent is one of the most essential of all substances required for life: water. Drinking half a liter of water increases activity of the sympathetic nervous system as measured by enhanced plasma norepinephrine levels (4) and muscle sympathetic nerve activity (5). Boschmann *et al.* (6) hypothesized that the sympathetic activation after water drinking might stimulate thermogenesis. They reported that drinking 500 ml of water increased resting energy expenditure by 30%. The response

started within 10 min of drinking the water, peaked at 30–40 min, and was sustained for more than an hour (6). The water-induced thermogenesis was attributed to sympathetic nervous system activation because ingestion of a β -adreno-receptor blocker before drinking almost completely abolished the response. Drinking water that had been heated to 37 C attenuated the thermogenic response by 40%, which led to the suggestion that water-induced thermogenesis could be partly attributed to the energy cost of warming the water to body temperature (6). The authors extrapolated that increasing daily water intake by 1.5 liters would augment energy expenditure by approximately 200 kJ/d (6). If confirmed, water-induced thermogenesis would have important implications for weight control programs. However, the concept of water-induced thermogenesis is controversial. Several studies in humans (7–16) have reported that water drinking has little or no effect on resting energy expenditure (Table 1).

We therefore undertook the present study to further investigate the concept of water-induced thermogenesis. To eliminate the possibility that dissolved impurities or other substances in tap or mineral water might contribute to thermogenesis, we used distilled water. On the basis that osmolality might have a role in water-induced thermogenesis, we also tested responses to physiological (0.9%) saline. As a positive control, thermogenic responses were measured after ingestion of the same volume of a 7% sucrose solution. Fi-

Abbreviations: BMI, Body mass index; RQ, respiratory quotient; VCO₂, carbon dioxide production; VO₂, oxygen consumption.

TABLE 1. Summary of several published studies measuring acute changes in energy expenditure after water drinking in humans

Study	Year of publication	Water volume	No. of subjects	Calorimetry method	Reported increase in energy expenditure?
Boschmann <i>et al.</i> (6)	2003	500 ml	14	Whole room	30% increase after 60 min
Brundin and Wahren (7)	1993	375 ml	7	Ventilated hood	2% increase over 2 h
De Jonge <i>et al.</i> (8)	1991	Not stated ^a	9	Ventilated hood	No
Dulloo and Miller (9)	1986	200 ml	8	Douglas bag	No
Felig <i>et al.</i> (10)	1983	400 ml	3	Ventilated hood	No
Gougeon <i>et al.</i> (11)	2005	750 ml	2	Ventilated hood	No
Komatsu <i>et al.</i> (12)	2003	300 ml	8	Douglas bag	2.7% increase over 2 h
LeBlanc <i>et al.</i> (13)	1984	600 ml	8	Pneumotachograph	No
Li <i>et al.</i> (14)	1999	280 ml	19	Ventilated hood	No
Paolisso <i>et al.</i> (15)	1997	Not stated	8	Ventilated hood	No
Sharief and Macdonald (16)	1982	4 ml/kg ideal body weight (average 292 ml)	6	Ventilated hood	No

^a But at least 410 ml.

nally, considering the suggestion that part of water-induced thermogenesis might be attributed to the energy required to warm the water to body temperature, we tested whether ingestion of cold water would augment the thermic response.

Subjects and Methods

Subjects

Responses to ingestion of distilled water, 0.9% saline, and 7% sucrose were measured in eight healthy volunteer subjects [six males, two females; aged 25 ± 1 yr; height 176 ± 3 cm; weight 69 ± 2 kg; body mass index (BMI) 22.5 ± 0.7 kg/m²]. Statistical analysis showed that this number of subjects was sufficient to detect, at a significance level (alpha) of 0.05 and power (1-beta) of 0.96, a change in resting energy expenditure of 6% with a SD of the response of 5%. Time-control experiments were performed in five subjects (three males, two females; aged 26 ± 2 yr; height 176 ± 3 cm; weight 64 ± 3 kg; BMI 20.8 ± 0.7 kg/m²). In six healthy subjects (five males, one female; age 27 ± 1 yr; height 179 ± 3 cm; weight 66 ± 3 kg; BMI 20.8 ± 0.6 kg/m²), we assessed responses to ingestion of cold water (3 C). None of the subjects had any diseases or were taking medications. The subjects were requested to avoid doing physical exercise, refrain from caffeine consumption for at least 24 h, and have nothing to eat for 12 h and nothing to drink for 2 h before the experiments. Written informed consent was obtained from each subject according to the Declaration of Helsinki. The study protocol was approved by the institutional ethics committee.

Protocol

All measurements were performed in the morning, starting at 0830–0930 h, in a temperature-controlled (21 C) quiet room with the subjects in a comfortable seated position.

Ingestion of distilled water, 0.9% saline, and 7% sucrose. The subjects attended three experimental sessions according to a randomized crossover design. Respiratory gas exchange was measured by indirect calorimetry using an open-circuit ventilated hood system (Deltatrac monitor; Datex, Helsinki, Finland). Resting energy expenditure and respiratory quotient (RQ) were derived from the rates of oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) using the Weir equation (17). The precision of the gas analyzers, the calibration procedure, and the accuracy of the entire ventilated-hood system were regularly determined by ethanol combustion tests lasting between 2 and 4 h. These ethanol tests yield values for RQ of 0.67, with a coefficient of variation less than 2% and differences between calculated and measured energy expenditure of less than 3%. After an initial resting period of 30–40 min to allow gas exchange values to reach a steady state, resting energy expenditure was measured over 30 min. Then subjects ingested 7.5 ml/kg body weight (mean volume 518 ± 16 ml) of distilled water, 0.9% saline, or a 7% sucrose solution over 3 min. The drinks were served at room temperature. Gas exchange measurements were continued for a further 90 min after the drink. Values were recorded every minute and then averaged over 10-min intervals.

Time-control experiment. In five volunteers we tested the effects of a sham drink. After resting energy expenditure was recorded over a 30-min period, the subjects raised a vessel containing water (7.5 ml/kg body weight) to their lips but did not ingest any of the water. Resting energy expenditure was recorded for a further 90 min after the sham drink.

Ingestion of cold water. In a subgroup of six subjects, we tested the effects of ingesting distilled water (7.5 ml/kg body weight, mean volume 4.95 ml) that had been cooled to 3 C.

Statistical analysis

All data are given as means \pm SEM. Responses to the distilled water, 0.9% saline, and 7% sucrose drinks were tested by ANOVA for repeated measures. The postdrink values at 10-min time intervals were compared with values recorded during the 30-min baseline period using Dunnett's test for multiple comparisons. Total changes in energy expenditure (areas under the curve) after the three drinks were compared by ANOVA for repeated measures with Tukey's *post hoc* test to compare pairs of drinks. Energy expenditure after ingestion of cold water was compared with the corresponding baseline value by a paired *t* test. The statistics were performed using statistical software (InStat version 3.01; GraphPad Software, San Diego, CA). The level of statistical significance was set at $P < 0.05$.

Results

Responses to distilled water, 0.9% saline, and 7% sucrose

Resting values of $\dot{V}O_2$, $\dot{V}CO_2$, resting energy expenditure, and RQ as recorded over 30 min before ingestion of each of the drinks were similar and are shown in Table 2. The time courses of the responses to the three drinks are shown in Fig. 1. Ingestion of distilled water did not significantly affect $\dot{V}O_2$ ($P = 0.20$), $\dot{V}CO_2$ ($P = 0.60$), resting energy expenditure ($P = 0.34$), or RQ ($P = 0.14$). Similarly, ingestion of 0.9% saline had no significant effects on $\dot{V}O_2$ ($P = 0.30$), $\dot{V}CO_2$ ($P = 0.15$), resting energy expenditure ($P = 0.33$), or RQ ($P = 0.14$). In contrast, ingestion of 7% sucrose resulted in a significant and sustained increase in $\dot{V}O_2$, $\dot{V}CO_2$, resting energy expenditure,

TABLE 2. Resting values of gaseous exchange parameters and resting energy expenditure recorded over 30 min immediately before ingestion of the water, saline, and sucrose drinks

	Water	Saline	Sucrose
$\dot{V}O_2$ (ml/min)	224 ± 14	218 ± 13	228 ± 14
$\dot{V}CO_2$ (ml/min)	199 ± 13	190 ± 11	193 ± 12
Resting energy expenditure (kJ/min)	4.53 ± 0.29	4.40 ± 0.26	4.58 ± 0.28
RQ	0.89 ± 0.02	0.87 ± 0.02	0.85 ± 0.02

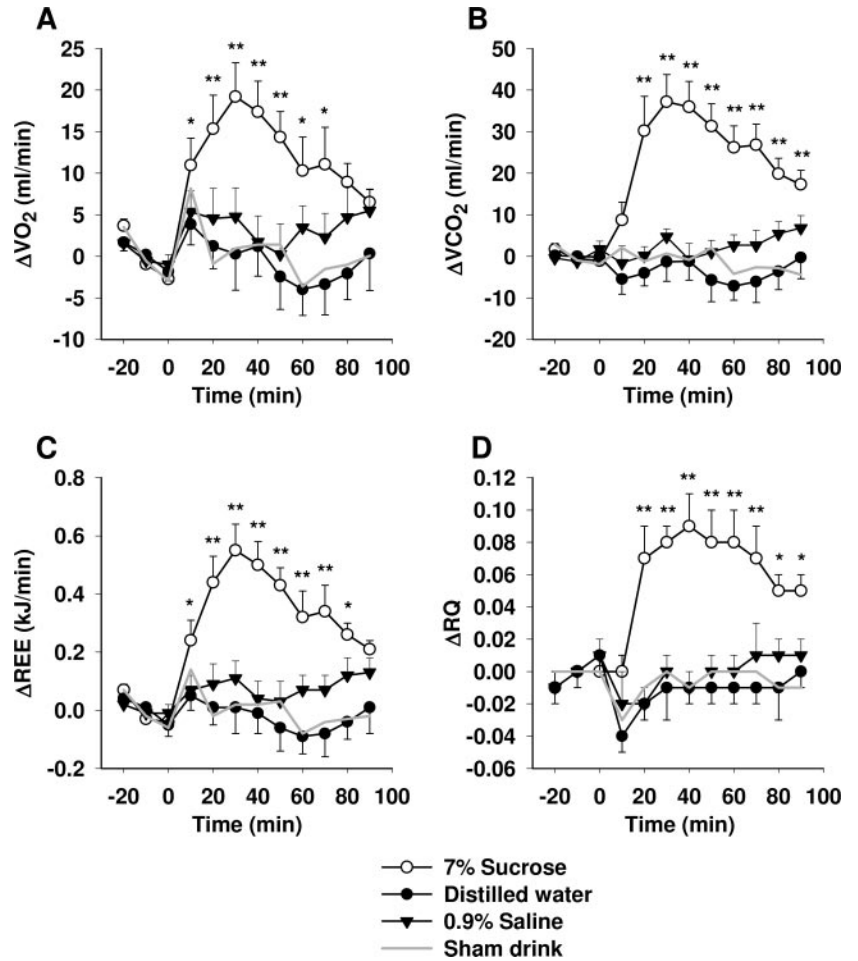


FIG. 1. Changes in VO_2 (A), VCO_2 (B), resting energy expenditure (REE) (C), and RQ (D) after drinking distilled water, 0.9% saline, or a 7% sucrose solution. Responses to a sham drink are also plotted. The drinks were ingested at time 0 min. Data points represent the mean value over the preceding 10 min (*, $P < 0.05$; **, $P < 0.01$).

ture, and RQ (all $P < 0.0001$). The average 90-min increase in resting energy expenditure (area under the curve) after the sucrose drink was 33 ± 4 kJ, compared with -2 ± 6 kJ after the distilled water drink and 7 ± 5 kJ after the saline drink (ANOVA, $P = 0.0002$; Tukey *post hoc* test, water vs. saline, $P > 0.05$; water vs. sucrose, $P < 0.001$; saline vs. sucrose, $P < 0.01$). Individual responses to ingestion of the water and sucrose drinks are compared in Fig. 2.

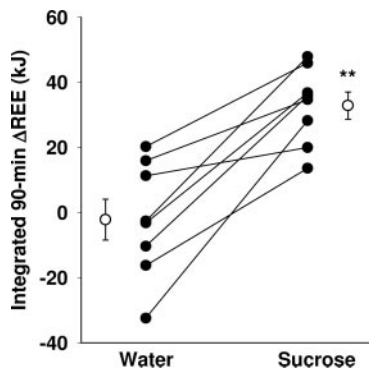


FIG. 2. Integrated changes in resting energy expenditure (REE) over 90 min in individual subjects after drinking water or a 7% sucrose solution (**, $P < 0.01$).

Time-control experiment

Responses to ingestion of the sham drink are included in Fig. 1. The coefficient of variation for resting energy expenditure over the entire 2-h recording period was 2.3% (1.9% before the sham drink and 2.4% after the sham drink). By comparison, for the water drink, the coefficient of variation for resting energy expenditure was 3.0% over the 2-h recording period (2.0% before the drink and 2.7% after the drink).

Responses to cold water

Drinking distilled water that had been cooled to 3 C increased resting energy expenditure in all six subjects (Fig. 3), from 4.48 ± 0.24 to 4.69 ± 0.23 kJ/min ($P = 0.0068$) over 60 min. The total increase in resting energy expenditure (area under the curve) after 3 C water ingestion was 13 ± 3 kJ after 60 min and 15 ± 3 kJ after 90 min.

Discussion

It was reported that drinking half a liter of water at room temperature increased resting energy expenditure by 30% after an hour (6). This previously unrecognized thermogenic property of water was suggested as a potential means for increasing energy expenditure in the treatment of obese and

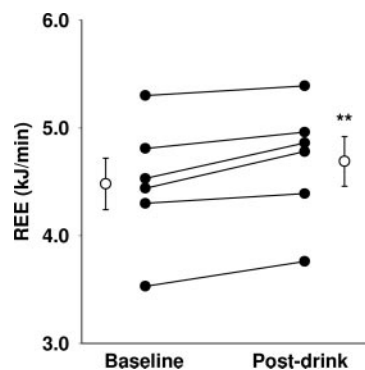


FIG. 3. Effect of drinking cold water (3 C) on resting energy expenditure (REE) in individual subjects. Postdrink values represent the average REE over 60 min (*, $P < 0.05$).

overweight individuals (6). The current study was designed to reassess water-induced thermogenesis and investigate whether osmolality or water temperature might influence energy expenditure after drinking. Our results are, however, inconsistent with the concept of water-induced thermogenesis. Resting energy expenditure remained unchanged after drinking distilled water or a 0.9% saline solution. Drinking water that had been cooled to 3 C increased resting energy expenditure by only about 5%. In contrast, ingestion of a 7% sucrose solution increased resting energy expenditure by 33 kJ over 90 min. This amounts to about 5% of the energy content of the sucrose and is in line with the dietary-induced thermogenesis reported elsewhere after carbohydrate ingestion (7, 14).

Boschmann *et al.* (6) reported that drinking water that had been warmed to 37 C reduced the postdrink increase in energy expenditure by 40%. The extent of this attenuation in water-induced thermogenesis closely matched the calculated energy required to heat the water from room temperature to body temperature (about 30 kJ for 500 ml) (6). On that basis, drinking cold water should augment the thermogenic effect. Indeed, we found that drinking distilled water that had been cooled to 3 C slightly increased resting energy expenditure by an average of 15 kJ over 90 min. However, this is substantially lower than the calculated energy required to heat the water from 3 to 37 C ($495 \text{ ml} \times 34 \text{ C} = 16830 \text{ cal} = 70 \text{ kJ}$), suggesting that most of the energy required for warming the water to body temperature is more likely to be met by a reduction in body heat loss, probably by the peripheral vasoconstriction that occurs after water drinking (5).

The 30% increase in energy expenditure after water drinking reported by Boschmann *et al.* (6) is impressive, but it is not supported by previously published studies (7–16) or the results of the current study. What could be the explanation for the apparent water-induced thermogenesis? One clue could be in the fact that Boschmann *et al.* (6) measured energy expenditure by whole-room indirect calorimetry, as opposed to the ventilated hood or mouthpiece techniques that we and others (7–16) have used. Ventilated hood and mouthpiece apparatus have a small dead space, thereby permitting rapid attainment of steady-state gas concentrations. In contrast, whole-room calorimeters may require 1 h or more to attain steady-state conditions because of their large size in relation to ventilation rate (18) and are therefore less suitable

for acute measurements. Boschmann *et al.* (6) used a run-in period of only 15 min before starting to measure resting energy expenditure. It is possible that their data reported from 30 min later, after water drinking, might be a consequence of a slow response time of the whole-room calorimeter and simply reflect earlier activities within the chamber. Unfortunately, Boschmann *et al.* (6) did not provide any information about the size of their chamber or the time required to reach a steady state.

Water-induced thermogenesis might also result from substances dissolved in the water. Boschmann *et al.* (6) did not state in their paper whether they used tap water, bottled water, or distilled water. Tap water and bottled water contain a number of dissolved electrolytes and impurities that could potentially stimulate a thermogenic effect. In the current study, we excluded this possibility by using distilled water.

Various studies have indicated that drinking half a liter of water increases the activity of the sympathetic nervous system (4, 5). The hypothesis that water drinking might also stimulate the metabolism therefore seems reasonable. It is, however, important to note that sympathetic activation does not uniformly affect all physiological functions. Sympathetic nervous system activity to several organs (such as heart, liver, kidneys, and pancreas) is increased in response to diet, but it is uncertain as to whether they contribute to dietary-induced thermogenesis (19). An increase in sympathetic activity to skeletal muscle has been reported after water drinking (5). Yet in humans, infusion of norepinephrine does not result in a detectable increase in thermogenesis in forearm skeletal muscle (20). Therefore, sympathetic activation observed after water drinking (particularly to skeletal muscle) might not necessarily lead to an increase in metabolic rate. Instead, the increase in sympathetic neural activity after water drinking is accompanied by peripheral vasoconstriction and a reduction in limb blood flow (5).

The confirmation of water-induced thermogenesis would have important public health implications, not least because water drinking might be useful as a safe, cheap, and non-pharmacological method of reducing weight. Using an experimental setup capable of rapidly detecting small changes in energy expenditure, we were unable to find a thermogenic effect of distilled water at room temperature. Consequently, our results cast doubt on a role for water as a thermogenic agent in the management of obesity.

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