# Strength training increases resting metabolic rate and norepinephrine levels in healthy 50- to 65-yr-old men

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**Pratley, R., B. Nicklas, M. Rubin, J. Miller, A. Smith, M. Smith, B. Hurley, and A. Goldberg.** Strength training increases resting metabolic rate and norepinephrine levels in healthy 50- to 65-yr-old men. *J. Appl. Physiol.* 76(1): 133–137, 1994.—Resting metabolic rate (RMR) decreases with age, largely because of an age-related decline in fat-free mass (FFM). We hypothesized that a strength-training program capable of eliciting increases in FFM would also increase RMR in older individuals. To test this hypothesis, RMR, body composition, and plasma concentrations of certain hormones known to affect RMR were measured before and after a 16-wk heavy-resistance strength-training program in 13 healthy men 50–65 yr of age. Average strength levels, assessed by the three-repetition maximum test, increased 40% with training ($P < 0.001$). Body weight did not change, but body fat decreased (25.6 ± 1.5 vs. 23.7 ± 1.7%; $P < 0.001$) and FFM increased (60.6 ± 2.2 vs. 62.2 ± 2.1 kg; $P < 0.01$). RMR, measured by indirect calorimetry, increased 7.7% with strength training (6,449 ± 217 vs. 6,998 ± 226 kJ/24 h; $P < 0.01$). This increase remained significant even when RMR was expressed per kilogram of FFM. Strength training increased arterialized plasma norepinephrine levels 36% (1.1 ± 0.1 vs. 1.5 ± 0.1 nmol/l; $P < 0.01$) but did not change fasting glucose, insulin, or thyroid hormone levels. These results indicate that a heavy-resistance strength-training program increases RMR in healthy older men, perhaps by increasing FFM and sympathetic nervous system activity.

basal metabolism; exercise; body composition; weight lifting; aging; catecholamines; thyroid tests

RESTING METABOLIC RATE (RMR) decreases with age. Both cross-sectional (18, 19, 22) and longitudinal (11, 23) studies indicate that RMR declines 1–3% per decade. These studies also suggest that the decline in RMR is largely due to an age-related loss of fat-free mass (FFM) (18, 23). Whether the decline in FFM and the decrease in RMR are effects of aging per se or are secondary to other factors, such as changes in lifestyle, is not known.

A decrease in habitual physical activity levels may contribute to the decline in FFM and RMR with age. Body density (10) and RMR levels (3, 16, 17) are generally higher in individuals who exercise regularly than in nonexercising control subjects. Studies also show a direct relationship between RMR and maximal aerobic power [maximal oxygen uptake ($\dot{V}O_{2\,max}$)] (17). In addition, aerobic exercise interventions that increase $\dot{V}O_{2\,max}$ but do not affect FFM increase RMR in younger (21) and older (15) individuals. Strength training, in contrast, does not substantially change $\dot{V}O_{2\,max}$ but can lead to increases in FFM (primarily muscle mass). These effects are seen in both younger (4) and older (8, 9) individuals. Because RMR is highly correlated with FFM, strength training might be expected to increase RMR. Despite this reasoning, few studies have examined the effects of strength training on RMR, and none have included older individuals in whom RMR may be lower initially.

We hypothesized that a heavy-resistance strength-training intervention would increase FFM and thereby increase RMR in sedentary older individuals. Because aerobic exercise apparently increases RMR by mechanisms other than by increasing FFM (15), we also sought to determine the effects of strength training on hormones known to regulate RMR.

## METHODS

*Subjects.* Thirteen healthy nonsmoking men between 50 and 65 yr of age [58 ± 1 (SE) yr] volunteered for the study. None of the men participated in a regular exercise program, and all were weight stable (±2.5 kg) for at least 6 mo before enrollment. All subjects provided written informed consent according to the guidelines of Institutional Review Boards for Human Studies at the University of Maryland and Francis Scott Key Medical Center before participation.

Subjects underwent a thorough medical screening including a history and physical examination, a fasting blood profile, a graded treadmill exercise test to exhaustion, and a 2-h oral glucose tolerance test. In two subjects with mild hypertension on monotherapy (a $Ca^{2+}$-channel blocker in one individual and an angiotensin-converting enzyme inhibitor in the other), medications were discontinued for 2 wk before testing at baseline and after training.

*Dietary control.* Subjects met with a nutritionist who instructed them in a weight-maintaining diet that followed American Heart Association (AHA) recommendations (1) and were weight stabilized on this diet for at least 6 wk before testing. During the training phase, subjects maintained this diet as verified by analysis of 7-day food records (Nutritionist III, Silverton, OR), periodic 24-h diet recalls, and weekly weights.

*Measurement of body composition and $\dot{V}O_{2\,max}$.* Body density was measured by hydrostatic weighing. Body fat and FFM were calculated (5) after correction for residual lung volume determined by the closed-circuit oxygen dilution method using an Airspec model 2000 mass spectrometer (Kent, UK). $\dot{V}O_{2\,max}$ was measured during a progressive treadmill test to subjective exhaustion as previously described (12). Fractional concentrations of oxygen and carbon dioxide in the expired gases were measured using an Airspec model 2000 mass spectrometer, and gas volumes were measured using a 120-liter Tissot spirometer (Collins, Boston, MA). In all subjects, at least two of the following three criteria were met to establish that a true $\dot{V}O_{2\,max}$ had been reached: *1*) a maximal heart rate within 10 beats/min of the age-predicted maximal value (220 − age), *2*) a respiratory exchange ratio of at least 1.10, and *3*) a plateau in oxygen up-

TABLE 1. *Body composition and $\dot{V}O_{2\,max}$ before and after strength training*

| | Before Training | After Training |
|---|---|---|
| Weight, kg | 82.4±3.5 | 82.1±3.3 |
| BMI, kg/m$^2$ | 26.3±1.1 | 26.4±1.0 |
| Body fat, % | 25.6±1.5 | 23.7±1.7* |
| FFM, kg | 60.6±2.2 | 62.2±2.1* |
| $\dot{V}O_{2\,max}$ | | |
| l/min | 2.57±0.12 | 2.61±0.11 |
| ml·kg$^{-1}$·min$^{-1}$ | 30.6±1.4 | 31.2±1.4 |
| ml·kg FFM$^{-1}$·min$^{-1}$ | 42.4±1.7 | 42.0±1.6 |

Values are means ± SE. $\dot{V}O_{2\,max}$, maximal aerobic power (maximal $O_2$ uptake); BMI, body mass index; FFM, fat-free mass. * Significantly different from before training ($P < 0.01$).

take (<2 ml·kg$^{-1}$·min$^{-1}$ difference) with increasing work loads.

*Measurement of RMR.* For 3 days before metabolic testing, subjects were provided isocaloric weight-maintaining diets based on 7-day food records. These diets, which followed AHA recommendations, provided ~50% of calories as carbohydrate, 30% as fat, and 20% as protein and contained 140–150 mmol sodium/day. All testing was performed in the morning after a 12-h overnight fast. Subjects were transported to the General Clinical Research Center on the morning of testing. A dorsal hand vein was cannulated with a 20-gauge intravenous catheter kept patent with a slow (<60 ml/h) infusion of 0.9% NaCl. The hand was then placed in a warming box thermostatically controlled at 70°C to arterialize the blood. Subjects rested quietly in the supine position in a thermoneutral environment (23 ± 1°C) for at least 30 min before blood sampling and determination of metabolic rate. Duplicate blood samples 15 min apart were obtained for baseline measurements. RMR was measured for 30 min by the open-circuit dilution technique using a Sensormedics model 2900 metabolic cart (Yorba Linda, CA) calibrated before each test using standard gases of known concentrations. Energy expenditure was calculated by the Weir equation (24) and expressed per 24 h. The coefficient of variation of RMR measured under these conditions is 1.1% with an intrasubject correlation coefficient of 0.95. RMR and plasma hormone levels were measured at baseline before any exercise and 22–24 h after the last training session on completing the 16-wk strength-training intervention.

*Strength testing and training program.* Strength testing and training were performed on Keiser K-300 variable-resistance exercise machines. Subjects were familiarized with the equipment for at least four sessions before testing. Strength was assessed before and after training by the three-repetition maximum (3RM) test, which was defined as the maximal resistance that can be moved through the full range of motion three times for each exercise tested. Six major muscle groups (4 upper body and 2 lower body) were tested as previously described (12), and total body strength (total 3RM) was taken to be the sum of the upper- and lower-body exercises.

The strength-training program consisted of 14 exercises performed using machines, dumbbells, and floor exercises. All training sessions began with a 3-min warm-up period of low-intensity cycling followed by 10 min of static stretching. Resistive exercises were performed at 90% of 3RM for the first three to four repetitions, after which the resistance was gradually reduced to permit the subject to complete 15 repetitions without interrupting the exercise. This procedure required subjects to exert near-maximal effort on every repetition. Weights were checked weekly and adjusted for strength gains as necessary. The following exercises were performed: leg press, chest press, leg curl, latissimus pulldown, leg extension, military press, thigh adductor, thigh abductor, upper back, triceps, lower back, upper abdominals, biceps curl (dumbbells), and lower abdominals (floor). One set of each exercise was performed except for the leg press, leg curl, and leg extension, which were repeated at the end of the set. A rest interval of ~90 s was allowed between exercises. Each training session lasted ~1 h. Subjects exercised three times per week for 16 wk. None of the subjects dropped out of the exercise intervention, and attendance at scheduled exercise sessions was >90%.

*Analytical techniques.* Fasting plasma glucose was measured using the glucose oxidase technique (Yellow Springs Instruments, Yellow Springs, OH). Fasting insulins were measured by radioimmunoassay (26), which has an intra-assay coefficient of variation of 8%. Plasma norepinephrine and epinephrine levels were measured using the single-isotope radioenzymatic method (14) with intra-assay coefficients of variation of 7 and 9%, respectively. Thyroxine, free thyroxine, triiodothyronine, free triiodothyronine, and thyroid-stimulating hormone were measured using commercially available radioimmunoassay kits (Diagnostic Products, Los Angeles, CA) and have intra-assay coefficients of variation of 3, 5, 6, 5, and 5%, respectively. Baseline samples and samples obtained after training were run in the same assays to eliminate interassay variation. Plasma catecholamines were not determined in two subjects because of technical problems.

*Statistical analyses.* All data were analyzed with commercial statistical software packages (SAS, Cary, NC; Statview, Abacus Concepts, Berkley, CA). Mean values of the duplicate samples of plasma hormones were used in all analyses. Data were checked for normality before parametric analyses. Plasma insulin levels were log normalized to achieve a normal distribution. The effects of strength training on strength, body composition, RMR, and plasma hormone levels were tested with paired $t$ tests. Pearson correlation coefficients were calculated to test for associations among RMR, body composition, and plasma hormone levels. All values are expressed as means ± SE.

## RESULTS

*Subject characteristics.* The 16-wk strength-training intervention increased total body strength by 40% (total 3RM, 571 ± 30 vs. 801 ± 43 kg; $P < 0.001$). Strength training did not change body weight; however, mean body fat decreased 1.9% ($P < 0.001$) and FFM increased 2.6% ($P < 0.05$) after training (Table 1). There was no change in $\dot{V}O_{2\,max}$ with the intervention.

*Dietary intake.* Seven-day food records obtained after dietary stabilization before and after training indicated that subjects were compliant with the AHA recommendations (Table 2). There were no significant changes in energy intake or diet composition during the strength-training intervention. In addition, 24-h dietary recalls and 1-day food records obtained throughout the study

TABLE 2. *Diet composition before and after strength training*

| | Before Training | After Training |
|---|---|---|
| Calories, kJ/24 h | 9,699±356 | 9,950±272 |
| Carbohydrate, % | 52±1 | 52±1 |
| Fat, % | 30±1 | 30±1 |
| Protein, % | 18±1 | 18±1 |

Values are means ± SE. Distribution of macronutrients is expressed as percentage of total energy intake. There were no significant differences after training.

TABLE 3. *Resting metabolic rate before and after strength training*

| | Before Training | After Training |
|---|---|---|
| RMR | | |
| ml $O_2$/min | 225.0±8.3 | 241.8±8.2* |
| kJ/24 h | 6,499±217 | 6,998±226† |
| kJ · $kg^{-1}$ · 24 $h^{-1}$ | 78.2±2.9 | 84.5±2.9† |
| kJ · kg $FFM^{-1}$ · 24 $h^{-1}$ | 108.4±5.9 | 113.6±4.1* |
| Respiratory quotient | 0.82±0.02 | 0.83±0.03 |

Values are means ± SE. RMR, resting metabolic rate. Significantly different from before training: * $P < 0.05$; † $P < 0.01$.

period verified that the results obtained from the 7-day food records reflected habitual intake.

*RMR (Table 3).* RMR (kJ/24 h) increased in 11 of 13 subjects with strength training (Fig. 1) and was, on average, 7.7% higher ($P < 0.001$) after training. Similar changes were seen when RMR was expressed per kilogram of body weight. When RMR was expressed in kilojoules per kilogram of FFM, the increase with strength training was smaller (5.2%) but remained significant. The fasting respiratory quotient did not change significantly with strength training.

At baseline, RMR (kJ/24 h) correlated with FFM ($r = 0.62$, $P < 0.05$) but not with %body fat, $\dot{V}O_{2\,max}$, or dietary energy intake. After training, RMR was no longer significantly related to FFM because of a disproportionate increase in RMR relative to FFM (Fig. 2). Changes in RMR did not correlate with changes in FFM, %body fat, or energy intake after the intervention.

*Plasma hormones (Table 4).* Resting supine arterialized plasma norepinephrine levels increased in 8 of 11 subjects after strength training by a mean of 36% ($P < 0.01$; Fig. 3). There was a similar trend toward higher arterialized plasma epinephrine levels; however, this did not reach statistical significance. In contrast, there were no significant changes in plasma thyroid hormone levels or fasting glucose and insulin levels with strength training.

RMR did not correlate with arterialized plasma norepinephrine or epinephrine levels or with thyroid hormone levels at baseline, nor were changes in any of these variables related after strength training.

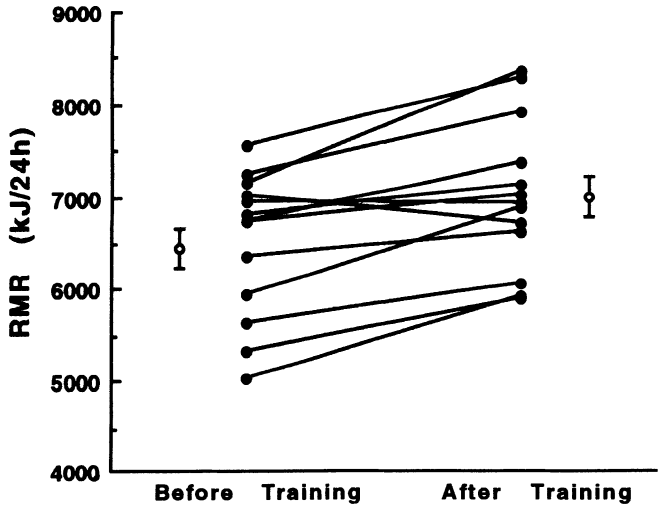


FIG. 1. Changes in resting metabolic rate (RMR) with strength training. Mean RMR increased from 6,499 ± 217 to 6,998 ± 226 kJ/24 h ($P < 0.001$).

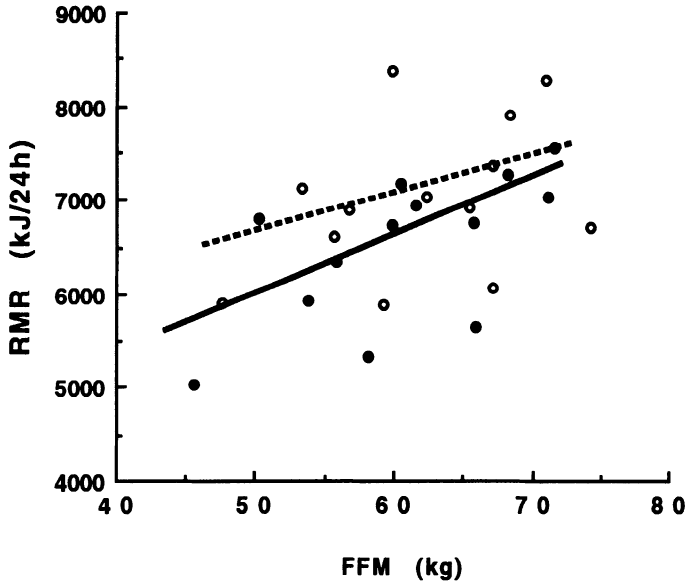


FIG. 2. Correlation between RMR and fat-free mass (FFM) before (solid circles and solid line; $r = 0.62$, $P < 0.05$) and after (open circles and dashed line; $r = 0.38$, $P$ = NS) 16 wk of strength-training intervention.

## DISCUSSION

This study demonstrates, for the first time, that strength training increases RMR in healthy older men and that this increase is accompanied by increases in FFM and plasma norepinephrine levels. These findings are consistent with the results of two recent cross-sectional studies that reported that RMR was 6.5% higher ($P < 0.05$) in 13 strength-trained young women than in 48 sedentary control subjects (3) and 13% higher ($P < 0.01$) in 18 strength-trained young men than in 42 sedentary control subjects (16). In the former study, RMR in the trained women was no longer significantly higher after adjustment for FFM, whereas in the latter study RMR remained ~5% higher ($P < 0.05$) in strength-trained men than in sedentary control men even after adjustment for FFM.

In contrast to these results, a 12-wk strength-training intervention in 13 young men produced only a 3% increase in RMR and no change in RMR adjusted for FFM

TABLE 4. *Plasma hormone and substrate levels before and after strength training*

| | Before Training | After Training |
|---|---|---|
| Norepinephrine, nmol/l | 1.1±0.1 | 1.5±0.1* |
| Epinephrine, nmol/l | 0.33±0.06 | 0.43±0.09 |
| Total $T_4$, nmol/l | 86.1±4.4 | 87.9±5.3 |
| Free $T_4$, nmol/l | 14.1±0.8 | 14.0±0.5 |
| Total $T_3$, nmol/l | 1.5±0.1 | 1.5±0.1 |
| Free $T_3$, pmol/l | 3.3±0.3 | 3.2±0.2 |
| TSH, mU/l | 1.6±0.4 | 1.6±0.2 |
| Glucose, mmol/l | 5.3±0.2 | 5.1±0.1 |
| Insulin, pmol/l | 64.8±9.6 | 58.8±8.4 |

Values are means ± SE. $T_4$, thyroxine; $T_3$, triiodothyronine; TSH, thyroid-stimulating hormone. * Significantly different from before training ($P < 0.01$).

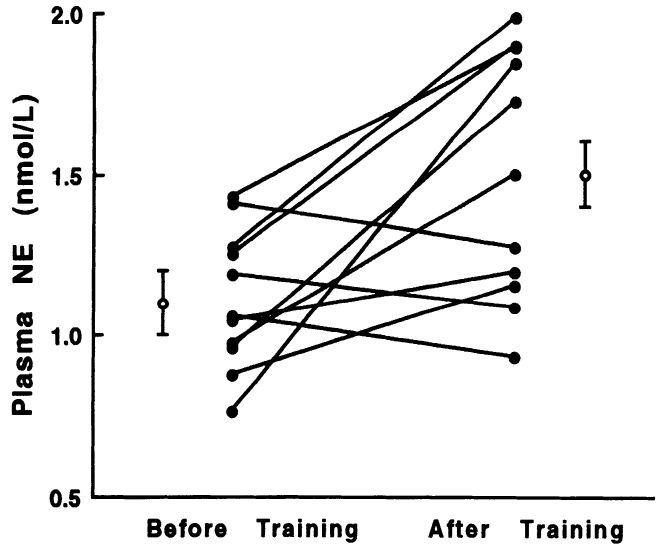


FIG. 3. Changes in arterialized plasma norepinephrine (NE) levels with strength training. Mean plasma NE levels increased from 1.1 ± 0.1 to 1.5 ± 0.1 nmol/l ($P = 0.01$).

(4). This disparity may reflect differences in the subjects selected and the design of the study. Sedentary older individuals who start with lower initial RMR values may have a more pronounced increase in RMR with training than do younger individuals. It also is possible that there are differences between younger and older individuals in the time course of adaptations to strength training. Although we were able to demonstrate increases in RMR after 16 wk of strength training in older individuals, it may be necessary for younger individuals to train for a longer period. The cross-sectional investigations that demonstrated a higher RMR in strength-trained younger individuals studied subjects who had trained for periods >2 yr before testing.

In this study, RMR was measured 22–24 h after the last training session, whereas Broeder et al. (4) measured RMR 48 h after the last exercise session; however, it is unlikely that a difference in the timing of the measurements accounts for the different results. Preliminary studies in our laboratory indicate that oxygen uptake returns to baseline within 3 h of an acute bout of resistive exercise. Furthermore, in the two cross-sectional studies that demonstrated higher RMR in strength-trained young individuals, RMR was measured 36–48 h after the last exercise session (3, 16).

Subjects in the present study were weight stable and were provided metabolic diets to ensure dietary compliance before measurement of RMR. This may have enhanced our ability to detect a change in RMR with training. In contrast, the younger individuals studied by Broeder et al. (4) may have been in a slightly negative caloric balance, as determined by food records, that may have blunted the increase in RMR with strength training. Further evidence of the importance of caloric balance comes from a recent study of strength training combined with a very-low-calorie diet in women that also failed to demonstrate a significant effect of strength training on RMR (6).

The increase in RMR with strength training in this study may be due, in part, to the increase in FFM; however, this is unlikely to be the only explanation for the following reasons. First, the average RMR of muscle is estimated to be 73.7 $kJ \cdot kg^{-1} \cdot 24\ h^{-1}$ (2). Assuming that the 1.6-kg increase in FFM observed in this study was entirely muscle, this would account for an increase of ~118 kJ/24 h or only ~24% of the observed increase of 499 kJ/24 h in RMR. Second, the increase in FFM did not correlate with the increase in RMR in these subjects. Third, RMR values after training were generally above, rather than on, the regression line relating baseline RMR to FFM, indicating that the increase in RMR with strength training is disproportionate to the increase in FFM. Finally, RMR was significantly higher after strength training even after normalization for FFM. Thus, mechanisms other than the increase in FFM may be important determinants of the increase in RMR with strength training.

Similar results have been observed with aerobic exercise training in older individuals. An 8-wk endurance-training program increased RMR ~10% but did not change FFM (15). Moreover, the 24% increase in arterialized norepinephrine levels reported with aerobic exercise training in this study (15) is similar to the 36% increase in resting arterialized plasma norepinephrine levels we found with strength training. Collectively, these data indicate that both strength training and aerobic exercise can increase RMR in older individuals and may do so by increasing basal sympathetic nervous system activity. In younger individuals, RMR was higher in endurance-trained subjects than in sedentary control subjects, and oral administration of propranolol, a nonselective $\beta$-adrenergic blocker, decreased RMR in the trained subjects but not in the control subjects (20). These results suggest that the effects of exercise to increase RMR are not limited to older individuals and, furthermore, may be mediated through specific $\beta$-adrenergic mechanisms. Notably, plasma norepinephrine levels were not higher in the young endurance-trained individuals than in sedentary control subjects. Poehlman et al. (17) also found no difference in plasma norepinephrine levels between young endurance-trained individuals and sedentary control subjects. Other studies have failed to show an effect of endurance training on resting plasma norepinephrine levels in both young (13) and older (7) individuals. These disparate results may reflect differences between younger and older individuals in the adaptive response of the sympathetic nervous system to exercise. They may also be due, in part, to methodological differences between studies. Studies in which venous, rather than arterialized, plasma norepinephrine levels were measured have tended not to show an effect of exercise (7, 13, 20). These studies also did not control dietary intake to the same degree as the present study did; thus, the effect of changes in diet may have obscured an effect of exercise on plasma norepinephrine levels.

Although not addressed in the present study, strength training could increase RMR also by increasing muscle protein turnover. A 12-wk strength-training program increased urinary 3-methyl-L-histidine by an average of 40% in older men, indicative of an increase in myofibrillar protein turnover (9). Because the energy costs of protein turnover may account for as much as 20% of RMR

(25), a similar increase in protein turnover stimulated by strength training could quantitatively account for the observed increment in RMR in this study.

Further studies that examine the effects of strength training on RMR, endocrine-metabolic function, and protein turnover as well as those that address the duration and intensity of strength training necessary to effect these changes are indicated for older individuals. Results of these studies not only will provide insight into the physiological benefits of strength training but may also improve our understanding of the aging process and lead to the development of interventions that could ameliorate some of the functional and metabolic declines observed with aging.

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