

THE EFFECT OF HABITUAL SNACKING ON DIET-INDUCED THERMOGENESIS IN YOUNG WOMEN

HIDEYUKI TOBE

Abstract

This study investigated the effect of habitual snacking on the diet-induced thermogenesis (DIT) in non-obese young women. Thirteen women aged 18~23 yrs old were divided into two groups: group one was of six women who consumed snacks; candy, cookies, chocolate, etc., "very frequently" between meals almost everyday, group two was of seven women who seldom consumed snacks. The BMI of both groups were matched ($19.7 \pm 1.0 \text{ kg/m}^2$). The DIT was measured 5 h after ingestion of a meal (2220 kJ=531 kcal) using the Douglas bag technique. Each subject had their DIT measured on eight occasions to obtain average DIT values because DIT had large intra-individual variability. Indeed, each subject's DIT values were markedly different (the mean intra-individual CV=39.6%). There was no significant difference between the two groups in percentage of body fat, resting metabolic rate (RMR), or RMR/body weight. The mean value of DIT (\pm SD) of group one ($6.4 \pm 1.2\%$) was lower than that of group two ($7.8 \pm 0.9\%$) ($p < 0.05$). Also, the main effects of the groups and the time after ingesting the test meal on postprandial energy expenditure were significant (two-way ANOVA; $p < 0.001$), but the interaction between the group and the time after ingesting the test meal was not significant. In conclusion, eating habits with habitual snacking blunt DIT in young women.

(Jpn. J. Phys. Fitness Sports Med. 2003, 52 : 249~254)

key word : Diet-Induced Thermogenesis, Snacking, Young Women

INTRODUCTION

Not only excessive energy intake, but also defective regulation of energy expenditure, is thought to be a responsible factor in human obesity. So, investigators have studied the factors of energy expenditure such as resting metabolic rate and diet-induced thermogenesis (DIT), which is defined as the increase in energy expenditure in response to food intake. Despite the fact that DIT is a minor fraction of total energy expenditure (3~10%), blunted DIT is supposed to play a role in development and/or maintenance of human obesity¹⁾.

Although the clinical application of improving DIT for obesity is expected, the role of DIT on weight regulation is still controversial. Several cross-sectional studies indicated that DIT is blunted in obese groups^{2~4)}, but others did not

show a relationship^{5,6)}. Also, there are some conflicting results for the effects of weight loss on DIT. Maffeis et al.⁷⁾ showed that DIT increased after a 5 kg-weight loss in obese children, and Thorne et al.⁸⁾ showed similar results after an 18 kg-weight loss by gastric banding. On the other hand, Garrow et al.⁹⁾ and Nichols et al.¹⁰⁾ indicated that DIT decreased inversely after weight loss. This disagreement in the results might be related to the high intra-individual DIT variability¹¹⁾. A more accurate estimation or higher statistical power should be obtained by repeating the measurements for each subject.

Several investigators have experimentally studied the effect of meal frequency on DIT^{12,13)} and circadian variation in DIT¹⁴⁾. Few studies have ever been done about the effect of daily eating habits such as snacking between meals. The purpose of this study is to investigate the effect of

habitual snacking on the DIT using data measured repeatedly for each subject.

Subjects and Methods

Subjects

Fourteen women aged 18~23 y were selected as subjects through an interview about their eating habits, habitual exercise, and BMI for the purpose of forming two groups. However, one subject could not complete the whole study, so data from thirteen women was eventually used. Group one was of six women who ate snacks between meals (candy, cookies, chocolate, etc.) "very frequently" almost everyday; they were almost always carrying candies, chocolates, and so on with them, and frequently eating them. Group two was of seven women who seldom ate snacks. All women were normal weight and the mean BMIs of the two groups were strictly matched to evaluate the effect of eating habits independent of BMI. Three women (one was in group one, two were in group two) were playing less intense recreational sports once or twice a week; the others did not exercise regularly. The exercise routine of subjects was not considered in the analysis because there was no unique trend in thermogenesis between the women playing sports and the ones who didn't. None of the subjects had a history of endocrine or metabolic disorders, nor did they take any drugs. Each woman was given an oral and written explanation of the study and possible risks and benefits involved before they signed an informed consent form.

Experimental Protocol

Subjects were instructed to abstain from vigorous physical activity and insufficient sleep the day before the experiment, and to finish their evening meal at least 12 h before the DIT measurement. The subjects were asked to come to the laboratory by train or car, and to walk slowly

to the test room.

On arrival at the laboratory between 8:00 and 9:00 a. m., the subjects were instructed to rest in the supine position in the environmentally controlled room in which the temperature was kept at 24~26°C. After 30 min of rest, the subjects were placed in a semirecumbent position, and the resting metabolic rate (RMR) was measured for 30 min by indirect calorimetry for the baseline measurement. The expired gas was collected in a Douglas bag through a facemask. After this measurement, subjects were served a test meal, which consisted of bread, butter, cheese, and milk. The energy provided was 2220 kJ (531 kcal), with 15% of the energy from protein, 36% from fat, and 49% from carbohydrates. The test meal was consumed within 30 min. After ingestion of the meal, the gas mask was replaced and postprandial energy expenditure was measured for 20 min followed by 20 min non-gas collecting period; these were continued for 5 h, for a total of 8 measurements ((20 + 20) × 8). Subjects were instructed to remain awake and motionless in the same semirecumbent position throughout the measurements, but reading or watching TV were permitted.

The collected expired gas was analyzed for oxygen and carbon dioxide concentrations with a gas analyzer (NEC Medical Systems, Inc., Tokyo) that used a polarographic electrode method for oxygen and infrared gas analysis for carbon dioxide. Calibration for oxygen and carbon dioxide was done with 100% nitrogen for the zero point and with a reference gas (mixture of 16.2% O₂ and 5.0% CO₂). The gas analyzer was checked by the nitrogen and the reference gas before every O₂ and CO₂ analysis of expired gas. The volume of gas was measured by a gas meter using a pump for aspirating it in equal velocity from the Douglas bag. The energy expenditure was calculated by Weir's equation¹⁵⁾. The cumulative increase of energy expenditure over 5 hours after meal inges-

tion was determined from the area under the curves of the energy expenditure using the pre-meal RMR as the baseline. The values of non-gas collecting periods were interpolated as means of before and after gas collecting period. The DIT was expressed as a percentage of the test-meal energy load.

Each subject was measured eight times according to the same procedure described above. All experiments were executed during three months. Height, body weight, and percentage of body fat were measured after each measurement of DIT. The bioelectric impedance method (Tanita TBF-511, Tokyo) was used for percentage of body fat measurement. Subjects were asked to maintain their body weight during the experiment period. The means of body weight, BMI and percentage of body fat of both groups did not change during the experimental period. The difference in DIT between groups was assessed by the t-test using the mean values of the eight measurements as each individual's data. The energy expenditure after meal ingestion was analyzed by using a two-way analysis of variation (ANOVA); the main effects of the groups and the time after meal ingestion, and the interaction between the two factors.

Results

Table 1 shows the characteristics of the subjects. The WT, BMI, and percentage of body fat were not different between both groups. The mean (\pm SD) of BMI was 19.7 ± 1.0 kg/m² in both groups.

There were marked differences in individual measured DIT values; intra-individual CV varied from 13.8% to 84.1%. The mean intra-individual CV of all subjects was 39.6%. On the other hand, the reproducibility of baseline RMR was relatively good; the mean intra-individual CV was 6.5%. Table 1 shows the differences in the metabolic indices between the groups. The mean RMR of group one was 5381 ± 510 kJ/day and that of group two was 5286 ± 602 kJ/day. There was no significant difference between the groups in the RMR, RMR/WT, or RMR/FFW.

The mean DIT of group one was $6.4 \pm 1.2\%$ as a percentage of energy of the test meal and that of group two was $7.8 \pm 0.9\%$, which is a significant difference (t-test; $p < 0.05$). The differences in DIT/WT and DIT/FFW were not significant. Figure 1 shows the change of energy expenditure after the test meal. The energy expenditure of group one (seldom snacking) increased rapidly

Table 1. Means and standard deviations of the physical characteristics and metabolic indices of subjects. Differences between the groups were examined by the t-test.

	Group 1 (n = 6) (Snacking)	Group 2 (n = 7) (Seldom snacking)	Differences between groups
Physical characteristics			
Body weight: WT (kg)	49.4 \pm 4.0	50.0 \pm 5.0	ns
Body mass index: BMI (kg/m ²)	19.7 \pm 1.0	19.7 \pm 1.0	ns
Percent of body fat: %fat (%)	26.5 \pm 2.3	24.8 \pm 2.6	ns
Fat-free weight: FFW (kg)	36.3 \pm 2.2	37.5 \pm 2.6	ns
Metabolic indices			
Resting metabolic rate: RMR (kJ/d)	5381 \pm 510	5286 \pm 602	ns
RMR/WT (kJ/kg)	108.8 \pm 4.7	106.1 \pm 10.3	ns
RMR/FFW (kJ/kg)	148.1 \pm 8.3	141.2 \pm 14.8	ns
Diet-induced thermogenesis: DIT (%)	6.4 \pm 1.2	7.8 \pm 0.9	$p < 0.05$
DIT/WT	0.13 \pm 0.03	0.16 \pm 0.02	ns
DIT/FFW	0.18 \pm 0.03	0.21 \pm 0.03	ns

ns : not significant

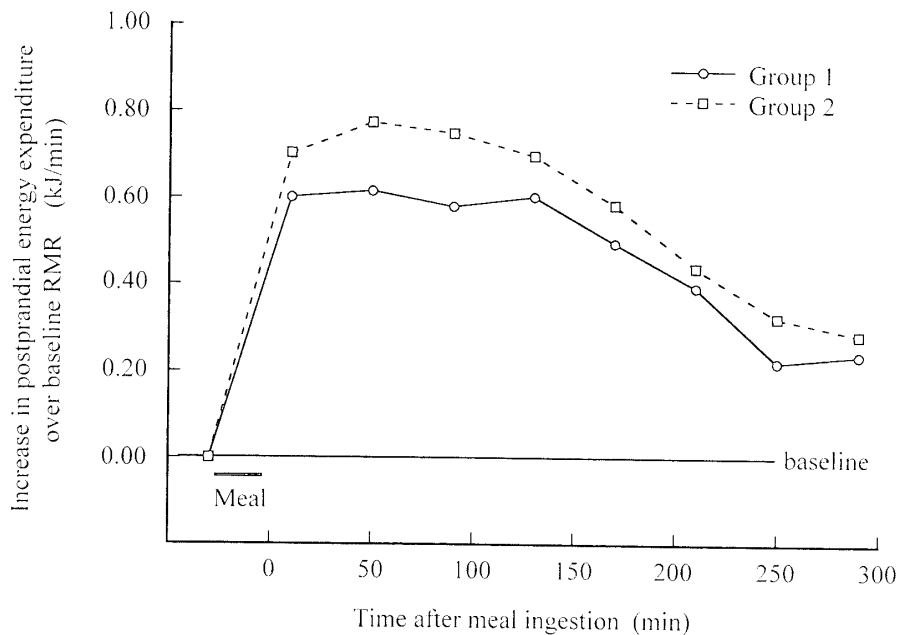


Figure 1. The increase of postprandial energy expenditure measured 5 h after the ingestion of the test meal. The data are shown as mean \pm SEM of group 1 (snacking) and 2 (seldom snacking). The main effect of the groups ($F=39.9$, $p<0.001$) and the time after meal ingestion ($F=63.1$, $p<0.001$) were significant, however the interaction between the two factors was not significant ($F=0.4$, $p>0.05$) by the two-way ANOVA.

after the meal, it reached the peak at 50 min and decreased gradually toward baseline after that. The energy expenditure of group two (frequently snacking) changed similarly to group one, but it ran under that of group one. The two-way analysis of variance (ANOVA) indicated that the main effects of the groups ($F=39.9$, $df=1$, $p<0.001$) and the time after meal ($F=63.1$, $df=7$, $p<0.001$) were significant, but the interaction between the two factors was not significant ($F=0.4$, $df=7$, $p>0.05$).

Discussion

Large intra-individual CVs of DIT have been reported so far (23~48% by a ventilated-hood^{11, 16)} or a metabolic chamber¹⁷⁾). The reproducibility in this study was also low (39.6%). However, by repeating measurements for individuals and using the mean value, relatively good precision was obtained; ie, the SEM (standard error of the mean) of individual DIT was $\pm 0.9\%$ as the per-

centage of the test-meal energy load on average.

Most studies about the effect of eating patterns on DIT have been executed in an experimental setting. The effect of meal frequency when total meal size is constant is especially interesting. LeBlanc et al.¹³⁾ showed the enhanced DIT in four small meals compared with one large meal with the same total caloric content, and said that the enhanced DIT in four small meals was due to lipid mobilization caused by repeated stimulation of the sympathetic nervous system. Tai et al.¹⁸⁾ examined the effect of frequency on DIT using the similar protocol as LeBlanc et al.¹³⁾, but they found that DIT was significantly higher when the one large meal was taken than when the same meal was taken in six portions at 30-min intervals. Young¹⁹⁾ and Molnar²⁰⁾ also observed enhanced DIT in one large meal compared with two or three small meals with the same total caloric content, respectively. Kinabo and Durnin¹²⁾ could not find a significant effect of meal frequency on DIT. Even though the findings are not consistent, they sug-

gest that the DIT might be lowered by increasing the frequency of meals even if the total caloric content is the same.

As far as I know, there is little information about whether habitual snacking affects DIT response or not. In the present study, significantly lower DIT was observed in women who habitually ate snacks compared with women who seldom did (Table 1). The results indicate the possibility that DIT could be lowered by increased snacking. The interaction between the group and the time after ingestion of the test meal was not detected.

There are studies suggesting that change in DIT might be related to activity in the sympathetic nervous system (SNS)^{21~23}. Schwartz et al.²⁴ reported that reduced DIT was associated with blunted SNS response to meals in older subjects. Bazelmans et al.²⁵ found the response to over-feeding of norepinephrine (NE) is blunted in subjects with obesity of childhood onset. Also, Welle²⁶ said that the thermogenic effect of norepinephrine accounted for approximately 20% of DIT using pharmacologic blockade of norepinephrine. These results suggest that the SNS induced increase in energy expenditure accounts for at least part of DIT. However, other authors could not find the effect of SNS on DIT^{27,28}. For example, Tappy et al.²⁸ stimulated SNS activity in the post-absorptive state by lower body negative pressure, and observed an increased plasma NE, but no significant effect on postprandial energy expenditure. The SNS activity by ingestion of nutrients might represent an important factor determining the magnitude of DIT, however no information was available on SNS activity in the present study. Further study is needed to clarify the effect of daily eating habits on DIT and the biological factor mediating them.

In conclusion, despite the high intra-individual variability of DIT, the data from repeated measurements on individuals suggested that habi-

tual snacking decreased the DIT in non-obese young women.

(Accepted Feb. 27, 2003)

Literature cited

- 1) Reed, G.W., and Hill, J. O. Measuring the thermic effect of food. *Am. J. Clin. Nutr.*, (1996), **63**, 164-169.
- 2) Laville, M., Cornu, C., Normand, S., Mithieux, G., Beylot, M., and Riou, J.P. Decreased glucose-induced thermogenesis at the onset of obesity. *Am. J. Clin. Nutr.*, (1993), **57**, 851-856.
- 3) Schutz, Y., Bessard, T., Jequier, E. Diet-induced thermogenesis measured over a whole day in obese and nonobese women. *Am. J. Clin. Nutr.*, (1984), **40**, 542-552.
- 4) Maffeis, C., Schutz, Y., Zocante, L., Micciolo, R., and Pinelli, L. Meal-induced thermogenesis in lean and obese prepubertal children. *Am. J. Clin. Nutr.*, (1993), **57**, 481-485.
- 5) Tounian, P., Girardet, J. P., Carlier, L., Frelut, M. L., Veinberg, F., and Fontaine, J.L. Resting energy expenditure and food-induced thermogenesis in obese children. *J. Pediatr. Gastroenterol Nutr.*, (1993), **16**, 451-457.
- 6) Weinsier, R. L., Nelson, K. M., Hensrud, D. D., Darnell, B. E., Hunter, G. R., and Schutz, Y. Metabolic predictors of obesity. Contribution of resting energy expenditure, thermic effect of food, and fuel utilization to four-year weight gain of post-obese and never-obese women. *J. Clin. Invest.*, (1995), **95**, 980-985.
- 7) Maffeis, C., Schutz, Y., and Pinelli, L. Postprandial thermogenesis in obese children before and after weight reduction. *Eur. J. Clin. Nutr.*, (1992), **46**, 577-583.
- 8) Thorne, A., Hallberg, D., and Wahren, J. Meal-induced thermogenesis in obese patients before and after weight reduction. *Clin. Physiol.*, (1989), **9**, 481-498.
- 9) Garrow, J. S., and Webster, J. D. Effects on weight and metabolic rate of obese women of a 3.4 MJ (800 kcal) diet. *Lancet*, (1989), **24**, 1429-1431.
- 10) Nichols, J. F., Bigelow, D. M., and Canine, K. M. Short-term weight loss and exercise training effects on glucose-induced thermogenesis in obese adolescent males during hypocaloric feeding. *Int. J. Obes.*, (1989), **13**, 683-690.
- 11) Weststrate, J. A. Resting metabolic rate and diet-induced thermogenesis: a methodological reappraisal. *Am. J. Clin. Nutr.*, (1993), **58**, 592-601.
- 12) Kinabo, J. L., and Durnin, J. V. Effect of meal frequency on the thermic effect of food in women. *Eur. J. Clin. Nutr.*, (1990), **44**, 389-395.
- 13) LeBlanc, J., Mercier, I., and Nadeau, A. Components

- of postprandial thermogenesis in relation to meal frequency in humans. *Can. J. Physiol. Pharmacol.*, (1993), **71**, 879-883.
- 14) Romon, M., Edme, J., Boulenguez, C., Lescroart, J., and Frimat, P. Circadian variation of diet-induced thermogenesis. *Am. J. Clin. Nutr.*, (1993), **57**, 476-480.
 - 15) Weir, J. B. New methods for calculating metabolic rate with special reference to protein metabolism. *J. Physiol.*, (1949), **109**, 1-9.
 - 16) Piers, L. S., Diggavi, S. N., Rijkskamp, J., Van Raaij, J. M. A., Shetty, P. S., and Hautvast, J. G. A. J. Resting metabolic rate and thermic effect of a meal in the follicular and luteal phases of the menstrual cycle in well-nourished Indian women. *Am. J. Clin. Nutr.*, (1995), **61**, 296-302.
 - 17) Tataranni, P. A., Larson, D. E., Snitker, S., and Ravussin, E. Thermic effect of food in humans: methods and results from use of a respiratory chamber. *Am. J. Clin. Nutr.*, (1995), **61**, 1013-1019.
 - 18) Tai, M. M., Castillo, P., and Pi-Sunyer, F. X. Meal size and frequency: effect on the thermic effect of food. *Am. J. Clin. Nutr.*, (1991), **54**, 783-787.
 - 19) Young, J. C. Meal size and frequency : effect on potentiation of the thermal effect of food by prior exercise. *Eur. J. Appl. Physiol. Occup. Physiol.*, (1995), **70**, 437-441.
 - 20) Molnar D. The effect of meal frequency on postprandial thermogenesis in obese children. *Pediatr. Padol.*, (1992), **27**, 177-181.
 - 21) Sims, E. A. H. Storage and expenditure of energy in obesity and their implications for management. *Med. Clin. North Am.* (1989), **73**, 97-110.
 - 22) Jung, R. T., Shetty, P. S., James, W. P. T., Barrand, M. A., Callingham, B. A. Reduced thermogenesis in obesity. *Nature*, (1979), **279**, 322-323.
 - 23) Acheson, K., Jequier, E., and Wahren, J. Influence of s-adrenergic blockade on glucose-induced thermogenesis in man. *J. Clin. Invest.*, (1983), **72**, 981-986.
 - 24) Schwartz R. S., Jaeger L. F., Veith R. C. The thermic effect of feeding in older men: the importance of the sympathetic nervous system, *Metabolism*, (1991), **39**, 733-737.
 - 25) Bazelmans J., Nestel P. J., O'Dea K. Blunted norepinephrine responsiveness to changing energy states in obese subjects. *Metabolism*, (1985), **34**, 154-160.
 - 26) Welle S. Sympathetic nervous system response to intake. *Am. J. Clin. Nutr.*, (1995), **62**, 1118-1122.
 - 27) Vaz M., Turner A., Kingwell B., Chin J., Koff E., Cox H., Jennings G., Esler M. Postprandial sympathetic-adrenal activity : its relation to metabolic and cardiovascular events and to changes in meal frequency. *Clin. Sci.*, (1995), **89**, 349-357.
 - 28) Tappy L., Girardet K., Schwaller N., Vollenweider L., Jequier E., Nicod P., Scherrer U. Metabolic effects of an increase of sympathetic activity in healthy humans. *Int. J. Obes. Relat. Metab. Disord.*, (1995), **19**, 419-422.