A Single Bout of Concentric Resistance Exercise Increases Basal Metabolic Rate 48 Hours After Exercise in Healthy 59–77-year-old Men

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Background. It has been shown that basal metabolic rate (BMR) decreases with age. The extent to which some of the decrease can be reversed by exercise in older men and women is unclear. Resistance exercise has been shown to significantly increase muscle mass in older individuals, and because muscle is a highly active metabolic tissue there is potential to increase BMR as a secondary outcome to the training adaptation.

Methods. Twelve healthy men aged 59–77 years performed single-leg knee extension exercise (right and left leg) and bench press lifts (16 sets, 10 reps/set with timed recovery between sets) at 75% of the individual’s 3RM. Subjects only performed the concentric phase of the lift. BMR was measured on two separate occasions, once after a nonexercise control period and again 48 hrs after a bout of resistance exercise.

Results. BMR was significantly increased (p < .006) 48 hrs after exercise (EX) compared to control (CON) (284.0 ± 34.0 vs 274.9 ± 34.0 kJ/hr, respectively). Calculated over a 24-hour period, the energy expenditure corresponded to 1570 ± 193 and 1627 ± 193 kcal/24 hr (p < .0002) for the CON and EX measures, respectively. VO₂ (L/min) was higher (p < .0002) 48 hrs after the EX bout compared to 48 hrs post-CON (0.232 ± 0.03 vs 0.225 ± 0.03 L/min, respectively).

Conclusion. We conclude that in healthy 59–77-year-old men, an acute bout of resistance exercise causes a sustained increase in BMR that persists for up to 48 hours after exercise.

Basal metabolic rate (BMR) decreases with increasing age (1–4). Aging is also associated with changes in body composition, specifically a reduction in muscle mass, which may contribute to the decrease in BMR (3). Resistance exercise provides a powerful stimulus to increase muscle protein turnover and synthesis, up to 109% at 24 hrs and 14% at 36 hrs after exercise (5). Because skeletal muscle is more metabolically active than fat, an increase in protein turnover resulting from resistance exercise may lead to a gain in muscle mass, attenuating some of the age-associated changes in BMR. Resistance exercise training has previously been shown to significantly increase muscle mass in elderly subjects (6–8). Exercise, both resistance and aerobic, has been used to reverse many of the negative physiological and metabolic outcomes related to aging. Cross-sectional and longitudinal studies suggest that both young and older subjects who participate in exercise training programs have higher resting metabolic rates when compared to inactive subjects (9,10). The increase in resting metabolic rate appears to be mediated to some extent by increased sympathetic nervous system activity (11–13). With resistance exercise training it is more likely that the increase in muscle protein turnover is a primary factor in elevating BMR. However, the extent to which an increase in BMR is due to some long-term physiological adaptation, or to the residual effects of the last bout of exercise, remains unclear.

The measurement of metabolic rate may be affected by age, exercise type, intensity (14–16), and/or control of BMR collection (17). The acute effects of resistance exercise on metabolic rate have been reported predominantly in a younger, trained population, and the results suggest there may be an increase in RMR for up to 15 hours post-exercise (4,18). There are no data to demonstrate the effects of an acute bout of resistance exercise on BMR in older healthy men. The purpose of the present study was to measure BMR under very controlled conditions 48 hr after a single bout of concentric resistance exercise in a group of healthy 59–77-year-old men.

METHODS

Subjects. — Twelve older, healthy male subjects (nonmedicated, nonhypertensive, nonhyperlipidemic, free of chronic infection or disease, nonsmokers) volunteered to participate in the study. The subjects had not participated in an exercise training program for at least one year. Each subject was informed of all procedures before written consent was obtained. The protocol and consent form were approved by the Internal Review Board of The Pennsylvania State University in accordance with the university guidelines for protection of human subjects.

Prescreening procedures. — Volunteers were admitted to the study after satisfying the criteria of a medical exam, a complete blood and urine chemistry, a 75 gm oral glucose
tolerance test (OGTT), resting electrocardiograph (ECG), and a resistance exercise stress test (RST). All subjects had a normal response to the OGTT by National Diabetes Data Group criteria (19). The RST was used to determine the individual's three repetition maximum (3RM) by graded design.

**Body composition.** — Body density was measured by hydrostatic weighing following the procedures of Akers and Buskirk (20). Percent body fat was calculated using the Siri equation (21). Residual lung volume was measured by open-circuit nitrogen washout technique, while the subjects were submerged under water.

**Resistance exercise.** — The exercise bout included 16 sets of 10 repetitions at 75% of the individual 3RM. The subjects performed the concentric phase of the lift, allowing only positive work on the bench press and each leg individually during the leg extension. Once the concentric phase of the lift was complete, the test supervisors lowered the weight to minimize any eccentric work that might be performed during the exercise bout. After the completion of each set, a predetermined timed recovery (30-sec, 30-sec, 1-min, 30-sec, 30-sec, 2-min, 30-sec, 30-sec, 1-min, 30-sec, 30-sec, 2-min, and 30-sec, 30-sec, 1-min) ensued. During the resistance bout, 75% of the individual's 3RM was maintained for as long as the subject could complete a set of 10 reps. In the event a subject could not lift the prescribed weight, the workload was decreased by 2.3 kg for the leg extension and 4.5 kg for the bench press, so that the subjects were able to complete the bout. The control (CON) and exercise (EX) trials were randomized and separated by at least one week.

**Diet and physical activity.** — Subjects resided in the General Clinical Research Center (GCRC) for a three-day familiarity period during the exercise and control trials, so as not to cause undue stress to the subjects the morning of the BMR collection (22). The subjects were provided a eucaloric diet (60% carbohydrate, 15% protein, 25% fat) to maintain energy balance. The diet was devoid of caffeine and alcohol. The total calories provided were based on height, weight, age, and activity level according to the Harris-Benedict equation (23). The diet consisted of normal foods and beverages and a supplemental formula beverage (Ensure, Ross Labs, Columbus, OH). During the three-day stay, exercise was monitored and daily activity was maintained within the subject's customary habits.

**Measurement of metabolic rate.** — On the test morning of each BMR measurement, the subjects were awakened, transported by wheelchair to void and to be weighed, and then reclined in a semi-darkened, thermoneutral (22 ± 1 °C) environment under a flow-thru plexiglas hood (Brooks Instruments, Hatfield, PA) for 30 minutes. Air was pulled through the hood at a rate of 50 L/min to maintain a slight negative pressure, allowing fresh air movement through the hood at all times. A continuous open-circuit expired gas collection was analyzed by Hartmann-Braun (Frankfurt, Germany) differential paramagnetic O2 (Magnos 4 G) and nondispersive infrared CO2 (Uras 4) analyzers. The analyzers were calibrated prior to the collection with known gas concentrations. Energy expenditure (kcal/min) was calculated using the Weir (24) equation, and substrate oxidation rates were calculated using 24 hr urinary urea nitrogen determinations to account for protein oxidation (25).

**Statistical analysis.** — Values were expressed as mean ± SE. A paired t-test analysis was used to test for differences in BMR between the control (no exercise) and the exercise trial. The level for statistical significance was set at p < .01.

**RESULTS**

Twelve 59–77-year-old men with normal glucose tolerance participated in the study. Subject characteristics are provided in Table 1. The average weight lifted during each knee extension exercise was 18 ± 4 kg for both the right and left leg, individually. An average of 36 ± 7 kg was lifted during the bench press. All subjects demonstrated an increase in BMR after the EX trial (Figure 1). When energy expenditure was calculated over a 24-hr period, the EX trial resulted in a 57 kcal/24 hr greater (p < .0002) expenditure (1570 ± 193 and 1627 ± 193 kcal/24 hr for the CON and EX measures, respectively. Oxygen consumption was significantly higher after EX compared to CON (Table 2); the difference corresponded to a 3% increase in VO2. There were no differences between trials for measurements of respiratory exchange ratio (RER), rates of carbohydrate oxidation, or fat oxidation (Table 2).

**DISCUSSION**

The principal finding of this study was that an acute bout of concentric resistance exercise caused an ~3% increase in BMR among a group of healthy 59–77-yr-old men 48 hr after the exercise bout. Previous investigations that have addressed the effects of resistance exercise on metabolic rate were completed as either cross-sectional studies looking at differences between trained and untrained young and middle-aged subjects (4,9,16,18), or examined the effects of 12–16-week resistance training programs (13,14,26). Some resistance training studies have found a 6–8% increase in RMR post-exercise (13,26), while others found no difference in RMR among young men after 16 weeks of training (14). However, these studies are not directly comparable to the present data, both because of the training effects, and because the studies measured RMR 20-min post-prandial (26) and 22–24 hr after the last bout of exercise (13). Nevertheless, based on our observations, it would appear that up to 50% of the increase in metabolic rate attributed to resistance training may be due to the residual
effects of the last exercise bout, at least among older healthy men.

Many factors could have contributed to the increase in BMR following exercise, including increased protein flux (27), glycogen repletion (28), increased cytokine activity (29), elevated sympathetic activity (11-13), and increased substrate cycling (30). In addition, greater oxygen consumption after exercise may be related to the intensity and duration of the exercise bout. Bahr et al. (15) and Gore and Withers (16) have shown that excess post-exercise oxygen consumption (EPOC) increases with longer duration exercise. In the present study the duration of the exercise session was ~90 min, which has been shown to be sufficient to deplete glycogen stores in concentrically exercised muscle (31). However, assuming adequate carbohydrate intake (60% of diet; refer to Methods), muscle glycogen would have been replenished and normalized within 48 hr after exercise (28). Indirectly, muscle glycogen depletion could increase BMR by shifting substrate use to lipids, thus increasing lipid oxidation. However, we did not observe a decrease in RER, or an increase in fat oxidation 48 hr after exercise (Table 2).

Subjects were only allowed to perform the concentric phase of the lift, in order to reduce the likelihood of skeletal muscle damage and consequent ultrastructural disruption of the myofibrils associated with eccentric contractions (32).

Table 2. Oxygen Consumption, Substrate Oxidation Rates and Respiratory Exchange Ratios at Rest, Measured 48 hrs Post-exercise Compared to a Nonexercise Control

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Exercise</th>
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<tbody>
<tr>
<td>VO₂ (L/min)</td>
<td>0.225 ± 0.3</td>
<td>0.232 ± 0.3*</td>
</tr>
<tr>
<td>Oxidation rates</td>
<td></td>
<td></td>
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<tr>
<td>CHO (g/min)</td>
<td>2.03 ± 0.8</td>
<td>2.25 ± 0.5</td>
</tr>
<tr>
<td>Fat (g/min)</td>
<td>0.65 ± 0.3</td>
<td>0.60 ± 0.1</td>
</tr>
<tr>
<td>Respiratory exchange</td>
<td>0.87 ± 0.05</td>
<td>0.87 ± 0.05</td>
</tr>
<tr>
<td>ratio (RER)</td>
<td></td>
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</tr>
</tbody>
</table>

Notes: Values are means ± SE; N = 12 men. VO₂, oxygen consumption; CHO, carbohydrate.
*Significantly higher than Control, p < .0002.

Eccentric work is also associated with a host of cytokine and other biochemical responses that may influence BMR (29). While concentric contractions have also been shown to induce endogenous pyrogen activity following cycling exercise, there is some uncertainty concerning the direct physiological effects of cytokines on BMR (29).

It has previously been shown that metabolic rate may be increased under conditions of increased protein turnover (27). However, the effects of resistance exercise, whether acute or chronic, on protein turnover and synthesis are unclear (27,33). Yarasheski et al. (33) have shown an increase in the rate of muscle protein synthesis among older subjects after 2 weeks of resistance exercise training. MacDougall et al. (5) likewise reported increases in muscle protein synthesis after an acute bout of resistance exercise, both at 24 and 36 hrs post-exercise. Thus, changes in protein synthesis rates may be relatively rapid and may be manifest in the early stages of a resistance training program. If the increase were to begin with the first bout of exercise, it could help explain the increase in BMR observed in our subjects.

An increase in sympathetic nervous system activity has also been shown to increase metabolic rate (12). Several investigators have now shown that both resistance and aerobic exercise training-related increases in metabolic rate occur in the presence of increased norepinephrine concentrations (11-13). However, older subjects do not consistently show an increased sympathetic response to exercise training (34). Since catecholamines were not measured in this study, we cannot comment directly on the contribution that increased β-adrenergic activity may have provided to the increase in BMR. However, it is likely that the contribution was small in light of the reports which show that catecholamine levels return close to resting levels within 2-4 hr after a single bout of exercise (35).

In conclusion, we have shown that in a group of healthy men aged 59–77 yr, an acute bout of resistance exercise results in a sustained increase in BMR, that persisted for up to 48 hr after the exercise had been completed. These findings suggest that an acute bout of concentric resistance exercise may attenuate the age-associated decrease in BMR and may also help regulate energy balance by increasing caloric expenditure for many hours after the exercise session is completed.
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