

Fat Oxidation Rate During and After Three Exercise Intensities in Non-Athlete Young Men

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Abstract: The purpose of present study was to compare fat oxidation rate during and after three different exercise intensities. Eight non-athlete male students (aged, 19.7 ± 1.8 yr; height, 174.3 ± 5.1 cm; weight, 64.5 ± 7 kg; VO_{2max} , 40.1 ± 4.4 ml/kg/min) participated in this study as subjects. Each subject was exercised at 55, 65 and 75% VO_{2max} on treadmill at three separate days those days which elapsed by at least one week. Experimental protocol consisted of a 15 min pre-exercise resting period, 60 min exercise period and 2-h recovery period. Indirect calorimetry was used throughout the experimental protocol to measure fat oxidation rate. The ANOVA with repeated measures and Bonferroni were employed to compare fat oxidation rate during and after different exercise intensities. The results of present study showed that absolute fat oxidation rates during and after 75% VO_{2max} and also total absolute fat oxidation rates were significantly higher than other intensities ($P \leq 0.05$). Relative fat oxidation rate during 55% VO_{2max} was significantly higher than other intensities ($P \leq 0.05$), but relative fat oxidation rate and total relative fat oxidation rate were not significantly different after three exercise intensities. Energy expenditure during 75% VO_{2max} and total Energy expenditure were significantly higher than other intensities ($P \leq 0.05$). It was concluded that exercise with 75% VO_{2max} can be beneficial to reduce and maintenance of the weight.

Key words: Fat Oxidation • Post Exercise Recovery • Exercise Intensity • Indirect Calorimetry

INTRODUCTION

Obesity has risen to epidemic proportions [1] and in adults, it is associated with development of Type 2 diabetes and other chronic diseases such as coronary heart disease (CHD) and some forms of cancer. Carbohydrate (CHO) and fatty acids are the dominant fuels oxidized by the muscle for energy production during and after exercise and that the absolute and relative contribution of these fuels can be influenced by diet [2, 3], muscle glycogen content [4, 5], exercise intensity [6, 7], duration [8]. One of the most important regulators of substrate oxidation is exercise intensity, because it has been demonstrated that increases in glycolytic flux will inhibit long-chain fatty acid transport into the mitochondria and therefore reduce long-chain fatty acid oxidation [9]. The intensity associated with the highest rate of fat oxidation is between 55 and 75% VO_{2max} , shown in several recent studies [8, 10, 11, 12, 5]. This wide range of exercise intensities may have been a

consequence of different study protocols, subject groups or type of exercise. At moderate intensities, lipolysis of subcutaneous fat is enhance, [1] while it is limited at high intensities of approximately 80-85% of maximal oxygen consumption (VO_{2max}) [13, 3]. Recent studies revealed that the optimal intensity for fat oxidation is at rather high intensities of 65 to 75% VO_{2max} in endurance-trained athletes [2, 3]. In earlier years, Romijn et al showed that endurance-trained men have their highest fat oxidation rate at 65% VO_{2max} [14]. They recently performed the same protocol with endurance-trained women and confirmed their prior results showing the highest fat oxidation at 65% VO_{2max} [12]. Different results were found by Astorino, who showed that endurance-trained women have their highest fat oxidation rate at 75% of peak oxygen consumption (VO_{2peak}) [10]. In more recent studies, highest fat oxidation rate is presumed to be at even lower intensities of 57% VO_{2max} [5] and 65% VO_{2max} [15], respectively. Whereas extensive studies have focused on the relative partitioning of lipids and

carbohydrates as fuels during exercise [10, 16, 12, 17], less effort has been aimed at uncovering the metabolic events during postexercise recovery. Results show that the relative contribution of lipids decreases as exercise intensity increases [$>60\%$ VO_2 peak] [18]. Thus, notwithstanding the preferential use of CHO during moderate- to high-intensity exercise, it is possible to hypothesize that lipid oxidation predominates during recovery, especially after physical exercise leading to glycogen depletion [19]. Although it was observed that the respiratory exchange ratio (RER) becomes very low during recovery from prolonged exercise [4], energy partitioning after exercise was largely ignored. Still, fewer have investigated substrate partitioning during postexercise recovery. Moreover, limited data exist on energy substrate partitioning in women during the recovery period [19, 20]. Although Kiens and Richter have provided data showing intramuscular triglyceride mobilization in the hours after high-intensity exercise [21] and Horton et al showed that lipid oxidation is promoted in men [22], but not women, during recovery from 2 h of mild- to moderate intensity (40% VO_2 peak) exercise, Melanson et al have concluded that physical activity bouts employing intensities and durations typically used in physical fitness and body weight management programs do not significantly impact lipid oxidation or daily total energy expenditure. The present research is done in order to compare fat oxidation rate during and after three different exercise intensities and to response to the following essential question:

Are there significant different on fat oxidation rate during and after three different exercise intensities or no?

MATERIALS AND METHODS

Subjects: Physiological characteristics of the subjects are presented in Table 1. Eight non athlete healthy young men gave informed written consent to participate in the study after gaining approval from the Human Ethics Committee of Guilan University. To determine the level of physical activity subjects completed a health and activity questionnaire prior to the study in which they had to rate the severity and amount of their physical activity over a typical 7 day period. Subjects with a history of cardiovascular symptoms, as well as those taking any medication were excluded.

Table 1: Mean (\pm SD) values of the subjects' characteristics

Variables	Mean \pm SD
Age (years)	19.75 \pm 1.83
Height (cm)	174.37 \pm 5.15
Body weight (kg)	64.56 \pm 7.02
BMI (kg/m^2)	21.27 \pm 1.86
Body fat (%)	12.58 \pm 2.80
VO_2max ($\text{ml}/\text{kg}/\text{min}$)	40.08 \pm 4.49

VO_2max , maximal oxygen uptake, BMI, Body Mass Index

Experimental Protocols: Subjects reported to the laboratory on four separate occasions. The first session was designed to familiarize the Subjects with the procedures and to determine their maximal oxygen consumption (VO_2max). Height, body weight and percent of body fat using bioelectrical impedance (InBody 3.0, Biospace, Korea) were also determined (Table 1). The subsequent three sessions involved three exercise trials, which were performed at 55, 65 and 75% VO_2max for 60 min on a treadmill [7]. All exercise trials were allocated randomly in a counterbalanced manner and separated by at least 7 days. Subjects completed one day food diary on the day before their first test and repeated this diet before the subsequent trials. In addition, subjects were requested to refrain from drinking alcohol, nor to engage in any kind of strenuous exercise in the 24 h before trials.

Determination of VO_2max : VO_2max was determined using Bruce protocol on treadmill until volitional fatigue. After 5 min warm-up and performing stretching exercises the test commenced and continued until the participant was fatigued. Expired gas analysis was acquired with a Gas analyzer (Quark b² 7.5, Cosmed, Italy) and VO_2max was calculated as the highest oxygen consumed over a 1 min period. For the duration of the test, heart rate was measured continuously using a heart rate monitor (PE30800, Polar Electro, Kempe, Finland). VO_2 max was confirmed using established physiological criteria from the British Association of Sport and Exercise Science (BASES) and included a respiratory exchange ratio (RER) above 1.15, oxygen uptake reaching a plateau with increasing work rate, a heart rate close to age predicated maximal values and a rating of perceived exertion (RPE) of 20.

Main Experiments: In general, the main experiments consisted of a preexercise rest period, an exercise period and a 2 h postexercise rest period. The preexercise rest period was of 15 min duration and it was initiated when the subjects seemed to be in a steady state with respect to whole body oxygen consumption. This period was

followed by an exercise period during which the subjects either exercised for 60 min at 55, 65 and 75 % of their VO_2max . After the exercise period, the subjects were studied for another 2 h during rest. Both the pre and postexercise periods were performed in a sitting position. Oxygen uptake and carbon dioxide output were measured continuously during the test by means of a Gas analyzer (Quark b² 7.5, Cosmed, Italy), using face mask and breath by breath techniques. The order of experiments was randomized. Subjects reported to the physiology laboratory at 08.00 h after an overnight fast. In the days prior to the experiments, they ate their habitual diet and they performed their habitual physical activities. However, from 24 h before the experiments they refrained from vigorous physical activity [19].

Statistical Analyses: The ANOVA with repeated measures was employed to evaluate the differences in the mean values of fat oxidation during and after exercise trials. When ANOVA indicated the presence of a significant difference, post-hoc comparisons using the Bonferroni's method were applied to determine pairwise differences. All data are given as Mean \pm SD and the level of significance was set at $P < 0.05$.

RESULTS

Energy Expenditure: Statistical analyses revealed a significant effect of exercise intensity on the rate of energy expenditure during exercise and total energy expenditure ($F = 32.274$, $P = 0.004$). Post-hoc analyses revealed a significant difference in rate of energy expenditure during exercise and total energy expenditure between 55 and 75 % VO_2max ($P < 0.05$). Furthermore, there was no significant difference between rate of energy expenditure after 55, 65 and 75 % VO_2max (Figure 1).

Fat Oxidation: Rate of absolute fat oxidation (g/min) during exercise and total absolute fat oxidation increased significantly with exercise intensity ($P < 0.05$). ANOVA with repeated measure revealed a significant difference between absolute fat oxidation during exercise and total absolute fat oxidation in three exercise intensity ($F = 53.33$, $P = 0.001$). Post-hoc analyses revealed that observant difference was significant between 55 and 75 % VO_2max only (Figure 2). In addition, relative fat oxidation during exercise decreased significantly with exercise ($P < 0.05$). ANOVA with repeated measure revealed a significant difference between relative fat oxidation during three intensity ($F = 18.009$, $P = 0.013$). Post-hoc analyses showed

that observant difference was significant between 55 and 75 % VO_2max only (Figure 4). In post exercise recovery period, absolute fat oxidation increased significantly with intensity ($P < 0.05$). Results indicated a significant difference between absolute fat oxidation after three exercise intensities ($F = 302.22$, $P = 0.00$). Post-hoc analyses revealed that observant difference was significant between 55 and 75 % VO_2max only (Figure 2). In addition, relative fat oxidation after exercise increased with intensity, although there was no significant difference between trials (Figure 4).

CHO Oxidation: Absolute CHO oxidation during exercise at the three intensities and total absolute CHO oxidation increased with intensity; however, there was no significant difference in absolute CHO oxidation during exercise at the three intensities (Figure 3). Statistical analyses indicated a significant increase in relative CHO oxidation during exercise at the three intensities ($F = 18.00$, $P = 0.013$). Post-hoc analyses revealed a significant difference in relative CHO oxidation during exercise at 55 and 75 % VO_2max (Figure 4). In post exercise recovery period, absolute CHO oxidation decreased with intensity ($P < 0.05$). ANOVA with repeated measure indicated that there was no significant difference in absolute CHO oxidation and also in relative CHO oxidation at three intensities (Figure 3).

DISCUSSION

One of the most important regulators of fat and carbohydrate oxidation during exercise and recovery is exercise intensity [6, 16, 11, 12, 5]. In this study, absolute fat oxidation during exercise increased with intensity, the greatest rates of absolute fat oxidation was observed during 75 % VO_2max but relative fat oxidation decreased with intensity and the greatest rates of relative fat oxidation was observed during 55 % VO_2max . One possible reason that relative fat oxidation decrease between 55 and 75 % VO_2max could be the effects of high intensity exercise on stimulation of muscle glycogenolysis and glucose uptake [14]. Another possible explanation could be the inhibition of long chain fatty acid entry into the mitochondria, possibly mediated by the increases in glycolytic flux during high intensity exercise [9], or down regulation of carnitine palmitoyl transferase I by either a decrease in free carnitine availability or a reduction in PH due to increase in lactate production during high intensity exercise [5]. Lipolysis rate, blood flow to adipose tissue and blood flow to skeletal muscle increased with intensity,

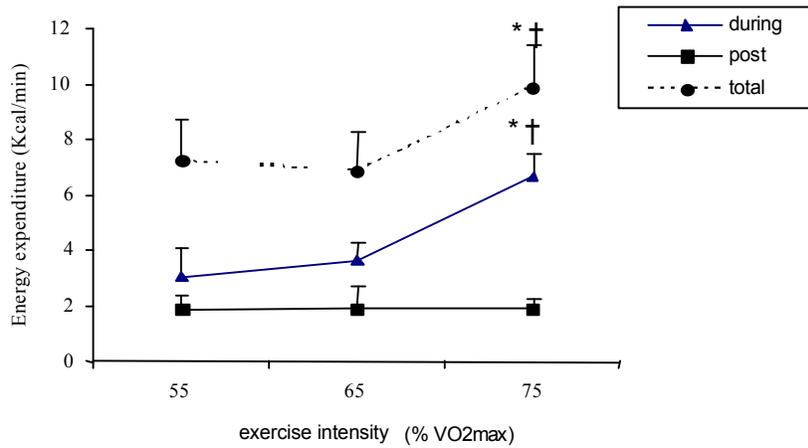


Fig. 1: Mean (SE) of energy expenditure (kcal/ min) at three different intensities (55%, 65% and 75%) of VO₂max
 * Significant different from 55% VO₂max (p<0.05) † Significant different from 65% VO₂max (p<0.05)

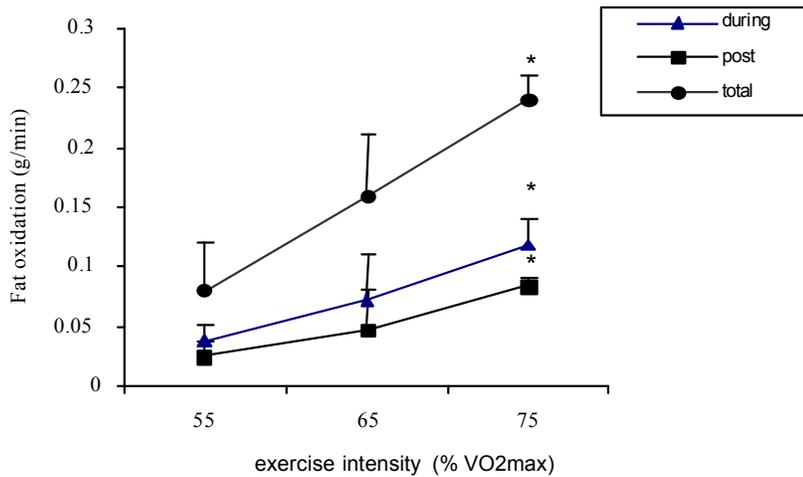


Fig. 2: Mean (SE) of absolute fat oxidation (g/ min) at three different intensities (55%, 65% and 75%) of VO₂max.
 * Significant different from 55% VO₂max (p<0.05)

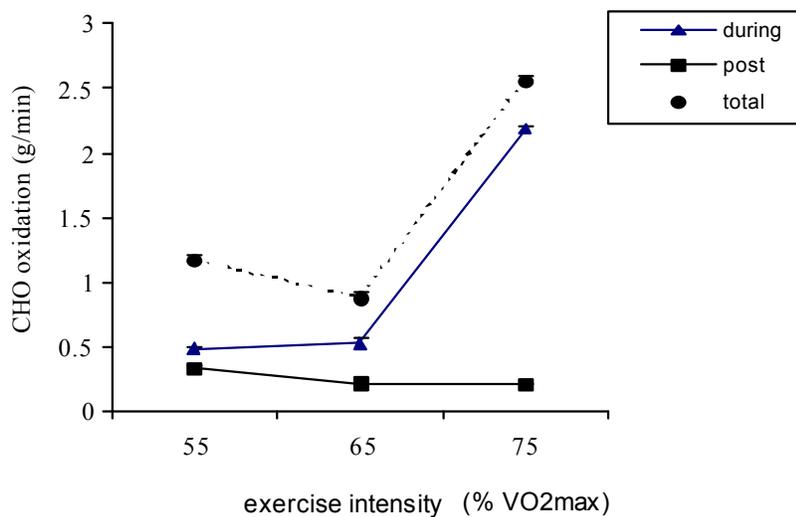


Fig. 3: Mean (SE) of absolute CHO oxidation (g/ min) at three different intensities (55%, 65% and 75%) of VO₂max.

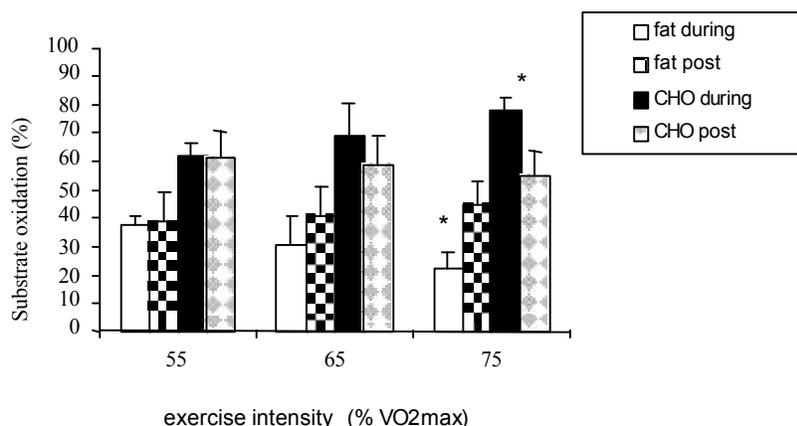


Fig. 4: Mean (SE) of absolute CHO oxidation (g/ min) at three different intensities (55%, 65% and 75%) of VO₂max
 * Significant different from 55% VO₂max (p<0.05)

that this result in fatty acid availability for skeletal muscle. There for, the rates of absolute fat oxidation increase [23]. With increase in exercise intensity above 80% VO₂ max, rates of fat oxidation declined and CHO oxidation increased that might be due to the increased glycolytic flux during high intensity exercise and inhibition of long fatty acid into mitochondria, their oxidation decreased. The increased fat oxidation for the transition from light intensity to moderate intensity is a direct result of the increased energy expenditure [8]. During exercise with 65-70 % VO₂ max, lipolysis increases approximately threefold, mainly because of an increased β-adrenergic stimulation by catecholamines and also a reduction in insulin. In addition, the blood flow to adipose tissue is reduced, resulting in an increase in the rate of re-esterification. Blood flow to skeletal muscle is increased dramatically and there for the delivery of fatty acid to the muscle is increased. In most studies, the greatest rate of fat utilization occurs at approximately 60-65 % VO₂max [12, 5]. If low and high intensity exercise equated in terms of time rather than total energy expenditure, high intensity exercise would use more grams of fat. Conversely, when total energy expenditure is held constant, low intensity exercise will utilize more fat grams compared with high intensity exercise.

In this study, absolute fat oxidation and also relative fat oxidation in post exercise recovery period increased with intensity and the greatest rate of absolute and relative fat oxidation observed in recovery from 75 % VO₂ max. our data corroborate results of previous studies showing that exercise raises total energy expenditure during exercise and shift the pattern of whole body metabolism toward lipid oxidation during the post exercise

recovery period [24, 21, 25, 20]. Although we did not sample tissue glycogen contents during our study, on the basis of extensive previous work [4, 26], our data can be interpreted to suggest that muscle and liver glycogen stores are mobilized to support a majority of energy expenditure during exercise.

It has been shown that the body prioritizes the resynthesis of glycogen during post exercise recovery [4, 26]. The increase in relative fat contribution during recovery from high intensity exercise in our study is consistent with the idea that the body will increase its fat oxidation for energy, while sparing CHO to minimize further challenge to glycemia and to facilitate subsequent restoration of glucose homeostasis and glycogen depletion. The mechanism for fat oxidation during recovery is not readily present. Previous studies [26, 4, 27] showed that IMTG content was unchanged during recovery, suggesting muscle lipid stores are not an appreciable source of fatty acids for increasing fat oxidation. There for, it is likely that elevated plasma fatty acids during recovery resulting in an increased uptake and oxidation of fatty acids within glycogen-depleted muscle. This is supported by the rapid decline in plasma fatty acid levels early in recovery, which can probably be attributed to both the rapid clearance by peripheral tissue, including skeletal muscle, as well as an insulin-mediated reduction in peripheral lipolysis [3, 4]. Norepinephrine hormone has a key role in elevated fat oxidation during recovery after exercise [28]. It may also be that the greater growth hormone response during exercise could explain the elevation of lipolysis in recovery [26]. Indeed, it has been proposed that growth hormone secretion during exercise could lead to elevated

lipolysis and fat oxidation in recovery. Growth hormone leads to decreased glucose uptake and increased fatty acid mobilization [29].

Additionally, the increased FA utilization in recovery may be related to depletion of CHO removing energy substrate competition between oxidative glycolysis and β -adrenergic [30, 31] or alternatively to stimulation of energy sensing pathways such as activation of AMPK in tissues such as muscle and liver [26].

Most studies showed that increased fat contribution after exercise in comparison with during exercise [24, 21, 20]. In addition to, most previous studies indicated that fat contribution to total energy expenditure during high intensity exercise further than low intensities exercise [32, 27]. This observed in this study, also. In this study, energy expenditure was significantly increased within 55 to 75 % VO_2 max, but energy expenditure during recovery, in spite of little increase in high intensities in high when energy expenditure during exercise with different intensities held constant, the rates of energy expenditure and total substrate utilization in recovery is same [23, 22, 19].

Unfortunately, none of these studies measured the degree of muscle glycogen depletion induced by the exercise. In order to more fully understand post exercise substrate utilization, the role of glycogen depletion needs additional clarification. In this study, in spite of higher energy expenditure during high intensities exercise, no significant difference between energy expenditure in recovery of 55, 65 and 75 % VO_2 max. The reason of it maybe shifts the pathway of substrate utilization, including decreased CHO oxidation and increased fat oxidation.

Generally, according to this study results it was concluded that exercise program with 75% $\text{VO}_{2\text{max}}$ can be beneficial to reduce the weight. Therefore, it seems that the 75% training program is the best exercise training for oxidation of fat and body weight control.

REFERENCES

1. Mokdad, A.H., E.S. Ford, B.A. Bowman, W.H. Diets, F. Vinicor, V.S. Bales and J.S. Marks, 2003. Prevalence of obesity, diabetes and obesity-related health risk factors. *JAMA*, 289: 76-79.
2. Coyle, E.F., A.E. Jeukendrup, M.C. Oseto, B.J. Hodgkinson and T.W. Zderic, 2001. Low-fat diet alters intramuscular substrates and reduces lipolysis and fat oxidation during exercise. *Am. J. Physiol. Endocrinol Metab.*, 280: E391-E398.
3. Horowitz, J.F., R. Mora-Rodriguez, L.O. Byerley and E.F. Coyle, 1997. Lipolytic suppression following carbohydrate ingestion limits fat oxidation during exercise. *Am. J. Physiol.*, 273: E768-E775.
4. Kimber, N.E., G.J.F. Heigenhauser, L.L. Spriet and D.J. Dyck, Skeletal muscle fat and carbohydrate metabolism during recovery from glycogen-depleting exercise in humans. *J. Physiol.*, 548: 919-927.
5. Van Loon, L.J.C., A.E. Jeukendrup, W.H.M. Saris and A.J.M. Wagenmakers, 1999. Effect of training status on fuel selection during submaximal exercise with glucose ingestion. *J. Appl. Physiol.*, 87: 1413-1420.
6. Bassami, M., S. Ahmadizad, D. Doran and D.P.MN. MacLaren, 2007. Effects of exercise intensity and duration on fat metabolism in trained and untrained older males. *Eur. J. Appl. Physiol.*, 101: 525-532.
7. Bircher, S. and B. Knechtle, 2004. Relationship between fat oxidation and lactate threshold in athletes and obese women and men. *J. Sports Sci. and Med.*, 3: 174-181.
8. Achten, J. and A.E. Jeukendrup, 2003. Maximal fat oxidation during exercise in trained men. *Int. J. Sports Med.*, 24: 603-608.
9. Coyle, E.F., A.E. Jeukendrup, A.J. Wagenmakers and W.H. Saris, 1997. Fatty acid oxidation is directly regulated by carbohydrate metabolism during exercise. *Am. J. Physiol. Endocrinol. Metab.*, 273: E268-E275.
10. Astorino, A., 2000. Is the ventilatory threshold coincident with maximal fat oxidation during submaximal exercise in women? *J. Sports Med. Phys. Fit.*, 40: 209-216.
11. Romijn, J.A., E.F. Coyle, L.S. Sidossis, A. Gastaldelli, J.F. Horowitz, E. Endert and R.R. Wolfe, 1993. Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity. *Am. J. Physiol.*, 265: E380-E391.
12. Romijn, J.A., E.F. Coyle, L.S. Sidossis, J. Rosenblatt and R.R. Wolfe, 2000. Substrate metabolism during different exercise intensities in endurance trained women. *J. Appl. Physiol.*, 88: 1707-1714.
13. Blair, S., 2002. The public health problem of increasing prevalence rates of obesity and what should be done about it. *Mayo. Clin Proc.*, 77: 109-113.
14. Romijn, J.A., E.F. Coyle, X.J. Zhang, L.S. Sidossis and R.R. Wolfe, 1995. Fat oxidation is impaired somewhat during high-intensity exercise by limited plasma FFA mobilization. *J. Appl. Physiol.*, 79: 1939-1945.

15. Pillard, F., C. Moro, I. Harant, E. Garrigue, M. Lafontan, M. Berlan, F. Crampes, I. De-Glisezinski and D. Riviere, 2007. Lipid Oxidation According to Intensity and Exercise Duration in Overweight Men and Women. *Obesity*, 15: 2256-2262.
16. Kang, J., J.R. Hoffman, N.A. Ratamess, A.D. Faigenbaum, M. Falvo and M. Wendell, 2007. Effect of exercise intensity on fat utilization in males and females. *J. Sports Med.*, 15: 175-188.
17. Venables, M.C., J. Achten and A.E. Jeukendrup, 2005. Determinants of fat oxidation during exercise in healthy men and women: a cross-sectional study. *J. Appl. Physiol.*, 98: 160-167.
18. Friedlander, A.L., G.A. Cassaza, M.A. Horning, A. Usaj and G.A. Brooks, 1999. Endurance training increases fatty acid turnover, but not fat oxidation, in young men. *J. Appl. Physiol.*, 86: 2097-2105.
19. Kuo, C.C., J.A. Fattor, G.C. Henderson and G.A. Brooks, 2005. Lipid oxidation in fit young adults during post-exercise recovery. *J. Appl. Physiol.*, 99: 349-356.
20. Pruett, E.D.R., 1970. FFA mobilization during and after prolonged severe muscular work in men. *J. Appl. Physiol.*, 29: 809-815.
21. Kiens, B. and E.A. Richter, 1998. Utilization of skeletal muscle triacylglycerol during post-exercise recovery in humans. *Am. J. Physiol. Endocrinol. Metab.*, 275: E332-E337.
22. Horton, T.J., M.J. Pagliassotti, K. Hobbbs and J.O. Hill, 1998. Fuel metabolism in men and women during and after long- duration exercise. *J. Appl. Physiol.*, 85: 1823-1832.
23. AL Mulla, N., L. Simonsen and J. Bulow, 2000. Post-exercise adipose tissue and skeletal muscle lipid metabolism in humans: the effects of exercise intensity. *J. Appl. Physiol.*, 524: 919-928.
24. Bielinski, R., Y. Schutz and E. Jequier, 1985. Energy metabolism during the post-exercise recovery in man. *Am. J. Clin Nutr.*, 42: 69-82.
5. Melanson, E.L., T.A. Sharp, H.M. Seagle, T.J. Horton, W.T. Donahoo, G.K. Grunwald, J.T. Hamilton and J.O. Hill, 2002. Effect of exercise intensity on 24-h energy expenditure and nutrient oxidation. *J. Appl. Physiol.*, 92: 1045-1052.
26. Henderson, G.C., J.A. Fattor, M.A. Horning, N. Faghihnia, M.L. Johnson, T.L. Mau, M. Luke-Zeitoun and G.A. Brooks, 2007. Lipolysis and fatty acid metabolism in men and women during the postexercise recovery period. *J. Physiol.*, 584: 963-981.
27. Thompson, D.L., K.M. Townsend, R. Boughey, K. Patterson and D.R. Bassett, 1998. Substrate use during and following moderate- and low-intensity exercise: Implications for weight control. *Eur. J. Appl. Physiol.*, 78: 43-49.
28. Arner, P., E. Kriegholm, P. Engfeldt and J. Bolinder, 1990. Adrenergic regulation of lipolysis in situ at rest and during exercise. *J. Clin Invest.*, 85: 893-898.
29. Robergs, R.A. and S.O. Roberts, 1997. Exercise physiology: Exercise, performance and clinical application. Boston: WCB McGraw-Hill.
30. Ashley, C.D., M.L. Kramer and P. Bishop, 2000. Estrogen and substrate metabolism. *Sports Med.*, 29: 221-227
31. Sedlock, D.A., 1991. Effect of exercise intensity on postexercise energy expenditure in women. *Br. J. Sp. Med.*, 25: 221-223.
32. Steffan, H.G., W. Elliott, W.C. Miller and B. Fernhall, 1999. Substrate utilization during submaximal exercise in obese and normal-weight women. *Eur. J. Appl. Physiol.*, 80: 233-239.