Increased thermogenic response to food and fat oxidation in female athletes: relationship with $\dot{V}O_2$ max

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Received 10 August 1999; accepted in final form 20 April 2000

López, Pilar, Marielle Ledoux, and Dominique R. Garrel. Increased thermogenic response to food and fat oxidation in female athletes: relationship with $\dot{V}O_2$ max. Am J Physiol Endocrinol Metab 279: E601–E607, 2000.—The thermogenic response to food (TRF) and substrate oxidation were studied in 12 endurance-trained and 13 untrained female subjects. Energy expenditure and substrate oxidation were calculated by indirect calorimetry before and for 6 h after an oral test meal and after the same meal given intragastrically on a separate occasion. The TRF was calculated after the oral meal, the obligatory component after the intragastric meal (OTRF), and the facultative component from the difference between the two. $\dot{V}O_2$ max was measured on a treadmill and body composition by underwater weighing. The TRF and OTRF were significantly higher in trained than in untrained subjects: $223 \pm 63$ vs. $185 \pm 50$ kcal/6 h ($P < 0.03$) and $174 \pm 38$ vs. $131 \pm 37$ kcal/6 h ($P < 0.01$) for the TRF and OTRF in trained vs. untrained subjects, respectively. Multiple regression analysis showed that maximum $O_2$ consumption ($\dot{V}O_2$ max), but not percentage of body fat, was significantly related to OTRF ($r = 0.68$, $P < 0.01$). Trained subjects had higher fatty acid oxidation than untrained subjects before (0.6 vs. 0.4 mg kg$^{-1}$ min$^{-1}$, $P < 0.05$) and after the oral meal (13 $\pm$ 6 vs. 8 $\pm$ 4 g/6 h $P < 0.05$). These results demonstrate that 1) TRF is higher in trained than in untrained women; 2) this is due to a higher cost of nutrient digestion, absorption and storage; 3) the difference is related to higher $\dot{V}O_2$ max; and 4) fatty acid oxidation is greater in trained women in both the postabsorptive and postprandial states. These observations suggest that endurance training induces metabolic changes that favor leanness.

energy expenditure; endurance training; women

THE THERMOGENIC RESPONSE to food (TRF) is an increase in resting energy expenditure (REE) after ingestion of food. In rats and dogs, TRF has two components, an obligatory component, which includes the process of digestion, absorption, and storage of nutrients, and a facultative component, which is linked with oropharyngeal stimulation (11, 12, 15). In humans, we have shown that 30–40% of TRF is related to oropharyngeal stimulation (10) and that this component is under the control of the sympathetic nervous system (9). These observations support the concept that a facultative component of the TRF exists in humans and that it is regulated in a manner similar to that in rodents. We have postulated that the existence of these two components of the TRF in humans may explain some of the discrepancies observed in the literature when only the whole thermic response to feeding is studied. Factors that have been found to influence TRF, such as energy content of the meal, obesity, or exercise may affect either component. If only one component is affected, analytical variability arising from the nonaffected component may impair the ability to show a difference between individuals. For instance, we have found that only the facultative component of TRF was affected by obesity (13).

The effects of exercise training on the TRF are poorly understood. Some research suggests higher TRF in trained subjects (8, 14, 37, 43), whereas other studies have found lower TRF (21, 22, 39) or no difference between trained and sedentary individuals (4, 28, 38). Understanding the relationships between physical training and TRF is important for the evaluation of long-term benefits of exercise for weight regulation. Although TRF represents a small proportion of 24-h energy expenditure, a permanent increase in this component of the energy balance equation is likely to be significant.

The objective of this study was, therefore, to measure both components of the TRF in trained and untrained women and to calculate the relationship of these measurements to $\dot{V}O_2$ max and body fat.

SUBJECTS AND METHODS

Subjects. Twenty-five (12 trained and 13 untrained) young (aged 18–35 yr), healthy female subjects were recruited for this study. The trained subjects were distance runners or triathletes currently performing $\geq 8$ h/wk of aerobic exercise, who had been training for at least two years previously, and who had engaged in provincial and national competitions. This group was selected from three university sports centers in Montreal. The untrained subjects had not performed aerobic exercise more than 1.5 h/wk during the previous two years and had never been enrolled in any athletic competition involving endurance activities; they were selected...
through city newspaper ads and ads in local universities. All subjects were nonsmokers, were weight stable (±2.5 kg during the 6 mo before the study), were not taking any medication known to affect metabolic rate, did not present any family history of diabetes or obesity, and had a regular menstrual cycle (28–35 days). Trained and untrained groups were matched for body size [body mass index (BMI) 18–22 kg/m²]. This was done to facilitate analysis of the effect of body composition on TRF. Informed consent was obtained from each subject, and the ethics committee of our institution approved the study.

Procedures for admission into the study. Before their engagement in the study, all subjects went through a preliminary session, during which they were informed of all the investigation procedures. During this session, a nasogastric tube (Flexiflo no. 8 enteral feeding tube, Ross Laboratories, Columbus, OH) was inserted into the stomach without nasal anesthesia and left in place for ~10 min. The participants were also familiarized with the measurement of indirect calorimetry and had a physical examination.

Glucose tolerance test. Because insulin resistance is associated with inhibition of the TRF (33), serum glucose and insulin concentrations were measured as markers of insulin sensitivity during a 75-g oral glucose challenge in all subjects.

Subjects arrived at the clinic after a 12-h fast, and a first blood sample was taken; then they ingested 75 g of glucose (Glucotrol, Rougié, Chambly, Canada), and a second blood sample was collected 120 min later. Serum glucose was measured by the glucose oxidase method. Insulin level was quantified by radioimmunoassay (SERONO RIA kit, Immunoacorp, Montreal, Canada).

Body composition. Body fat was assessed by densitometry and skinfold measurements. Body density was measured by underwater weighing (18); a maximum of seven trials was performed for each subject. Three trials with the least variability and providing the highest body density were averaged to obtain final body density. Residual lung volume was measured using the closed-circuit helium-dilution method (26). Skinfold thicknesses were measured at 10 sites on the left side of the body with a Lange caliper (Cambridge Scientific Industries, Cambridge, MD); the average of triplicate measurements was used to calculate body density. Fat-free mass (FFM) and fat mass (FM) were calculated from body density values according to the equations of Siri (34) and from skinfold values with the equations of Allen et al. (2).

Maximal treadmill exercise test. Maximal aerobic capacity was measured on a treadmill (Quinton Q65); the Léger and Boucher (23) protocol was selected using the modified formula of Léger and Mercier (24). All subjects were asked to abstain from drinking alcohol or products containing caffeine 24 h before the test and not to eat a large meal 3–4 h before the evaluation; trained subjects were instructed to abstain from intense exercise 24 h before the test. The test began with a 5-min run at 8 km/h, after which the speed was increased by 1 km/h every 2 min until exhaustion. Expired gases were measured using a breath by breath system (mass spectrometer, Marquette 1100 medical gas analyzer with a ventilation measurement module and the Marquette Calcul Vo₂ software). Heart rate (HR) was monitored continuously with a portable chest strap monitor (Polar CIC, Port Washington, NY).

Preexperimental procedures before TRF tests. All subjects completed a 3-day dietary recall (2 weekdays and 1 weekend day) to obtain data on mean total caloric intake and percent-
age of energy nutrients. The day before each TRF test, they were asked to eat a high-carbohydrate diet (60% carbohydrate, 18% protein, and 22% fat). To attain this objective, each subject was given a list of equivalents to follow during meals. This was done to reflect the usual proportion of energy derived from fat and carbohydrates by the subjects as determined from the 3-day food records. Trained subjects were instructed to avoid physical activity for 24 h before the REE and TRF tests.

On two nonconsecutive days during the follicular phase (i.e., between days 5 and 10 of the menstrual cycle, day 1 being the first day of bleeding), all subjects were asked to arrive at the laboratory with minimum effort at 8:00 AM after a 12-h fast. They rested for 20 min before the procedures.

REE and TRF tests. REE was measured for 30 min before each test meal. The test meals were assigned randomly (see test meal composition in Table 1).

Test 1 was the oral ingestion of a standard meal; for test 2, the same meal was blended and administered through a nasogastric tube within 20 min. The tube was removed after the procedure. Energy expenditure was then measured for 30 min every hour for 6 h. The subjects were allowed to sit or stand and stretch between measurements. Walking or the ingestion of any food or beverage was not permitted. This procedure prevents an increase of REE due to fidgeting when the subjects are asked to maintain a supine position for 6 h (29). The first postprandial measurement began upon completion of the meal.

Energy expenditure was measured by continuous indirect calorimetry with a ventilated hood system (Deltatrac Metabolic Monitor, SensorMedics, Anaheim, CA). Oxygen consumption (VO₂) and carbon dioxide production (VCO₂) were calculated from continuous measurements of oxygen and CO₂ concentrations in inspired and expired air diluted in a constant flow generated by the analyzer. The accuracy and precision of the system were calibrated in vitro in our laboratory. Differences between values predicted from the injection of nitrogen and CO₂ into a sealed tank and measured values were <2%, and coefficients of variation for repeated measures were <1% (17).

Urine collection was initiated after voiding at 8:20 AM and lasted until the end of the procedure. Total nitrogen concentrations were measured by chemiluminescence with an AN-TEK analyzer (ANTEK, Houston, TX) and were used for the calculation of energy substrate oxidation.

Table 1. Composition of test meals

<table>
<thead>
<tr>
<th></th>
<th>Oral</th>
<th>Liquid</th>
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</thead>
<tbody>
<tr>
<td>Milk 2%, ml</td>
<td>200</td>
<td>200</td>
</tr>
<tr>
<td>Orange juice, ml</td>
<td>125</td>
<td>125</td>
</tr>
<tr>
<td>Cream 10%, ml</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Butter, g</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Eggs</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>White bread, g</td>
<td>60</td>
<td>265</td>
</tr>
<tr>
<td>Cream of wheat, g</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sugar, g</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Carbohydrate, g</td>
<td>60.1</td>
<td>60.5</td>
</tr>
<tr>
<td>%Energy</td>
<td>40.4</td>
<td>40.5</td>
</tr>
<tr>
<td>Protein, g</td>
<td>29.9</td>
<td>29</td>
</tr>
<tr>
<td>%Energy</td>
<td>20.1</td>
<td>19.5</td>
</tr>
<tr>
<td>Fat, g</td>
<td>26.1</td>
<td>26.5</td>
</tr>
<tr>
<td>%Energy</td>
<td>39.5</td>
<td>40.0</td>
</tr>
<tr>
<td>Energy, kJ</td>
<td>2,490</td>
<td>2,497</td>
</tr>
</tbody>
</table>
Calculations and statistics. Energy expenditure was calculated from \( V\dot{O}_2 \), \( V\dot{O}_2 \), and urinary nitrogen by the Weir equation (42)

\[
EE (kJ/24 h) = (3.941 \cdot V\dot{O}_2 + 1.106 \cdot V\dot{CO}_2 - 2.17 \cdot N) \cdot 4.18
\]

TRF was calculated as the increase in energy expenditure above the fastest value, measured on the test day. The thermogenic response measured after the intragastric meal was considered the obligatory component of TRF (OTRF) and the difference between TRF and OTRF the facultative component.

Substrate oxidation was quantified with the coefficients of Consolazio, as described previously (10). Data were analyzed by repeated-measures ANOVA. When the differences were significant, multiple comparisons between groups (Tukey’s “B” method) were conducted. Paired \( t \)-tests were employed to compare substrate oxidation between meals (oral vs. intragastric) within individuals. Non-paired \( t \)-tests were used to compare substrate oxidation between groups (trained vs. untrained). REE was compared between the two groups of subjects after analysis of covariance (ANCOVA). REE was the dependent variable and FFM the covariate. This was possible because the correlation between the two variables in our population was 0.62 (\( P < 0.01 \)). Calculations were performed using the Statistical Package for Social Sciences (SPSS V4.01, SPSS, Chicago, IL).

RESULTS

Subjects. The average duration of aerobic exercise in trained subjects was 13.6 ± 2.8 h/wk, and the average time since involvement in competitive activities was 4.3 ± 1.6 yr. The physical characteristics of the trained and untrained subjects are shown in Table 2. There was no difference in age, weight, height, and BMI between the groups. Body fat, FM, and resting HR were lower in trained subjects; FFM was higher in trained subjects. There were no significant differences in plasma insulin and glucose concentrations (data not shown).

The 3-day dietary recall revealed no significant differences between untrained and trained subjects in the percentage of total energy from carbohydrate and protein consumption: carbohydrate, 55 ± 4 vs. 59 ± 6% for untrained and trained subjects, respectively; protein, 20 ± 6 vs. 21 ± 5% for untrained and trained subjects, respectively. Energy derived from lipids was lower (20 ± 5 vs. 25 ± 4%, \( P = 0.05 \)), and total energy intake was higher (7,460 ± 1,389 vs. 6,663 ± 853 kJ/day, \( P = 0.01 \)) in trained subjects.

Thermogenic response to food. Figure 1, A and B, presents the response of each group (trained and untrained) to oral and tube feeding. Both groups exhibited significantly lower energy expenditure after tube feeding (−22.2 and −29.0%, respectively, \( P < 0.01 \) for both comparisons).

Figure 1, C and D, shows the TRF and OTRF in both groups of subjects. Trained subjects had higher TRF and OTRF than untrained subjects: TRF, 223 ± 63 and 185 ± 50 kJ/6 h in trained vs. untrained subjects, respectively (\( P < 0.05 \)); OTRF, 173 ± 38 vs. 131 ± 36 kJ/6 h in trained and untrained subjects, respectively (\( P < 0.05 \)). The facultative component of TRF was 54 ± 14 and 50 ± 13 kJ/6 h in untrained and trained subjects, respectively.

Multiple regression analysis with TRF as the dependent variable revealed no relationship between TRF, \( V\dot{O}_2 \) max, and percent body fat. OTRF, however, was significantly related to \( V\dot{O}_2 \) max (\( r = 0.68, P = 0.05 \); see Fig. 2) but not to percentage of body fat.

Substrate oxidation. Results of substrate oxidation are presented in Table 3. At rest and after a 12-h fast, fatty acid oxidation was significantly higher in trained subjects (+37% between the average values of both study days): 0.40 ± 0.2 vs. 0.55 ± 0.3 mg·kg⁻¹·min⁻¹ in untrained and trained subjects, respectively. Glucose oxidation was not different between groups.

In untrained subjects, postprandial oxidation of glucose was higher and fatty oxidation was lower after the oral meal than after the intragastric meal (59 ± 12 vs. 49 ± 10 and 8 ± 4 vs. 11 ± 5 g) for glucose and fatty acid oxidation, respectively (\( P < 0.05 \) for both comparisons). There was no difference in substrate oxidation between the two postprandial periods in trained subjects.

Comparisons of untrained and trained subjects showed that fatty acid oxidation was significantly higher in trained subjects after the oral meal (13 ± 6 vs. 8 ± 4 g, \( P < 0.05 \)) but not after the intragastric meal. All comparisons yielded similar results when the data were expressed as milligrams per kilogram of FFM per minute. Postprandial protein oxidation was not different between groups or between the two modes of feeding: trained subjects, 0.8 ± 0.2 and 0.8 ± 0.3 kJ·kg⁻¹·day⁻¹ for trained and untrained subjects, respectively (\( P < 0.05 \)).  

Table 2. Characteristics of the study population

<table>
<thead>
<tr>
<th></th>
<th>Trained</th>
<th>Untrained</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>25.6 ± 4.3</td>
<td>23.8 ± 4.4</td>
<td>NS</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>56.5 ± 5.8</td>
<td>55.7 ± 5.3</td>
<td>NS</td>
</tr>
<tr>
<td>Height, cm</td>
<td>164.9 ± 6.3</td>
<td>163.9 ± 5.5</td>
<td>NS</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>21.0 ± 2</td>
<td>20.8 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>Hydrodensity, %BF</td>
<td>13 ± 4</td>
<td>17 ± 5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>10 Skinfolds, %BF</td>
<td>13 ± 4</td>
<td>16 ± 3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Triceps, mm</td>
<td>10 ± 2</td>
<td>13 ± 4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>71 ± 2</td>
<td>9.5 ± 3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>V\dot{O}_2 max, ml·kg⁻¹·min⁻¹</td>
<td>53.6 ± 5</td>
<td>42.45 ± 5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Resting HR, beats/min</td>
<td>59 ± 7</td>
<td>70 ± 8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>REE, kJ·kg⁻¹·BW⁻¹·day⁻¹</td>
<td>101 ± 12</td>
<td>97 ± 12</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>corrected for FFM</td>
<td>100 ± 10</td>
<td>92 ± 10</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

BMI, body mass index; FM, fat mass; FFM, fat-free mass; \( V\dot{O}_2 \) max, maximum oxygen consumption; HR, heart rate; REE, resting energy expenditure; BW, body weight; NS, not significant.
DISCUSSION

The results of this study show that TRF is higher in endurance-trained than in sedentary women. In addition, they show that the obligatory component of TRF, not the facultative one, is responsible for this effect. We believe that intragastric feeding results in the blunting of the facultative component of TRF, because we have found that intravenous administration of propranolol, an inhibitor of the sympathetic nervous system, eliminates the difference in TRF between intragastric and oral feeding and has no effect on TRF after intragastric feeding (9). Indeed, it was shown by Acheson et al. (1) that facultative thermogenesis induced by the intravenous administration of glucose can be suppressed by propranolol infusion. Taken together with our findings, these results suggest that intragastric feeding abolishes a component of TRF that is dependent on the autonomic nervous system. Insulin secretion is unlikely to be responsible for the facultative component of TRF, because no difference was found in the area under the curve of plasma insulin after intragastric vs. oral feeding (10).

Previous investigations measuring TRF in trained and untrained subjects at rest presented discrepant data. Methodological issues, such as heterogeneity of the group, the small number of subjects studied, “mixed” gender, various time lapses between testing and the last bout of exercise, inconsistent duration of TRF measurement, and variable quantity and composition of test meals may explain these discrepancies (8, 14, 21, 22, 29, 36, 41). The literature is limited and controversial concerning TRF in female athletes, as well (4, 28, 43).

In this study, the trained and untrained subjects were matched for age, weight, height, and BMI to minimize the possible influence of these parameters on TRF, which resulted in recruiting control subjects with low body fatness, and these subjects may not represent the general sedentary population. Because excess body fatness is associated with decreased insulin sensitivity (33) and lower TRF, our subject selection is likely to have led to a smaller between-group difference than would have been expected from a sedentary group with more body fat. In addition, subjects followed a specific nutrient partitioning for 3 days before testing. This was done to avoid the possible influence of an habitual pretest diet on TRF. Whether TRF is influenced...
by the energy partitioning of a meal is controversial, and there are no data on the influence of diet composition before measuring TRF. The difference between the two groups of subjects regarding food energy partitioning was small and is unlikely to have affected the results.

Our results show a significantly higher TRF (+22%) in trained subjects, and when both TRF components are taken into consideration, the obligatory but not the facultative component is higher (+32%) in these subjects than in the untrained controls. Our data on the TRF are in agreement with those of authors who measured TRF in athletes 36 h or more after the last exercise bout (8, 14, 43) and in contradiction with those where the TRF was measured 12–16 h after exercise (21, 39). The importance of the length of time without exercise before measuring TRF in trained subjects has been stressed (30), but only one study has specifically addressed this issue. In that investigation (41), the thermogenic response to an oral glucose load was significantly higher 3 days after the last exercise bout compared with 16 h. These results suggest that a detraining phenomenon may have the same effect as a stressful situation on TRF in athletes and favor a 24-h lag time between the last bout of exercise and TRF measurements in trained individuals. On the other hand, it is possible that TRF would also be influenced by an exercise bout close to the time of measurement. The lower TRF found in trained subjects 12–16 h postexercise might represent an adaptive phenomenon with increased efficiency of nutrient storage during this period of time. The phenomenon must be transient, however, because our study and investigations with TRF measurements performed more than 24 h postexercise showed heightened TRF in trained subjects.

In addition to demonstrating a higher TRF in trained women, our data suggest that this difference is related to an enhancement of the cost of digestive absorption and metabolic storage of nutrients (i.e., the “obligatory component”) and not through an increment of the “facultative component” of the TRF. Whether this increased cost of metabolic digestion, absorption, and storage of nutrients is related to carbohydrate or protein metabolism, or both, cannot be evaluated from our data. In the postabsorptive state, no difference in leucine turnover was found between endurance-trained and sedentary subjects (19), but postprandial measurements of protein turnover have not been made under these circumstances.

We found a strong correlation between %body fat and VO_{2\,max} (−0.68, P < 0.005); the correlation between OTRF and VO_{2\,max} did not change with %body fat as a covariate. This indicates that, at least in subjects of comparable body fatness, body composition does not influence the energy cost of nutrient storage. In the literature, investigations looking at VO_{2\,max} and TRF have conflicting results, with observations of an increase in both parameters in trained subjects (8, 14) as well as an inverse relationship between the TRF and VO_{2\,max} in trained subjects (22, 30). Poehlman et al. (30), studying male subjects, proposed a curvilinear relationship between VO_{2\,max} and TRF, with highly trained subjects having a lower TRF when VO_{2\,max} exceeded 70 ml·kg\(^{-1}\)·min\(^{-1}\). These discrepancies may be related to the methodological issues already discussed, but they may also arise from the fact that both components of TRF were not measured in these experiments.

The finding of a relationship between VO_{2\,max} and the cost of nutrient metabolism and storage deserves further investigation. Consistent with our findings of a positive relationship between VO_{2\,max} and TRF, it is the observation that VO_{2\,max} is positively and independently related to insulin sensitivity (7). Indeed, low insulin sensitivity has been reported to be an independent factor related to low TRF (33). The influence of the genetic background of an individual vs. the effect of endurance training on VO_{2\,max} cannot be inferred from our data, because the relationship between VO_{2\,max} and OTRF was correlated in our subjects irrespective of their training status. Whether endurance training increases the obligatory component of TRF remains to be studied.

In addition to higher TRF, we found that athletes had a higher resting metabolic rate (RMR), even after the data were adjusted for FFM. We used FFM as a...
covariate instead of dividing RMR by FFM, because the regression line between the two variables did not intercept zero. Most studies on the relationship between the RMR and endurance training have been conducted in men, and the results of those that have investigated women are conflicting. No difference was observed in RMR between trained and untrained women by LeBlanc et al. (22) and Owen et al. (28), but exercise training in combination with weight reduction has been found to increase RMR in women (20, 25). Basal metabolic rate was reported to be higher in four female cross-country skiers measured at ≥39-h postexercise compared with control subjects matched for lean body mass (35). These discrepancies may be related to the time elapsed after the last exercise bout, phase of the menstrual cycle (36), physical activity, or food intake, all of which may influence RMR. We attempted to minimize these factors by controlling for body size and composition, pretest food intake, and phase of the menstrual cycle. As these factors are likely to influence RMR, optimal experimental conditions for such studies should measure either small numbers of subjects with strictly standardized settings or large numbers of subjects allowing for multivariate analyses.

In the present investigation, differences in FFM were accounted for by ANCOVA, and it was shown that RMR was higher in trained subjects after this correction. Thus our data indicate that endurance training per se increases RMR in women.

Studies of RMR in healthy men suggest that strenuous exercise (i.e., >70% VO₂ max) can augment RMR up to 24 h postexercise (30), but the relationship between aerobic fitness and RMR in men is not clear. A positive relationship between RMR and aerobic fitness was found in some investigations (3, 30, 40) but not in others (9, 14, 21). The conflicting results on relationships between the RMR and physical fitness observed in both men and women suggest that longitudinal studies are warranted in subjects showing large variations in VO₂ max through physical training.

The data on substrate oxidation show that fatty acid oxidation was 37% higher in trained than in untrained subjects in the postabsorptive state. This difference was more pronounced during the postprandial phase, with the average value for fatty acid oxidation in trained subjects being 65% above that of untrained subjects. The physiological regulation of fatty acid oxidation at rest and the effects of endurance training have not been well characterized. Calles-Escandon and Driscoll (5) found no difference in fatty acid oxidation in trained subjects being 65% above that of untrained subjects. The physiological regulation of fatty acid oxidation at rest and the effects of endurance training have not been well characterized. Calles-Escandon and Driscoll (5) found no difference in fatty acid oxidation in trained subjects being 65% above that of untrained subjects.

REFERENCES

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