Effects of endurance training on total fat oxidation in elderly persons

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Poehlman, Eric T., Andrew W. Gardner, Paul J. Arciero, Michael I. Goran, and Jorge Calles-Escandon. Effects of endurance training on total fat oxidation in elderly persons. J. Appl. Physiol. 76(6): 2281-2287, 1994.—We examined the influence of 8 wk of endurance training on basal levels of fat oxidation and its association with changes in norepinephrine (NE) kinetics, resting metabolic rate (RMR), and body composition in 18 healthy elderly persons (66.1 ± 1.4 yr; 10 men, 8 women). Fatty acid appearance rate and total body fat oxidation were determined from \(^{14}C\)palmitate infusion and indirect calorimetry. NE kinetics were determined from infusions of \(^{3}H\)NE, RMR was determined from the ventilated hood technique, and body composition was determined from underwater weighing. Endurance training increased peak oxygen consumption by 11% (1.9 ± 0.1 to 2.1 ± 0.1 l/min; \(P < 0.01\)) and increased RMR by 7% (1.20 ± 0.02 to 1.28 ± 0.02 kcal/min; \(P < 0.01\)). Endurance training increased NE appearance rate by 35% (0.51 ± 0.04 to 0.69 ± 0.04 \(\mu g/min; P < 0.01\)), whereas no change in NE clearance was noted. Endurance training increased fat oxidation by 22% (201.0 ± 11.2 vs. 244.0 ± 15.2 \(\mu mol/min; P < 0.01\)) but did not alter fatty acid appearance rate. Approximately two-thirds of the variation (\(r^2 = 0.65\)) for the increase in fat oxidation was explained by increased NE appearance rate (\(r^2 = 0.51; P < 0.01\)) and changes in fat-free weight (\(r^2 = 0.14; P < 0.01\)). We conclude that 1) endurance training shifts in vivo basal substrate utilization toward greater fat oxidation in elderly individuals and 2) enhanced fat oxidation is associated with increased activity of the sympathetic nervous system and alterations in fat-free mass.

exercise; sympathetic nervous system

FATTY ACID MOBILIZATION and oxidation from adipose tissue triglyceride stores is generally considered to predominate as a source of fuel in working muscles, particularly during long-term exercise (9). However, it is unclear as to which in vivo factors are involved in the regulation of fat oxidation in response to endurance training in the elderly. Although sympathetic nervous system activity probably plays a regulatory role in fat mobilization and utilization in adult humans (1), the metabolic link between changes in fat oxidation and sympathetic nervous system activity in response to an exercise intervention paradigm in the elderly has not been examined. A reduced capacity to oxidize stored lipid has been found in the elderly in response to short-term fasting and catecholamine stimulation (21, 22). It is possible that this may partially contribute to the increase in adiposity with advancing age. Thus, clinical interventions, such as endurance training, which may serve to enhance lipid oxidation, could have long-term benefits in the regulation of body composition and the restoration of energy balance in elderly persons.

The majority of knowledge, however, regarding the effects of endurance training on fat metabolism has been derived from in vitro studies or from measurements of circulating concentrations of substrates considered to be representative of lipolytic action. It has been shown that endurance training increases the lipolytic response to catecholamines in isolated adipocytes from experimental animals and human subjects (3, 5–7), although this finding is not unanimous among investigators (31). It is unknown, however, as to the effects of chronic endurance exercise on in vivo fatty acid availability and fat oxidation in elderly persons. At present, discrepant results have been found regarding the effects of endurance training on resting sympathetic nervous system activity (18, 26, 27). Because alterations in sympathetic nervous system are involved in the regulation of patterns of fat mobilization and utilization, we found it of interest to examine the influence of endurance training on in vivo changes in total body fat oxidation and its relationship with changes in sympathetic nervous system activity and metabolic rate in elderly persons.

Thus, the first objective in the present study was to examine the effects of endurance training on in vivo basal rates of fatty acid availability and whole body fat oxidation in previously sedentary elderly persons. Our second objective was to examine the metabolic determinants of the changes in total fat oxidation by examining its association with changes in norepinephrine (NE) appearance rate (NE\(_{\text{app}}\)), resting metabolic rate (RMR), and body composition.

MATERIALS AND METHODS

Subjects. Eighteen older individuals (10 males, 8 females) in excellent general health participated in these studies. Criteria for subject selection were 1) no clinical symptoms or signs of heart disease, 2) resting blood pressure of <140/90, 3) a normal electrocardiogram at rest and during an exercise stress test, 4) absence of any prescription or over-the-counter medication that could affect cardiovascular function or lipolysis, 5) no family medical history of diabetes, and 6) weight stability (±2 kg) by medical history within the past year. At the time of the study, no individual was participating in a formal exercise program. The energy expenditure in leisure time physical activity by questionnaire (38) was estimated at 334 ± 61 kcal/day, indicating that subjects were physically active but were not involved in formal exercise programs. The nature, purpose, and possible risks of the study were carefully explained to each subject before they gave consent to participate. The experimental protocol was approved by the Committee on Human Research for the Medical Sciences.

Endurance training program. The supervised endurance training program consisted of cycling exercise three times per
week for 8 wk. The training sessions consisted of 10 min of flexibility exercises followed by cycling at an individually prescribed duration and intensity to expend a given amount of calories. Cycling exercise was chosen to reduce the chance of musculoskeletal injury and to strictly control exercise intensity and duration. Exercise prescriptions were derived with the net energy expenditure (total cost of exercise - RMR) during each training session. Net energy expenditure during training was estimated from the linear relationship between heart rate and oxygen consumption (V\text{O}_{2}) derived during a peak V\text{O}_{2} test preceding the exercise program, as previously described (10). The exercise program was closely supervised by an exercise physiologist and two research assistants, who monitored heart rate every 5 min to ensure adherence to the exercise prescription. Volunteers were frequently provided feedback and encouragement to maintain exercise time and duration. All volunteers began the 1st wk with an exercise prescription designed to generate a net energy expenditure of 150 kcal at 60% of peak V\text{O}_{2} three times per week. During week 2, the net energy expenditure was increased to 200 kcal/session at 60% of peak V\text{O}_{2}.

During week 3, the intensity of the exercise program was increased to 65% of peak V\text{O}_{2} and the net exercise expenditure was maintained at 200 kcal/session. Volunteers exercised at 70% of their peak V\text{O}_{2} during weeks 4 and 5, and the net energy expenditure was increased to 250 kcal/session. During weeks 6–8, the exercise intensity was increased to 75% of peak V\text{O}_{2} and volunteers expended 250 kcal/session during week 6 and 300 kcal/session during weeks 7 and 8. Exercise prescriptions were adjusted during the 4th wk of the program to take into account increases in peak V\text{O}_{2} to maintain an appropriate exercise intensity. The goal of the exercise program was to increase cardiovascular fitness without having large changes in body weight and composition, which may tend to confound the exercise effect on RMR and NE kinetics (25).

**Timing of metabolic measurements.** A detailed description of indirect calorimetry, body composition, peak V\text{O}_{2}, and NE kinetic measurements and their reproducibility in our laboratory have been previously reported (29, 30). All measurements were performed ~36 h after the last exercise session, since this time period has been shown to eliminate the residual effects of the last exercise bout on RMR and plasma NE (28). Volunteers were admitted to the Clinical Research Center the afternoon before their metabolic testing at 1600 h. Volunteers were fed a standardized meal (1,000 kcal; 55% carbohydrates, 30% fat, and 15% protein) at 1730 h. All volunteers followed an identical test sequence in which RMR was measured after an overnight fast, followed by determination of NE kinetics, lipid kinetics, plasma hormones, and substrate concentrations. Thereafter, body composition and peak V\text{O}_{2} were determined.

**Metabolic measurements.** RMR was measured using a ventilated hood technique after an overnight fast in which volunteers slept in the Clinical Research Center. An overnight timed urine collection was used to estimate the rate of urinary nitrogen excretion. Substrate oxidation rates were calculated with use of stoichiometric equations (2). Body fat was estimated from body density before and after exercise training by underwater weighing with simultaneous measurement of residual lung volume by helium dilution using the Siri equation (35). Fat-free mass (FFM) was estimated as total body mass − fat mass. Peak V\text{O}_{2} was assessed by a progressive and continuous test to exhaustion on a cycle ergometer using an open-circuit gas analysis system.

**Measurement of NE kinetics.** Volunteers were admitted to the Clinical Research Center the day before infusion studies were performed. The subjects were prohibited from the use of known modulators of catecholamine release and metabolism, including nicotine and caffeine, for 12 h before the studies. All studies were performed after an overnight fast with the subjects strictly recumbent. Volunteers were awakened at 0700 h, and an intravenous line was placed in a forearm vein for tracer infusion. A second intravenous line was placed in a dorsal vein of the opposite hand, which was placed in a warming box and used for blood sampling. NE kinetics (NE\text{app} and NE clearance rate) were performed under steady-state conditions using a modification of the tritiated isotope dilution method of EULER et al. (8) and as previously described in our laboratory (29). [\text{H}]NE was infused continuously for 60 min at 0.71 µCi/min. Arterialized blood samples were drawn at 50, 55, and 60 min of the infusion for determination of plasma NE and calculation of plasma NE\text{app} and NE clearance rate. The assumptions and limitations underlying the isotope dilution method for calculation of NE kinetics have been previously described (8).

**Analytic methods.** Arterialized plasma catecholamine samples were drawn into iced containers containing EDTA and glutathione, quickly spun at 4°C, separated, and stored at −80°C until they were assayed. Plasma NE was extracted with alumina and then eluted with 0.1 M perchloric acid. An internal standard, 3,4-dihydroxybenzylamine, was added to plasma and NE before extraction (Bioanalytical Systems, West Lafayette, IN). NE concentrations were obtained by high-performance liquid chromatography (HPLC) with electrochemical detection (Bioanalytical Systems). Aliquots of the extracts were counted for radioactivity of [\text{H}]NE (Triarc 3000, Packard Instruments, Downers Grove, IL).

Steady-state concentrations of NE were found before (211 ± 61, 207 ± 55, and 218 ± 56 pg/ml) and after (257 ± 54, 244 ± 52, and 245 ± 50 pg/ml) endurance training during the infusion at 50, 55, and 60 min, respectively. Similarly, steady-state concentrations were achieved for tracer concentrations before [186 ± 66, 195 ± 75, and 195 ± 71 counts per minute (cpm)/ml] and after [163 ± 35, 168 ± 34, and 171 ± 36 cpm/ml] endurance training, as measured during the infusion at 50, 55, and 60 min, respectively. NE plasma clearance rates (in l/min) were calculated as infusion rate (in cpm/min) divided by plasma (in cpm/ml) (mean of 3 samples corrected for extraction recovery). NE\text{app} (in µg/ml) was calculated as clearance (in l/min) × plasma NE concentration (in µg/l). Subjects were instructed not to restrict sodium intake 1 wk before performing the metabolic studies and during the exercise program. No differences were noted for sodium concentrations before (140 ± 6 meq/l) and after (138 ± 8 meq/l) training nor in dietary sodium intake before (2,862 ± 82 meq/day) and after endurance training (3,125 ± 310 meq/day).

**Method of preparation for [\text{14C}]palmitate.** A nonprimed constant infusion of [\text{14C}]palmitic acid (0.27 µCi/ml) was performed for 90 min. Samples for determination of plasma specific activity of palmitic acid were taken at 60, 70, 80, and 90 min after the start of the infusion. The tracer was prepared by dissolving 5 mCi of [\text{14C}]palmitic acid (57 mCi/mmol, New England Nuclear Products, Boston, MA) in a solution containing human albumin. Initially, the tracer was dried under a stream of nitrogen and was reconstituted by adding small amounts of albumin solution (5 ml at a time) in a sonicator maintained at 37°C until the palmitic acid was completely dissolved. The target final concentration of albumin was 0.075 g/ml for this stock solution (40 ml final volume), which was further diluted with saline up to a volume with a palmitic acid concentration ([\text{PAL}]) of 12.5 µCi/ml. The solution was passed through a Millipore filter (no. 22) and was checked for sterility and pyrogenicity, and 25 µCi of the solution were aliquoted in vials. The purity of the tracer was checked in our laboratory by HPLC and beta scintillation counting. More than 99.5% of the counts in the final infusates could be accounted for by the peak, which in the HPLC was identified as palmitic acid. The final concentration of each infusate for each subject was measured in our laboratory. Plasma samples were spiked with a known amount...
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### Table 1. Physical characteristics of older individuals before and after 8-wk endurance training program

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Before Training</th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>66.1±1.4</td>
<td>66.0±1.3</td>
</tr>
<tr>
<td>Height, cm</td>
<td>169±9.0</td>
<td>167±8.9</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>70.8±2.2</td>
<td>70.8±2.1</td>
</tr>
<tr>
<td>Fat-free mass, kg</td>
<td>53.0±2.1</td>
<td>52.6±2.2</td>
</tr>
<tr>
<td>Fat mass, kg</td>
<td>17.7±1.1</td>
<td>18.2±1.2</td>
</tr>
<tr>
<td>Peak V\textsubscript{O\textsubscript{2}}, l/min</td>
<td>1.9±0.1</td>
<td>2.1±0.1*</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 18. Peak V\textsubscript{O\textsubscript{2}}, maximal O\textsubscript{2} consumption. Because no differences were noted between men and women, data are pooled and presented as 1 group. * P < 0.01.

### Table 2. Resting energy metabolism and substrate oxidation of older individuals before and after 8-wk endurance training program

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Before Training</th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting metabolic rate, kcal/min</td>
<td>1.20±0.02*</td>
<td>1.28±0.02+</td>
</tr>
<tr>
<td>Urinary nitrogen excretion, mg/min</td>
<td>5.5±0.03</td>
<td>7.0±0.07+</td>
</tr>
<tr>
<td>Carbohydrate oxidation, mg/min</td>
<td>120±8</td>
<td>110±8</td>
</tr>
<tr>
<td>Fat oxidation, mg/min</td>
<td>58±3</td>
<td>70±4†</td>
</tr>
<tr>
<td>Protein oxidation, mg/min</td>
<td>34±2</td>
<td>44±4*</td>
</tr>
<tr>
<td>Nonprotein respiratory quotient</td>
<td>0.84±0.01</td>
<td>0.82±0.01†</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 18. * P < 0.05; † P < 0.01.
NE and fatty acid kinetics of older individuals before and after 8-wk endurance training program

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Before Training</th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>NE concentration, pg/ml</td>
<td>211 ± 13</td>
<td>249 ± 10*</td>
</tr>
<tr>
<td>NE appearance, µg/min</td>
<td>0.51 ± 0.04</td>
<td>0.69 ± 0.04†</td>
</tr>
<tr>
<td>NE clearance, l/min</td>
<td>2.6 ± 0.2</td>
<td>2.9 ± 0.3</td>
</tr>
<tr>
<td>Total body fat oxidation, µmol/min</td>
<td>201 ± 11.2</td>
<td>244 ± 15.2†</td>
</tr>
<tr>
<td>Free fatty acid appearance, µmol/min</td>
<td>891 ± 85</td>
<td>907 ± 114</td>
</tr>
<tr>
<td>Free fatty acid concentration, µmol/l</td>
<td>906 ± 43</td>
<td>765 ± 36</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 9 males and 8 females. NE, norepinephrine. * P < 0.05; † P < 0.01.

**TABLE 3.**

**FIG. 1.** Relationship between change in resting metabolic rate (RMR) with change in norepinephrine (NE) appearance in older individuals. ●, Males (n = 9); ◇, females (n = 8). r = 0.81; P < 0.01. One data point is missing due to technical difficulties with infusion.

**FIG. 2.** Relationship between change in fat oxidation with change in NE appearance in 17 individuals. ●, Males (n = 9); ◇, females (n = 8). r = 0.69; P < 0.01. One data point is missing due to technical difficulties with infusion.

Drake was noted, whereas increases in the oxidation of fat (P < 0.01) and protein (P < 0.05) were found.

NE and fatty acid kinetics. As shown in Table 3, fasting levels of arterialized plasma NE concentrations were 18% higher (P < 0.05) after training due to a 35% increase (P < 0.01) in NEappearance, whereas no change was noted in NE clearance rate. Total body fat oxidation increased by 22% (P < 0.01) after endurance training, whereas no significant change in FFAappearance was found. Our fasting levels of [FFA] were similar to other values reported in the literature for older fasted individuals (4). We noted no change in [FFA] after training.

Plasma hormones, substrates, and energy intake. There was no effect of endurance training or gender on fasting plasma concentrations of glucagon (97 ± 4 vs. 98 ± 4 pg/ml, before and after training, respectively) or insulin (53 ± 6 vs. 50 ± 4 pmol/l, before and after training, respectively). Fasting glucose concentrations significantly decreased after training (103 ± 2 vs. 98 ± 1 mg/dl, before and after training, respectively; P < 0.05). Energy intake increased from 1,878 ± 138 to 2,212 ± 137 kcal/day (P < 0.05). Exercise training did not change the relative intakes of protein (17 ± 0.8 vs. 16 ± 1.2%, before and after training, respectively), carbohydrate (53 ± 2 vs. 52 ± 3.4%, before and after training, respectively), or fat (29 ± 2.0 vs. 30 ± 2.5%, before and after training, respectively) (data not shown).

Univariate analysis. We examined the associations between changes in fat oxidation, NEappearance, body composition, and RMR. Figure 1 shows that changes in NEappearance were strongly associated with changes in RMR (r = 0.81; P < 0.01). Figure 2 shows that the increase in total body fat oxidation was positively related to NEappearance (r = 0.71; P = 0.51; r² = 0.51) of the variation in fat oxidation, and changes in FFM accounted for an additional 14% (r = 0.81; r² = 0.65; P < 0.05). The partial correlation between changes in fat oxidation and FFM (with the effects of NE appearance removed) was r = 0.54 (P < 0.05). No independent contribution of protein, carbohydrate, or fat was found. Changes in fat oxidation were positively related to NEappearance (r = 0.69; P < 0.01) of the variation in fat oxidation: increases in NEappearance alone accounted for 51% of the increase in fat oxidation. Changes in FFM accounted for an additional 14% (r = 0.81; r² = 0.65; P < 0.05). The partial correlation between changes in fat oxidation and FFM (with the effects of NE appearance removed) was r = 0.54 (P < 0.05). No independent contribution of protein, carbohydrate, or fat was found. Changes in fat oxidation were positively related to NEappearance (r = 0.69; P < 0.01) of the variation in fat oxidation: increases in NEappearance alone accounted for 51% of the increase in fat oxidation. Changes in FFM accounted for an additional 14% (r = 0.81; r² = 0.65; P < 0.05). The partial correlation between changes in fat oxidation and FFM (with the effects of NE appearance removed) was r = 0.54 (P < 0.05). No independent contribution of protein, carbohydrate, or fat was found. Changes in fat oxidation were positively related to NEappearance (r = 0.69; P < 0.01) of the variation in fat oxidation: increases in NEappearance alone accounted for 51% of the increase in fat oxidation. Changes in FFM accounted for an additional 14% (r = 0.81; r² = 0.65; P < 0.05). The partial correlation between changes in fat oxidation and FFM (with the effects of NE appearance removed) was r = 0.54 (P < 0.05). No independent contribution of protein, carbohydrate, or fat was found.
DISCUSSION

Our primary objective in the present study was to examine the effects of endurance training on total body fat oxidation in elderly persons. The new findings are that 1) endurance training shifts in vivo basal substrate metabolism toward greater fat oxidation and 2) the enhanced fat oxidation is associated with an increase in NE_app and alterations in FFMA in older persons.

The present study replicates our earlier findings (26) regarding the linear relationship between changes in RMR and NE_app in response to endurance training in the elderly. Recently, Tremblay et al. (40) found that RMR was higher in trained than in untrained men and that after oral administration of propranolol, the higher RMR in younger trained men was reduced to a level comparable to that of untrained men. Collectively, results from the present study and those of Tremblay et al. suggest that increased sympathetic nervous system activity plays a role in the enhancing effect of exercise on RMR in younger and older persons.

Our study showed that endurance training altered basal substrate utilization patterns in healthy elderly persons by shifting disposal of fatty acids from nonoxidative to oxidative pathways. We noted that fat oxidation increased by 22% in response to endurance training, despite no change in FFMA, an estimate of fatty acid availability. This finding was noted in 15 of 17 volunteers measured. Increased basal levels of lipid oxidation, as derived from a lower fasting respiratory quotient, have been previously found in younger trained men compared with untrained men (40).

We had originally predicted that an increased FFMA would have coincided with an increase in fat oxidation (11). Although our study cannot directly address the mechanism for the dissociation between fatty acid availability and oxidation in response to training, several possibilities may be operative. First, it is possible that an increased rate of lipolysis could have elevated the rate of reesterification to triglycerides within the adipocyte (e.g., intra-adipocyte recycling), thus masking any net change in FFMA. Second, Hurley et al. (15) found that, despite decreased availability and oxidation of plasma free fatty acids, fat oxidation was higher in subjects during exercise after endurance training. The source of the additional fatty acids used in the trained state was postulated to be the intramuscular triglyceride stores. Thus, the hydrolysis of the intramuscular stores with their subsequent intramuscular oxidation could partially explain the discrepancy between the absence of changes in basal lipolysis and fat oxidation. It has also been postulated (13) that plasma [FFA] is only one of several factors that determine the level of fat oxidation. It is possible that [FFA] to which the mitochondria are exposed in the cytoplasm may also determine the rate of fat oxidation. Third, the absence of change in the basal FFMA may relate to the well-known antilipolytic effect of insulin. Insulin is an important hormone-regulating fatty acid release from triglyceride stores. It is possible that an enhancement in insulin sensitivity due to exercise training may have blunted FFMA in the elderly individuals.

Our findings of enhanced fat oxidation in response to endurance training are supported by several in vitro and in vivo studies. Endurance training increases the aerobic capacity of muscle by increasing the number of mitochondria and mitochondrial respiratory and citrate cycle enzymes (14, 17, 24). It has also been shown that skeletal muscle of elderly humans retains the ability to adapt to endurance exercise with an increase in the capacity for aerobic metabolism (3). Romijn et al. (34) found that younger trained males exhibited higher rates of fat oxidation than young untrained males. Because their infusion studies were performed within proximity to the last exercise bout (12–18 h), it was unclear, however, whether the observed differences in fatty acid flux between young trained and untrained men reflected the carryover effects of the last exercise bout and/or an adaptation to exercise training. In the present study, the increase in fat oxidation was smaller in magnitude (−22%) compared with the differences observed between trained and untrained men (−47%) observed in the work by Romijn et al. Although these studies are not directly comparable, differences in the magnitude of fat oxidation may be attributable to 1) variations in experimental design (cross-sectional vs. longitudinal), 2) age of the subjects (younger vs. older), and 3) timing of the metabolic measurements relative to the last exercise bout (12–18 vs. >36 h postexercise in the present study).

Preliminary evidence from this study provides support for the independent involvement of sympathetic nervous system activity and body composition mediating changes in total body fat oxidation in response to endurance training in elderly persons. Using stepwise multiple regression analysis, we found that approximately two-thirds of the increase in fat oxidation was accounted for by changes in NE_app and FFMA. The increase in NE appearance alone in response to training accounted for ~50% of the increase in fatty acid oxidation. Tremblay et al. (40) also support the concept that the higher resting levels of lipid oxidation in trained young men are sympathetically mediated. They found that, after oral administration of propranolol, the previously higher lipid oxidation noted in trained men was reduced to a level comparable to that of untrained men. These findings suggest that higher levels of β-adrenergic stimulation in trained individuals may be contributory to their higher basal level of lipid oxidation.

The increase in FFMA in response to endurance training was an independent variable, accounting for 15% of the explained variance in fat oxidation. Despite no mean group change in FFMA (range of changes: -2.0 to 1.1 kg), those individuals who did increase FFMA increased basal total fat oxidation. The partial correlation between changes in fat oxidation and fat-free weight after statistically controlling for the effects of NE appearance was significant (r = 0.54; P < 0.05). These findings suggest that even small changes in the “consumer” of fatty acids...
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(i.e., FFM), in response to exercise training, influence the level of fat oxidation in elderly persons. In other words, the energy needs of FFM may be one mechanism regulating the level of fat oxidation in the elderly.

We examined the possibility that a shift in macronutrient intake could have influenced basal levels of fat oxidation, as previous work has shown that an elevation in fat intake for 3-6 days (>60% of calories from fat sources) increases fat oxidation (16). However, we consider it unlikely that our observed increase in fat oxidation in the present study was related to a shift in macronutrient selection, as no changes were noted in the relative ingestion of the macronutrients in response to endurance training. Furthermore, in a previous study from our laboratory, in which food intake was covertly measured in elderly persons, endurance training did not change macronutrient selection (27). Collectively, our results and those of others (40) favor the interpretation that increased activity of the sympathetic nervous system and alterations in body composition favor a chain of metabolic events leading to increased fat oxidation in elderly individuals in response to endurance training.

Two lines of evidence from the present study suggest that regular endurance training may be of benefit in the regulation of energy balance and body composition with advancing age via its impact on RMR and substrate utilization patterns. Ravussin et al. (33) have shown that a low RMR after normalization for differences in body composition was associated with increased prospective body weight gain in Pima Indians. We have consistently found that endurance training increases RMR on the order of 8-10% in older persons independent of changes in body composition (26, 27). Second, a low ratio of fat oxidation to carbohydrate oxidation, as reflected in a higher 24-h respiratory quotient (41), has been found to be an additional risk factor predisposing individuals to subsequent gain in body weight. Our results point toward a lowering of the fasting nonrespiratory quotient in response to endurance training.

We have paid close attention to the following methodological issues that we feel render a clearer interpretation of our data. First, we conducted our metabolic studies ±36 h after the last exercise session. Thus, our findings probably reflect chronic adaptations to endurance training rather than an acute exercise phenomenon. Second, we believe that the expression of data in an absolute position (39). The need to normalize data in the present study was obviated because of the longitudinal design and the absence of significant changes in body composition (39). The need to normalize data in the present study was obviated because of the longitudinal design and the absence of significant changes in body composition (39). Third, we have provided data (see MATERIALS AND METHODS) indicating high reproducibility of FFA_app in older individuals in our laboratory.

In conclusion, our results suggests that 1) endurance training shifts basal substrate utilization toward greater fat oxidation and 2) the enhanced fat oxidation is associated with an increased NE_app and alterations in FFM in the elderly.

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