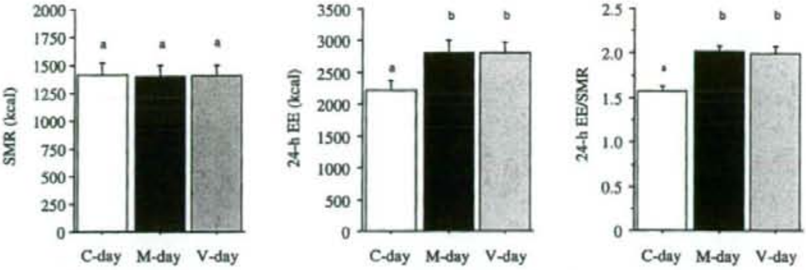

**FIGURE 2.** Sleeping metabolic rate (SMR), 24-h energy expenditure (24-h EE), and 24-h EE/SMR for each modeling day ($n = 11$). C-day, a control day; M-day; a day with high-frequency, moderate-intensity physical activity; V-day, a day with high-frequency vigorous-intensity physical activity. Error bars indicate SD. Bars with the same letter were not significantly different by one-way ANOVA with Scheffe's post hoc test ($P < 0.05$).

calculation, we determined that ≥9 subjects were needed. All values are presented as means ± SDs. Differences were considered to be statistically significant if the $P$ value was $<0.05$. The SMR, 24-h EE, and 24-h EE/SMR values obtained in the 3 protocols were compared by one-way analysis of variance, and significant differences were analyzed by using Scheffe's post hoc test. Differences between actual 24-h EE and predicted 24-h EE without EPEE on M-day or V-day were assessed by the paired $t$ test. Differences in any variable between M-day and V-day were assessed by the paired $t$ test. Correlations between EPEE on the M-day or the V-day and $\dot{V}O_{2\text{peak}}$ or body composition were assessed by Pearson's correlation coefficients ($r$). All statistical analyses were performed by using SPSS version 14.0J for WINDOWS (SPSS Inc, Chicago, IL).

## RESULTS

All subjects completed the 3-d metabolic chamber measurements according to prescribed timetables. The subjects' average total energy intake was 2685 ± 303 kcal, and this did not differ between the 3 d in each subject, because they ate all provided meals completely. Mean $\dot{V}O_{2\text{peak}}$ was 47.3 ± 8.3 $\text{mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$. Relative physical activity intensities for slow walking, brisk walking, and jogging were 21.4 ± 5.1%, 33.3 ± 7.0%, and 65.0 ± 14.1% of $\dot{V}O_{2\text{peak}}$, respectively.

Mean SMR, 24-h EE, and 24-h EE/SMR are shown in **Figure 2**. Twenty-four–hour EE for C-day, M-day, and V-day was 2228 ± 143 kcal, 2816 ± 197 kcal, and 2813 ± 163 kcal, respectively. No significant differences were observed in 24-h EE values between M-day and V-day, although there were significant differences between C-day and M-day or V-day. There were no significant differences between SMR values (or BMR values) for the 3-d periods for the subjects. CVs for SMR and BMR over 3 d were 1.0% and 1.7%, respectively. Twenty-four–hour EE/SMR for 3 d was 1.58 ± 0.06 for C-day, 2.02 ± 0.07 for M-day, and 2.00 ± 0.08 for V-day.

There were no significant differences between the measured 24-h EE value and the predicted 24-h EE value without EPEE for M-day or V-day (**Table 3**). Mean EPEE values for M-day and V-day were not significantly different. Relative EPEE values to measured 24-h EE values were 1.2 ± 2.7% and 1.0 ± 0.8%. Furthermore, relative EPEEs to ΔPAEEs were 6.2 ± 13.9% and 5.1 ± 9.2%, respectively.

Mean percentages of spontaneous physical activity during prescribed physical activity (slow walking, brisk walking, and jogging) for the 3 d were ≈100%. There were no significant differences in mean percentages of spontaneous physical activity for resting periods for the 3 d (C-day: 38.1 ± 9.0%; M-day: 43.8 ± 5.6%; V-day: 40.4 ± 8.2%).

The relations between EPEE/24-h EE and $\dot{V}O_{2\text{peak}}$ or fat-free mass for M-day or V-day are shown in **Figure 3** and **Figure 4**. EPEE/24-h EE for V-day was negatively correlated with $\dot{V}O_{2\text{peak}}$, whereas no significant correlation between these variables was observed for EPEE/24-h EE for M-day. As for the relation between EPEE/24-h EE or EPEE and fat-free mass, no significant correlations were observed for either M-day or V-day. Also, fat mass and body mass indexes were not significantly correlated with EPEE/24-h EE or EPEE for either day. The correlation coefficients for EPEE versus between-day SMR difference were 0.31 (NS) and 0.63 ($P < 0.05$) for M-day and V-day, respectively.

## DISCUSSION

This investigation examined the effects of EPEE on 24-h EE by modeling normal living activities in a metabolic chamber.

**TABLE 3**

Elevated post–physical activity energy expenditure (EPEE) for the M-day and the V-day[1]

| | M-day[2] | V-day[2] |
|---|---|---|
| Predicted 24-h EE without EPEE (kcal)[3] | 2781 ± 185 | 2784 ± 167 |
| Measured 24-h EE (kcal) | 2816 ± 197[4] | 2813 ± 163[4] |
| Δ PAEE (kcal)[5] | 553 ± 53 | 556 ± 49 |
| EPEE (kcal)[6] | 35 ± 78 | 29 ± 53 |
| EPEE/measured 24-h EE × 100 (%) | 1.2 ± 2.7 | 1.0 ± 0.8 |
| EPEE/Δ PAEE × 100 (%) | 6.2 ± 13.9 | 5.1 ± 9.2 |

[1] All values are $\bar{x}$ ± SD. M-day, a day with high-frequency, moderate-intensity physical activity; V-day, a day with high-frequency, vigorous-intensity physical activity; 24-h EE, 24-h total energy expenditure; Δ PAEE, additional physical activity–induced energy expenditure.

[2] There was no significant difference in each variable between the M-day and the V-day by paired $t$ test ($P < 0.05$).

[3] Values were calculated on the basis of a control day.

[4] There was no significant difference between predicted 24-h EE without EPEE and measured 24-h EE on the M-day or the V-day by paired $t$ test ($P < 0.05$).

[5] Predicted 24-h EE without EPEE minus control day.

[6] Measured 24-h EE minus predicted 24-h EE without EPEE.

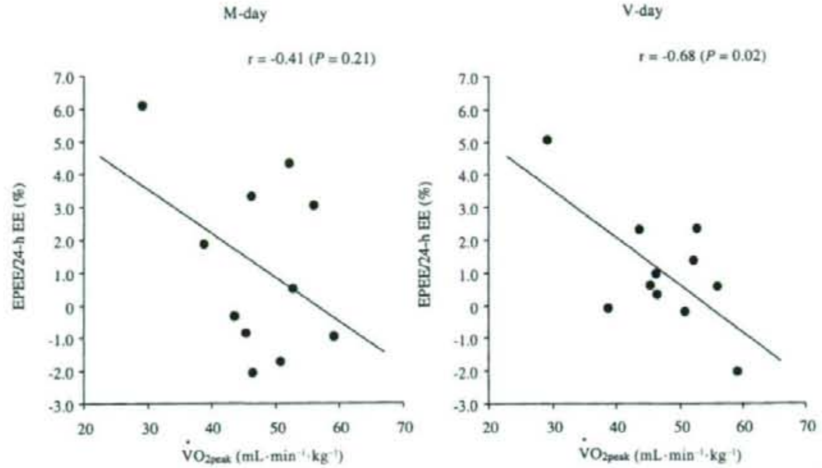

**FIGURE 3.** Relation between elevated post–physical activity energy expenditure (EPEE) and peak oxygen uptake ($\dot{V}O_{2peak}$) for the M-day (a day with high-frequency, moderate-intensity physical activity) and the V-day (a day with high-frequency, vigorous-intensity physical activity). 24-h EE, 24-h energy expenditure.

There was no significant additional EE as EPEE in total 24-h EE when the subjects spent 24 h in a metabolic chamber under conditions that were ≈2.0 for 24-h EE/SMR (similar to PAL) for high-frequency, moderate-intensity or vigorous-intensity physical activity. However, subjects with low physical fitness may produce significant additional EE as EPEE by increasing daily vigorous-intensity physical activity.

Although EPOC has been studied in depth, most analyses examined the effects of EPOC after a single bout of exercise (4, 6). However, if one is to understand the effects of EPOC in weight reduction or maintenance, it is important to investigate the magnitude of EPOC after physical activity under normal living conditions. Previous studies suggested that EPOC after a single bout of exercise was generated in proportion to exercise duration, when exercise intensity exceeds about 50–60% $\dot{V}O_{2max}$ (6–8). Almuzaini et al (29) reported that dividing a 30-min exercise session into 2 parts significantly increased the magnitude of EPOC. However, this study was limited to the 40-min period after exercise, and the difference between EPOC values was only ≈10 kcal. Normal daily activity conditions evidently differ from those experimental conditions, because in the normal course of daily activity, people engage in a wide range of physical activity, the intensity of which vary from light to vigorous (26). The duration of most of these activities

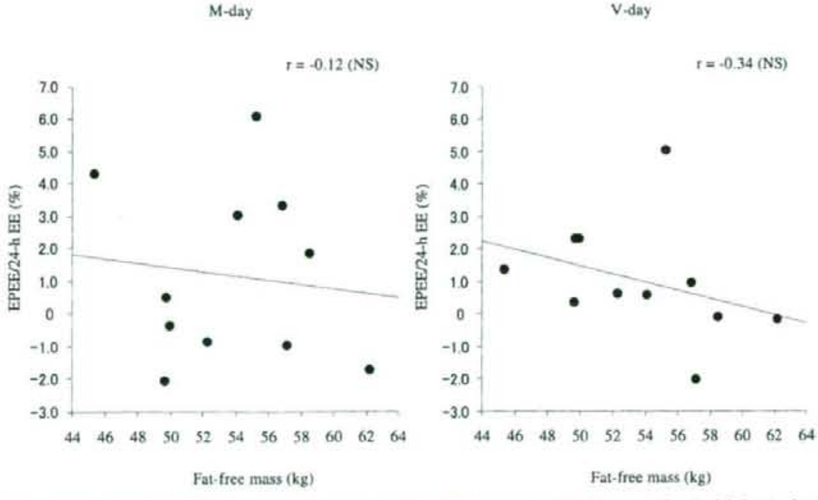

**FIGURE 4.** Relation between elevated post–physical activity energy expenditure (EPEE) and fat-free mass for the M-day (a day with high-frequency, moderate-intensity physical activity) and the V-day (a day with high-frequency, vigorous-intensity physical activity). 24-h EE, 24-h energy expenditure. There were no significant correlations between EPEE and fat-free mass for any day ($P < 0.05$).

and the intervals between moderate-to-vigorous physical activity are short, but their frequencies are high. This situation requires examination of the effects of EPEE (similar to, but a broader component than EPOC) on 24-h EE under normal activity conditions.

If significant EPEE by combined physical activity in daily living exists, one reason might be that the total duration of all physical activity amounted to several hours per 24 h, even though each activity was short. Furthermore, short intervals between physical activities with high frequencies may produce higher EPEE than the EPEE induced by a prolonged, single bout of exercise. However, the present study failed to identify any significant additional EE as EPEE in the 24-h EE. That is, multiplying short-duration physical activities with short intervals between activities may not contribute much toward increasing the 24-h EE, compared with prolonged physical activity, even if persons perform these short-duration physical activities with high frequencies during a 24-h period. The Institute of Medicine has proposed that 15% of EE as EPOC to ΔPAEE from sedentary condition should be added in estimating total energy expenditure (14). This proposition is based on the report by Bahr et al (13), which compared EPOC magnitudes between 20, 40, and 80 min of ergometer exercise at 70% $\dot{V}O_{2max}$. As a result, each EPOC was ≈15% of exercise-induced EE for 12 h after the exercise sessions. However, this evidence was obtained from a single-bout trial. In our experimental design, normal daily activity conditions were modeled, and EPEE resulted in 6.2 ± 13.9% (M-day) and 5.1 ± 9.2% (V-day) of ΔPAEE. Therefore, the equation estimating TEE proposed by the Institute of Medicine, which adds 15% EE as EPOC to ΔPAEE, would overestimate 24-h EE.

Previous studies using a single round of exercise suggest that one possible explanation for not observing significant EPEE in daily activity was that many daily activities do not reach the intensity threshold of 50–60% $\dot{V}O_{2max}$ (7, 8). We adopted brisk walking (3.8 METs) as a moderate-intensity physical activity and jogging (8.0 METs) as a vigorous-intensity physical activity. Note that Pate et al (30) have defined that moderate-intensity physical activity ranged from 3 to 6 METs and vigorous-intensity physical activity was >6 METs. The relative exercise intensities of brisk walking and jogging were 33.3 ± 7.0% and 65.0 ± 14.1%, respectively. Brisk walking did not reach 50–60% $\dot{V}O_{2max}$ as the intensity threshold for producing EPOC. On the other hand, Melanson et al (31) reported that if energy expenditures were matched between high-intensity exercise and low-intensity exercise with well-controlled conditions in a metabolic chamber, no significant difference was observed in 24-h EE values between these 2 conditions. Saris and Schrauwen (32). reported that in obese subjects, no significant differences in 24-h EE values were observed between the day with high-intensity interval exercise and the day with low-intensity endurance exercise when the exercise sessions were equicaloric in energy expenditure. From our results and previous studies, it appears that exercise or physical activity intensity does not influence the contribution of EPOC to 24-h EE. However, a significant difference of EPOC magnitude may be found between high- and low-intensity exercise when one measures EPOC within several hours of a single-bout trial.

The inclusion (or lack thereof) of oxygen deficit in calculations of EPOC may contribute to discrepancies among studies. Most studies with a single-bout trial compared EPOC to oxygen consumption during an exercise bout (4, 6), in which case oxygen deficit is not taken into account. On the other hand, we discuss EPEE without EE equivalent to oxygen deficit, by extrapolating steady state EE (excluding EE in the first 3 min and the last 1 min) to the whole EE in each physical activity period. Although total EPOC is greater than oxygen deficit, a large part of the rapid component of EPOC is explained by oxygen deficit during the exercise (5). If we consider the extra energy expenditure due to exercise or physical activity in 24-h EE, such as the estimation of TEE in the Institute of Medicine (14), EE corresponding to oxygen deficit should be excluded. However, the study by Bahr et al (13) discussed the contribution of EPOC to EE during an exercise bout, so adding 15% as EPOC to ΔPAEE should lead to overestimation of TEE.

Although EPEE did not contribute to a significant increase in 24-h EE in the present study, EPEE/24-h EE for V-day was significantly correlated with $\dot{V}O_{2peak}$. That is, a person with low fitness may generate more EPEE by increasing daily physical activity. Short and Sedlock (33) reported that even though EE during exercise in trained subjects was much higher than that in untrained subjects, EPOC was similar between the groups if different subjects exercised at the same relative intensity of 70% $\dot{V}O_{2peak}$. When compared with subjects with higher $\dot{V}O_{2max}$, those with lower oxygen uptake produced more lactate, a major biochemical component of EPOC (34). Therefore, a negative relation between $\dot{V}O_{2peak}$ and EPEE/24-h EE observed in the present investigation is reasonable.

Trained individuals have better thermoregulatory capacities than do untrained individuals because physical training enhances the sweating mechanism at a given level of the central sweating drive (35). Therefore, elevated body temperature in untrained individuals could last longer than in trained individuals (36). These phenomena require extra oxygen consumption for recovery. Therefore, fitness level may contribute to the magnitude of EPEE. On the other hand, there was no significant relation between EPEE/24-h EE for the M-day and $\dot{V}O_{2peak}$. In other words, it is assumed that exercise duration (with relative intensity over the threshold for producing the slow component of EPOC) may be longer in less fit subjects than in fit subjects, leading to more slow-component production in less fit subjects. However, the relative intensities of the 3 physical activities in this study were different in individuals, because these physical activities were prescribed by the same absolute intensities. Interestingly, EPEE was negative on both the M-day and the V-day in several subjects. One possible reason may have been interday variations in resting metabolic rate. In the present study, SMR values on M-day or V-day were lower than the SMR values on C-day in some cases, and the correlation coefficients for EPEE versus between-day SMR difference were 0.31 and 0.63 on M-day and V-day, respectively. Therefore, the negative values of EPEE may reflect interday variations in resting metabolic rate. In addition, people may compensate for an increase in EE due to activity by decreasing their nonexercise EE (37). In the present study, the total duration of prescribed physical activity was 240 min on the M-day. That is, the duration on the M-day was 60 min longer than on the V-day. Therefore, the total duration or frequency of physical activity during the day may affect the observed decrease in nonexercise EE.

In the present study, there were no significant correlations between EPEE for M-day (or V-day) and physical status (fat-free mass, fat mass, and body mass index). Crommett and Kinzey (38) have reported similar results. Although body composition may not influence EPEE, many obese persons are untrained and have a low level of fitness. Therefore, our results suggest that obese persons with a low fitness level could expect additional EE as EPEE to assist in weight reduction, provided that they perform high-frequency, relatively vigorous physical activity in the course of daily living. Otherwise, persons with a high fitness level may not expect significant EPEE through daily physical activity, because daily activity is difficult to adjust to high intensity, like typical exercise activity.

This study had several limitations. First, we could not measure actual daily physical activity because we conducted the measurements in a metabolic chamber. Additionally, the duration of each physical activity under normal living conditions may be shorter than 15 min in many cases, whereas 15 min was the minimum duration of activity required for accurate measurement values in this study. Even though we gathered data under well-controlled conditions, our experimental protocol could not detect any EPEE on the control day. However, control day EPEE values should be added to the calculated EPEE values on M-day and V-day. Although we believe that control day EPEE values would not be large, the actual ΔPAEE in normal daily living may thus be slightly higher than our calculated results. Furthermore, future studies are needed to clarify the association between fitness and EPEE, because inclusion of a single subject in our data set, the one with the lowest fitness, strongly contributed to the significant (but modest) inverse association observed between these variables.

In conclusion, we found that EPEE has a only small effect on 24-h EE under normal living conditions. Therefore, adding 15% EE as EPOC to ΔPAEE, in accord with the Institute of Medicine recommendation for estimating TEE, would overestimate 24-h EE. However, persons with a low physical fitness level may produce additional EE as EPEE by increasing relatively vigorously intense daily living physical activity.

We express our heartfelt thanks to the subjects who participated in this study. We thank the members of the National Institute of Health and Nutrition, especially Hiroko Kogure, Emiko Taguri, and Rieko Miyake, for their help in data acquisition and analyses.

The responsibilities of the authors were as follows—KO: study design, data acquisition, data analysis, data interpretation, and writing the manuscript; ST: study design, data acquisition, data analysis, data interpretation, and writing the manuscript; KI: study design and editing the manuscript, IT: study design and editing key aspects of the manuscript. None of the authors had a personal or financial conflict of interest.

## REFERENCES

1. World Health Organization. Diet, nutrition and the prevention of chronic diseases. World Health Organ Tech Rep Ser 2003;916:1–149.
2. Klem ML, Wing RR, McGuire MT, Seagle HM, Hill JO. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. Am J Clin Nutr 1997;66:239–46.
3. Hill JO, Wyatt HR. Role of physical activity in preventing and treating obesity. J Appl Physiol 2005;99:765–70.
4. LaForgia J, Withers RT, Gore CJ. Effects of exercise intensity and duration on the excess post-exercise oxygen consumption. J Sports Sci 2006;24:1247–64.
5. Gaesser GA, Brooks GA. Metabolic bases of excess post-exercise oxygen consumption: a review. Med Sci Sports Exerc 1984;16:29–43.
6. Borsheim E, Bahr R. Effect of exercise intensity, duration and mode on post-exercise oxygen consumption. Sports Med 2003;33:1037–60.
7. Bahr R, Sejersted OM. Effect of intensity of exercise on excess postexercise $O_2$ consumption. Metabolism 1991;40:836–41.
8. Gore CJ, Withers RT. Effect of exercise intensity and duration on postexercise metabolism. J Appl Physiol 1990;68:2362–8.
9. Levine JA, Eberhardt NL, Jensen MD. Role of nonexercise activity thermogenesis in resistance to fat gain in humans. Science 1999;283:212–4.
10. American College of Sports Medicine. ACSM's guideline for exercise testing and prescription. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2006.
11. Ministry of Health Labour and Welfare of Japan. Exercise and physical activity reference for health promotion 2006. Internet: http://www.nih.go.jp/eiken/programs/pdf/epar2006.pdf (accessed 2 June 2007).
12. Levine JA, Lanningham-Foster LM, McCrady SK, et al. Interindividual variation in posture allocation: possible role in human obesity. Science 2005;307:584–6.
13. Bahr R, Ingnes I, Vaage O, Sejersted OM, Newsholme EA. Effect of duration of exercise on excess postexercise $O_2$ consumption. J Appl Physiol 1987;62:485–90.
14. Institute of Medicine. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty, acids, cholesterol, protein and amino acids. Washington, DC: National Academy Press, 2005.
15. Leonard WR, Galloway VA, Ivakine E. Underestimation of daily energy expenditure with the factorial method: implications for anthropological research. Am J Phys Anthropol 1997;103:443–54.
16. Warwick PM. Factorial estimation of daily energy expenditure using a simplified method was improved by adjustment for excess post-exercise oxygen consumption and thermic effect of food. Eur J Clin Nutr 2006;60:1337–40.
17. Seale JL, Klein G, Friedmann J, Jensen GL, Mitchell DC, Smiciklas-Wright H. Energy expenditure measured by doubly labeled water, activity recall, and diet records in the rural elderly. Nutrition 2002;18:568–73.
18. Walsh MC, Hunter GR, Sirikul B, Gower BA. Comparison of self-reported with objectively assessed energy expenditure in black and white women before and after weight loss. Am J Clin Nutr 2004;79:1013–9.
19. Borg G. Borg's perceived exertion and pain scales. Champaign, IL: Human Kinetics, 1998.
20. Futami J, Tanaka S, Yamamura C, Oka J, Ishikawa-Takata K, Kashiwazaki H. Measurement of energy expenditure by whole-body indirect human calorimeter—evaluation of validity and error factors. J Jpn Soc Nutr Food Sci 2003;56:229–36.
21. Yamamura C, Tanaka S, Futami J, Oka J, Ishikawa-Takata K, Kashiwazaki H. Activity diary method for predicting energy expenditure as evaluated by a whole-body indirect human calorimeter. J Nutr Sci Vitaminol (Tokyo) 2003;49:262–9.
22. Weir JB. New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol 1949;109:1–9.
23. Ministry of Health Labour and Welfare Japan. Dietary reference intakes for Japanese, 2005. Tokyo, Japan: Daiichi Shuppan, 2005.
24. Meijer EP, Goris AH, Wouters L, Westerterp KR. Physical inactivity as a determinant of the physical activity level in the elderly. Int J Obes Relat Metab Disord 2001;25:935–9.
25. Westerterp KR. Pattern and intensity of physical activity. Nature 2001;410:539.
26. Ainsworth BE, Haskell WL, Whitt MC, et al. Compendium of physical activities: an update of activity codes and MET intensities. Med Sci Sports Exerc 2000;32:S498–504.
27. Hughson RL, O'Leary DD, Betik AC, Hebestreit H. Kinetics of oxygen uptake at the onset of exercise near or above peak oxygen uptake. J Appl Physiol 2000;88:1812–9.
28. Koga S, Poole DC, Shiojiri T, et al. Comparison of oxygen uptake kinetics during knee extension and cycle exercise. Am J Physiol Regul Integr Comp Physiol 2005;288:R212–20.
29. Almuzaini KS, Potteiger JA, Green SB. Effects of split exercise sessions on excess postexercise oxygen consumption and resting metabolic rate. Can J Appl Physiol 1998;23:433–43.
30. Pate RR, Pratt M, Blair SN, et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. JAMA 1995;273:402–7.
31. Melanson EL, Sharp TA, Seagle HM, et al. Effect of exercise intensity on 24-h energy expenditure and nutrient oxidation. J Appl Physiol 2002;92:1045–52.

32. Saris WH, Schrauwen P. Substrate oxidation differences between high- and low-intensity exercise are compensated over 24 hours in obese men. Int J Obes Relat Metab Disord 2004;28:759–65.
33. Short KR, Sedlock DA. Excess postexercise oxygen consumption and recovery rate in trained and untrained subjects. J Appl Physiol 1997;83:153–9.
34. Hermansen L, Stensvold I. Production and removal of lactate during exercise in man. Acta Physiol Scand 1972;86:191–201.
35. Astrand P, Rodahl K. Textbook of work physiology: physiological bases of exercise. 2nd ed. New York, NY: McGraw-Hill Book Company, 1977.
36. Frey GC, Byrnes WC, Mazzeo RS. Factors influencing excess postexercise oxygen consumption in trained and untrained women. Metabolism 1993;42:822–8.
37. Leon AS, Casal D, Jacobs D Jr. Effects of 2,000 kcal per week of walking and stair climbing on physical fitness and risk factors for coronary heart disease. J Cardiopulm Rehabil 1996;16:183–92.
38. Crommett AD, Kinzey SJ. Excess postexercise oxygen consumption following acute aerobic and resistance exercise in women who are lean or obese. J Strength Cond Res 2004;18:410–5.

ANTI-AGING MEDICINE

Received: Oct 19, 2007
Accepted: Dec 5, 2007

*Original Article*

# Accuracy of Predictive Equations for Basal Metabolic Rate and Contribution of Abdominal Fat Distribution to Basal Metabolic Rate in Obese Japanese People

Shigeho Tanaka [1], Kazunori Ohkawara [1], Kazuko Ishikawa-Takata [1], Akemi Morita [2], Shaw Watanabe [3]

1) Health Promotion and Exercise Program, National Institute of Health and Nutrition
2) Nutritional Education Program, National Institute of Health and Nutrition
3) National Institute of Health and Nutrition

## *Abstract*

**BACKGROUND:** Large errors may occur when predicting basal metabolic rate (BMR) based on physical characteristics in obese people. In addition, the contribution of abdominal visceral fat to BMR remains controversial. This study examined the accuracy of several predictive equations for BMR and the contribution of abdominal fat distribution to BMR in obese Japanese participants in the Saku Control Obesity Program (SCOP).

**METHODS:** BMR was determined using a mask and Douglas bag in adult males ($n = 12$) and females ($n = 11$). We measured abdominal subcutaneous and visceral fat areas using computerized tomography.

**RESULTS:** All the equations, with the exception of Bernstein's, overestimated BMR in obese males. Some equations, including the Japan-Dietary Reference Intakes and the Food and Agriculture Organization of the United Nations/World Health Organization/United Nations University (FAO/WHO/UNU) equations, overestimated BMR in obese females, while the Harris-Benedict and Henry equations provided relatively accurate predictions of BMR in obese females. We found no correlation between abdominal visceral fat area and BMR when adjusted for sex, fat-free mass, and abdominal subcutaneous fat area (partial $r = -0.022$). Abdominal subcutaneous fat area correlated significantly with BMR when adjusted for sex, fat-free mass, and abdominal visceral fat area (partial $r = 0.732$), although this correlation was no longer significant after adjustment for total fat mass (partial $r = 0.266$).

**CONCLUSIONS:** In obese Japanese subjects, most the predictive equations overestimated BMR in males, whereas some equations were relatively accurate for females. Our findings indicate abdominal fat distribution may not be independently related with BMR.

***KEY WORDS:*** Basal metabolic rate, obese, predictive equation, abdominal visceral fat

## *Introduction*

Basal metabolic rate (BMR) constitutes the largest component of total energy expenditure in the majority of people. Because BMR can be predicted from simple anthropometric measurements, it is often used to estimate total energy expenditure.

Many equations have been developed for estimating basal or sleeping metabolic rates based on anthropometric measurements, age, and sex.[1,2] These equations can be helpful when actual metabolic measurements are not available. It has been shown in Caucasians, however, that BMR is considerably more difficult to predict in obese than in normal-weight subjects.[2-5] Studies have found that predictive equations overestimate BMR and/or that large prediction errors may occur in obese subjects.[2-5] In addition, most of the equations currently available apply only to Caucasians. The validity of the predictive equations has not been examined in obese Japanese subjects, despite several studies showing that some of the predictive equations are not applicable to nonwhite populations.[6-9]

In addition, the contribution of abdominal fat distribution to BMR remains controversial. Some studies have shown a relationship between abdominal visceral fat (AVF) area and BMR or resting metabolic rate,[10-14] whereas others have not.[15-17] To our knowledge, no study has examined these relationships in Japanese subjects using computerized tomography (CT), with the exception of Okura et al, who investigated the relationship in healthy elderly subjects.[14] They reported that adjusted resting energy expenditure correlated inversely with AVF but not with abdominal subcutaneous fat (ASF). While significant, this relationship with AVF was relatively weak ($r = -0.131$).

In the present study, we examined the validity of predictive equations for BMR in obese Japanese men and women. The contribution of abdominal fat distribution, as measured by CT, to BMR was also examined.

Anti-Aging Medicine 5 (1) : 17-21, 2008

Dr. Shigeho Tanaka / Health Promotion and Exercise Program,
National Institute of Health and Nutrition
1-23-1 Toyama, Shinjuku-ku, Tokyo 162-8636, Japan

## *Methods*

### *Subjects*

The subjects in the study were 50- to 54-year-old obese subjects (12 males and 12 females) residing in Saku city. They were randomly selected from among the participants in the Saku Control Obesity Program (SCOP), the details of which are described elsewhere in this supplement.[18,19] The measurements of BMR for one of the female subjects failed; therefore, data for 12 males and 11 females were used in the present study.

The study protocol was approved by the Ethics Committees of the National Institute of Health and Nutrition and Saku Central Hospital. The study protocol was explained to the subjects prior to enrollment, and all subjects provided their informed consent.

### *Basal Metabolic Rate*

The subjects reported to the hospital for the series of measurements at approximately 8 am on the study day. BMR was measured in the supine position and in the post-absorptive state (12 hours or longer after the last meal). The temperature in the room was controlled at 24–26°C. The measurement was performed using a mask and Douglas bag for 20 minutes with 1 minute of intermission. The volume of expired air was measured with a certified dry gas meter (Shinagawa DC-5, Tokyo, Japan). The expired air was sampled and the $O_2$ and $CO_2$ concentrations were measured using a gas analyzer (Arco System, AR-1, Kashiwa, Japan) with a galvanic $O_2$ sensor and an infrared $CO_2$ sensor. For each of the consecutive measurements, the gas analyzer was calibrated initially using atmospheric air. The values of $O_2$ consumption and $CO_2$ production were expressed under standard temperature, pressure, and dry air conditions. BMR was estimated from $O_2$ consumption and $CO_2$ production using Weir's equation.[20]

### *Anthropometric Measurements*

Body weight was measured to the nearest 0.1 kg and height to the nearest 0.1 cm using a stadiometer. The measurements were performed in light clothing and underwear. Body mass index (BMI) was calculated as weight (kg) divided by square of height ($m^2$). Percentage body fat was evaluated by the bioelectric impedance method (Tanita, BF-220, Tokyo, Japan).

To assess ASF and AVF levels, a CT scan was performed at the level of the umbilicus, with the subject in the supine position. ASF and AVF areas were determined using commercially available software (Fat Scan; N2 System Corp., Osaka, Japan).[21] The attenuation range of CT numbers for ASF was set as the mean ± 3 standard deviation (SD).

### *Predictive Equations of Basal Metabolic Rate*

The predictive equations of Japan-Dietary Reference Intakes (DRI),[22] Harris and Benedict,[23] the Food and Agriculture Organization of the United Nations/World Health Organization /United Nations University (FAO/WHO/UNU),[24] Henry,[25] Owen,[26,27] Mifflin,[28] and Bernstein[29] were evaluated (***Table 1***). For the Japan-DRI equations, the Ministry of Health and Welfare proposed adjusting for body weight.[30] Therefore, the equations including this adjustment were also examined. For the FAO/WHO/UNU equations, those using body weight only are often used. However, in the present study, equations using body weight and height were also examined.

### *Statistical Analyses*

The results are presented as the mean ± SD. The % difference of the prediction error was calculated as the residual divided by the measured value for each subject. The relationship between measured and predicted values of BMR and anthropometric measurements was examined using Pearson's correlation. Sex was treated as a binomial variable (0 for males, 1 for females) and was adjusted for in the partial correlation analysis. Adjustment for age was not performed because the range was small (50–54 years). Statistical significance was set at $p < 0.05$ for all predictors. The statistical analyses were performed using SPSS® for Windows (version 14.0; SPSS Inc., Chicago, IL, USA).

***Table 1 Predictive equations for basal metabolic rate used in the present study***

| Predictive equations (kcal/day) | Males | Females |
|---|---|---|
| Japan-DRI [22] | weight × 21.5 | weight × 20.7 |
| Japan-DRI with adjustment for body weight [22,30] | [21.5 + (10.8 - 0.173 × weight)] × weight | [20.7 + (8.9 - 0.172 × weight)] × weight |
| Harris and Benedict [23] | 66 + (13.7 × weight) + (5.0 × height) − (6.8 × age) | 665 + (9.6 × weight) + (1.8 × height) − (4.7 × age)] |
| FAO/WHO/UNU (body weight) [24] | 879 + (11.6 × weight) | 829 + (8.7 × weight) |
| FAO/WHO/UNU (body weight and height) [24] | 901 + (11.3 × weight) + (16.0 × height/100) | 865 + (8.7 × weight) − (25.0 × height/100) |
| Henry [25] | [(59.2 × weight + 2480)]/4.184 | [(40.7 × weight + 2900)]/4.184 |
| Owen [26,27] | 879 + (10.20 × weight) | 795 + (7.18 × weight) |
| Mifflin [28] | 5 + (9.99 × weight) + (6.25 × height) − (4.92 × age) | −161 + (9.99 × weight) + (6.25 × height) − (4.92 × age) |
| Bernstein [29] | −1032 + (11.0 × weight) + (10.2 × height) − (5.8 × age) | 844 + (7.48 × weight) − (0.42 × height) − (3.0 × age) |

weight: kg, height: cm, age: year.
Predictive equations for 50 to 54-yr-old obese subjects were used.

## *Results*

The physical characteristics of the subjects are summarized in ***Table 2***. There was a similar degree of correlation between the measured and predicted values of BMR for the various predictive equations ($r = 0.839–0.859$). The relationships between measured and predicted BMR based on the Japan-DRI, DRI-adjusted, Harris-Benedict, and Bernstein equations are shown in ***Figures 1 and 2***. In obese males, the majority of equations overestimated BMR, particularly for those with lower BMR (***Figure 1***), whereas Bernstein's equation significantly underestimated BMR (***Table 3***). In particular, the Japan-DRI and FAO/WHO/UNU equations overestimated BMR to the greatest extent. The Mifflin equation provided a better prediction of BMR, while the equation overestimated BMR. In obese females, the Japan-DRI and FAO/WHO/UNU equations overestimated BMR, whereas the Harris-Benedict and Henry equations provided a relatively accurate prediction of BMR (***Figure 2***). In both sexes, adjustment for body weight in the Japan-DRI equations attenuated the overestimation of BMR, although the value still remained too high. In addition, the FAO/WHO/UNU equations with or without body height provided almost identical values. The SD of the % difference of the predicted BMR was comparable between the various equations.

***Table 2 Physical characteristics of subjects***

| | Mean± SD | Range |
|---|---|---|
| **Males** | | |
| Age (year) | 52 ± 1 | 50.0 - 54.0 |
| Body height (cm) | 172.8 ± 3.9 | 168.8 - 179.2 |
| Body weight (kg) | 91.3 ± 10.0 | 79.1 - 116.5 |
| Body mass index (kg/m$^2$) | 30.6 ± 3.3 | 27.7 - 39.2 |
| Percentage of body fat (%) | 28.3 ± 4.9 | 23.4 - 39.2 |
| Abdominal subcutaneous fat area (cm$^2$) | 272 ± 80 | 163 - 436 |
| Abdominal visceral fat area (cm$^2$) | 165 ± 51 | 98 - 289 |
| **Females** | | |
| Age (year) | 53 ± 2 | 50.0 - 54.0 |
| Body height (cm) | 158.6 ± 5.8 | 152.0 - 169.3 |
| Body weight (kg) | 82.5 ± 12.2 | 69.3 - 109.7 |
| Body mass index (kg/m$^2$) | 32.7 ± 3.8 | 28.4 - 40.0 |
| Percentage of body fat (%) | 44.3 ± 7.0 | 34.7 - 62.5 |
| Abdominal subcutaneous fat area (cm$^2$) | 383 ± 96 | 250 - 619 |
| Abdominal visceral fat area (cm$^2$) | 140 ± 57 | 84 - 266 |

SD: standard deviation

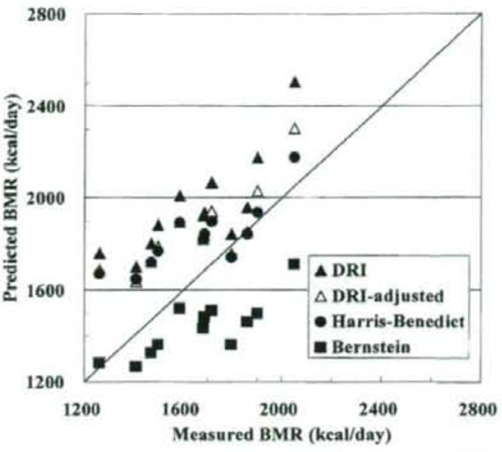

***Fig. 1.*** Relationships between measured and predicted basal metabolic rate (BMR) in obese males. DRI: Japan-Dietary Reference Intakes.

Following adjustment for sex and fat-free mass, BMR correlated significantly with ASF ($r = 0.806$, $p < 0.001$) and AVF ($r = 0.493$, $p < 0.05$). When sex, fat-free mass, and ASF were adjusted for, BMR was not correlated with AVF ($r = -0.022$, n.s.). The relationships between AVF and residual of BMR adjusted for sex, fat-free mass, and ASF is shown in ***Figure 3***. On the other hand, BMR correlated significantly with ASF when adjusted for sex, fat-free mass, and AVF ($r = 0.732$, $p < 0.001$); however, this correlation was no longer significant after additional adjustment for total fat mass ($r = 0.266$, n.s.).

***Table 3 Measured and predicted basal metabolic rate in obese people***

| | **Values(kcal/day)** | | **%difference(%)** | |
|---|---|---|---|---|
| | Mean± SD | Range | Mean± SD | Range |
| **Males** | | | | |
| **Measured** | 1659 ± 226 | 1262 - 2051 | | |
| **Predicted** | | | | |
| Japan-DRI 22) | 1963 ± 216 | 1701 - 2505 | 19.2 ± 9.8 | 2.7 - 39.6 |
| Japan-DRI with adjustment for body weight 22,30) | 1856 ± 178 | 1639 - 2304 | 12.8 ± 9.7 | -2.1 - 33.8 |
| Harris and Benedict 23) | 1831 ± 142 | 1648 - 2178 | 11.4 ± 10.0 | -2.9 - 32.5 |
| FAO/WHO/UNU (body weight) 24) | 1938 ± 116 | 1797 - 2230 | 18.2 ± 12.0 | 4.2 - 45.0 |
| FAO/WHO/UNU (body weight and height) 24) | 1961 ± 113 | 1822 - 2245 | 19.6 ± 12.3 | 5.4 - 46.9 |
| Henry 25) | 1885 ± 142 | 1712 - 2241 | 14.8 ± 10.7 | 0.7 - 38.8 |
| Owen 26,27) | 1811 ± 102 | 1686 - 2067 | 10.5 ± 11.4 | -2.7 - 35.9 |
| Mifflin 28) | 1744 ± 108 | 1601 - 1996 | 6.3 ± 10.3 | -6.4 - 28.2 |
| Bernstein 29) | 1436 ± 125 | 1267 - 1713 | -12.7 ± 7.4 | -24.0 - 1.7 |
| **Females** | | | | |
| **Measured** | 1477 ± 210 | 1192 - 1895 | | |
| **Predicted** | | | | |
| Japan-DRI 22) | 1709 ± 253 | 1435 - 2271 | 15.8 ± 7.2 | -1.8 - 24.5 |
| Japan-DRI with adjustment for body weight 22,30) | 1599 ± 210 | 1372 - 2064 | 8.6 ± 6.6 | -6.2 - 18.5 |
| Harris and Benedict 23) | 1496 ± 126 | 1367 - 1777 | 2.1 ± 8.0 | -7.5 - 15.7 |
| FAO/WHO/UNU (body weight) 24) | 1547 ± 106 | 1432 - 1783 | 5.8 ± 9.2 | -5.9 - 21.9 |
| FAO/WHO/UNU (body weight and height) 24) | 1543 ± 106 | 1430 - 1778 | 5.6 ± 9.2 | -6.2 - 21.6 |
| Henry 25) | 1496 ± 119 | 1367 - 1760 | 2.2 ± 8.2 | -7.1 - 16.7 |
| Owen 26,27) | 1388 ± 88 | 1293 - 1583 | -5.0 ± 8.6 | -16.5 - 9.9 |
| Mifflin 28) | 1396 ± 149 | 1240 - 1719 | -4.9 ± 7.0 | -15.0 - 5.9 |
| Bernstein 29) | 1370 ± 94 | 1273 - 1581 | -6.3 ± 8.1 | -16.6 - 7.7 |

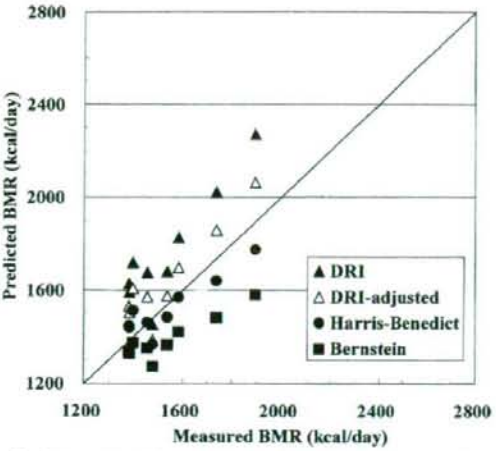

*Fig. 2.* Relationships between measured and predicted basal metabolic rate (BMR) in obese females. DRI: Japan-Dietary Reference Intakes.

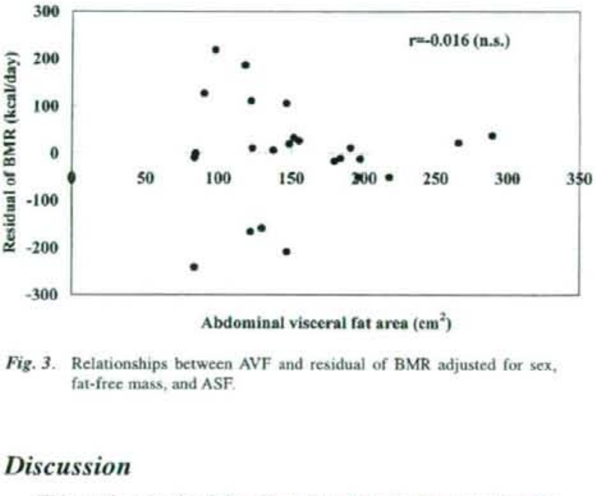

*Fig. 3.* Relationships between AVF and residual of BMR adjusted for sex, fat-free mass, and ASF.

## *Discussion*

This study examined the accuracy of predictive equations for BMR in obese Japanese people. The findings indicate that many of the equations, including the Japan-DRI and FAO/WHO/UNU equations, overestimate BMR, particularly in obese males. Similar results have been reported in many studies on Caucasians. However, several of the equations provided accurate estimates of BMR, mainly in obese females. Among the equations, the Mifflin equation in males, and the Harris-Benedict and Henry equations in females, provided a better accurate prediction of BMR.

The Japan-DRI equations are simple multiples of body weight (21.5 × body weight for males, 20.7 × body weight for females). The other equations include an intercept in addition to a term for body weight and in some cases terms for body height and age as well. Considering these differences, it is understandable that the Japan-DRI equations overestimate BMR in obese subjects of both sexes. To improve this, a term for adjustment of body weight was provided for each sex.[30] While this term reduced the overestimation of BMR, a large overestimation of BMR (12.8 ± 9.7 % for males and 8.6 ± 6.6 % for females) still remained. The suggested values for adjustment of body weight were obtained in rather lean Japanese subjects, with fat-free mass contributing considerably more to BMR than fat mass. Therefore, a low fat-free mass relative to body weight (indicated as a high percentage of body fat) may explain the overestimation, even after additional adjustment for body weight.[1] In contrast, Bernstein's equation, which was developed for obese Caucasians, underestimated BMR, especially in obese males.

The other equations incorporate an intercept, and some of them have terms for body height and age. Thus, it is expected that the terms adjust BMR for body composition to some degree, similar to BMI. However, the FAO/WHO/UNU equations that include terms for body weight and height provided comparable values to those with a term for body weight only. Considering the mean values of % difference, inclusion of terms for body height and/or age is not likely to improve the prediction of BMR in obese people, whereas the existence of an intercept or a curvilinear term would be expected to improve the predictive ability of the equation.

With the exception of the Japan-DRI equation, the predictive equations did not overestimate BMR to a large extent in obese females. This sex difference was not observed in previous studies. Female subjects in the present study had slightly higher BMI than male subjects, and their percentage body fat was also greater. While these differences should have been associated with overestimation of BMR considering the result of the present study, this can not explain the sex difference. Thus, the reason for the observed sex difference in the present study remains unclear.

The SD values of the % difference ranged from 7.4% to 12.3% in obese males and 6.6% to 9.2% in obese females. Previous studies reported that the interindividual coefficient of variation was about 8-13% in healthy people,[31,32] although interindividual variability of sleeping metabolic rate was less, at least for Japanese subjects.[9] The values calculated in the present study were within this range, but different from those of a previous study of obese subjects.[3] A possible reason for this difference may have been the uniformity of body composition, although the range of percentage body fat in the present study was large in both sexes, suggesting that this was unlikely to be the reason.

It remains controversial whether AVF is related to BMR.[10-17] Because AVF is related to sex, fat mass, and ASF, it is necessary to adjust for these variables in order to examine the relationship between AVF and BMR. In the present study, we adjusted not only for sex and fat-free mass but also for ASF to clarify the independent contribution of abdominal fat distribution. As a result, ASF but not AVF correlated independently with BMR after adjustment for sex and fat-free mass. However, this significant correlation disappeared after additional adjustment for fat mass, indicating that the independent correlation between ASF and BMR may actually reflect the relationship between fat mass and BMR. Adipose tissue has a small but definite contribution to BMR, while ASF and fat mass are correlated with each other, particularly in obese people, who have a large fat mass. If the relationship between ASF and BMR reflects the relationship between fat mass and BMR, this implies that abdominal fat distribution is not associated with BMR in obese Japanese people.

One of the limitations of the present study was the relatively small sample size. However, to obtain values of % difference when using predictive equations of BMR, the sample size used should provide relatively stable results. In the present study, normal-weight subjects were not included. To clarify the characteristics of obese subjects, this may be another problem. In addition, the analysis evaluating the contribution of abdominal fat distribution was performed in all subjects with adjustment for the effect of sex. As a consequence, comparison with the results of earlier studies could not be undertaken. Some of these previous studies reported the results for separate age categories, sex, and menopausal status.

In conclusion, the majority of the predictive equations overestimated BMR in obese Japanese males, whereas some equations were relatively accurate for obese females. In obese people, overestimated BMR may lead to overestimation of total energy expenditure. Caution is therefore needed when selecting predictive equations of BMR for obese Japanese people. Our results indicate abdominal fat distribution was not independently related to BMR.

## *Acknowledgments*

The authors thank the study subjects for their cooperation. We thank the other staff of the Saku Control Obesity Program, especially Hiroko Kogure for her help in data acquisition and analyses.

## *References*

1) Cunningham JJ. Body composition as a determinant of energy expenditure: a synthetic review and a proposed general predictive equation. Am J Clin Nutr 54:963-969, 1991.
2) Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. J Am Diet Assoc 105:775-789, 2005.
3) Foster GD, McGuckin BG. Estimating resting energy expenditure in obesity. Obes Res 9(Suppl.5):S367-S372, 2001.
4) Siervo M, Boschi V, Falconi C. Which REE prediction equation should we use in normal-weight, overweight and obese women? Clin Nutr 22:193-204, 2003.
5) Frankenfield DC, Rowe WA, Smith JS, et al. Validation of several established equations for resting metabolic rate in obese and nonobese people. J Am Diet Assoc 103:1152-1159, 2003.
6) Case KO, Brahler CJ, Heiss C. Resting energy expenditures in Asian women measured by indirect calorimetry are lower than expenditures calculated from prediction equations. J Am Diet Assoc 97:1288-1292, 1997.
7) Liu HY, Lu YF, Chen WJ. Predictive equations for basal metabolic rate in Chinese adults: a cross-validation study. J Am Diet Assoc 95:1403-1408, 1995.
8) Yamamura C, Kashiwazaki H. Factors affecting the post-absorptive resting metabolic rate of Japanese subjects: reanalysis based on published data. Jpn J Nutr Diet 60:75-83, 2002.
9) Ganpule AA, Tanaka S, Ishikawa-Takata K, et al. Interindividual variability in sleeping metabolic rate in Japanese subjects. Eur J Clin Nutr 61:1256-1261, 2007.
10) Leenen R, van der Kooy K, Deurenberg P, et al. Visceral fat accumulation in obese subjects: relation to energy expenditure and response to weight loss. Am J Physiol 263:E913-E919, 1992.
11) Busetto L, Perini P, Giantin V, et al. Relationship between energy expenditure and visceral fat accumulation in obese women submitted to adjustable silicone gastric banding (ASGB). Int J Obes Relat Metab Disord 19:227-233, 1995.
12) Armellini F, Zamboni M, Mino A, et al. Postabsorptive resting metabolic rate and thermic effect of food in relation to body composition and adipose tissue distribution. Metabolism 49:6-10, 2000.
13) Sharp TA, Bell ML, Grunwald GK, et al. Differences in resting metabolic rate between white and African-American young adults. Obes Res 10:726-732, 2002.
14) Okura T, Koda M, Ando F, et al. Relationships of resting energy expenditure with body fat distribution and abdominal fatness in Japanese population. J Physiol Anthropol Appl Human Sci 22:47-52, 2003.
15) Armellini F, Robbi R, Zamboni M, et al. Resting metabolic rate, body-fat distribution, and visceral fat in obese women. Am J Clin Nutr 56:981-987, 1992.
16) Nicklas BJ, Goldberg AP, Bunyard LB, Poehlman ET. Visceral adiposity is associated with increased lipid oxidation in obese, postmenopausal women. Am J Clin Nutr 62:918-922, 1995.
17) Macor C, Ruggeri A, Mazzonetto P, et al. Visceral adipose tissue impairs insulin secretion and insulin sensitivity but not energy expenditure in obesity. Metabolism 46:123-129, 1997.
18) Watanabe S, Morita A, Aiba N, et al. Study design of the Saku Control Obesity Program (SCOP). Anti-Aging Med 4:70-73, 2007.
19) Morita A, Ohmori Y, Suzuki N, et al. Anthropometric and clinical findings in obese Japanese: the Saku Control Obesity Program (SCOP). Anti-Aging Med 5:13-16, 2008.
20) Weir JB. New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol 109:1-9, 1949.
21) Yoshizumi T, Nakamura T, Yamane M, et al. Abdominal fat: standardized technique for measurement at CT. Radiology 211:283-286, 1999.
22) Ministry of Health, Labour and Welfare. Japan Dietary Reference Intakes for Japanese, 2005. Daiichi shuppan: Tokyo, 29, 2005. (in Japanese)
23) Harris JA, Benedict GF. A Biometric Study of Basal Metabolism in Man. Publication no. 297. Carnegie Institute, Washington DC, 1919.
24) FAO/WHO/UNU. Energy and protein requirements: report of a joint FAO/WHO/UNU expert consultation. WHO Tech Rep Ser 724:1-206, 1985.
25) Henry CJ. Basal metabolic rate studies in humans: measurement and development of new equations. Public Health Nutr 8:1133-1152, 2005.
26) Owen OE, Holup JL, D'Alessio DA, et al. A reappraisal of the caloric requirements of men. Am J Clin Nutr 46:875-885, 1987.
27) Owen OE, Kavle E, Owen RS, et al. A reappraisal of caloric requirements in healthy women. Am J Clin Nutr 44:1-19, 1986.
28) Mifflin MD, St Jeor ST, Hill LA, et al. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr 51:241-247, 1990.
29) Bernstein RS, Thornton JC, Yang MU, et al. Prediction of the resting metabolic rate in obese patients. Am J Clin Nutr 37:595-602, 1983.
30) Ministry of Health and Welfare, Japan. Recommended Dietary Allowances for the Japanese, revision in 1975. Ministry of Health and Welfare, Tokyo, 23-48, 1975. (in Japanese)
31) Shetty PS, Henry CJ, Black AE, et al. Energy requirements of adults: an update on basal metabolic rates (BMRs) and physical activity levels (PALs). Eur J Clin Nutr 50:S11-S23, 1996.
32) Muller MJ, Bosy-Westphal A, Klaus S, et al. World Health Organization equations have shortcomings for predicting resting energy expenditure in persons from a modern, affluent population: generation of a new reference standard from a retrospective analysis of a German database of resting energy expenditure. Am J Clin Nutr 80:1379-1390, 2004.

特集　新しい健康づくりのための運動基準・指針

# 生活習慣病予防のための身体活動・運動量

田中　茂穂

## 1. 「運動所要量」改定の必要性

日本では，「健康づくりのための運動所要量」が1989年に策定された[1]．その特徴と問題点を，表1にまとめた．身体活動・運動と生活習慣病や総死亡率に関する科学的研究は，この四半世紀に急速に発展し，冠状動脈疾患ばかりでなく，糖尿病などの生活習慣病罹患に対する身体活動・運動の予防効果が科学的に明らかにされている．そこで，今回の「健康づくりのための運動基準」では，これらの蓄積されたエビデンスを対象に系統的レビューを行ない，それをもとに，生活習慣病予防のために必要な身体活動・運動量を示すこととした．レビューを担当したのは，「厚生労働省運動所要量ワーキンググループ」のメンバーであった国立健康・栄養研究所の田畑泉，宮地元彦，髙田和子，筆者の4名である．

## 2. レビューの目的

生活習慣病の予防に有効な身体活動量の境界値を決定するために，身体活動と生活習慣病の発症に関する観察研究について系統的レビューを行なった．

今回は「生活習慣病の予防」を検討の対象としており，すでに生活習慣病を有する人のための「運動療法」とは異なる．また，日本においても，運動の効果やそのメカニズムに関する生理学的研究が数多く行なわれてきた．しかし，それらの多くが，たとえば血圧や血液生化学検査値といった生活習慣病のリスクファクターの変化を平均としてみており，長期間にわたる観察に基づいて疾病の発症そのものについて効果があるかどうかを検討した研究はきわめて少ない．

そこで，主な生活習慣病の発症，肥満の発現，および死亡をアウトカムとした．また，健常者あるいは一般の住民を対象とした観察研究について検索を行なった．研究の質という点からは，本来はRandomized Controlled Trial（RCT）が望ましいと考えられる．しかし，倫理的な問題から，このような内容の研究についてのRCTはきわめて少ない．そこで，因果関係を検討するためにより ふさわしい方法として，同一コホートを追跡した「観察研究」を扱うこととした．

## 3. 身体活動・運動の定量化の方法

身体活動量は，

$$\Sigma\ (\text{活動強度} \times \text{時間} \times \text{頻度})$$

と表すことができる．

エネルギー消費量（kcal）も身体活動量を表す単位である．しかし，エネルギー消費量は体格に

---

筆者：たなか　しげほ（独立行政法人　国立健康・栄養研究所）

0039-8985/06/¥250/JCLS

表1 これまでの運動所要量の特徴と問題点

**特徴**

- 50% $VO_2max$ 強度の運動と生活習慣病との関連から，有酸素運動の必要性を提唱している．
- 体力（最大酸素摂取量）と生活習慣病のリスクファクター（血液性状および血圧，体脂肪率）の異常値との関係から体力の基準値を求めた．
- 「生活習慣病のリスクファクターの異常値」→「目標の体力」→「そのために必要な運動量」という一連の流れがある．
- 一研究室の一貫した測定結果に基づいて作成された．

**課題**

- 「生活習慣病の発症」そのものではなく，「そのリスクファクターの異常値」から基準値を作成している．
- さまざまな運動・身体活動に関する知見が得られつつあるが，それに対応していない．
- 横断的なデータ解析に基づいて基準値が得られているが，本来は，縦断的な観察が必要である．
- 年齢階級による最大酸素摂取量の変化が，その他の報告と比較して小さい．

表2 検索方法

**対象としたデータベース**：PubMed と医学中央雑誌

**対象とした期間**：2005 年 4 月 11 日まで

**検索式**：PubMed では，("physical activity" OR exercise OR "physical training" OR fitness) AND（疾病毎に選択）AND（follow* OR observation* OR prospective OR longitudinal OR retrospective）

**検索制限**：human（人を対象とした研究）

**対象とした報告**：原著論文

**年齢**：学童期（6 歳以上）から高齢期

**対象とした生活習慣病等**：肥満，高血圧症，高脂血症，糖尿病，脳血管疾患，循環器病による死亡，骨粗鬆症，ADL，総死亡

**採択基準**：

原則として重度の疾病を有していない者（健康，または軽度の症状で運動が可能な者）を長期（原則 2 年以上）観察し，死亡率や発症率を身体活動・運動量別に分析した研究．

定量的方法で評価された身体活動・運動量に関する情報（種類・強度，時間：分 / 週または分 / 日，頻度：回 / 週）を明示した研究．この情報がない場合，「種類・強度と分 / 週」の情報から計算．

身体活動・運動量の群分けや区分けの方法，カットオフラインの設定が論理的な研究．

身体活動・運動単独の効果を分析［身体活動・運動以外の要因（性・年齢・喫煙・代謝性危険因子…）を統計的に補正］した研究．

対象者の人数が十分かどうかは，分析法や測定精度等から判断．

⬇

身体活動量の群間差が得られた最低値から，生活習慣病の予防に有効な身体活動量の境界値を決定する．

かなり依存し，たとえばアメリカ人と日本人が同様の活動を行なった場合でも，エネルギー消費量に大きな差が生じる．そこで，体格の違いを考慮することができ，また，今回レビューした文献でも多く用いられていた

METs・時/週＝1週間当たりのΣ METs×時間

でまとめていくこととした．

たとえば，速歩（4METs）を30分/日×3日/週とジョギング（7METs）を30/日×2日/週実施した場合，

4×30/60×3+7×30/60×2＝13METs・時/週

と計算できる．

## 4. 検索方法

検索方法の詳細は，表2の通りである．それぞれの文献について，もっとも身体活動・運動量の少ない群に比べて，生活習慣病の発症等が有意に低下する群の下限値を抽出した．たとえば，身体活動量により5群に分け，もっとも身体活動量の

少ない群と比べて，少ない方から3番目の群ではじめて有意差がみられた場合は，その群における身体活動量の下限値を用いて，生活習慣病の予防に有効な身体活動量の境界値を決定することとした．なお，身体活動量の境界値を検討する上で明らかに不適切な群分けをしていると考えられる文献は削除した．

## 5. 選択された報告における身体活動・運動の概観

検索式でヒットした件数は，体力と生活習慣病の発症・死亡に関する文献とあわせて8,134本であった．さらに，タイトルと抄録による一次スクリーニングにより794本に絞った．これらの全文を取り寄せ精読したところ，上記の採択基準に該当する文献数は，身体活動・運動に関して36本であった（否定的な結果の得られた文献，および高齢者に関する文献等も含む）．性や年齢，あるいは疾病によって区別するには文献数が少なかったため，これらをまとめて検討することとした．

## 6.「運動」か？「身体活動」か？

身体活動や運動については，1984年に行なわれたアメリカ疾病予防センター（CDC）のワークショップで採用された以下のような定義[2]が，広く受け入れられている．

- **運動**（Exercise）

一つ以上の体力要素を維持あるいは改善するために行なわれる，計画的・組織的・継続的な身体の動作．

- **身体活動**（physical activity）

骨格筋の活動により安静時よりも多くのエネルギー消費を伴う身体の状態．

ロンドンの2階建てバスの車掌と運転手を比較したモリスや，ハーバード大学の卒業生を対象として縦断的に観察したパッフェンバーガーらの研究に代表されるように，必ずしも運動に限定せず身体活動量をとらえようとした調査は古くから存在した[3]．しかし，ジョギングやウォーキング等の有酸素運動の効果やメカニズムが明らかになるとともに，特に1980年代は，身体活動の中でも有酸素運動に焦点が当てられるようになっていた．

しかし，1990年代に入ったあたりから，必ずしも運動ではなく日常的な活動でも，エネルギー消費量を増加させれば生活習慣の予防や改善などに効果があることを重要視するようになった[3~5]．それは，パッフェンバーガーらを含む疫学的な研究の裏づけがあると同時に，しっかりした有酸素運動を定期的に行なうように訴えても，実現の可能性が低いということがその背景にある．そこで，国際的には運動のみならず，より広く身体活動をとらえる方向に変わってきている．なお，体力増進を目的とした運動のガイドラインも別途存在する[6]．

身体活動には，運動やスポーツの他，労働や家事，余暇活動など（運動以外を「生活活動」と呼ぶこととした），日常生活におけるすべての活動が含まれる（図1）．しかし，実際には，運動の他，ガーデニングのような余暇活動や，掃除などの家事，力仕事，通勤等による歩行などが調査対象となっている場合が多い．これらの活動は，およそ3～6METsの中強度（moderate）あるいは6METsを越える高強度のいずれかに相当する[4]．厳密なものではないが，今回のレビューにおいても，多くの場合，およそ3METs以上の身体活動・運動が対象となっていると考えられた．なお，多くの文献で用いられたそれぞれの質問紙は，一回当たりの最低持続時間や頻度について限定していなかったため，今回の基準で規定する根拠はなかった．

今回，レビューの結果をまとめていく上で大きな問題となったのは，調査対象としている身体活動の範囲が研究によって大きく異なることであった．実際のところ，身体活動の分類法は質問紙の数だけあるといってもよいが，大別すると，以下のようになる．

①運動のみ

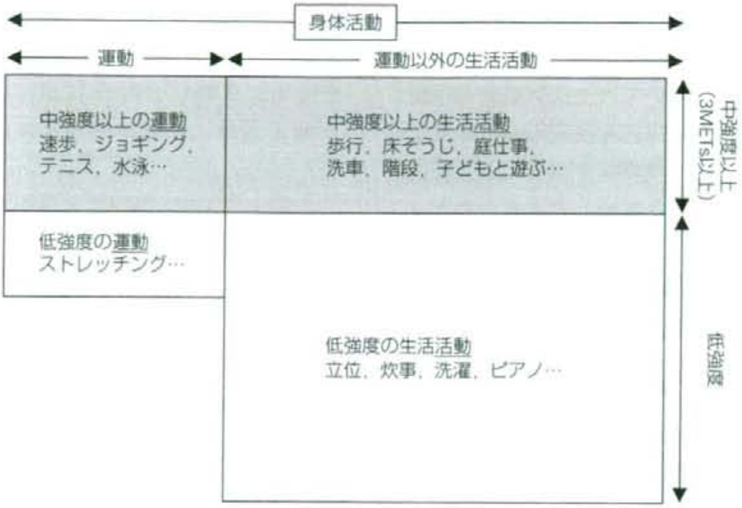

図1 身体活動と運動

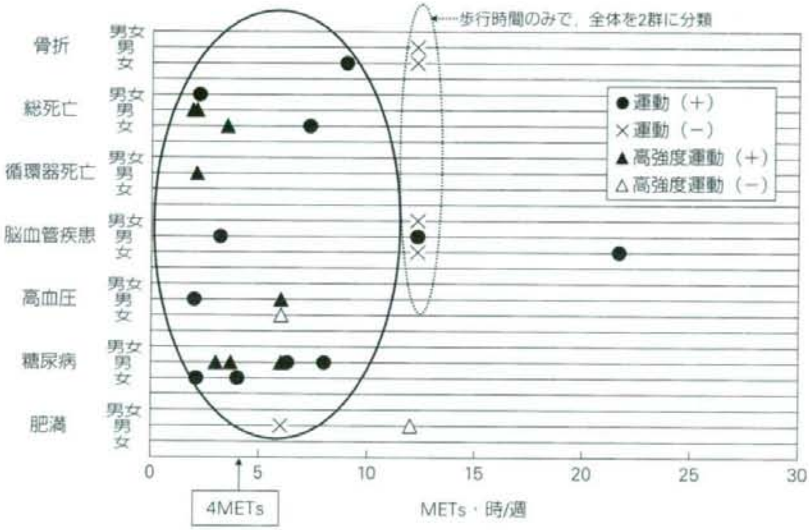

図2 “運動”の境界値（METs・時/週）

②運動＋一部の余暇活動（多くの場合，ガーデニングのみ）

③運動＋余暇活動（含；ガーデニング）＋移動（日常的な歩行や階段）

④あらゆる身体活動（立位，炊事，洗濯なども含む）

このうち，④については，1日だけで30METs・時を越える．しかし，そのような調査に基づく報告は少なかったため，除外した．

①に該当する値を図2に，②と③に該当する値を図3に示した．各研究において，有意差のみられた身体活動量のもっとも少ない群の下限値

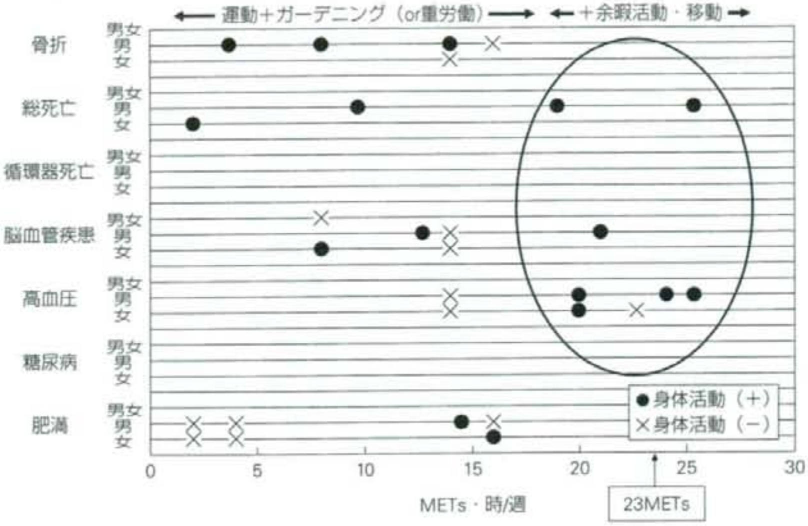

図3　"身体活動"の境界値（METs・時/週）

を＋，みられなかった場合は，身体活動量のもっとも大きな群の下限値の値を－として表示してある．＋が多く，－がほとんど存在しない値の範囲を基準値と設定することとなる．また，図2では，ジョギング程度かそれ以上の比較的高強度の運動に限定したもの（「高強度運動」と表示）と，速歩程度の中強度以上の運動をすべてまとめたもの（「運動」と表示）に分類した．

表3　身体活動・運動量の基準値（対象：20～69歳）

| 身体活動 | 運動 |
|---|---|
| 23METs・時/週<br>≒3.3METs・時/日<br>毎日約60分程度の中強度活動（ふつう歩行，床そうじ，庭仕事等）<br>歩行中心の活動であれば，1日当たりおよそ8,000～10,000歩に相当 | 4METs・時/週<br>（範囲：2～10METs・時/週）<br>例：<br>・速歩：約60分/週<br>・ジョギング：約35分/週<br>・テニス：約35分/週 |

## 7．「運動量」の基準値

図2において約12METs・時/週に相当するいくつかのプロットは，歩行時間で全体を2つの群に分けて（境界値は1日30分），群間の差を検定した研究から得られたものである．人数が少ない結果が含まれていることもあり，有意差の得られていないケースがいくつかみられる．しかし，それらを除くと，ほとんどの報告で有意な境界値が得られた．それらのほとんどは2～10METs・時/週に分布しており，平均をとると4METs・時/週であった．また，高強度運動のみの場合と中強度を含む場合とでそれらの値の平均を比較すると，差は1に満たなかった．そこで，特に運動の強度を区別することなく，約2～10METs・時/週に分布する値から，基準値を決定することとした．その結果，基準値とその範囲をそれぞれ4METs・時/週，2METs・時/週～10METs・時/週とした（表3）．

現在の運動量に応じて，基準値，あるいは基準値の範囲の値を上回ることを目指すようにする．すなわち，運動習慣がまったくない人は2METs・時/週に，運動量が基準値以下の人は基準値を目指して，さらに基準値よりも運動量が多い人は10METs・時/週を目指すようにする．その結果，生活習慣病の発症リスクが低くなることが期待される．

具体的な運動の例としては，速歩，体操（動き

のあるもの），ジョギング，ランニング，水泳，球技などが，3METs以上の運動に含まれる．たとえば，速歩は約4METs（分速90～100m）の強度である．したがって，4METs・時/週を速歩で換算した場合は，約60分/週に相当する．同様に，ジョギングやテニス（約7METs）の場合は，約35分/週に相当する．先に述べた理由により，頻度や持続時間は問題としない．

## 8．「身体活動量」の基準値

「身体活動」においては，基準値を23METs・時/週とした（表3）．

図3において，図の左側に存在する点のほとんどは，先に述べた運動の他，限定した屋外活動あるいは余暇活動（多くの場合，ガーデニングのみ）の実施状況もたずねた質問紙から得られた結果である．それらの結果は，得られた活動量の境界値のバラツキが大きく，群間の有意差が得られたかどうかもまちまちである．

それに対して右側に位置する点は，スポーツはもちろん，屋外での歩行（健康増進のための速歩に限らず，日常生活における歩行を含む）や階段の利用，その他の中・高強度活動を対象としている．それらの結果は，約19METs・時/週から約26METs・時/週の間に分布しており，ほとんどが有意な結果となっている．そこで，これらの値から，身体活動量の基準値を決定することとした．ただし，この値に相当する週当たりの身体活動時間は，3METsの強度（普通歩行）で1日当たり54～74分の幅がある．しかし，国民にとって，3METs以上に該当する活動時間の20分の違いを十分に区別できるものではない．そこで，身体活動量の基準値は，よりわかりやすいように1つの値，すなわち系統的レビューで抽出された論文の値の平均値を基準とした．

強度が3METs以上の身体活動としては，日常的な歩行（買い物，通勤など），床そうじ，庭仕事，物を運ぶ，子どもと遊ぶといった活動があげられる．日常的な歩行をはじめとするこれらの活動の強度は3METs程度であるので，23METs・時/週（≒3.3METs・時/日）は，3METs以上の強度の身体活動で1日当たり約60分に相当する．ここでの身体活動は，必ずしも歩行を伴うとは限らないが，一般に3METs以上の強度の身体活動の多くは歩行を伴っている．そこで，歩行中心の活動で構成されている場合を考えると，1日当たり約60分（10分当たり1,000歩とすると，約6,000歩に相当）に相当する．日常生活の中では，屋内での歩行など，低強度で意識されない歩数が2,000～4,000歩程度みられるので[7]，1日当たりの歩数の合計としては，およそ8,000～10,000歩に相当すると考えられる．

## 9．国際的な身体活動ガイドラインとの比較

国際的な身体活動ガイドラインは，特に体重増加の予防を目的としたものが多い[5,8]．たとえば，国際肥満学会は，大規模観察研究における質問紙調査の結果[9]や，二重標識水法を用いた減量後女性における体重増加と身体活動レベル（PAL）との関係[10,11]などから，「およそ1.7以上のPALが必要」とし，そこから「毎日45～60分の中強度活動」という結論を導き出している[12]．ただし，日本人の場合は，半数以上がこの値をすでに上回っている[13]．欧米と日本で，生活環境や遺伝的な背景が多少なりとも異なることから，「必要な身体活動量」には民族差がある可能性も否定できない．今後，日本人を対象として，できれば客観的な方法に基づいた観察研究が待たれる．

## 10．問題点および今後の課題

### 1）境界値の決定法

今回は，有意差の得られた最低の境界値から基準値を決定し，群間における発症率の差は考慮していない．この方法では，境界値は，対象者の人数や分け方等の影響を受ける．ただし，今回抽出された研究間で，相対危険度に大きな違いはみられなかった．

### 2）身体活動の評価法

質問紙によって，扱っている身体活動・運動の内容や量的な換算法に差がみられる．また，こうした質問紙による評価法は，活動内容を区別する上では有用であるが，被験者の主観に左右され，必ずしも十分な妥当性があるわけではない[14]．加速度計法・歩数計法等の，より客観的な方法を用いる必要がある[15]．

### 3）対象特性や疾病の区別

今回，性や年齢階級，疾病別に基準値を決定するほどの根拠がなかったため，これらをまとめて検討した．また，基準値を決定する際に直接利用された日本人の研究は2件のみ[16,17]であった．

### 4）身体活動量の上限値

今回は，身体活動の上限値を決定する根拠は見当たらなかった．ただし，上限値の存在を示唆する報告もあり，「運動のし過ぎ」に関する検討も必要である．

### ［文　献］

1）進藤宗洋ほか：日本の厚生省の"健康づくりのための運動所要量"，pp202—208．日本臨牀増刊号「身体活動と生活習慣病」，日本臨牀社，2000

2）Caspersen CJ, et al.: Physical activity, exercise, and physical fitness : definitions and distinctions for health-related research. Public Health Rep, 100 : 126—131, 1985

3）Blair SN, et al.: The evolution of physical activity recommendations : how much is enough? Am J Clin Nutr, 79 : 913S—920S, 2004

4）Pate RR, et al.: Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. JAMA, 273 : 402—407, 1995

5）Brooks GA, et al.: Chronicle of the Institute of Medicine physical activity recommendation : how a physical activity recommendation came to be among dietary recommendations. Am J Clin Nutr, 79 : 921S—930S, 2004

6）American College of Sports Medicine Position Stand : The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. Med Sci Sports Exerc, 30 : 975—991, 1998

7）波多野義郎：ウォーキングと歩数の科学．不昧堂出版，1998

8）FAO/WHO/UNU. Human Energy Requirements : Report of a Joint FAO/WHO/UNU Expert Consultation, Rome, 17-24 October 2001, FAO Food and Nutrition Technical Report Series 1, 2004

9）Fogelholm M, et al.: Does physical activity prevent weight gain-a systematic review. Obes Rev, 1 : 95—111, 2000

10）Schoeller DA, et al.: How much physical activity is needed to minimize weight gain in previously obese women? Am J Clin Nutr, 66 : 551—556, 1997

11）Weinsier RL, et al.: Free-living activity energy expenditure in women successful and unsuccessful at maintaining a normal body weight. Am J Clin Nutr, 75 : 499—504, 2002

12）Saris WH, et al.: How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. Obes Rev, 4 : 101—114, 2003

13）第一出版編集部編：厚生労働省策定　日本人の食事摂取基準（2005年版），第一出版，2005

14）山村千晶ほか：身体活動量に関する質問票の妥当性について．栄養誌，60：265—276, 2002

15）Wareham NJ, et al.: Physical activity and obesity prevention : a review of the current evidence. Proc Nutr Soc, 64 : 229—247, 2005

16）Hayashi T, et al.: Walking to work and the risk for hypertension in men : the Osaka Health Survey. Ann Intern Med, 131 : 21—26, 1999

17）Okada K, et al.: Leisure-time physical activity at weekends and the risk of Type 2 diabetes mellitus in Japanese men : the Osaka Health Survey. Diabet Med, 17 : 53—58, 2000

体力科学（2006）**55**, 527～532

**【教育講座】**

# 間接熱量測定法による１日のエネルギー消費量の評価

田中茂穂

# EVALUATION OF TOTAL ENERGY EXPENDITURE USING INDIRECT CALORIMETRY

SHIGEHO TANAKA

現在，エネルギー消費量を推定するのに，いくつかの方法が利用されている．しかし，特に「１日当たりのエネルギー消費量」（total energy expenditure：TEE）については，必ずしもそれらの精確度や特徴を十分に吟味して使用されているわけではないようである．そこで，本稿では，間接法に基づく TEE の測定・推定法について述べる．

## I．直接法と間接法

### A．直接法

消費されたエネルギーは熱となって放散されるため，その熱量を直接測ればよい．代表的な直接法の測定機器である Atwater-Rosa-Benedict human calorimeter の場合，測定室内の被験者が放射する熱を，室内に張りめぐらされた管を流れる水の温度から測定する．また，室内で発生した水蒸気量から呼気等の水蒸気の気化熱を測定するとともに，体温の変化も考慮して，エネルギー消費量を測定する．

このように，装置が大がかりで，活動内容も限定されるため，最近はほとんど使用されていない．

### B．間接法

エネルギーを生み出す際，食物からとりこんだ栄養素が酸素と反応（消費）し，二酸化炭素を産生する．これらの化学式に基づいて，酸素摂取量と二酸化炭素産生量，および尿中窒素量が正確に得られれば，多くの場合１％程度かそれ以下の誤差で，エネルギー消費量が推定できる[1]．例えば，最もよく利用される Weir[2]の式は，以下の通りである．

EE（kcal）＝3.941×酸素摂取量＋1.106×二酸化炭素産生量－2.17×尿中窒素排泄量

また，三大栄養素のうち，摂取エネルギーに占めるたんぱく質の割合は比較的安定している．そこで，たんぱく質の占める割合を12.5％と仮定すると，先の Weir の式は以下のようになる．

EE（kcal）＝3.9×酸素摂取量＋1.1×二酸化炭素産生量

たんぱく質の占める割合が20％を大きく越えるような極端に偏った食事であったり，激しい運動中に限定したりしなければ，尿中窒素排泄量を考慮しないことによる誤差の影響は１％未満であり，呼気分析だけでも十分に正確に測定することができる．

間接法は，直接法と比べて簡便に実施できる上に，ズレの小さい仮定に基づいており，直接法による測定と非常によく一致する．しかも，エネルギー基質の評価が可能である．したがって，しっかりとした呼気分析が行われるのであれば，非常に正確かつ有用な方法である．

## II．TEE の内訳

TEE の構成要素の標準的な割合やその変動について，表１にまとめた．値は，体重 60 kg 程度の，標準的な日本人の体格をもとに試算したものである．

基礎代謝量（basal metabolic rate：BMR）は，一般に TEE の中で，最も大きな構成成分である．標準

---

(独)国立健康・栄養研究所　健康増進プログラム
(エネルギー代謝プロジェクトリーダー)
〒162-8636 東京都新宿区戸山1-23-1

*National Institute of Health and Nutrition, Health Promotion and Exercise Program, Project for Energy Metabolism (Project Leader)*

表1．　総エネルギー消費量の内訳とバラツキ

| 成分 | 割合（%） | 個人差（kcal/日） | 備　考 |
|---|---|---|---|
| **基礎代謝量** | **60** | **±100** | **割合は大きいが、体格でおおよそ決定** |
| **食事誘発性体熱産生** | **10** | **± 50** | **割合も変動幅も小さいが、相対的な測定誤差が大きい** |
| **運動** | **0~10** | **± 50~100** | **日本人で週2日以上の運動を実施している者は30%弱** |
| **運動以外の身体活動** | **20~30** | **±200~300** | **PALの大きな個人差(1.4~2.2)を生じる主な原因** |

数値は，標準的な体格の日本人（スポーツ選手等は除く）における，おおよその推定値．個人差は，標準偏差あるいは推定の標準誤差からの概算

的な日本人における身体活動レベル（physical activity level：PAL＝総エネルギー消費量÷BMR）は1.75程度と考えられている[3]ので，逆算すると，BMR は TEE の約60％弱（＝1÷1.75）となる．しかし，性・年齢や体重などから BMR を推定する場合，国際的に用いられている推定式でも[4]，日本の基礎代謝基準値でも[5]，推定誤差はおよそ±8～13％程度である．除脂肪量あるいは各臓器重量から推定できる場合は，更に推定誤差が小さくなる[6]．したがって，BMR は，TEE に占める割合や体格による変動幅は大きいものの，体格が決まれば，個人差はそれほど大きいものではないと考えられる．

食事誘発性体熱産生については，これまでの知見によると，TEE の約10％程度と，絶対量が小さい[7]．そのため，個人間の変動係数は約20％程度と大きな値が報告されているものの[8]，TEE の中では，相対的に小さな変動にしかならないと考えられる．

運動については，どこまでを運動ととらえるかによって，値が大きく異なる．国民健康・栄養調査[9]によると，運動の定義が示されていないものの，1回30分以上，週2日以上，1年以上の運動を続けている人の割合が国民の30％に満たない．すなわち，70％以上の人は，運動習慣を有していないことになる．一方，週2日以上運動している人でも，運動していない日が週に数日あるはずである．また，運動している日について考えると，例えば速歩の場合，安静時からの付加エネルギーは，30分間で 100 kcal 弱，1時間で 200 kcal 弱である．したがって，TEE に占める運動の割合は，運動している日でさえ10％以下，多くの場合は5％以下であると考えられる．

平均60％程度の BMR と約10％の食事誘発性体熱産生，および多くの場合0～10％程度と考えられる運動を除いた残り20～30％は，運動以外の身体活動（nonexercise activity thermogenesis：NEAT）である[10]．NEAT は，姿勢の保持（座位や立位を含む）や，掃除・洗濯を含む家事，買い物・通勤などにおける歩行，庭仕事などの余暇活動，仕事中における荷物の運搬など，低～中強度を中心に様々な活動が含まれる．しかし，運動をしていない人を中心とした一般の集団でも，PAL の標準偏差は0.2を越える（～0.3）．この値は，食事誘発性体熱産生や運動の個人間変動，および測定誤差の影響も受けるが，先に述べた点を考えると，主に NEAT によるものであると考えられる．標準的な体格（男女平均して BMR≒1400 kcal/日）であれば，±200～300 kcal に及ぶ．

したがって，TEE を正確に推定するためには，BMR の測定・推定に加え，NEAT をいかに評価するかが，非常に大きな問題となる．

## Ⅲ．1日のエネルギー消費量の測定法

短時間の場合，マスクを装着し，ダグラスバッグまたは携帯式の代謝測定装置を使用して測定できる．しかし，食事中を含むいくつかの活動の測定は不可能である．また，不快感や見た目の違和感などから，外出や一部の日常活動が制限される．したがって，長時間連続して測定することは，現実的には

不可能である．

１日あるいはそれ以上の長時間にわたるエネルギー消費量を推定するには，以下のような方法がある．

1）エネルギー代謝測定室
2）二重標識水法
3）心拍数法
4）加速度計法
5）生活活動記録に基づく要因加算法

「エネルギー代謝測定室」（ヒューマンカロリメーターあるいはメタボリックチャンバー）とは，人が数時間～数日間生活できる部屋（机やベッド，トイレなど）（図１）と，ガス濃度や流量等の測定機器を備えた設備である11)．被験者は，滞在中に酸素を消費し二酸化炭素を排出するが，それによる室内の濃度変化からエネルギー消費量を測定する間接法によるものがほとんどである．測定機器を含む設備全体が十分に管理されれば，既存の設備の中では，数時間に及ぶエネルギー消費量を，最も正確に測定することができる．例えば，国立健康・栄養研究所のエネルギー代謝測定室の場合，６時間のアルコール燃焼試験の結果は，エネルギー消費量の真値に対して−0.2±0.5%である．

ただし，生活の場所が室内に限定されるため，個人の生活実態を反映した日常の TEE とは異なる．したがって，実験的に再現したある条件下（活動内容，食事，その他の室内環境など）でのエネルギー消費量を測定したり，他の方法の妥当性の検討に利用される．

二重標識水（doubly labeled water : DLW）法は，$^{2}$H と $^{18}$O を摂取し，１週間～２週間程度の期間にわたる二酸化炭素産生量の推定値と food quotient から，平均のエネルギー消費量を推定する11,12)．したがって，この方法も，いわゆる「間接法」の一種である．

DLW 法は，測定機器を携帯する必要がなく，測定期間中の制限がほとんどない．しかし，二重標識水が非常に高価で，しかも入手しにくいこと，安定同位体の濃度を正確に測定するのは容易ではないことから，ごく限られた研究グループがこの方法を用いた研究を実施しているに過ぎない．

また，サンプルの分析誤差に加え，呼吸商や水分代謝について，いくつかの仮定をおいてエネルギー消費量を推定している11,12)．したがって，日常生活における TEE を測定する方法として最も信頼のできる値が得られるとされてはいるものの，TEE の推定精度は，エネルギー代謝測定室を基準とした場合，確度・精度ともに±５％程度は覚悟する必要がある（表２）．

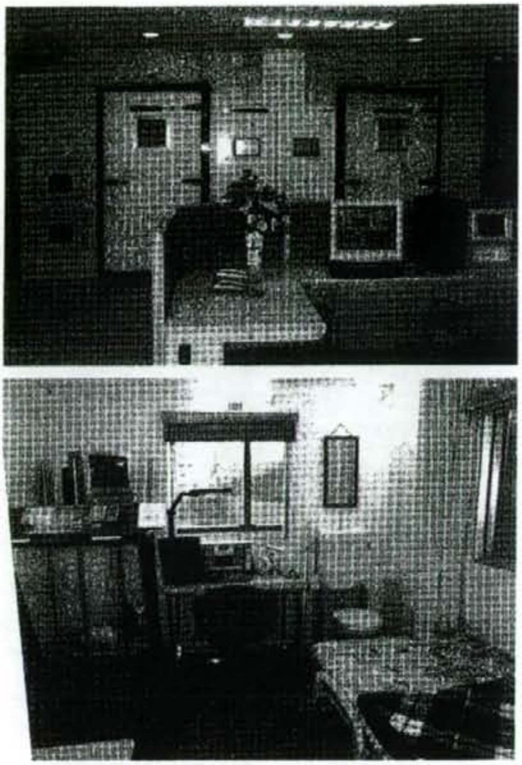

図１．　エネルギー代謝測定室の外観と室内

これら２つの方法は，酸素摂取量と二酸化炭素産生量の両方またはいずれかを測定しているので間接法であるが，3)～5) は，酸素摂取量を“推定”していることから，いわば「間接法の間接法」と言える．

## Ⅳ．運動以外の身体活動の重要性

このように，TEE を測定・推定する方法はいくつか存在する．しかし，エネルギー代謝測定室や二重標識水法を妥当基準とすると，簡便な方法は，一般に非常に大きな誤差を有する13)．日本人を対象とした結果をまとめたのが，表３である．

米国/カナダの食事摂取基準14)においては，二重