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

R. PRATLEY, B. NICKLAS, M. RUBIN, J. MILLER, A. SMITH, M. SMITH, B. HURLEY, AND A. GOLDBERG

Division of Gerontology, Department of Medicine, University of Maryland at Baltimore, Baltimore Veterans Affairs Medical Center, Baltimore 21201; and Departments of Kinesiology and Human Nutrition and Food Systems, University of Maryland, College Park, Maryland 20742

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 (23.6 ± 1.6 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.

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 Ca2+-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 VO2max. 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). VO2max 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 VO2max 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 $\bar{V}O_2\text{max}$ before and after strength training

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Before Training</th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, kg</td>
<td>82.4±3.5</td>
<td>82.1±3.3</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.3±1.1</td>
<td>26.4±1.0</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>25.6±1.5</td>
<td>23.7±1.7*</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>60.6±2.2</td>
<td>62.2±2.1*</td>
</tr>
<tr>
<td>$\bar{V}O_2\text{max}$, l/min</td>
<td>2.57±0.12</td>
<td>2.61±0.11</td>
</tr>
<tr>
<td>ml·kg$^{-1}$·min$^{-1}$</td>
<td>30.6±1.4</td>
<td>31.2±1.4</td>
</tr>
<tr>
<td>ml·kg FFM$^{-1}$·min$^{-1}$</td>
<td>42.4±1.7</td>
<td>42.0±1.6</td>
</tr>
</tbody>
</table>

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

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 $\bar{V}O_2\text{max}$ 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

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Before Training</th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calories, kcal/24 h</td>
<td>9,689±356</td>
<td>9,950±272</td>
</tr>
<tr>
<td>Carbohydrate, %</td>
<td>52±1</td>
<td>52±1</td>
</tr>
<tr>
<td>Fat, %</td>
<td>30±1</td>
<td>30±1</td>
</tr>
<tr>
<td>Protein, %</td>
<td>18±1</td>
<td>18±1</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th></th>
<th>Before Training</th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMR (ml O₂/min)</td>
<td>225.0±8.3</td>
<td>241.8±8.2*</td>
</tr>
<tr>
<td>RMR (kJ/24 h)</td>
<td>6,499±217</td>
<td>6,998±220†</td>
</tr>
<tr>
<td>RMR (kJ·kg⁻¹·24 h⁻¹)</td>
<td>78.2±2.9</td>
<td>84.5±2.9†</td>
</tr>
<tr>
<td>RMR (kJ·kg FFM⁻¹·24 h⁻¹)</td>
<td>108.4±5.9</td>
<td>113.6±4.1*</td>
</tr>
<tr>
<td>Respiratory quotient</td>
<td>0.82±0.02</td>
<td>0.83±0.03</td>
</tr>
</tbody>
</table>

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, VO₂ 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.

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

<table>
<thead>
<tr>
<th></th>
<th>Before Training</th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norepinephrine, nmol/l</td>
<td>1.1±0.1</td>
<td>1.5±0.1*</td>
</tr>
<tr>
<td>Epinephrine, nmol/l</td>
<td>0.33±0.06</td>
<td>0.43±0.09</td>
</tr>
<tr>
<td>Total T₄, nmol/l</td>
<td>86.1±4.4</td>
<td>87.9±5.3</td>
</tr>
<tr>
<td>Free T₄, nmol/l</td>
<td>14.1±0.8</td>
<td>14.0±0.5</td>
</tr>
<tr>
<td>Total T₃, nmol/l</td>
<td>1.5±0.1</td>
<td>1.5±0.1</td>
</tr>
<tr>
<td>Free T₃, pmol/l</td>
<td>3.3±0.3</td>
<td>3.2±0.2</td>
</tr>
<tr>
<td>TSH, mU/l</td>
<td>1.6±0.4</td>
<td>1.6±0.2</td>
</tr>
<tr>
<td>Glucose, mmol/l</td>
<td>5.3±0.2</td>
<td>5.1±0.1</td>
</tr>
<tr>
<td>Insulin, pmol/l</td>
<td>64.8±9.6</td>
<td>58.6±8.4</td>
</tr>
</tbody>
</table>

Values are means ± SE. T₄, thyroid; T₃, triiodothyronine; TSH, thyroid-stimulating hormone. * Significantly different from before training (P < 0.01).
combined with a very-low-calorie diet in women that also
training on RMR (6).
study may be due, in part, to the increase in FFM; how-
failed to demonstrate a significant effect of strength
training. Further evidence of the importance of caloric bal-
caloric balance, as determined by food records, that may
have blunted the increase in RMR with strength train-
ing. In contrast, the younger individuals studied by
Broeder et al. (4) demonstrated higher RMR in strength-trained
individuals 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 measure-
ments accounts for the different results. Preliminary
studies in our laboratory indicate that oxygen uptake re-
turns to baseline within 3 h of an acute bout of resistive
exercise. Furthermore, in the two cross-sectional studies
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 measure-
ments reflects differences in the subjects selected and the design of the study. Sedentary older indi-
viduals 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. Al-
though 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.
Subjects in the present study were weight stable and
were provided metabolic diets to ensure dietary compli-
ance before measurement of RMR. This may have en-
hanced our ability to detect a change in RMR with train-
ing. 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 train-
ing. Further evidence of the importance of caloric bal-
ance comes from a recent study of strength training com-
bined 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; how-
ever, 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·kg⁻¹·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 exer-
cise 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 in-
dicate 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
β-adrenergic blocker, decreased RMR in the trained sub-
jects 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 β-adrenergic mechanisms. No-
tably, plasma norepinephrine levels were not higher in
the young endurance-trained individuals than in seden-
tary control subjects. Poehlman et al. (17) also found no
difference in plasma norepinephrine levels between
young endurance-trained individuals and sedentary con-
tral 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 be-
 tween studies. Studies in which venous, rather than ar-
terialized, 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 in-
creased urinary 3-methyl-L-histidine by an average of
40% in older men, indicative of an increase in myofibril-
lar protein turnover (9). Because the energy costs of pro-
tein 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.

The authors thank K. Vaitkevicius, J. Hagberg, K. H. Koffler, A. Menkes, R. A. Redmond, R. Rogus, and the staffs of the General Clinical Research Center and the Johns Hopkins Academic Nursing Home project for invaluable assistance.

This research was supported by National Institute on Aging Clinical Investigator Award K08-AG-00494 to R. Pratley, the Johns Hopkins Academic Nursing Home Awards PO1-AG-04402 and RO1-AG-07660 to A. Goldberg, and the General Clinical Research Center and the Johns Hopkins Academic Nursing Home Diabetes and Nutrition Section, NIDDK/NIH, 4212 North 16th St., Washington, DC 20205.

Investigator Award K08AG-00494 to R. Pratley, the Johns Hopkins Academic Nursing Home Awards PO1-AG-04402 and RO1-AG-07660 to A. Goldberg, and the General Clinical Research Center at Francis Scott Key Medical Center M01-RR-02719.

Present address and address for reprint requests: R. Pratley, Clinical Diabetes and Nutrition Section, NIDDK/NIH, 4212 North 16th St., Phoenix, AZ 85016.

Received 12 May 1993; accepted in final form 17 August 1993.

REFERENCES