Active Recovery Counteracts the Post-Exercise Rise in Plasma-Free Fatty Acids

Ine Wigernæs, Sigmund B. Strømme, and Arne T. Høstmark

The present study investigated the effect of active recovery (AR) as compared to rest recovery (RR) upon FFA concentrations following moderate- (MI) or high-intensity (HI) running. Fourteen well-trained males (23.7 ± 6 years, $\dot{VO}_{2\text{max}} = 69.5 \pm 1.8\text{ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$) were randomly assigned into two trials (HI = 30 min at 82% of $\dot{VO}_{2\text{max}}$, MI = 60 min at 75% of $\dot{VO}_{2\text{max}}$). Within each group, the subject completed two sets of experiments of running followed by either AR (15 min running at 50% of $\dot{VO}_{2\text{max}}$) or RR (complete rest in the supine position). Plasma volume changes after the exercise did not deviate between the AR or RR trials. In both the HI and MI trials, AR resulted in lower FFA peaks and lower overall FFA concentrations while performing AR ($p < .05$). However, upon discontinuing AR, there was a rise in the FFA concentration. At 120-min post-exercise, the FFA concentrations after AR and RR were not significantly different. The changes in the FFA/albumin ratio were similar to the FFA responses. It is concluded that AR may counteract the rise in FFA 5–15 minutes after exercise.

Key Words: free fatty acids, albumin, endurance exercise, recovery, young trained males

Introduction

The major part of fatty acids in humans are stored as triglycerides in adipose tissue, while some are found within the myocardium and in skeletal muscle (6, 12). Triglycerides are also associated with the plasma lipoproteins. In the blood, the metabolically active non-esterified "free" fatty acids (FFA) are bound to serum albumin, except for 0.01%, which are dissolved. Plasma FFA are released from adipose tissue triglycerides to be used as substrate for energy production (2).

Wolfe et al. (31) found that after a 4-hour walk on a treadmill at an intensity of 40% of $\dot{VO}_{2\text{max}}$, the FFA concentration had increased from less than 0.5 mmol/L at start to 1.6 mmol/L, exceeding 2 mmol/L 10 min post-exercise. Bahr et al. (3) observed in fasting subjects that the concentration of FFA in the first 15 min after exhaustive workouts (80 min at 70–75% of $\dot{VO}_{2\text{max}}$) rose to an average concentration.

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of 2 mmol/L. For some of the subjects, the peak FFA concentration was even above 3 mmol/L. A linear relationship was found between peak FFA concentrations and both intensity and duration of the exercise. Bangsbo (5) reported that FFA concentrations in soccer players stayed relatively constant during warm-up and the first half (0.2–0.4 mmol/L) but were increasing after 20 min in the second half, reaching 1.2 mmol/L 5 min after the match. The rise in postexercise FFA concentrations was also observed by Pruett (25).

It has been suggested that high concentrations of FFA in plasma may have negative health effects (15, 22, 23). An increased risk of fatal myocardial infarction, probably due to ventricular arrhythmias, has been related to FFA concentrations above 1.2 mmol/L (19, 20). In vitro, FFA may inhibit cell growth (8) and cause hemolysis of erythrocytes, but the presence of albumin protects against these effects (16).

The plasma concentration of FFA stays relatively constant during light, short-term exercise, rises slowly during endurance type exercise, and is suppressed if the exercise intensity is above 85% of \( \text{VO}_{2\text{max}} \) (28). Immediately after exercise, however, the FFA concentrations are appreciably increased.

We reasoned that a continued FFA utilization by active recovery (AR) might counteract the plasma FFA increase after exercise. El-Sayed et al. (11) conducted a study where the subjects performed AR, or complete rest in supine position, rest recovery (RR) at an intensity of 30% of the individual \( \text{VO}_{2\text{max}} \) after cycling 90-min at an intensity of 70% of \( \text{VO}_{2\text{max}} \). Surprisingly, they did not find any significant reduction in plasma FFA after AR as compared with RR. Presumably, an intensity of 30% of \( \text{VO}_{2\text{max}} \) might not have been sufficient to prevent the rise in plasma FFA. Thus, it seemed pertinent to reinvestigate the effect of AR on the FFA concentration, using a higher exercise intensity during AR. We accordingly studied the plasma FFA concentration in response to 15 min of AR at 50% of \( \text{VO}_{2\text{max}} \) following a moderate- (MI, 60 min at 75% of \( \text{VO}_{2\text{max}} \)) or high-intensity (HI, 30 min at 82% of \( \text{VO}_{2\text{max}} \)) trial, as compared with RR. Thiobarbituric acid reactive substances (TBARS) and albumin were also determined. The former is an indicator of lipid peroxidation, and the latter is involved in transport of FFA.

**Methods**

**Subjects**

Fourteen healthy, non-smoking, college-aged males participated. The subjects were randomly assigned into two groups. Seven subjects completed two 30-min sets of running at 82% of \( \text{VO}_{2\text{max}} \) (high intensity group, HI). One set was followed by RR and the other by AR. Another 7 subjects completed two 60-min sets of running at 75% of \( \text{VO}_{2\text{max}} \) (moderate intensity group, MI) followed by either RR or AR. All the subjects were well-trained for endurance activities, mainly running, and were active throughout the year. Average age was 23.4 ± 1.1 years. Mean height and weight was 178.9 ± 2.4 cm, and 74.3 ± 2.8 kg. \( \text{VO}_{2\text{max}} \) exceeded 60 ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\) for all subjects. The mean value for the HI-trial subjects was 69.8 ± 1.6 ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\) and for the MI-trial subjects 69.2 ± 2.0 ml kg\(^{-1}\) \cdot min\(^{-1}\). Lactate thresholds were 1.9 ± 0.1 and 2.5 ± 0.4 mmol/L, respectively.

None of the participants suffered from any upper respiratory tract infections or had been using antibiotics the last 4 weeks prior to, or during, the experimental period. There were no dropouts during this period.
The study was approved by the Norwegian Institutional Review Board for Human Research. An individual consent form explained the test procedures and that handling of personal data was strictly confidential, including substitution of all names with codes in all written material.

**Procedures**

The subjects had a carbohydrate rich meal consisting of white bread, jam, and water 3–4 hours pre-exercise. Only water was allowed to be consumed before, during, and after the running trials. The subjects reported that the pre-exercise meals were identical before all laboratory procedures. The subjects had refrained from intense exercise the previous 36 hours.

All running trials were carried out using a motorized treadmill (Woodway®). Prior to all exercise trials, a standard 10-min warm-up at an uphill grade of 3° (5.3%) was performed. The heart rates were recorded by a Polar Sport Tester®. Rating of perceived exertion (RPE 7) and oxygen uptake were also assessed several times during the trials but not during the recovery period.

**Lactate Thresholds/Maximal Oxygen Uptake**

A total of four or five intervals of 5-min running at 6° (10.5%) were sufficient to establish the lactate thresholds. Each subject started at 8 km/hour, increasing 1 km/hour at each step until reaching 10 km/hour, then the increase was reduced to 0.5 km/hour. Heart rates and RPE were recorded every minute throughout the interval. Between 2:30 and 4:30, the subject wore a mouthpiece and a nose clip in order to analyze expired air and determine the oxygen consumption (\(\dot{V}O_2\)). Blood lactate was assessed after fingertip sampling of blood into heparinized capillary tubes and the sample was immediately injected into a YSI 23L/YSI Sports lactate analyzer®. The lactate assessments took no longer than 30 s, then the subject continued on the treadmill at an increasing speed. Following the incremental lactate threshold test, a 3-min recovery run was permitted before a 4–6-min \(\dot{V}O_2^{\text{max}}\) test was carried out (1, 14). The subjects started running at the intensity where lactate accumulated in the lactate threshold assessment test. Oxygen consumption was printed every 10 s, using a Jaeger® metabolic chart (±3%).

Daily calibration procedures for the mixing chamber involved a comparison of the concentrations of gases analyzed compared to a known concentration of inert gases. The lactate analyzer was calibrated between each subject, using a known lactate concentration of 5 mmol/L and 15 mmol/L from YSI®.

**HI and MI Running Trials**

The order of the two recovery experiments was randomized for each individual. Each subject performed this exercise twice, where the only variable was the type of recovery in the first 15 min after completing the workout—in other words, RR (resting in the supine position) or AR, consisting of continuous jogging at 50% of \(\dot{V}O_2^{\text{max}}\) at an increment of 3°. Both MI and HI trials were performed with a 6° increment. The intensity of the submaximal exercise (75 or 82% of \(\dot{V}O_2^{\text{max}}\)) was characterized by the speed of the treadmill (for HI = 9.5 ± 0.3 km/hour, for MI = 8.6 ± 0.3 km/hour). The reasoning behind the choice of intensities was to compare a
subthreshold workout with a more anaerobic workout, which therefore had to be of a shorter duration. Both training intensities are relevant in order to improve endurance capacity and is not exhaustive. It is worth noticing that the RPE recorded during the trials was not statistically different between the two intensities (vide infra). However, considering the monotony in the 60-min trial, local fatigue and discomfort might explain the lack of difference between perceived effort even though lactate concentration in the HI trial was higher ($p < .05$). RPE was not recorded in the RR or AR intervention period. The trials were completed during a 4-day period to reduce possible interference with altered fitness during the testing period.

**Blood Sampling**

Prior to the running trials, a flexible catheter was inserted into an antecubical vein, and a blood sample was drawn after 15 min of rest in the supine position. Another sample was taken immediately after terminating the MI or HI trial, then at 5-, 10-, and 15-min post-exercise (either AR or RR), and 30, 60, and 120 min after cessation of the MI or HI trial. Heparinized vacutainers were used and immediately put on ice after sampling. No heparin was used in the catheter, due to its effect on lipolysis. In separate experiments, we tested the possibility of in vitro lipolysis in tubes. Blood from 10 rested and fasted subjects was collected to obtain (a) EDTA-plasma, (b) heparin plasma, and (c) serum. FFA values were (mean ± SE) 0.210 ± 0.040, 0.205 ± 0.045, and 0.226 ± 0.032 mmol/L, respectively. Only 0.9% NaCl solution was used to prevent coagulation in the catheter. The time schedule indicating the trials and blood sampling are presented in Figure 1.

**Analyses**

The concentration of FFA was assessed enzymatically as described by Jebens and Sejersted (17). Albumin was measured using the bromcresol green (BCG) method (10). The hematology variables were analyzed by Technicon H-1 and H-2. Plasma-, blood-, and cell-volume changes were calculated according to Dill and Costill (9).

<table>
<thead>
<tr>
<th>Blood sample#</th>
<th>ACTIVITY</th>
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<th>2-5</th>
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<td>EXERCISE</td>
<td>RR or AR</td>
<td>REST</td>
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<td>75</td>
<td>90</td>
<td>120</td>
<td>180 min</td>
</tr>
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<td>HI (82% of VO$_2$max)</td>
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<td>30</td>
<td>45</td>
<td>60</td>
<td>90</td>
<td>150 min</td>
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</table>

*AR=active recovery, RR=rest recovery, MI=moderate intensity, HI=high intensity*

**Figure 1** — Time schedule for blood sampling.
Design and Statistical Analysis

The project was an experimental randomized cross-over study. The independent variable in this study was the type of recovery (AR or RR). Dependent variables were the blood variables measured throughout the steady-state workouts. The results were analyzed using the SPSS® (v. 6.1 for Windows) statistical computer package. For each experiment (AR and RR) the immediate postexercise value was first subtracted from the succeeding ones. Then the non-parametric Wilcoxon signed rank test for area under the curve (0–15-min, 15–30-min, and 15–120-min post-exercise) was used to estimate whether AR differed significantly from RR. Wilcoxon signed rank test was also performed on the resting values (sample 1) in order to evaluate possible initial differences between the AR and RR experiments. Mean values and standard errors are presented in the result section. A significance level of $p < .05$ was set for the present study.

Results

Variables During High (HI) and Moderate (MI) Intensity Trials

Mean speed, $\dot{V}O_2$, heart rate, and lactate concentration were higher in the HI trial compared to the MI trial, but did not differ between the RR or AR experiments except for lactate in the HI experiment (Table 1). Before any recovery regimen was initiated, there was no difference in RPE, whether between the HI and MI trials, or between AR and RR experiments. When measured 10 min after the onset of exercise,

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Variables During the High (HI)- and Moderate (MI)-Intensity Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>HI ($n = 7$)</td>
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<tr>
<td>Speed (km/h)</td>
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<tr>
<td>RR</td>
<td>9.6 ± 0.0</td>
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<tr>
<td>AR</td>
<td>9.5 ± 0.0</td>
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<tr>
<td>$\dot{V}O_2$ (ml · min⁻¹ · kg⁻¹)</td>
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<tr>
<td>RR</td>
<td>57.4 ± 0.6</td>
</tr>
<tr>
<td>AR</td>
<td>57.1 ± 0.5</td>
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<tr>
<td>Heart rate (bpm)</td>
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<tr>
<td>RR</td>
<td>166 ± 2</td>
</tr>
<tr>
<td>AR</td>
<td>165 ± 2</td>
</tr>
<tr>
<td>RPE (1–10)</td>
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</tr>
<tr>
<td>RR</td>
<td>5.7 ± 0.4</td>
</tr>
<tr>
<td>AR</td>
<td>5.6 ± 0.3</td>
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<tr>
<td>Lactate (mmol/L)</td>
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</tr>
<tr>
<td>RR</td>
<td>2.1 ± 0.1**</td>
</tr>
<tr>
<td>AR</td>
<td>1.9 ± 0.1</td>
</tr>
</tbody>
</table>
lactate concentration stayed fairly constant throughout the rest of the trials. RPE and heart rates increased throughout the experiments for all subjects (data not shown).

**Plasma FFA**

**High Intensity Trial.** Figure 2 (upper panel) illustrates FFA concentrations in response to the HI exercise session lasting 30 min (EX), followed by either 15-min RR or AR, and then rest until 2 hours post-exercise for both experiments. The pre-exercise value for FFA was lower in the AR than in the RR experiment \( (p < .05) \). The increase in FFA concentration was higher after RR \( (0.40 \pm 0.07 \text{ to } 0.67 \pm 0.08 \text{ mmol/L}) \) than after AR \( (0.20 \pm 0.05 \text{ to } 0.30 \pm 0.06 \text{ mmol/L}) \) during the first 5-min PE. At the end of the intervention period, 15-min post-exercise, the FFA concentration after AR was almost 87\% larger than after performing RR. Adjusted for differences immediately PE, AR resulted in lower overall FFA concentrations in the 15-min period of AR, compared to the RR experiment \( (\text{Wilcoxon signed rank test for the area under the curve in the recovery period}) \). After discontinuing the AR, however, the FFA concentrations increased also for this experiment. After RR, there was a reduction in FFA 15–30-min PE. During RR, the return in FFA concentration seemed to start at least 5-min PE. In the time period 15–120-min after exercise, there was in both experiments a trend towards a steady increase in FFA so that approximately the same FFA concentrations were reached for both experiments 120-min PE.

**Moderate Intensity Trial.** There was no difference in the resting FFA concentrations between the RR and AR experiments prior to this trial (Figure 2, middle panel). However, immediately after exercise, the FFA concentrations differed \( (p < .05) \). Again, there was a more rapid increase in FFA concentration in RR than in the AR immediately after the MI trial. Also in this trial, the FFA concentration after RR reached its peak value within 5 min post-exercise. For RR, the area under the curve in the time period 0–15 min post-exercise was significantly higher than the corresponding area for the AR experiment \( (p < .02) \). After cessation of AR, there was an increase in FFA 15–30 min post-exercise. In the RR experiment, the FFA concentration continued to decrease until 30-min of rest. During the rest of the observation period, there were no major changes in the FFA curve for both experiments.

Since the FFA responses to the HI and MI trials were similar, a panel of the pooled HI + MI data is shown in Figure 2, lower panel. For both trials, the FFA/albumin ratio seemed to react upon the different recovery regimens in a similar way as FFA, due to minor changes in the albumin concentration (data not shown).

**Other Variables.** Plasma volume change for the HI trial was \( 12.1 \pm 0.8\% \) after RR and \( 11.4 \pm 1.7\% \) after AR. For the MI trial, the plasma volume change was \( 8.2 \pm 1.7\% \) after RR and \( 10.2 \pm 2.0\% \) after AR. There were no significant differences in the changes of plasma, blood, or red cell volume between RR and AR experiments. Hb values and erythrocyte counts were slightly higher after AR than RR in the 15-min PE sample in the HI trial and MI trial, respectively \( (p = .03) \). Except for this, there were no significant differences in Hb, Hct, or erythrocytes between the recovery regimens in any of the two trials.

In the HI trial, pre-exercise glucose concentrations were significantly higher \( (4.84 \pm 0.16 \text{ vs. } 4.09 \pm 0.18 \text{ mol/L}) \) in the RR experiment than in the AR, and also 15-min post-exercise \( (4.71 \pm 0.26 \text{ vs. } 4.13 \pm 0.15 \text{ mol/L}) \). Plasma glucose concentrations did not differ significantly between RR or AR in the MI trial.
Figure 2 — Free fatty acids (FFA) after high- and moderate-intensity trials, as influenced by active and passive recovery. Seven subjects completed two sets of 30-min running (EX) at 82% of $\dot{V}O_{2\text{max}}$ (high intensity group, HI). One set was followed by rest recovery (RR, — • — ), and the other by active recovery (AR, — □ — ). Another 7 subjects completed two 60-min sets of running at 75% of $\dot{V}O_{2\text{max}}$ (moderate intensity group, MI) followed by either RR or AR. In the lower panel, the pooled HI + MI response is shown. In all three panels, a significant difference ($p < .05$, Wilcoxon signed rank test) was found for the difference between AR and RR as regards the area under the curves during the time periods 0–15 and 15–30 min after exercise. In the HI trial (top), the initial value differed between the AR and RR experiment ($p < .05$).
We also determined plasma TBARS, a measure of lipid peroxidation (29, 30). Baseline values were \( 2.84 \pm 0.3 \) and \( 2.95 \pm 0.30 \) \( \mu \text{mol/L} \) for the RR and AR experiments, respectively. No differences in TBARS were observed after the two recovery regimens. Five, 10, and 30 min post-exercise, plasma TBARS for both recovery regimens were significantly lower than in the 0- and 15-min samples after exercise (results not shown).

**Discussion**

In the present work, the immediate rise in plasma FFA concentration regularly found after exercise was appreciably attenuated by active recovery, as observed in our two exercise trials. Additionally, during active recovery at \( 50\% \text{ of } \dot{V}O_{2\text{peak}} \), 0–15 min post-exercise as compared with rest recovery, there was a lower overall FFA concentration in all subjects. After cessation of the active recovery, however, an increase in FFA concentrations was observed.

FFA are released in order to supply working muscles with substrate for energy production. The hormone sensitive lipase is working on both adipose tissue and intramuscular triglycerides releasing FFA to meet the energy requirements during exercise (18, 24). Additionally, lipoprotein lipase catalyzes the release of FFA from circulating very low density lipoproteins and chylomicrons (13, 21, 24). Immediately post-exercise, however, it has been observed that the FFA concentration may rise appreciably, especially after long-term or high-intensity endurance work (3, 5, 11, 25, 27). This increase is probably reflecting an imbalance between FFA release and uptake during the first minutes after cessation of exercise, where the utilization of FFA is profoundly reduced, while the lipolysis is maintained. A peak FFA is seen 10–15 min post-exercise (11, 31). The FFA increase is related to the prandial state of the individuals, where fasting subjects may obtain higher FFA concentrations than their fed counterparts (18). This difference is presumably attributed to the antilipolytic action of insulin (26). The immediate FFA increase after exercise also depends on the severity of the exercise (25). Exercise of long duration leads to a larger FFA increase than short duration exercise, and high-intensity exercise results in higher concentrations of FFA than low-intensity workouts, the first minutes post-exercise. These differences are probably related to catecholamine concentrations.

In the high-intensity trial, the resting baseline values for FFA and glucose were different. We suggest that this difference may be attributed to some variations in carbohydrate intake. Fat restriction in the 3-hour pre-exercise meal was strongly encouraged, and it was emphasized that only a meal consisting of white bread and jam should be taken. However, it cannot be ruled out that the subjects in the AR experiment had ingested more carbohydrate than in the RR experiment. Consequently, the insulin response and inhibition of lipolytic hormones would be greater in the AR experiment, giving a lower initial FFA value. It could be argued that variations in carbohydrate-stimulated insulin concentrations also governed the FFA curve during AR and RR. However, the rise in FFA concentrations upon discontinuing AR works against this possibility. Also, similar patterns of FFA responses after exercise were observed in the two trials in spite of differences in basal FFA values. Thus, it seems unlikely that differences in basal FFA levels in the high intensity trial had a major influence on the post-exercise FFA response.

In the “moderate” intensity experiment, the immediate post-exercise FFA values were different in the AR and RR experiments. Our explanation for this is that
it took about 30–40 s before blood was collected from the RR group in the first sample. The FFA uptake by the cells would level off immediately at the cessation of exercise due to a rapid decrease in the β-oxidation rate within the previously active muscle cells, and thereby result in a sharp rise in the FFA concentration when the lipolytic rate is not decelerated to the same degree. The subjects in the AR condition were still moving on the treadmill while blood was sampled, and the first sample was drawn quicker compared to the RR experiment.

Looking at the training intensities with respect to percentage of VO₂max, the difference between the high- and moderate-intensity trials might seem small. However, the moderate intensity trial was ensured to be below the lactate threshold for the individuals, while the high intensity trial sought to be within or even above the threshold. This represents a major physiological difference.

Oxidation in muscle and reesterification in adipose tissue of the released FFA (triacylglycerol-FFA cycling) govern the plasma FFA concentration. At rest, about 75% of the FFA is reesterified, and by training 4 hours at 40% of VO₂max, reesterification decreases to 25%. In resting recovery after exercise, reesterification is rapidly increased to 90% (31).

The finding that FFA 0–15 min post-exercise is lowered by AR as compared with RR seems contradictory to the study by El-Sayed et al (11). They detected a sixfold increase in FFA reaching 1.8 mmol/L 10 min into the recovery period after performing a 90-min cycle workout at 75% of VO₂max. Active recovery for 15 min at an intensity of 30% of VO₂max did not attenuate this elevation in FFA. A likely explanation could be that active recovery at an intensity of only 30% of VO₂max might not have been sufficient to achieve the “down-training” effect, especially related to blood flow, to attenuate the post-exercise increase in FFA.

In the present experiments, FFA concentrations did not rise to the high concentrations reported previously (3, 11), probably due to shorter duration of the exercise and the consumption of a light carbohydrate meal 3–4 hours pre-exercise. Unfortunately, the exact energy intake was not recorded. However, in both experiments, the subjects reported that they followed the instructions to have a low fat meal containing bread, jam, and water. In any instance, there was an effect of AR on plasma FFA in the time period 0–15 min post-exercise. The “moderate” intensity trial resulted in slightly higher post-exercise FFA concentrations than the high-intensity trial, probably due to higher reliance on fat as the energy source, since the “moderate” intensity trial was more aerobic and of longer duration than the high-intensity trial. However, the mean FFA concentration for both trials were still below 1 mm, and the FFA/albumin ratio was similar after moderate- and high-intensity exercise.

Exercise is accompanied by increased formation of reactive oxygen species (4), which might promote lipid peroxidation. The present finding that TBARS, a measure of lipid peroxidation (29, 30), did not differ between active and passive recovery, does not support that the observed differences in plasma FFA altered lipid peroxidation.

Active recovery seems to be a favorable way to “down-train” or gradually “deactivate” the body after high-intensity endurance exercise, and its benefits might be more extensive than to stimulate lactate elimination and promote the return of creatine kinase to baseline levels. The type of active recovery used in the present work did not cause permanent FFA lowering. Whether the observed transient reduction in plasma FFA by active recovery may be considered beneficial for particular
types of exercise remains to be settled. Further studies are required to determine which type of active recovery has the greatest impact on the long term FFA concentration after exercise.

References


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**Acknowledgments**

The technical assistance by Eva Kristensen is gratefully acknowledged. We thank professor of statistics, Ingar Holme, Ph.D. for statistical advice, and Dr. Jostein Hallen for valuable discussion in planning the laboratory procedures.