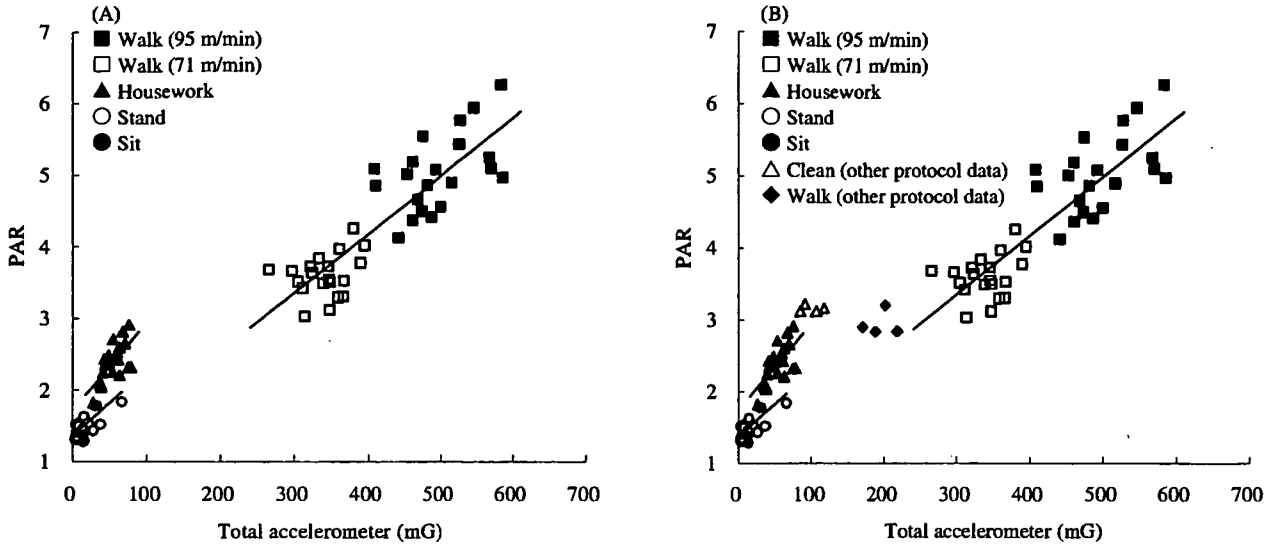

Figure 1: Relationship between total acceleration and PAR. Original data (A) with additional protocol data (B).

Therefore, the classification of daily lifestyle PA in our study could be a significant contribution to weight management, especially in the area of clinical practice.

Additionally, we found a high validation of predicting EE in low-intensity PA. Our results indicate that EE measured by chamber was closely correlated with EE estimated using the three equations and one constant value (percentage difference, 4.4%; correlation coefficient, 0.94; SEE, 61 kcal/10.5 hours). Although a previous study that estimated daily EE using triaxial accelerometry was limited, the percentage difference between EE measured by chamber and EE estimated by the developed non-linear model using Tritrac (triaxial accelerometer) was small (16). Moreover, Plasqui et al. (17) observed the relationship between total EE measured by the doubly labeled water technique for 15

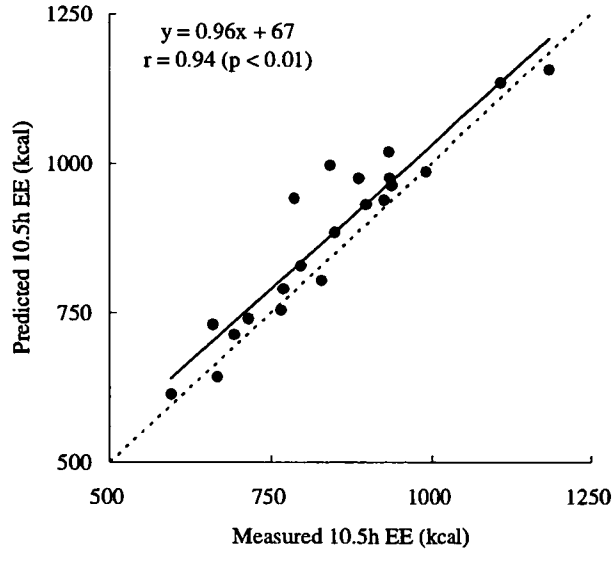

Figure 2: Relationship between measured and predicted 10.5-hour EE.

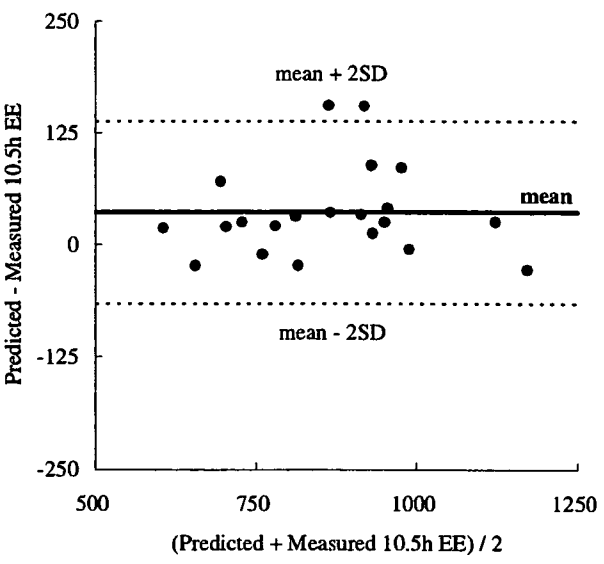

Figure 3: Bland and Altman analysis. The differences between measured and predicted 10.5-hour EE are plotted against the measured and predicted mean 10.5-hour EE.

consecutive days and the predicted EE using the equation of counts for Tracmor (triaxial accelerometer), age, weight, and height as parameters. These authors indicated that the correlation coefficient was 0.90, and SEE was 167 kcal/d between measured and predicted EE. Our study presents a novel method to objectively evaluate EE of low-intensity PA under close-to-normal living conditions using triaxial accelerometry that compares favorably with the previous study.

We believe that our highly accurate prediction of EE for low-intensity PA is due to the method used to develop each equation for standing, housework, and walking. A previous study reported that an equation based on the acceleration of walking underestimated EE of moderate-intensity lifestyle activities (7). Recently, Crouter et al. (18) found that the estimation of EE both in walking and lifestyle activity could be improved by the two regression lines. In the present study, if only one equation was developed from the relationship between total acceleration and PAR of all plots, including sitting, standing, housework, and walking [EE kcal = 0.0068 × acceleration count (mG) + 1.5509], the predicted EE of residual time (10.5 hours) would be overestimated (931 ± 155 kcal/10.5 hours, $p < 0.01$, 10.3 ± 5.2% difference). One possible explanation for this overestimation is that EE of static body posture such as sitting and standing may be overestimated by all plots included in the equation. Thus, the developed equations for each daily lifestyle PA are a novel method for predicting EE.

A previous study compared the ability to predict EE using uniaxial and triaxial accelerometry (7,19). The results indicated that triaxial accelerometry had higher accuracy of estimating EE than uniaxial accelerometry. However, as Plasqui et al. (17) pointed out, because two devices from different manufacturers were used, no conclusions can be drawn regarding the possible benefits of triaxial vs. uniaxial accelerometry. When Plasqui et al. (17) initially observed the contributions of vertical and horizontal acceleration to total EE per day adjusted for weight, height, and age, vertical acceleration explained an additional 16% of the variation in total EE. Furthermore, because horizontal acceleration contributed another 5%, it was concluded that triaxial accelerometers are more suitable than uniaxial accelerometers for estimating daily life activities. Similarly, the present study also compared the ability to quantitate low-intensity PA using either triaxial acceleration or only vertical acceleration from a triaxial accelerometer. Our results demonstrate that EE equations developed using only vertical acceleration overestimated EE by 135 kcal/10.5 hours. Further analysis of our data shows that there is no difference in EE for sitting and standing between equations using triaxial acceleration and only vertical acceleration (triaxial, 681 kcal/10.5 hours vs. uniaxial, 672 kcal/10.5 hours, $p = 0.06$), whereas the equation using only vertical acceleration overestimated the EE of housework periods by 109 kcal/10.5 hours (triaxial, 195 kcal/10.5 hours vs. uniaxial, 304 kcal/10.5 hours, $p < 0.01$). Therefore, we conclude that a triaxial accelerometer has a higher ability to predict EE of low-intensity PA, especially when the activity includes a large variation in horizontal acceleration, such as housework. Additionally, the technique of using not only total acceleration but also the vertical-to-horizontal acceleration ratio can be emphasized as a merit of the three-dimensional accelerometer.

There are some limitations of this study. The first limitation concerns the validity of the equations developed by comparing the EE measured by IHC with the EE estimated using developed equations for the residual time (i.e., 630 minutes = 10.5 hours). It is noted that this approach tends to overestimate the validity of the methods developed. We need to test the prediction equations of the present study in free-living conditions using the doubly labeled water method. The second limitation was that total acceleration data from 100 to 250 mG were blank during the chamber stay, although the relationship between PAR and total acceleration allowed for the development of EE equations for each activity. However, the plots describing the relationship between PAR and total acceleration for housework and walking in the supplemental experiment were likely to be an extension of the regression line, explaining this relationship in both activities in the present study. The results indicate that either of the equations for housework and walking can be applied to the range of 100 to 250 mG for total acceleration. Another limitation is that we did not develop an equation for cycling, which is a very popular lifestyle PA. Future studies should apply to all types of lifestyle activities. Lastly, the reason for the slight overestimation of the EE/10.5 hours in the present study should be clarified.

In conclusion, we identified low-intensity PA with high accuracy using total acceleration and the vertical-to-horizontal acceleration ratio obtained from a triaxial accelerometer. Notably, the use of the vertical-to-horizontal acceleration ratio is a novel method. Due to the classification of low-intensity PA, it is possible to accurately predict EE using equations for each activity. We demonstrated that triaxial accelerometry, when the total, vertical, and horizontal accelerations are utilized, can effectively evaluate different types of activities and estimate EE for low-intensity physical activities associated with modern lifestyles. In combination with measured or a highly accurately predicted sleeping metabolic rate (20), EE in sedentary lifestyle can be obtained.

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Relationship between Blood Adipocytokines and Resting Energy Expenditure in Young and Elderly Women

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Summary It has been demonstrated in a previous study that resting energy expenditure (REE) is associated with adiponectin levels in the blood. However, body composition was not taken into consideration in that study. The purpose of the present study was to again investigate the relationship between blood adipocytokines and REE, adjusted by body composition, in both young and elderly women. REE and blood adipocytokines were measured in 115 young (age: 22.3±2.1 y, BMI: 21.3±1.9 kg/m^2) and 71 elderly (63.4±6.5 y, 22.9±2.3 kg/m^2) women. Dual energy X-ray absorptiometry was used to measure percent body fat. Fat mass and fat free mass (FFM) were calculated. REE (kcal/d and kcal/kg BW/d) was lower in elderly women than in young women, but no significant difference was observed in REE, expressed as kcal/kg FFM/d, between the two groups. Although elderly women had a higher percent body fat and higher serum leptin concentrations than young women, plasma adiponectin concentrations did not differ between young and elderly women. In elderly women, REE (kcal/d) was significantly and inversely correlated with plasma adiponectin concentration ($r=-0.386$, $p<0.001$), but REE expressed per kilogram of BW or FFM was not significantly correlated. Furthermore, no significant correlation was observed between REE (kcal/d) and concentrations of plasma adiponectin or serum leptin, after adjusting for potential confounders such as body composition and hormones, in either age group. These results suggest that adipocytokines do not influence REE in adult women.

Key Words resting energy expenditure, adiponectin, leptin, age, female adults

Resting energy expenditure (REE) accounts for 60 to 80% of total daily energy expenditure and is the basis for estimating energy requirements. In the field of energy metabolism, early investigators showed intense interest in establishing the factors contributing to REE (*1–3*).

Recent progress has shown that adipocytokines, which are bioactive substances secreted from adipocytes (*4, 5*), play an important role in regulation of basal metabolic rate (*6*). In particular, much attention is paid to adiponectin, which is suggested to play a role in improving insulin resistance and protecting against arteriosclerosis (*7–9*). In addition, rodent studies suggest that energy expenditure is regulated by adiponectin (*6, 10–13*). Further, Ruige et al. (*14*) recently demonstrated that a low resting metabolic rate (RMR; kcal/d) is strongly and inversely associated with high adiponectin levels in overweight or obese humans. Together these studies suggest that there is a relationship between adiponectin and REE.

It is well known that body mass, especially fat free mass (FFM), is a useful parameter for estimating REE (*15–17*). Fat mass (FM) is also an important predictor of REE in elderly people even though it is low-metabolic-rate tissue (*17–21*). However, in a previous study conducted to investigate the relationship between adiponectin and REE, body composition (FFM and FM) was not taken into consideration (*14*). It is therefore necessary to assess the effect of adiponectin on REE after adjusting for the confounding influence of body composition.

Therefore, we re-evaluated the relationship between blood adipocytokines and REE adjusted by body compo-

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sition in young and elderly women. We did not find any significant relationship between plasma adiponectin concentration and REE adjusted by body composition in either age group.

MATERIALS AND METHODS

Subjects. One hundred and fifteen young women (22.3±2.1 y) and 71 elderly women (63.4±6.5 y), who were at least 3 y (13.5±7.5 y) post-menopause, were recruited for the study. Women who had BMI (kg/m^2) which fell outside the range BMI<18.5, BMI>30 were excluded. There were 4 young and 17 elderly subjects who were overweight (25≤BMI<30). Included participants were not using any medications, including estrogen-replacement drugs. All subjects were informed of the purpose and possible risks of the study and then provided written informed consent, as approved by the Ethical Committee at the National Institute of Health and Nutrition in Japan.

Study protocol and indirect measurement of REE. Participants came to the National Institute of Health and Nutrition in the early morning. They were asked to minimize any walking prior to their laboratory visit for the REE measurement. REE was measured directly by open-circuit indirect calorimetry. Measurements were performed between 0700 and 0900 h in a room at constant temperature (23–25°C). After entering the laboratory, subjects rested in the supine position for at least 30 min, and wore a Hans-Rudolph full face mask (Hans Rudolph Inc., Kansas City, MO, USA). Two samples of expired air were collected in Douglas bags over each of two 10 min periods, and the mean of the two values was used for analysis. For young subjects, all measurements were made during the follicular phase of the menstrual cycle.

An oxygen and carbon dioxide analyzer (Arco-1000A, Arco System, Japan) was used to analyze the rate of oxygen consumption and carbon dioxide production. The volume of expired air was determined using a dry gas volume meter (DC-5, Shinagawa, Japan) and converted to standard temperature, pressure and dry gas (STPD). Gas exchange results were converted to REE (kcal/d) using Weir's equation (22).

Body composition analysis. Body weight (BW) was measured to the nearest 0.1 kg using an electronic scale (Inner Scan BC-600, TANITA Co., Japan), and height (Ht) was measured to the nearest 0.1 cm using a stadiometer (YL-65, YAGAMI Inc., Japan). Body mass index (BMI) was calculated by dividing BW in kilograms by the Ht in meters squared (kg/m^2). The percentage of whole body fat (% body fat) was measured using dual energy X-ray absorptiometry (Hologic QDR-4500 DXA Scanner, Hologic Inc., Waltham, MA, USA). Manufacturer's software version 11.2 for Windows was used to analyze the % body fat. FFM and FM were calculated by BW and % body fat.

Blood samples. Venous blood samples (fasting for at least 12 h) were collected for measurements of serum glucose, glycosylated hemoglobin (HbA_{1c}), total cholesterol, HDL-cholesterol, triglycerides, estradiol (E_2), total triiodothyronine (T_3), leptin, and plasma adiponectin. Serum and plasma samples were stored at −80°C for subsequent analysis. All blood parameters were analyzed by SRL, Inc. (Tokyo, Japan) and Mitsubishi Chemical Medience Corporation (Tokyo, Japan).

Statistical analysis. The data were presented as mean±standard deviation (SD). Statistical analyses were carried out with the Sigma Stat 2.03 (Systat Software Inc., California, USA). Statistical analysis was performed using the Student's *t*-test for parametric variables and the Mann-Whitney rank sum test for non-parametric variables to determine differences between young and elderly women. To determine the associations between REE and adipocytokines, partial correlation coefficients were used after adjusting for the potential confounding influence of FM (kg), FFM (kg), E_2 (pg/mL), and T_3 (ng/dL). For all the statistical analyses, the level of significance was defined as a p value of less than 0.05.

RESULTS

Table 1 presents comparisons of characteristics, whole body composition, and blood biochemical profiles. Ht was significantly lower in the elderly women than in young women. However, no significant difference in BW was noted between the two groups. The elderly women had significantly higher levels of % body fat and FM, and lower levels of FFM than the young group.

REE (kcal/d and kcal/kg BW/d) in the elderly group was significantly lower than in the young group (Table 2). When REE is expressed in terms of kcal/kg FFM/d, however, no significant difference in REE was found between the two groups. No significant difference in plasma adiponectin concentration was noted between the two groups, whereas serum leptin concentration was significantly higher in elderly women than in young women.

There was no significant relationship observed between REE (kcal/d, kcal/kg BW/d and kcal/kg FFM/d) and plasma adiponectin concentration in young women (Fig. 1, a-1–a-3). In contrast, in elderly women, REE (kcal/d) was significantly and inversely correlated with plasma adiponectin concentration ($r=-0.386$, $p<0.001$, Fig. 1, b-1), but REE expressed per kilogram BW and FFM were not significantly correlated (Fig. 1, b-2, b-3). Significant and inverse relationships between REE adjusted by BW and concentrations of serum leptin were observed in the two groups (young: $r=-0.318$, elderly: $r=-0.426$, $p<0.001$, respectively, Fig. 2, a-2, b-2). When REE was expressed relative to FFM, however, no significant relationships were obtained for either group (Fig. 2, a-3, b-3).

A partial correlation coefficient calculated after adjusting for the confounding influence of FM (kg), FFM (kg), E_2 (pg/mL), and T_3 (ng/dL) did not demonstrate any significant relationship between REE and concentrations of plasma adiponectin and serum leptin (Table 3).

Table 1. Physical and biochemical characteristics in young and elderly women.

	Young (n=115)		Elderly (n=71)	
Age (y)	22.3±2.1	(19.1–29.5)	63.4±6.5†	(50.2–77.0)
Ht (cm)	161.3±6.7	(142.2–181.0)	153.8±5.2*	(141.3–164.7)
BW (kg)	55.4±6.5	(41.7–73.7)	54.2±6.0	(41.4–72.2)
BMI (kg/m^2)	21.3±1.9	(18.5–26.6)	22.9±2.3*	(19.4–28.9)
% body fat	24.0±4.4	(14.3–35.7)	30.2±4.8*	(18.6–38.9)
FM (kg)	13.3±3.0	(7.5–24.8)	16.5±4.0*	(8.2–26.7)
FFM (kg)	42.1±5.5	(31.4–57.5)	37.7±3.5†	(28.2–38.9)
Glucose (mg/dL)	87±5	(69–100)	94±8†	(78–115)
HbA_{1c} (%)	4.8±0.3	(3.9–5.7)	5.1±0.3†	(4.6–6.0)
Total cholesterol (mg/dL)	178±26	(121–249)	216±27*	(158–282)
HDL-cholesterol (mg/dL)	69±13	(40–100)	66±15	(34–107)
Triglycerides (mg/dL)	60±24	(25–182)	93±44†	(34–280)
E_2 (pg/mL)	75±60	(10–295)	11±3†	(10–29)
T_3 (ng/dL)	108±16	(61–150)	112±18	(80–160)

Values are means±SD (range; minimum–maximum), Ht: height, BW: body weight, BMI: body mass index, FM: fat mass, FFM: fat free mass, HbA_{1c}: glycosylated hemoglobin, E_2: estradiol, T_3: total triiodothyronine, *p<0.001 vs. young group (Student's t-test), †p<0.001 vs. young group (Mann-Whitney rank sum test).

Table 2. Resting energy expenditure and adipocytokines in young and elderly women.

	Young (n=115)		Elderly (n=71)	
REE (kcal/d)	1,190±154	(830–1,622)	1,085±109†	(913–1,459)
(kcal/kg BW/d)	21.5±1.9	(17.8–27.5)	20.1±1.9*	(16.2–25.1)
(kcal/kg FFM/d)	28.4±2.3	(24.0–33.7)	28.9±2.4	(23.5–35.7)
Adiponectin (μg/mL)	9.9±3.8	(2.9–22.1)	9.9±4.1	(2.6–20.8)
Leptin (ng/mL)	6.1±3.1	(1.1–19.8)	7.8±4.4††	(1.5–25.7)

Values are means±SD (range; minimum–maximum), REE: resting energy expenditure, *p<0.001 vs. young group (Student's t-test), †p<0.001 and ††p<0.05 vs. young group (Mann-Whitney rank sum test).

DISCUSSION

The present study demonstrates that plasma adiponectin and serum leptin concentrations are not associated with REE in either young or elderly women when confounding factors, such as FM and FFM, are taken into account.

Elderly women recruited in this study were at least 3 y post-menopause. Although no significant difference in BW was noted between the young and elderly women, the elderly women had significantly higher levels of % body fat and FM, and lower levels of FFM than the young group (Table 1). These results are consistent with previous studies, the results of which indicated that adipose tissue mass is controlled by steroid hormones and that menopause is also associated with increased body mass accompanied by elevated adiposity in females (*23*, *24*).

In the present cross-sectional study, REE (kcal/d and kcal/kg BW/d) in the elderly group was significantly lower than in the young group. However, when REE was expressed per kilogram of FFM, no significant difference was observed between the two groups (Table 2). This evidence suggests that the specific metabolic rate per FFM does not decline with advancing age, and that REE is regulated mainly by the mass of the tissue-organs with both lower and higher metabolic rates, including skeletal muscle, intestinal organs and residuals in adults (*25–28*).

It is generally well known that thyroid function is associated with REE (*29*). In addition, previous studies in premenopausal women (*30*, *31*) have shown that REE is lower during the early follicular phase of menstrual cycle, when E_2 is low, than in the midluteal phase. The present investigation demonstrated a significant relationship between serum T_3 concentration and REE in terms of kcal/kg FFM/d in all subjects (young: r=0.493, elderly: r=0.385, all subjects: r=0.456, p<0.001, respectively). In the young group but not the elderly group, serum E_2 concentration significantly correlated with REE (kcal/kg FFM/d), which was measured during the follicular phase of the menstrual cycle (r=0.222, p<0.05). These results suggest that concentrations of serum T_3 and E_2 may have a role in the regulation of REE in adult women.

Previous animal studies demonstrated that adiponec-

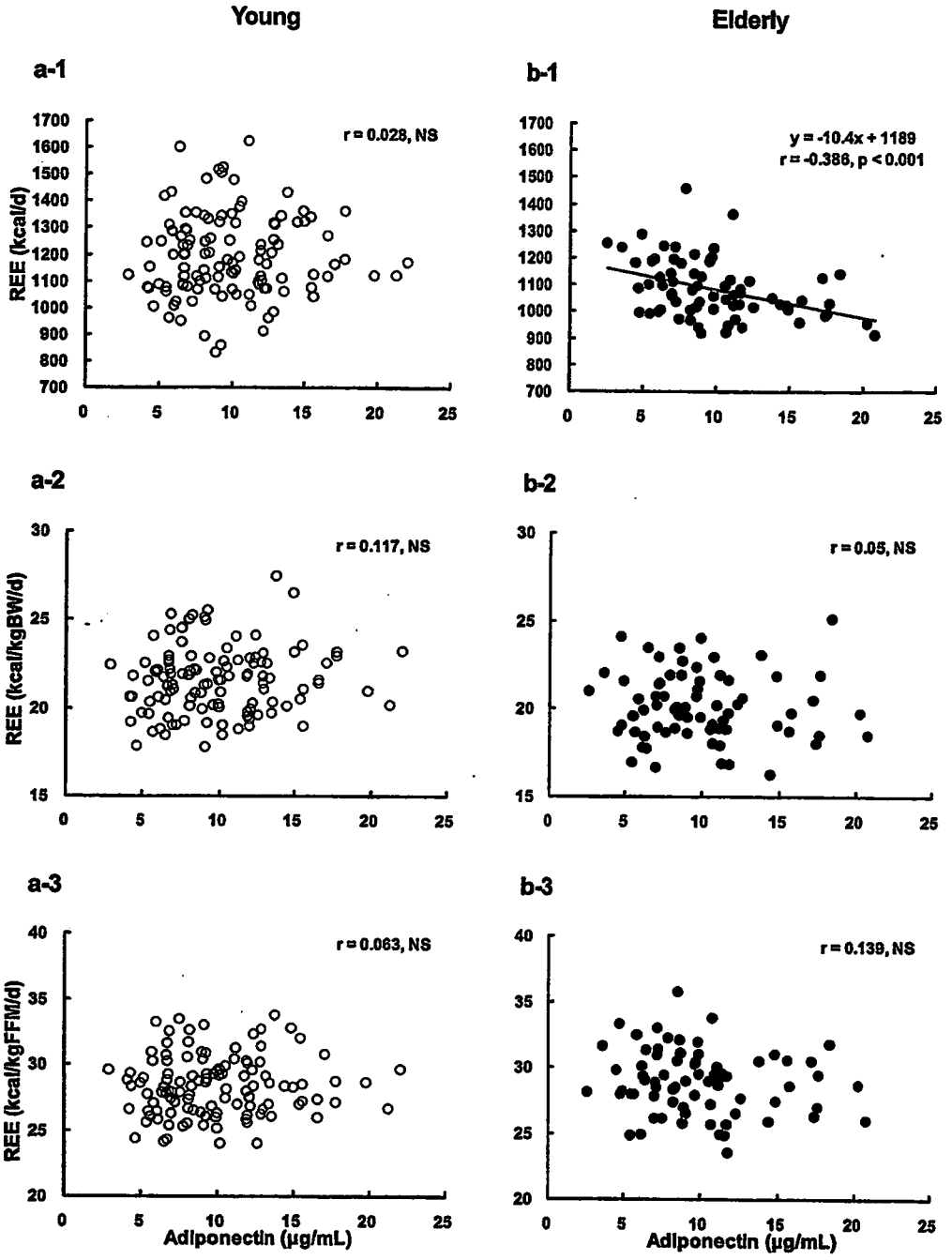

Fig. 1. Relationship between REE (kcal/d, kcal/kg BW/d, kcal/kg FFM/d) and adiponectin.

tin increases β-oxidation through AMP-kinase activation, suggesting that adiponectin plays crucial and central roles in the regulation of energy expenditure (*32*). In contrast, we demonstrate that a significant inverse link exists between plasma adiponectin concentrations and REE in elderly women ($r=-0.386$, $p<0.001$, Fig. 1, b-1). This is consistent with the results observed in overweight and obese males and females (*14*). These results might provide the possibility that protection by adiponectin against obesity-related disorders is especially important for human subjects with low RMR, and it is tempting to suggest plasma adiponectin as a valuable predictor for RMR, or vice versa.

We previously demonstrated that both FFM and FM are important predictors of REE in elderly people (*20*, *21*). Thus, the inverse association between REE (kcal/d) and concentrations of plasma adiponectin in the present investigation in the elderly group may reflect spurious correlations. Therefore, for the purpose of determining the precise relationship between plasma adiponectin concentrations and REE, we adopted a partial correlation coefficient after adjusting for confounding influences of FM (kg), FFM (kg), E_2 (pg/mL), and T_3 (ng/dL). As a consequence, we have demonstrated that REE is not significantly correlated with plasma adiponectin concentrations (Table 3). These results suggest that adiponectin is not involved in the regulation of REE in young or elderly women.

Jørgensen et al. (*33*) demonstrated that serum leptin was a strong positive determinant for RMR in men. However, in their study, no adequate adjustment of RMR was made for either FFM or FM. In the present

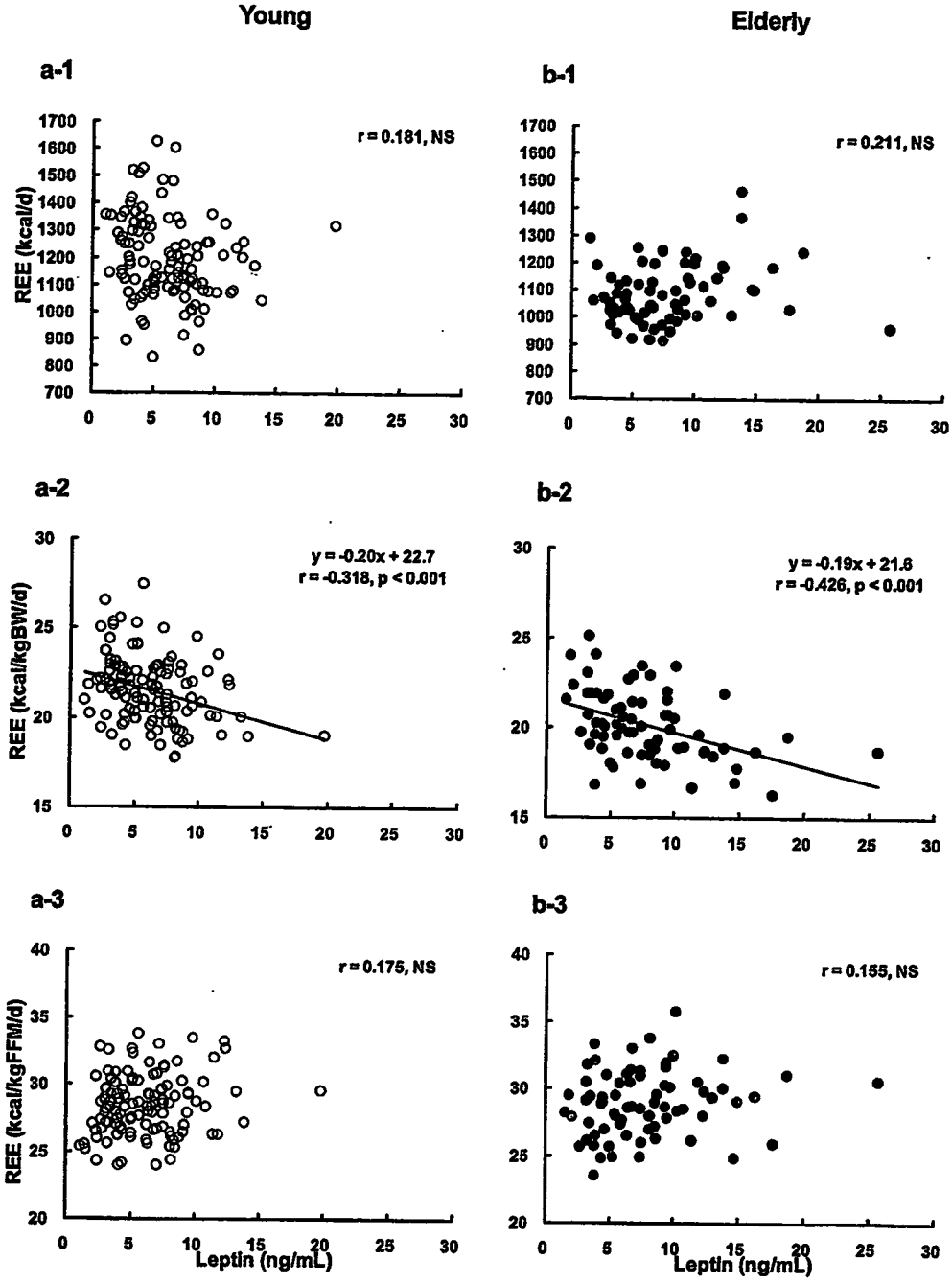

Fig. 2. Relationship between REE (kcal/d, kcal/kg BW/d, kcal/kg FFM/d) and leptin.

Table 3. Partial correlation coefficient to REE in female subjects.

	Young (n=115)		Elderly (n=71)	
Variable	β	p	β	p
REE (kcal/d)				
Adiponectin (μg/mL)	0.138	0.148	−0.200	0.104
Leptin (ng/mL)	−0.006	0.947	0.067	0.588

β: partial correlation coefficient: controlling for FM (kg), FFM (kg), E_2 (pg/mL), and T_3 (ng/dL).

investigation, no significant relationship between leptin and REE adjusted by body composition was observed (Table 3). This result is consistent with the report of Neuhäuser-Berthold et al. (34), suggesting that leptin might not have a significant role in the regulation of REE.

Our investigation has a few limitations. First, we did not test middle-aged (30–49 y) adults. Second, we did not include male subjects. Third, although adipocytokines such as adiponectin and leptin were related to the levels of body fat mass, we did not observe for lean and obese subjects. Future studies are needed to investigate this association in lean and obese adults and middle-aged adults.

In conclusion, the present investigation provides evi-

dence to suggest that adipocytokines, such as adiponectin and leptin, do not influence REE in adult women.

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Experimental Physiology

Resistance training in men is associated with increased arterial stiffness and blood pressure but does not adversely affect endothelial function as measured by arterial reactivity to the cold pressor test

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Resistance training is a popular mode of exercise, but may result in stiffening of the central arteries. Changes in carotid artery diameter were determined using the cold pressor test (CPT), which results in production of nitric oxide via sympathetic activation and is one of the novel methods available for assessing endothelial function in the carotid artery. To investigate the effect of resistance training on endothelial function, we designed a cross-sectional study of carotid arterial vasoreactivity to CPT in men participating in regular resistance training with increased carotid arterial stiffness compared with age-matched control subjects. Twelve resistance-trained middle-aged men (age 38.7 ± 1.7 years) and 17 age-matched control subjects (age 36.8 ± 1.2 years) were studied. The direction and magnitude of changes in carotid artery diameter were measured by B-mode ultrasonography during sympathetic stress induced by submersion of the foot in ice slush for 90 s. Carotid arterial β-stiffness index, and systolic and mean arterial blood pressure were higher (7.7 ± 0.7 *versus* 6.0 ± 0.4 arbitrary units, 116 ± 2 *versus* 131 ± 4 mmHg and 86 ± 2 *versus* 95 ± 2 mmHg, respectively, all $P < 0.05$) in the resistance training group compared with control subjects. There were, however, no significant differences in the amount or percentage change in carotid artery diameter in CPT between the two groups (resistance training group, 0.33 ± 0.07 mm and 5.2 ± 1.1%; control group, 0.37 ± 0.06 mm and 5.8 ± 0.9%, respectively). These findings suggest that while carotid arterial stiffening and higher blood pressure are observed in regular resistance-trained men, these are not associated with abnormalities in carotid arterial vasoreactivity to sympathetic stimulus, which implies intact endothelial function.

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Resistance training is a popular form of exercise, and has become an integral component of exercise recommendations endorsed by a number of national health organizations (American College of Sports Medicine Position Stand, 1998; Pollock *et al.* 2000). Resistance training has favourable effects on the musculoskeletal system, thereby contributing to maintenance of functional capacity and prevention of sarcopenia and osteoporosis. In contrast, resistance training may be associated with reduction of compliance and increases in arterial stiffness in the central elastic artery (carotid artery; Bertovic *et al.* 1999; Miyachi *et al.* 2003, 2004; Cortez-Cooper *et al.* 2005; Kawano *et al.* 2006).

Increased arterial stiffness and reduced arterial compliance may be associated with endothelial dysfunction (Lind *et al.* 1999; Cheung *et al.* 2002; Nakamura *et al.* 2004). Indeed, impaired endothelial function and arterial stiffening are induced with advancing age and in the presence of cardiovascular diseases (Zeiher *et al.* 1989; O'Rourke, 1990; Taddei *et al.* 1995; Tanaka *et al.* 2000; Najjar *et al.* 2005). Therefore,

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Table 1. Subject characteristics

	Control	Resistance trained
Number of subjects	17	12
Age (years)	36.8 ± 1.2	38.7 ± 1.7
Height (cm)	171.0 ± 1.2	171.0 ± 1.8
Body weight (kg)	71.9 ± 1.9	74.9 ± 2.1
Percentage body fat (%)	19.4 ± 1.2	12.3 ± 0.9*
Total cholesterol (mmol l^{-1})	5.0 ± 0.2	4.7 ± 0.2
HDL cholesterol (mmol l^{-1})	1.3 ± 0.1	1.6 ± 0.1*
Plasma glucose (mmol l^{-1})	5.0 ± 0.1	5.1 ± 0.1
Triglycerides (mmol l^{-1})	1.5 ± 0.3	0.9 ± 0.1
Resting heart rate (beats min^{-1})	58 ± 2	56 ± 2
Maximal heart rate (beats min^{-1})	186 ± 3	183 ± 4
$\dot{V}_{O_2max}$ (l min^{-1})	2.7 ± 0.1	2.8 ± 0.1
$\dot{V}_{O_2max}$/body weight (ml kg^{-1} min^{-1})	37.7 ± 1.4	36.9 ± 1.3
Leg extension power (W)	1719 ± 91	2293 ± 155*
Handgrip (kg)	45.6 ± 1.6	51.0 ± 2.0*

Data are means ± S.E.M.; $\dot{V}_{O_2max}$, maximal oxygen consumption. *$P < 0.05$ *versus* control subjects.

impaired endothelial function is thought to be one of the physiological mechanisms underlying the reduction in carotid arterial compliance with resistance training. In this context, we hypothesized that resistance training would cause impairment of endothelial function in the carotid artery.

Local endothelial function in humans can be estimated by flow-mediated dilatation (Corretti *et al.* 2002) and/or vasoreactivity in response to medication with acetylcholine, etc. (Ludmer *et al.* 1986). Since it is difficult to determine endothelial function of the carotid artery in healthy humans using these methods, the cold pressor test (CPT), which results in production of nitric oxide (NO) via sympathetic activation (Nase & Boegehold, 1996; Tousoulis *et al.* 1997) is one of the novel methods (Rubenfire *et al.* 2000; Lavi *et al.* 2006) available for assessing endothelial function in the carotid artery.

To evaluate our hypothesis, we designed a cross-sectional study in which carotid arterial vasoreactivity to receptor-mediated sympathetic cold stimulus in regular resistance-trained men with reduced carotid arterial compliance was compared with age-matched sedentary control subjects.

Methods

Subjects

A total of 29 healthy men, 28–49 years of age, participated in the present study (Table 1). The sedentary subjects were recruited through various forms of advertisement and had not participated in a regular exercise programme for at least the previous 2 years. The resistance-trained men were recruited from various fitness clubs and had been performing vigorous resistance training for > 10 years. All resistance-trained men had been performing moderate- to high-intensity 'full-body' resistance exercise involving large muscle groups. To better isolate the effects of resistance exercise training, those who had been concurrently performing regular aerobic exercise (i.e. 'cross-training') were excluded from the study. All subjects were normotensive (< 140/90 mmHg), non-obese and free of overt chronic diseases as assessed by medical history, physical examination and complete blood chemistry and haematological evaluation. Candidates who smoked in the past 4 years, were taking medications, had ever used anabolic steroids or other performance-enhancing drugs, or who had significant femoral intima–media thickening (< 1.1 mm), plaque formation and/or other characteristics of atherosclerosis [ankle–brachial index (ABI) < 0.9] were excluded. All subjects gave their written, informed consent to participation in this study. All procedures were reviewed and approved by the Human Research Committee of the National Institute of Health and Nutrition.

Measurements

Before testing, subjects abstained from caffeine and fasted for at least 4 h (a 12 h overnight fast was used for determination of metabolic risk factors). All measurements were performed under comfortable laboratory conditions in the morning. Tests of resistance-trained men were conducted 20–24 h after their last exercise training session to avoid the immediate (acute) effects of exercise, but they were still considered to be in their normal (i.e. habitually exercising) physiological state.

Body composition

Body composition was determined using dual-energy X-ray absorptiometry (DEXA; model DPX-IQ, Lunar

Radiation) with subjects in the supine position. Measurement of fat mass using DEXA has been well validated against other standards (Haarbo *et al.* 1991).

Carotid arterial intima–media thickness (IMT)

Carotid artery IMT was measured from the images obtained using a SonoSite 180 PLUS ultrasound system (SonoSite, Bothell, WA, USA) equipped with a high-resolution linear-array broad-band transducer as previously described (Miyachi *et al.* 2004). Ultrasound images were analysed using image analysis software (NIH Image 1.63, Bethesda, MD, USA). At least 10 measurements of IMT were taken at each segment, and the mean values were used for analysis. This technique has excellent day-to-day reproducibility (coefficient of variation, $3 \pm 1\%$) for the carotid IMT.

Carotid arterial compliance

A combination of ultrasound imaging of the pulsatile common carotid artery with simultaneous applanation of tonometrically obtained arterial pressure from the contralateral carotid artery permits non-invasive determination of arterial compliance (Tanaka *et al.* 2000). The carotid artery diameter was measured from images obtained using an ultrasound system (Sonosite, Bothell, WA, USA) equipped with a high-resolution linear-array transducer. A longitudinal image of the cephalic portion of the common carotid artery was acquired 1–2 cm proximal to the carotid bulb. All image analyses were performed by the same investigator who was blinded to the group assignments.

Pressure waveforms and amplitudes were obtained from the common carotid artery with a pencil-type probe incorporating a high-fidelity strain-gauge transducer (SPT-301; Millar Instruments, Houston, TX, USA; Kelly *et al.* 1989; Tanaka *et al.* 2000). Since baseline levels of blood pressure are subjected to hold-down force, the pressure signal obtained by tonometry was calibrated by equating the carotid mean arterial and diastolic BP to the brachial artery value (Tanaka *et al.* 2000; Miyachi *et al.* 2004). In addition to arterial compliance (Van Merode *et al.* 1988), we also calculated the β-stiffness index, which provides an index of arterial compliance adjusted for distending pressure (Hirai *et al.* 1989). The arterial compliance and the β-stiffness index were calculated using the following equations:

$$\text{arterial compliance} = \frac{[(D_1 - D_0/D_0)]}{2\,(P_1 - P_0)} \times \pi \times D_0^2$$

and

$$\beta - \text{Stiffness index} = \frac{\ln\,(P_1/P_0)}{[(D_1 - D_0)\,/D_0]}$$

where D_1 and D_0 are the maximal and minimal diameters, and P_1 and P_0 are the highest and lowest blood pressures, respectively. The day-to-day coefficients of variation were 2 ± 1, 7 ± 3 and $5 \pm 2\%$ for the carotid artery diameter, pulse pressure and arterial compliance, respectively.

Cold pressor test

The CPT was performed by submersion of the right foot up to the ankle in ice slush for 90 s, a modification of the method published previously (Corretti *et al.* 1995*b*; Rubenfire *et al.* 2000). The foot was chosen to maximize the haemodynamic and sympathetic responses (Seals, 1990). Subjects were instructed to avoid breath-holding, muscle contractions and Valsalva's manoeuvre. Measurements of carotid arterial geometry were obtained before (baseline) and for 10 s during CPT. The day-to-day coefficient of variation for the change in carotid arterial diameter response to CPT was $4 \pm 1\%$.

Maximal oxygen uptake

We measured maximal oxygen consumption ($\dot{V}_{O_2max}$) during incremental cycle ergometer exercise (Miyachi *et al.* 2001). Oxygen consumption (coefficient of variation, $4 \pm 1\%$), heart rate and ratings of perceived exertion were measured throughout the protocol (Miyachi *et al.* 2001).

Metabolic risk factors for coronary heart disease

To screen for the presence of coronary heart disease, concentrations of fasting serum lipids and plasma glucose were determined with enzymatic techniques (Tanaka *et al.* 2000).

Arterial blood pressure at rest

Chronic levels of arterial blood pressure at rest were measured with a semi-automated device (Form PWV/ABI; Colin Medical, Komaki, Japan) over the brachial and dorsalis pedis arteries. Recordings were made in triplicate with subjects in the supine position (Miyachi *et al.* 2005).

Muscle strength

Leg extension power was determined using a dynamometer (Anaero Press 3500; Combi Wellness, Tokyo, Japan) in the sitting position. The subjects were fastened with a seat belt to a chair. In the starting position, the feet were placed on a sliding plate with the knee angle adjusted to 90 deg. Subjects were advised to vigorously extend their legs. Five trials were performed at 15 s intervals and the average of the two highest recorded power outputs (in W) was taken as the definitive measurement (Yoshiga *et al.* 2002).

Table 2. Cardiovascular measures

	Control	Resistance trained
Brachial systolic BP (mmHg)	116 ± 2	131 ± 4*
Brachial mean BP (mmHg)	86 ± 2	95 ± 3*
Brachial diastolic BP (mmHg)	71 ± 2	74 ± 3
Brachial PP (mmHg)	45 ± 1	57 ± 2*
Carotid systolic BP (mmHg)	104 ± 2	123 ± 5*
Carotid PP (mmHg)	33 ± 2	48 ± 4*
Carotid artery diameter (mm)	6.4 ± 0.1	6.2 ± 0.1
Carotid artery IMT (mm)	0.64 ± 0.02	0.65 ± 0.03

Data are means ± S.E.M.; BP, blood pressure; PP, pulse pressure; IMT, intima–media thickness. *$P < 0.05$ *versus* control subjects.

Handgrip strength of the right arm was measured with a hand-held dynamometer, with the subject standing and the arms extended by their sides. The subjects then gripped the dynamometer as strongly as possible for 3 s without pressing the instrument against their body or bending at the elbow, and values (in kg) were recorded as the averages of two trials.

Statistics

Statistical analyses were performed using statistical software (StatView, SAS, Cary, NC, USA). All data are presented as means ± S.E.M. Mean differences between resistance-trained and control men were examined using Student's unpaired *t* test. Analysis of covariance (ANCOVA) was used to test for differences in carotid arterial compliance and β-stiffness index between resistance-trained men and control subjects, with mean arterial blood pressure as a covariate.

Statistical significance was set *a priori* at $P < 0.05$ for all comparisons.

Results

Subject characteristics are presented in Table 1. Body fat was lower in the resistance-trained men compared with the control subjects. Although all metabolic risk factors were well within clinically normal levels in both groups, high-density lipoprotein (HDL) cholesterol levels were higher in resistance-trained men compared with control subjects. Muscle strength, assessed by leg extension power and handgrip strength, was higher in resistance-trained men than in the control subjects. There were no significant differences in other parameters between the two groups.

Table 2 shows cardiovascular measures. With the exception of diastolic blood pressure in the brachial artery, blood pressure parameters of brachial and carotid arteries were higher in resistance-trained men compared with control subjects. Ankle–brachial index was lower in resistance-trained men than control subjects. There were no significant differences in the diameter or IMT in the carotid artery between the two groups.

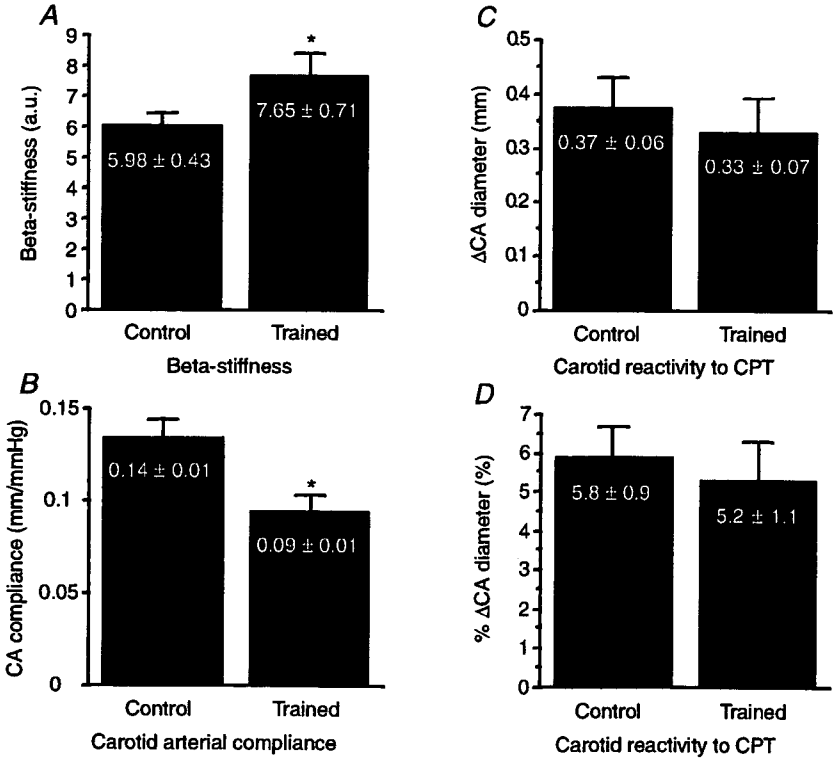

Figure 1. Carotid arterial β-stiffness index (*A*), carotid arterial (CA) compliance (*B*), and amount (*C*) and percentage change (*D*) in carotid artery diameter in response to CPT in resistance-trained men and control subjects
Values are means ± S.E.M. *$P < 0.05$ *versus* control subjects.

Carotid arterial β-stiffness (Fig. 1*A*) was higher and compliance (Fig. 1*B*) was lower in resistance-trained men compared with control subjects. There were no significant differences in the amount (Fig. 1*C*) or percentage change (Fig. 1*D*) of carotid artery diameter in response to CPT between resistance-trained men and control subjects. The differences in carotid arterial compliance and β-stiffness index between resistance-trained men and control subjects disappeared after normalizing carotid arterial compliance and β-stiffness index relative to mean arterial blood pressure (ANCOVA; $P = 0.081$ and $P = 0.101$, respectively).

Discussion

The results of the present study indicated that, although the carotid arterial compliance was lower in resistance-trained men compared with age-matched control subjects, there were no significant differences in the amount or percentage change of carotid arterial diameter in CPT between resistance training and control groups. In contrast to our original hypothesis, these findings suggest that while regular resistance training can increase carotid arterial stiffness, this is not associated with abnormalities of carotid arterial vasoreactivity to sympathetic physiological stress induced by cold.

The endothelial function of conduit arteries is one of the vascular functions, and has been identified as a primary target of injury from mechanical forces and processes that increase cardiovascular risk, such as hypertension (Moyna & Thompson, 2004). Owing to the clinical and functional importance of health of the endothelium, we examined the impact of resistance training on endothelial function. As a primary approach to resolve this issue, we performed a cross-sectional study. To isolate the effects of resistance training as much as possible, resistance-trained men and control subjects were carefully matched for age, height, body weight, aerobic capacity and metabolic risk factors. Although subjects were recruited carefully, as described in the Methods, blood pressure in resistance-trained men was higher than that in the control subjects. As a result, we found a 30% reduction in central arterial compliance in resistance-trained men compared with control subjects. These results are consistent with those of a previous cross-sectional study (Bertovic *et al.* 1999). Differences in carotid arterial compliance and β-stiffness index between resistance-trained men and control subjects were affected after normalizing carotid arterial compliance and β-stiffness index relative to mean arterial blood pressure. Given this association between blood pressure and arterial compliance, higher blood pressure may lead to lower arterial compliance in resistance-trained men than in control subjects due to equation using arterial distensibility and blood pressure. However, we feel that the higher blood pressure in resistance-trained men may be induced by greater arterial stiffening associated with the resistance training. Nevertheless, despite the higher arterial stiffness and blood pressure in resistance-trained men than in control subjects, there was no difference in carotid arterial vasoreactivity to CPT between the two groups.

The response of conduit arteries to systemic cold may be the result of the balance between adrenergic vasoconstriction and vasodilatation, with the latter being mediated by endothelial function (Nabel *et al.* 1988; Zeiher *et al.* 1989; Vita *et al.* 1992; Corretti *et al.* 1995*a*). The normal coronary vasodilator response to CPT can be blocked by competitive inhibition of L-arginine, a substrate for NO synthase (Tousoulis *et al.* 1997), and L-arginine can normalize the vasoconstrictor response to CPT in coronary artery disease (Gellman *et al.* 1996). In addition, both endogenous NO and exogenously administered NO donors suppress sympathetic outflow at the prejunctional level, and NO may exert a tonic influence on the discharge of sympathetic efferents (Zanzinger *et al.* 1994; Nase & Boegehold, 1996). Therefore, the endothelial function, via NO, may play an important role in changing the conduit artery diameter response to sympathetic stimulation by the CPT. We first examined the impact of resistance training with arterial stiffening on endothelial function of the carotid artery using CPT, and found that there were no significant differences in the amount or percentage change in carotid arterial diameter in response to CPT between resistance-trained men and control subjects. Our results were consistent with those of a previous study, which demonstrated that resistance training did not affect endothelial function in the peripheral muscular artery evaluated by flow-mediated dilation (FMD) (Rakobowchuk *et al.* 2005). These findings are consistent with the posit that regular resistance training may protect against the adverse effects of resistance load associated hypertension by preserving arterial endothelial function (Jurva *et al.* 2006).

The results of the present study indicated that carotid arterial compliance in resistance-trained men was lower than that in control subjects, and blood pressure was significantly higher in resistance-trained men compared with control men. In contrast, HDL cholesterol level was higher in resistance-trained men than in control subjects, and there were no differences in other lipid profiles or IMT between the two groups. Considering the relationships between reduction in arterial compliance and impaired endothelial function, hypertrophied IMT or abnormal lipid profile with advancing age and/or the presence of cardiovascular disease (Zeiher *et al.* 1989; O'Rourke, 1990; Taddei *et al.* 1995; Tanaka *et al.* 2000; Najjar *et al.* 2005), the decrease in carotid arterial compliance induced by resistance training may be different from vascular alterations seen in ageing or in the presence of

cardiovascular disease. Arterial compliance is affected by endothelial function as well as by sympathetic vascular tone, arterial calcification, elastin-to-collagen ratio and IMT, and correlates with clinical parameters, such as aerobic capacity, age, blood pressure, body fat, waist circumference and lipids (Nichols & O'Rourke, 1998; Tanaka *et al.* 2000). The degree to which these other factors affect the relationship between training-associated decrease in arterial compliance independent of endothelial function will require further studies in a larger cohort.

Rubenfire *et al.* (2000) reported that the direction and magnitude of the change in carotid artery diameter in response to CPT are altered based on the presence of risk factors and coronary disease independent of IMT. The carotid artery vasoreactivity to CPT may have a valuable role in coronary risk assessment and in predicting response to therapy. The present study revealed that there were no significant differences in carotid arterial vasoreactivity to CPT and IMT between resistance-trained men and control subjects, suggesting that regular resistance training may not affect at least two of the cardiovascular disease risk factors. In addition, HDL cholesterol, leg extension power and handgrip strength were higher in resistance-trained men than in control subjects. Given these functional and physiological benefits of resistance training, we should emphasize that the practice of resistance training should not be discouraged.

Limitations

Endothelial function assessed by FMD should optimally be adjusted by shear stress, shear rate or blood flow velocity (Pyke & Tschakovsky, 2005; Rakobowchuk *et al.* 2005). However, it is technically difficult to determine the blood velocity or shear stress during the relatively short period (90 s) of CPT used in our study. Further, in contrast to the occlusion release technique for assessing brachial endothelial function, the carotid artery vasoreactivity to CPT is a complex interaction between clinical, adrenergic nerve and hormonal responses and endothelial function.

Conclusion

The results of the present study showed that regular resistance training is associated with reduction of central arterial compliance as measured using a combination of ultrasound images and applanation tonometry. However, there were no differences in carotid arterial vasoreactivity to CPT between resistance-trained men and sedentary control subjects. These findings suggest that while carotid arterial stiffening and higher blood pressure are observed in regular resistance-trained men, they are not associated with impaired vasoreactivity to sympathetic stimulus, which implies intact endothelial function. Nevertheless, the results of the present cross-sectional study must be confirmed in future prospective exercise intervention studies.

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報　文

若年成人女性の基礎代謝量と身体組成

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Basal Metabolic Rate and Body Composition of Japanese Young Adult Females

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The basal metabolic rate (BMR) of Japanese females in their twenties (F2006) was compared with that of females of the same age measured in the 1950s (F1950 ; Nagamine and Suzuki, 1964). The subjects measured during 2004～2006 were 83 females (F2006) with no exercise habits. BMR was measured by using indirect calorimetry, and the body composition was assessed by dual-energy X-ray absorptiometry (DXA).

While the height and weight of F2006 were respectively significantly higher and heavier than those of F1950, BMR of F2006 (1,110±112kcal/day) was not significantly different from that of F1950 (1,132kcal/day). In addition, there was no difference in lean body mass (LBM) between the two groups. On the other hand, BMR per body weight of F2006 (21.5±2.1kcal/kg/day) was significantly lower than that of F1950 (23.1kcal/kg/day). BMR per body weight of F2006 was correlated with LBM per body weight (%LBM, r=0.51, p<0.001), although BMR per LBM of F2006 was not different from that of F1950.

These data suggest that BMR per body weight of the current young females of Japan is lower than that of the young females of the same age measured in the 1950s, during which data for establishing the BMR reference value in "Dietary Reference Intakes for Japanese, 2005" was obtained. Furthermore, the difference in BMR per body weight between the two groups can be explained by the difference in %LBM.

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Key words : Japanese young adult female, basal metabolic rate, lean body mass

緒　言

「日本人の食事摂取基準（2005年版）」(Dietary Reference Intakes for Japanese, 2005：以下DRIs-J）では1日の推定エネルギー必要量を算出する際に，各自の体重に基礎代謝基準値を掛け合わせて求めた基礎代謝量（Basal metabolic rate：BMR）に身体活動レベル（Physical activity level：PAL）を乗じて求めている[1]。このように，1日の推定エネルギー必要量を求めるためにはBMRが主な要因であり，重要である[2]。

現在使用されている基礎代謝基準値は，1969年に改定された日本人の栄養所要量の値で示されたものを使っている[3]が，この基礎代謝基準値は主に1950年代に測定されたデータを基に定められている。当時（1950, 57, 59年）の20歳代女性の体重と身長の平均値はそれぞれ49.5kg，151.0cm[4]であったが，現代の20歳代女性ではそれぞれ50.9kg，158.3cm[5]であり，体重には差がない

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が，身長は高くなっている。BMRには身体組成が大きな影響を与えることが知られているが[6~8]，このような体格の変化による身体組成の変化により，BMRが変化している可能性がある。

そこで，本研究では現代の若年女性のBMRと身体組成を測定し，DRIs－Jの基礎代謝基準値策定の基となった基礎代謝量測定[9~11]と同時期の1958～1960年の間に行われたNagamine and Suzuki[12]によって報告されている若年女性の身体組成と，BMRのデータ（以下先行研究）を比較することにより，DRIs－Jで用いられている基礎代謝基準値の妥当性を検討することを目的とした。

方　法

1. 被験者

本研究の被験者は，事前の聞き取りおよびアンケート調査において定期的な運動習慣のない健康な20歳代女性83名であった。その内訳は大学生および大学院生75名，事務職員8名であった。

本研究は，独立行政法人国立健康・栄養研究所「人間を対象とする生物医学的研究に関する倫理委員会」の承認を得てヘルシンキ宣言の精神に則り，実施した。測定に当たって，対象者に測定の目的，利益，不利益，危険性，データ管理や公表について説明を行い，書面による同意を得た。

本研究の結果との比較に用いた先行研究[12]の被験者は，東京在住の大学生が中心の平均年齢21歳の女性112名であった。

2. 身体組成

本研究では身長，体重は排尿を済ませた早朝空腹時に測定し，Body mass index（BMI）を算出した。除脂肪量（Lean body mass：LBM）は，二重エネルギーX線法（Dual－energy X－ray absorptiometry：DXA法，QDR－4500，Hologic社製）を用いて，軟部組織のうちLBM（骨塩量を除いた）と骨塩量（kg）の和として求めた。%LBM（%）は体重に対するLBMのパーセンテージとして表した。

先行研究[12]における被験者の身体組成の測定は水中体重法であった。

3. 基礎代謝量（Basal metabolic rate：BMR）

本研究のBMRの測定は個々人の月経周期に関する事前の聞き取りを行い，実験日を調節し，月経第1日目から起算して第5～14日の卵胞期に実施した。測定前日は激しい運動を避け，前夜の午後9時までに通常通りの夕食を摂り，その後は水以外の飲食はしないように指示した。測定日当日は朝食を食べずに被験者室に来所してもらい排尿後，室温20～25℃の条件下において，覚醒・仰臥安静状態で基礎体温，心拍を計測した後に，BMRを測定した。

BMRの測定は，ルドルフマスクを装着後30分以上仰臥させた後，仰臥位のまま，ダグラスバッグに呼気を10分間，5分のインターバルをおいて2回採取した。呼気はガスメーター（DC－50，品川製作所製）にて換気量を測定するとともに，質量分析計（ARCO－1000，アルコシステム社製）を用いて酸素および二酸化炭素量の濃度を分析して酸素摂取量（*l*/分）を算出し，Weir[13]の式により1分当たりのBMR（kcal/分）を求めた。さらに1,440（分）を乗じ，1日当たりのBMRとした。

先行研究[12]のBMRの測定はダグラスバッグ法を用いていた。酸素摂取量の計算式については記載がなかった。先行研究[12]のBMRは体表面積（Body surface area：BSA）当たりのBMRのみ記載があり，BSA当たりのBMRは，32.4 ± 2.4kcal/m²BSA/時であった。BSAの計算式（BSA＝体重（kg）$^{0.425}$ × 身長（cm）$^{0.725}$ × 71.84）を用いて，先行研究のBMRの絶対値および体重当たりのBMRを求めた（表2）。

4. 血液検査

採血は早朝空腹時に実施し，ヘモグロビン，ヘマトクリット，総コレステロール，HDLコレステロール，中性脂肪，トリヨードチロニン（Triiodothyronine：T_3）を測定した。血液分析は㈱三菱化学ビーシーエルに委託した。ヘモグロビンおよびヘマトクリットは自動血球分析装置を使用し，総コレステロール，HDLコレステロールおよび中性脂肪は酵素法，T_3はECLIA法を用いた。

5. 最大酸素摂取量

モナーク社製自転車エルゴメーターを用いた漸増負荷法により，最大酸素摂取量を測定した。ペダルの回転数は60rpmとし，60 Wで5分間のウォーミングアップを行わせた後，1分毎に，15 Wずつ負荷を増加させ，疲労困憊まで至らしめた。呼気ガスの採取は，ダグラスバッグに摂取し，ガス分析は質量分析計，換気量計測はガスメーターを用いた。

6. 統計処理

すべてのデータは，平均値と標準偏差（Mean ± SD）で表した。本研究で得られた各指標の統計処理は，Microsoft Excel XP（Microsoft社），Sigma Stat2.0（SPSS社）にて行った。項目間（先行研究[12]のBMRと本研究のBMR）における平均値の差の検定には，対応のない*t*－testを用いた。

さらにBMRに影響を及ぼす因子を確認するために，本研究の被験者のデータを対象としてステップワイズの重回帰分析を行った。従属変数をBMR，説明変数を

表1 被験者の身体的特徴と基礎代謝量

年齢	(歳)	22.9 ± 2.0
身長	(cm)	159.6 ± 5.8
体重	(kg)	51.9 ± 5.8
BMI	(kg/m²)	20.4 ± 2.0
除脂肪率 (%LBM)	(%)	75.8 ± 4.1
体脂肪率 (%Fat)	(%)	25.2 ± 4.0
LBM	(kg)	39.2 ± 4.9
最大酸素摂取量	(ml/kg/min)	34.2 ± 4.9
BMR	(kcal/日)	1,110 ± 112
	(kcal/kg 体重/日)	21.5 ± 2.1
	(kcal/kg LBM/日)	28.4 ± 2.3
	(kcal/m² BSA/時)#	31.3 ± 2.6

若年成人女性 (83名) の身体的特徴と基礎代謝量を平均値±標準偏差で示したもの。
n=83
BMI : Body mass index, LBM : 除脂肪量, BMR : 基礎代謝量, BSA : 体表面積
#体表面積の計算式 : BSA = 体重(kg)$^{0.425}$ × 身長(cm)$^{0.725}$ × 71.84

体重，身長にした場合と説明変数をLBM，脂肪量，身長にした場合のステップワイズ重回帰分析（変数増加法）を行い，BMRに対する説明変数の寄与率を求めた。

また，現代若年女性と先行研究[12]のBMRの差を議論することを目的として，現代若年女性を%LBM（平均値（75.8%），標準偏差（4.1%））で%LBMが高値群（%LBM ≧平均値＋1/2標準偏差），中間値群（平均値－1/2標準偏差＜%LBM＜平均値＋1/2標準偏差），低値群（%LBM ≦平均値－1/2標準偏差）の3群に分け，それぞれの値を示した。%LBMの3群間の比較は，一元配置分散分析後，多重比較（Tukey－test）を行った。

すべて有意水準は5%未満とした。

結　　果

1. 被験者の身体的特徴

本研究で対象とした被験者の特徴を表1に示した。本研究で対象とした20歳代の運動習慣のない女性83名の身体的計測値を，平成16年国民健康・栄養調査報告[5]で報告されている20歳代女性の値（身長：158.3 ± 5.4cm，体重：50.9 ± 6.9kg）と対応のないt－testで比較をしたところ，いずれの項目においても有意な差は認められなかった。したがって，本研究の被験者は現代の20歳代女性の標準的な体位を有する集団であったと考えられる。

先行研究[12]の被験者の特徴と本研究との比較を表2に示した。本研究の被験者の身長，体重および体脂肪率（%Fat）は，先行研究[12]よりも有意に大きかった。

2. 基礎代謝量（Basal metabolic rate : BMR）

本研究で測定した被験者のBMRは1,110 ± 112kcal/日，体重当たりのBMRは21.5 ± 2.1kcal/kg体重/日であった。BMR（kcal/日）と体重（kg）には，有意な正の相関関係が認められた（図1：r = 0.58, p<0.001）。さらに，BMR（kcal/日）とLBM（kg）との間にも有意な正の相関関係が認められた（図2：r = 0.66, p<0.001）。

ステップワイズの重回帰分析の結果，BMRに対して体重の寄与率が34%であり，LBMが43%であった。体重の推定標準誤差（Standard estimated error : SEE）は91.9kcal/日，LBMは85.4kcal/日であった。

体重当たりのBMRと体重の間では有意な負の相関関係が認められた（図3：r = 0.51, p<0.001）。

さらに，%LBMの異なる3群間の比較の結果，%LBMの高値群は，低値群，中間値群と比較して，有意に体重，脂肪量が小さく，LBMには3群間で差はなか

表2 先行研究と本研究の被験者の身体的特徴と基礎代謝量の比較

		1958～1960年 先行研究[12] (n=112)		2004～2006年 本研究 (n=83)	
		Mean	SD	Mean	SD
年齢	(歳)	21.3	—	22.9	2.0
身長	(cm)	155.3	4.7	159.6	5.8**
体重	(kg)	48.9	5.6	51.9	5.8**
LBM	(kg)	39.0	4.3	39.2	4.9
体脂肪率 (%Fat)	(%)	19.9	6.6	25.2	4.0**
BMR	(kcal/日)	1,132	—	1,110	112
	(kcal/kg 体重/日)	23.1	—	21.5	2.1
	(kcal/kg LBM/日)	29.0	—	28.4	2.3
	(kcal/m² BSA/時)#	32.4	2.4	31.3	2.6*

LBM : 除脂肪量，BMR : 基礎代謝量，BSA : 体表面積
#体表面積の計算式 : BSA=体重(kg)$^{0.425}$× 身長(cm)$^{0.725}$ × 71.84
**p<0.001, *p<0.05