ABSTRACT

This study was undertaken to investigate three aspect of the adaptations that occur in skeletal muscle as a result of endurance training. First, rats were trained at three different intensities [20 m/min (TI), 27 m/min (TII), and 35 m/min (TIII) for 1 hr./day during weeks 5 and 6 of training] to ascertain the influence of intensity of training on the activity of succinate dehydrogenase and on the endurance (run time to exhaustion) of the rats. Training increased muscle succinate dehydrogenase and endurance to approximately the same extent in all three trained groups which suggest that the intensity of exercise training does not greatly alter the muscle respiratory capacity nor the endurance of trained rats.

Secondly, rats trained on training schedule TIII were sacrificed at 2 and 6 weeks of training and at 1, 2, and 3 weeks after termination of training to determine how quickly the succinate dehydrogenase adaptation occurs with training, and how quickly it disappears upon detraining. At 2 weeks of training, an approximately 10% increase in succinate dehydrogenase was observed, and at 6 weeks about a 30% increase. At one week of detraining, the difference in muscle respiratory capacity had dropped to approximately half that found at the end of training, but then remained at about the level through the third week of detraining.

In the third series of experiments, rats were trained on training schedule TIII in order to determine the effect of exercise training on the oxidation of both saturated and unsaturated fatty acids. Training increased the oxidation rates of both saturated and unsaturated fatty

acids, and there was no alteration in the preferential order of oxidation, i.e., oleate > palmitate > linoleate > stearate.

A STUDY
OF SOME BIOCHEMICAL
ADAPTATIONS TO EXERCISE TRAINING

A Thesis

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the Faculty of the Department of Biology

East Carolina University

In Partial Fulfillment

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Master of Arts in Biology

by

Thomas Patrick Stephenson

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Dedicated

to

My Father

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INTRODUCTION

It is generally accepted that the immediate energy source for muscular contraction comes from the hydrolysis of the terminal phosphate bond of adenosine triphosphate (ATP). ATP is supplied to the contractile tissue primarily from the oxidation of the carbohydrates and fats stored in the muscle or brought to it by the circulatory system. Therefore, there are two factors which primarily regulate the ATP supply to the muscle tissue during exercise; (1) the levels of activity of the enzymes which ultimately leads to the production of ATP, i.e., the level of enzymes present and the modulation of their activity (allosteric regulation, etc.), and (2) availability of oxidizable substrates and oxygen, i.e., the quantity of substrates stored and/or the mobilization of these storage fuels which when metabolized leads to the production of ATP.

Chronic exercise has been shown to induce a variety of biochemical changes in skeletal muscle, which ultimately result in the trained individuals having a greater capacity of ATP production. Thus the trained individual is able to perform a heavier work load over a longer period of time.

In relation to the storage and catabolism of carbohydrates, Gollnick et al.(1) have reported that the muscle tissue of trained individuals contains greater stores of glycogen than the muscle tissue of untrained individuals. Huston et al. (2) reported that several key enzymes (phosphorlase, phosphofructokinase, hexokinase) of glycolysis undergo an adaptive increase in their activities with training of the individual. Thus the trained individual has obviously adapted to the

greater demand for energy during exercise by storing greater amounts of glycogen and by increasing its capacity to degrade glycogen and synthesize ATP.

The triglycerides stored in liver have been shown to decrease in response to exercise training (3), however, the storage of triglycerides in muscle tissue in response to exercise training is unclear as Morgan et al. (4) reported that triglycerides were increased by exercise training; Froberg et al. (3) reported that triglycerides were lowered; and Dohm et al. (5) reported that no alternation of triglycerides took place as a result of exercise training. Nevertheless, the level of fats available is sufficient to furnish a major portion of the greater demand for energy during exercise of the physically conditioned animal.

It has been demonstrated that an adaptation occurs in the lipolytic system of adipose tissue with physical training which is manifested by a greater rate of release of fatty acids from the adipose tissue in the trained than in the untrained animal during exercise (6,7). Likewise it has been demonstrated that training increases the rate of palmitate oxidation in muscle tissue (8,9). Since it has been shown that the relative amounts of carbohydrates and fats utilized at different work loads is dependent on the physical fitness of the animal, with fatty acid oxidation serving as a more important source of energy in the physically trained, than in the untrained (6,7,10,11), then it appears probable that the increase in palmitate oxidation acts in synergy with the increase in the rate of fatty acid mobilization to enable the physically trained animal to obtain a greater portion of energy required during exercise from fats.

It has previously been demonstrated that unsaturated fatty acids are oxidized at a faster rate than are saturated fatty acids (12,13). Therefore another possible adaptation that could occur in the muscle tissue of trained individuals would be to increase the rate of oxidation of saturated fatty acids in relation to the rate of oxidation of unsaturated fatty acids and/or to alter the degree of saturation of the fatty acids stored in the adipose tissue or muscle.

Skeletal muscle mitochondria undergo an adaptive increase in both size and number with exercise training (14,15). Accompanying this increase in size and number is an increased level of activity of the enzymes of the citric acid cycle (16,17,18) and of the mitochondrial respiratory chain (19,20,21,22). These adaptations better equip the trained individual to metabolize all sources of fuels, whether they be carbohydrate or fat.

Little attempt has been made to correlate biochemical adaptations to exercise with the change in the performance capacity of the individual, and several authors (20,23,24) have taken the point of view that unless biochemical adaptations can be shown to accompany a change in the performance capacity of the isolated muscle and/or the total animal, then their relevance to the general problem of exercise adaptation is dubious.

The purpose of the research reported in this thesis was to study three aspects of the adaptations that occur in muscle energy metabolism as a result of endurance training. The three aspects are (1) to determine how fast the previously reported (16,17) adaptation of an increase in succinate dehydrogenase activity with training occurs during the training schedule and how fast it disappears upon detraining of the animal, (2) to determine whether or not the increase in succinate

dehydrogenase activity is proportional to the amount of work done or to the intensity of the exercise training and (3) to determine if muscle oxidation of all fatty acids (saturated and unsaturated) is increased by exercise training.

MATERIALS AND METHODS

Care of Animals:

From previous experience, it had been found that Sprague-Dawley rats can be trained to run on a treadmill, therefore, male Sprague-Dawley rats initially weighing approximately 220g were choosen as the experimental animals. After having received the rats from Holzman Company, Madison, Wisconsin, the animals were randomized and placed into individual cages within the animal facility where temperature was held constant at 22°C. The animals were kept on a normal day-night cycle and allowed food (commerical lab chow) and water *ad libitum* throughout the experimental period. The animals were weighed and inspected weekly to ascertain whether or not they were in a healthy condition.

Training of Animals:

All animals placed into the untrained category (UT) were allowed to remain sedentary in their cages throughout the experimental period. Trained animals were run on a treadmill daily between the hours of 0800 and 1200 six days a week. Animals which could not keep up with their respective training schedule due to either injury or respiratory sickness were discarded from the experiment. Three training regimens designated as TI, TII, and TIII were used in the training of the rats. All trained groups were started on their respective regimen on Monday of week one at 15 m/min for 15 minutes and gradually increased in time, rate and grade as indicated in Table 1.

Table 1. The Speed, Time, and Grade for the Three Treadmill Training Regimens

Week*	Time (min)	Grade (%)	Treadmill Speed (m/min)		
			Trained I	Trained II	Trained III
1	30	0	20	20	20
2	40	0	20	27	30
3	50	0	20	27	35
4	50	8	20	27	35
5	60	8	20	27	35
6	60	8	20	27	35

* The speed and time are given for the last day of the week. Rats on all three regimens ran 6 days per week.

Preparation of Homogenate:

Rats were sacrificed in a rested condition (for trained rats, 24 hours after their last exercised bout) by decapitation, the gastrocnemius muscles were quickly excised and placed into an ice-cold isotonic solution. The muscle tissue was then minced and connective tissue removed by the use of a tissue press equipped with a pair of stacked sieves with 1.0 mm and 0.5 mm diameter holes respectively. The muscle tissue was weighed, and one part muscle was then homogenized with nine parts of an homogenization media containing 0.1 M potassium chloride, 0.5 M Tris-HCl (pH 7.5), 0.005 M $MgSO_4$ and 0.001 M EDTA, in a Ten-Brock glass-glass homogenizer. The homogenates being prepared for the succinate dehydrogenase assay were then filtered through two layers of coarse cheese cloth to remove large fibrous material which caused turbidity and interferred with the photometric assay. The homogenates prepared for the fatty acid oxidation assay were not filtered.

Succinate Dehydrogenase Assay:

The assay of succinate dehydrogenase activity was accomplished essentially as the method described by King (25). The assay mixture consisted of the following in a final volume of 2 ml: 100 mM potassium phosphate (pH 7.8), 0.1% bovine serum albumin, 20 mM succinate, 1 mM potassium ferricyanide, 0.4 ug rotenone and 200 ul of the homogenate.

The assay was performed on a Beckman Model DU Quartz Spectrophotometer connected to a Gilford 2000 multiple sample absorbance recorder. The steps of the reaction being measured may be observed in Figure 1.

Assay of Fatty Acid Oxidation

The oxidation rates of palmitate-1-C^{14}, stearate-1-C^{14}, oleate-1-C^{14},

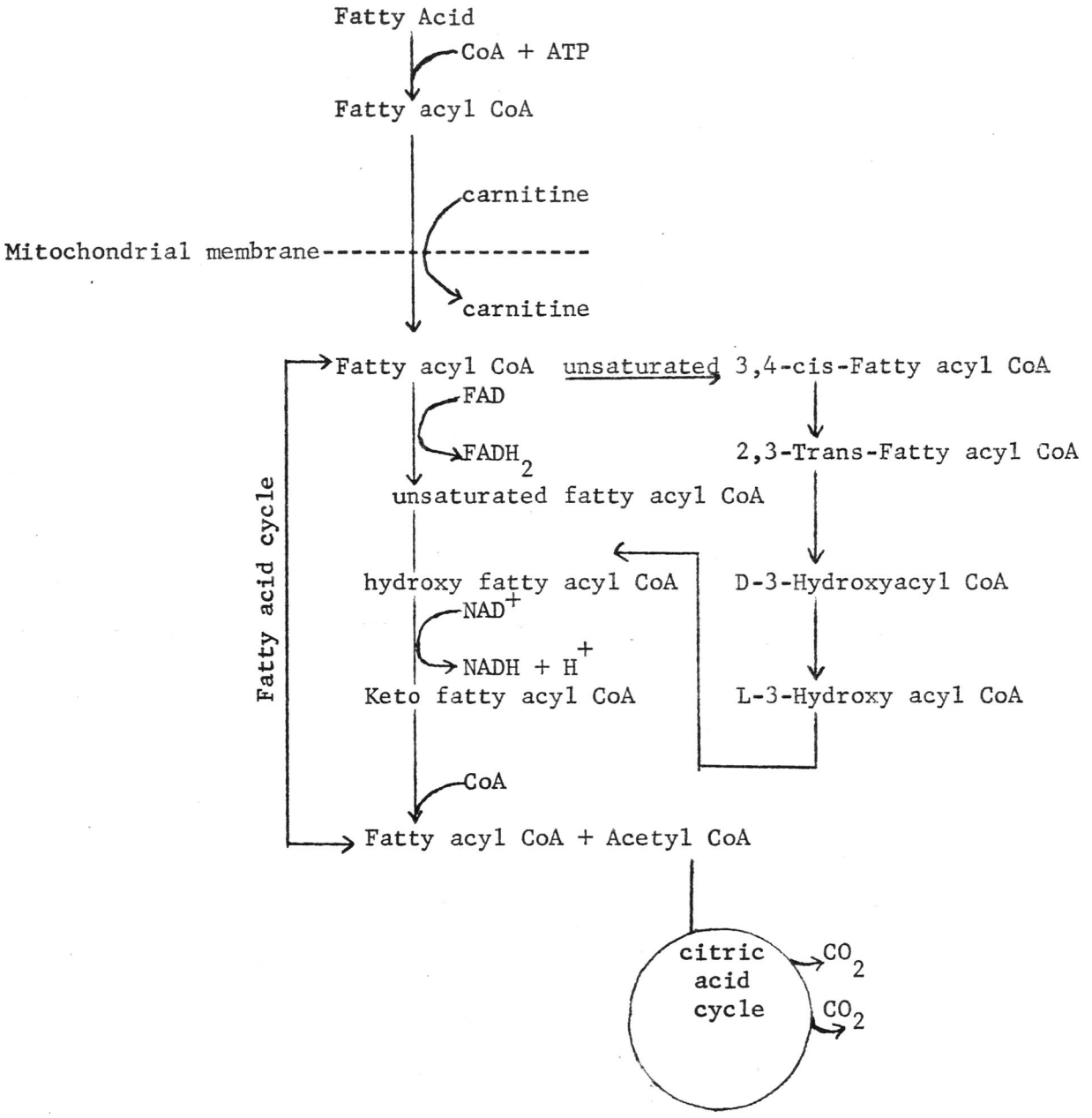
and linoleate-1-C^{14} were measured by collecting and counting the $^{14}CO_2$ produced during an incubation period as described by Dohm et al. (8). A 2.0 ml incubation mixture was used containing the following cofactors (pH 7.4) in millimoles per liter: DL. carnitine, 1.0; ATP,2.0; coenzyme A,0.05; dithiothereitol, 1.0; malate, 0.1; $MgCl_2$1.0; bovine serum albumin, 0.072; NAD^+, 0.1; sucrose, 100; K_2HPO_4,10; KCl,80; and EDTA, 0.1. The substrates used were 0.2 mM palmitate-1-C^{14} (0.1 uCi), 0.2 mM stearate-1-C^{14} (0.1 uCi), 0.2 mM oleate-1-C^{14}(0.1 uCi) and 0.2 mM linoleate-1-C^{14} (0.1 uCi). The reaction was initiated by the addition of 0.5 ml of the homogenate. The contents of the 25 ml Erlenmeyer flasks were then gassed with oxygen and quickly stoppered with a rubber-septum stopper containing a polyproplylene center well cup, and incubated with gentle shaking at 37°C for 30 minutes. Immediately prior to the termination of the incubation reaction, 0.2 ml of ethanolamine: methylcellosolve (1:2) was injected into the handing center well cup. The reaction was then terminated by injection of 0.2 ml of 4.0 N H_2SO_4 into the contents of the flask. The flask were then shaken for 60 minutes to collect evolved CO_2. Next a 0.15 ml aliquot of the center well contents was transferred to a scintillation vial containing 10 ml of scintillation fluid made up of one part Triton X-100 and two parts toluene scintillation solution composed of 8.25 g PPO and 0.25 g M_2POPOP per liter of toluene. The samples were then counted for 20 minutes in a Packard model 3320 liquid scintillation spectrometer.

The steps of the reaction being measured may be observed in Figure 2.

Figure 1. The Steps of the Reaction Being Measured in the Succinate Dehydrogenase Assay

Figure 2. The Steps of the Reaction Being Measured in the Fatty Acid Oxidation Assay

(yellow) Succinate Dehydrogenase (colorless)
Succinate + Ferricyanide → Fumarate + Ferroycyanide



Protein Assay:

A 0.5 ml aliquot of the homogenate was taken for the assay of proteins by the Biuret method as described by Gornall et al. (26).

Statistical Analysis:

The data were statistically analyzed by a one-way analysis of variance. In the study on three different training intensities, a one-way analysis of variance was run on the four groups, and group comparisons were made with the Neuman-Keul test (27).

RESULTS

Establishing Conditions of Succinate Dehydrogenase Assay:

The dependence of the rate of the reaction on the concentration of substrate was ascertained by determining the amount of ferricyanide reduced per minute at several different substrate (succinate) concentrations. The results of this study are shown in Figure 3. It should be noted that with a 20 millimolor concentration of succinate, which was the level chosen for the assay, the enzyme system is well above saturation with regard to substrate and, therefore, the reaction rate is zero-order with respect to substrate. Thus the reaction rates determined on the experimental animals were totally dependent on the concentration of the enzyme present in the muscle tissue of the individual animals.

To demonstrate the linearity of the reaction with time, the reaction was initiated with succinate and the reduction of ferricyanide was determined on the Gilford 2000 Multiple Sample Absorbance Recorder over an extended period of time. The results of the experiment are shown on Figure 4.

Linearity of the reaction with respect to different concentrations of enzyme was shown by quantitating the amount of ferricyanide reduced per minute per milligram of protein at several different concentrations of enzyme. The reaction mixtures were prepared as previously described except that the amount of homogenate added to different reaction mixtures was varied. The results of this experiment may be observed in Figure 5. It should be noted that the reaction is linear with respect to enzyme concentration; and, therefore, we may assume that

Figure 3. The Effect of Substrate Concentration on the Activity of Succinate Dehydrogeneas in Muscle Homogenates

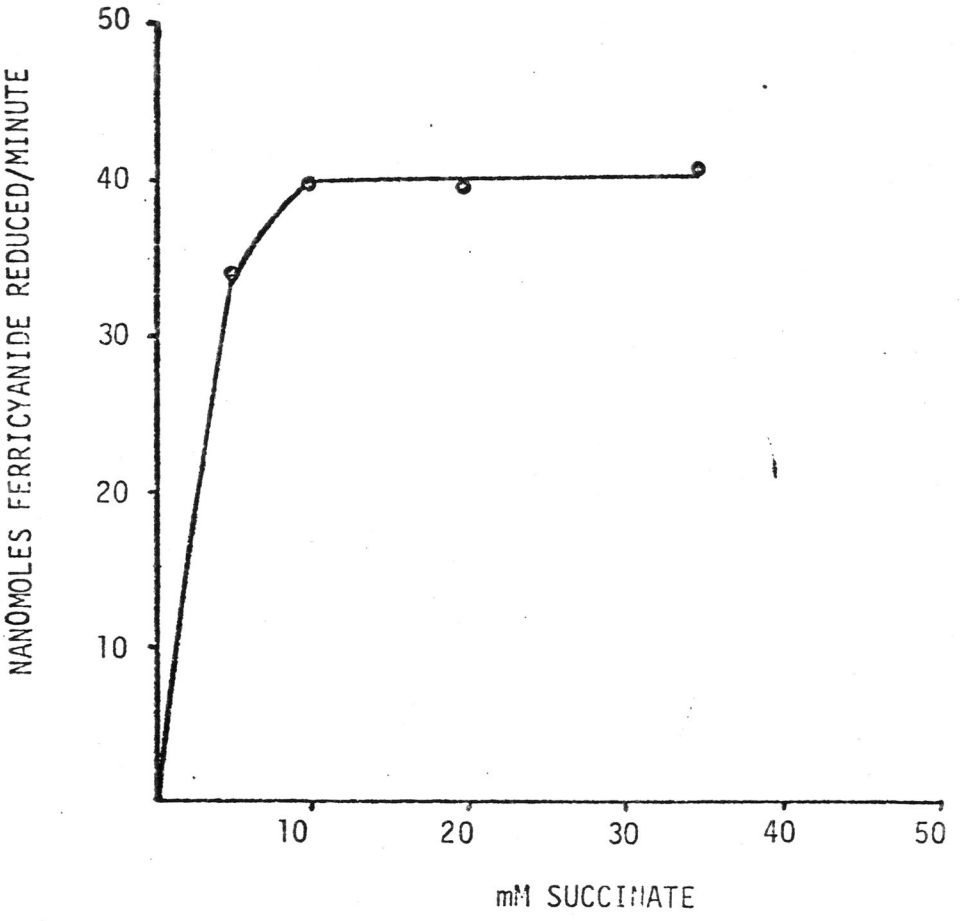
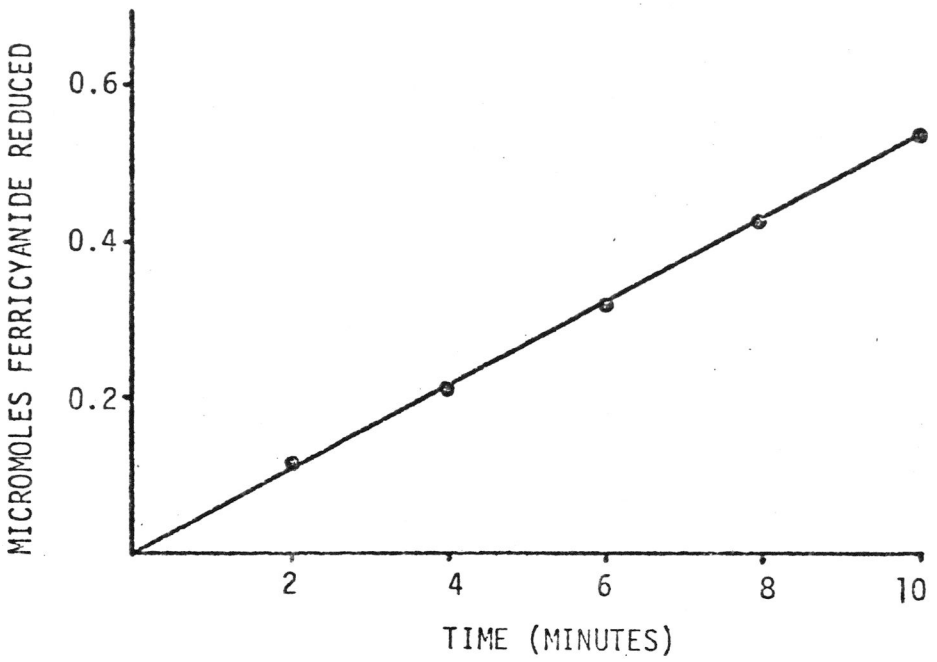
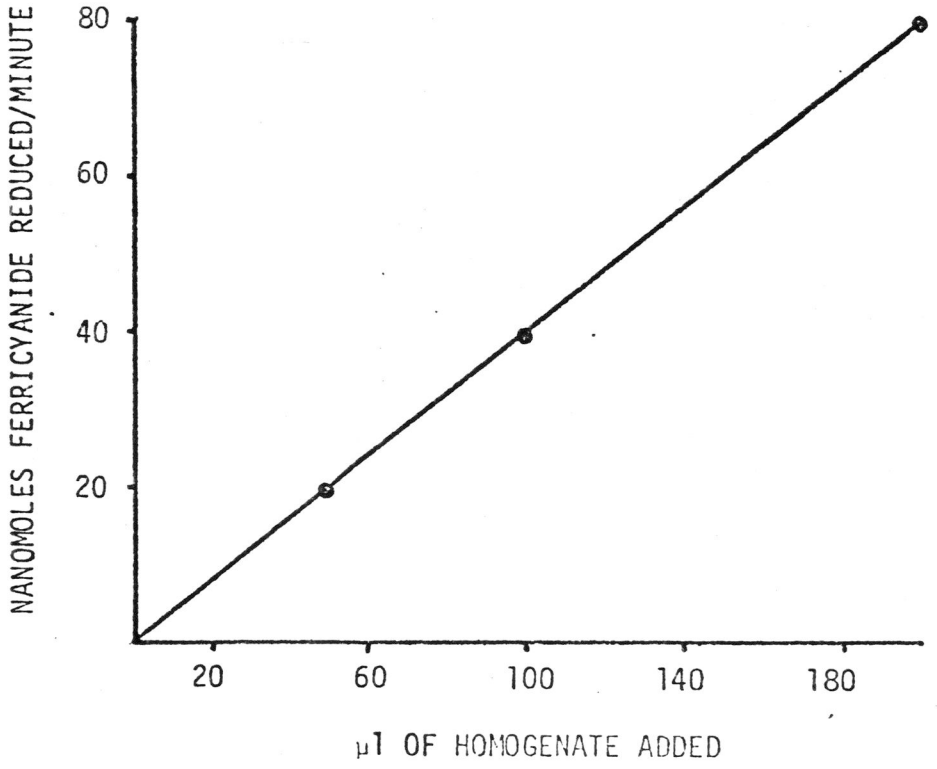

NANOMOLES FERRICYANIDE REDUCED/MINUTE
50
40
30
20
10
10
20
30
40
50
mM SUCCINATE

Figure 4. Proof of Linearity With Time of Succinate Dehydrogenase Reaction in Muscle Homogenates

Figure 5. Proof of Linearity of Succinate Dehydrogenase Activity With Enzyme Concentration in Muscle Homogenates


MICROMOLES FERRICYANIDE REDUCED
0.6
0.4
0.2
2
4
6
8
10
TIME (MINUTES)


NANOMOLES FERRICYANIDE REDUCED/MINUTE
80
60
40
20
20
60
100
140
180
µl OF HOMOGENATE ADDED

the differences in the rates of the reaction observed in the experimental animals were due to differences in the concentration of the enzyme within the muscle tissue of each animal.

Establishing the Conditions for the Assay of Fatty Acid Oxidation:

A quench curve for the counter was determined by using a known amount of radioactive material and thereafter determining the per cent efficiency of counting, using different concentrations of pyrimadine as a quenching agent. The results may be observed on Figure 6. During the assays of fatty acid oxidation, all samples gave an external standard count of approximately $3.4 \times 10^{+5}$ counts per minutes. It was; therefore, assumed that no quenching occurred from the samples; and, thus, no quench corrections were made.

The effects of different concentration of the necessary coenzymes and cofactors of the reaction were determined in order to ascertain their optimal concentrations. The results of these experiments may be noted on Table 2.

The effect of different concentrations of substrate was determined on each of the substrates used in this study; however, because each essentially demonstrates the same curve, only the data obtained using linoleic acid as the substrate are presented in Figure 7. It should be noted that with a substrate concentration of 0.2 millimolor, which was the level chosen for this study, the enzyme system is well above saturation with regard to substrate; therefore, the reaction rate is of zero-order for substrate. The graph also demonstrates that at higher concentrations, substrate inhibition begins to play a role in the reaction.

Linearity with respect to time of the reaction was demonstrated by initiating several reactions and allowing each to incubate for different

Figure 6. Graph of Quench Curve for the Packard Model 3320 Liquid Scintillation Spectrometer Used in This Research

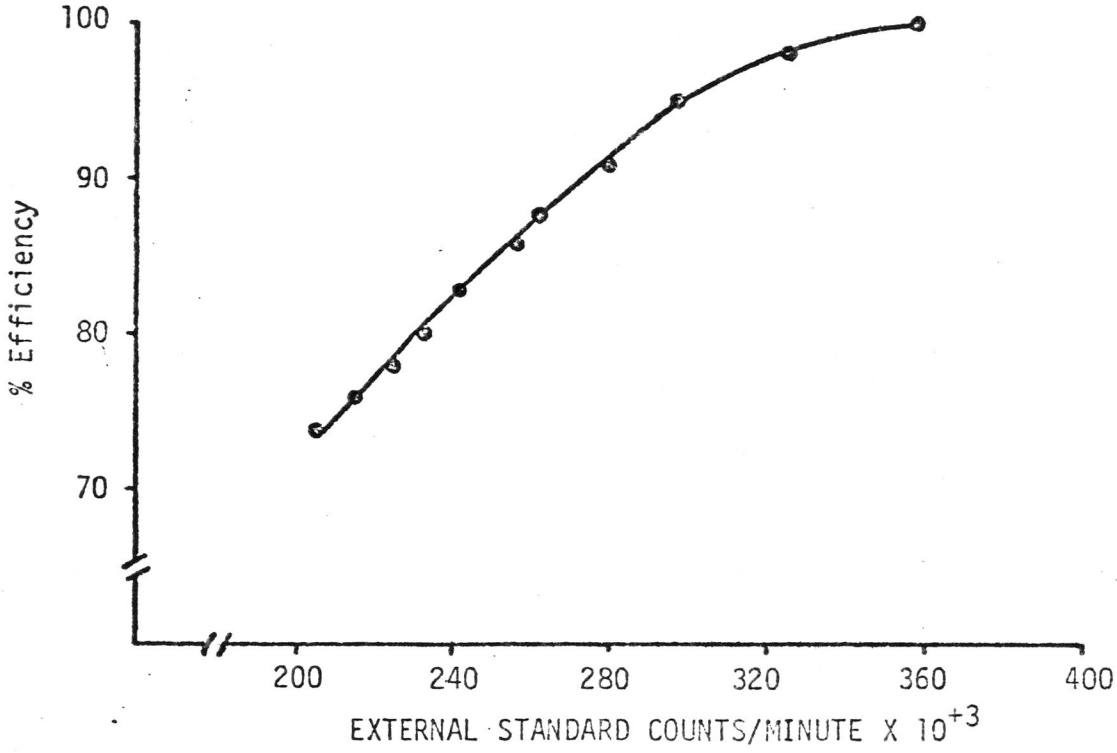
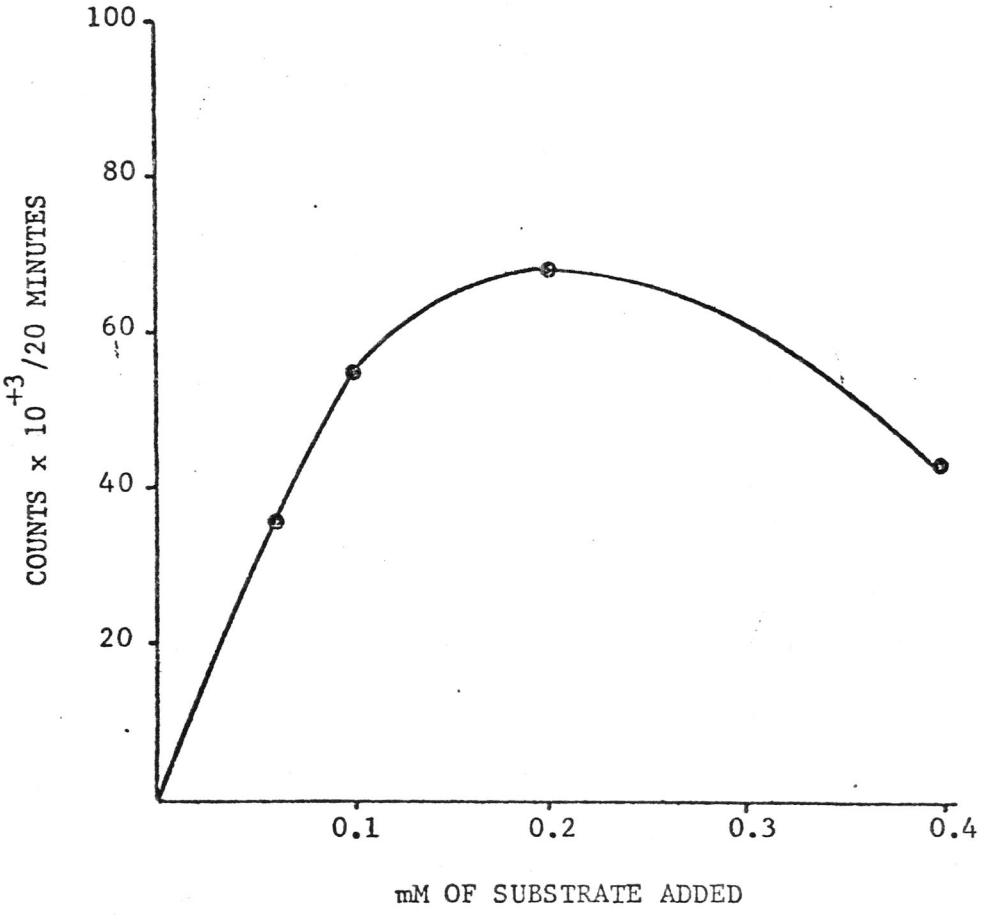

100
90
80
70
% Efficiency
200
240
280
320
360
400
EXTERNAL STANDARD COUNTS/MINUTE X 10^{+3}

Table 2. The Effect of Cofactor and Coenzyme Concentration on the Oxidation of Palmitic Acid in Muscle Homogenates

COFACTOR	CONCENTRATION (mM/L)	CPM
malate	0.05 0.1 0.2	5200 7600 3000
NAD^{++}	0.05 0.1 0.2	5600 7600 6600
carnitine	0.5 1.0 2.0	1500 7600 7400
coenzyme A	0.025 0.05 0.1	500 7600 7500
ATP	0.05 1.0 2.0 4.0	6000 9100 9200 6800
$MgCl_2$	0.5 1.0 2.0	7500 9200 9100

Figure 7. The Effect of Substrate (Linoleic Acid) Concentration on the Rate of Fatty Acid Oxidation in Muscle Homogenates


100
80
60
40
20
COUNTS x 10^{+3}/20 MINUTES
0.1
0.2
0.3
0.4
mM OF SUBSTRATE ADDED

lengths of time. The $^{14}CO_2$ evolved from each reaction was collected and counted. The results of this experiment may be observed on Figure 8.

The reaction was shown to be linear with respect to enzyme concentration by preparing several reaction mixtures as previously described and varying the amount of enzyme preparation (homogenate) added to each. The reactions were then allowed to proceed for 30 minutes, $^{14}CO_2$ was collected, and counts per minute were plotted against milliliters of homogenate. The results of this experiment may be observed on Figure 9. It should be noted that the rate of fatty acid oxidation (CO_2 release) is linear with respect to the enzyme concentration. Therefore, it may be assumed that the differences in the rate of fatty acid oxidation observed in the experimental animals were due to differences in the concentrations of the enzymes within the muscle tissue of each animal.

Figure 8. Proof of Linearity With Time of Oxidation of Linoleic Acid in Muscle Homogenates

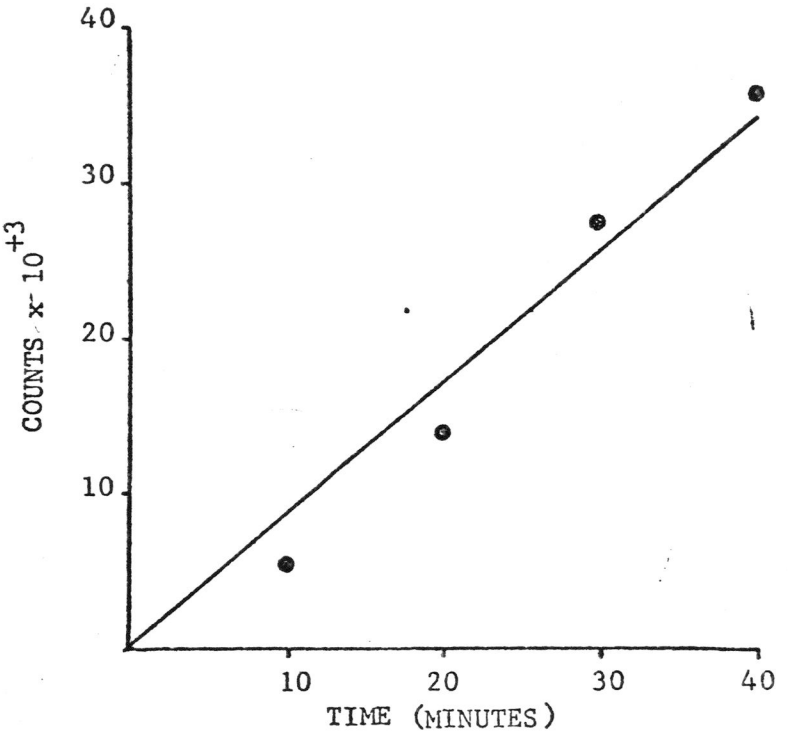
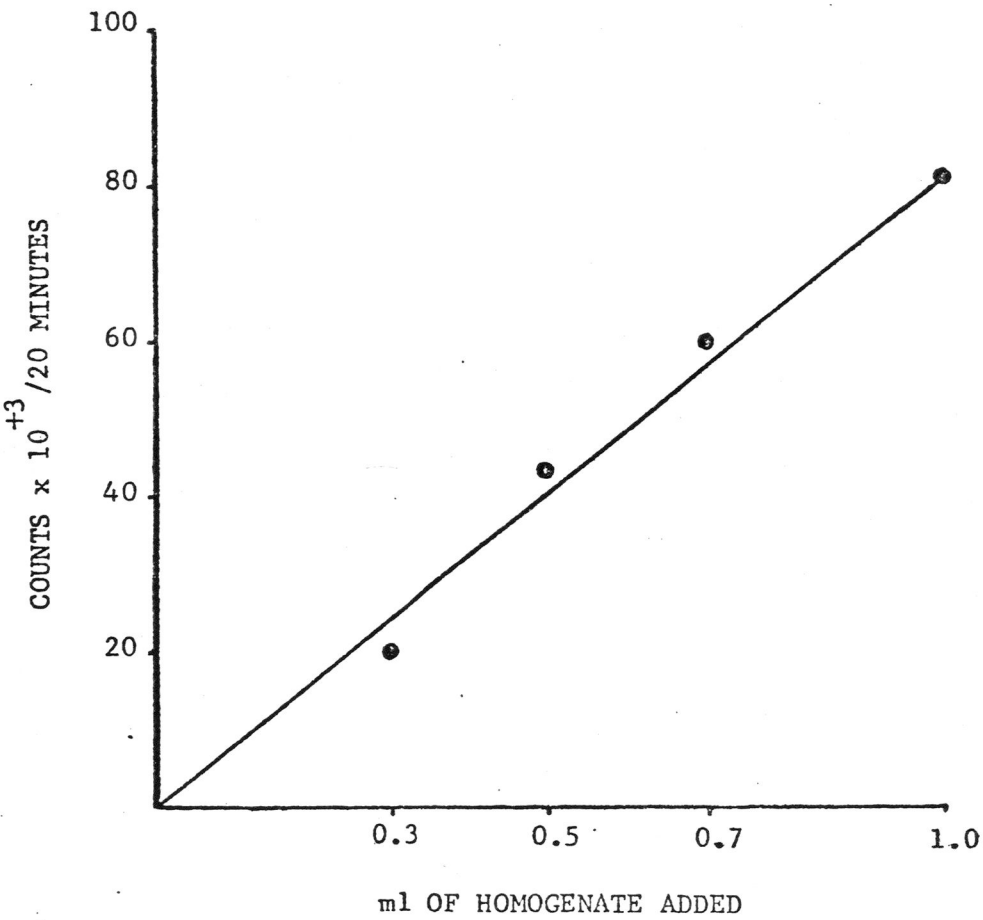

40
30
20
10
COUNTS x 10+3
10
20
30
40
TIME (MINUTES)

Figure 9. Proof of Linearity of the Rate of Oxidation of Linoleic Acid With Enzyme Concentration


100
80
60
40
20
COUNTS x 10+3 /20 MINUTES
0.3
0.5
0.7
1.0
ml OF HOMOGENATE ADDED

The Effect of Training at Three Different Intensities (Treadmill Speeds) on the Activity of Succinate Dehydrogenase in Muscle Homogenates:

The data in Table 3 shows that all three training regimens induced an increase in succinate oxidation by approximately 20-30%. The experiment was performed two times, and both trials demonstrated that succinate oxidation was not significantly altered as a result of different intensities of training; in other words, muscle succinate dehydrogenase acitvity was increased to the same extent in all trained groups.

The Effect of Training at Three Different Intensities on the Time to Exhaustion:

Exercised rats were judged to be exhausted when they could no longer run, even when electrically stimulated. This has been shown (8) to be a satisfactory method of determining the state of exhaustion. Even though the data is not complete because of a breakdown in the air conditioning system in the animal facility on the third day of the experiment, which resulted in different animals being exhausted at different temperatures, Table 4 shows that there is a trend toward increased endurance with an increase in the intensity of training; however, there was no statistically significant difference between any of the trained groups. Table 4 also shows that the temperature at which the animals were exhausted had a pronounced effect on endurance, reducing the time to exhaustion in all groups and eliminating any effect of intensity of training. These results suggest many possibilities for further research on heat stress in relationship to exercise training.

Table 3. The Effect of Training at Three Different Intensities on the Activity of Succinate Dehydrogenase in Muscle Homogenates

Groups	Nanomoles of Ferricyanide Reduced/Min/Mg Protein	
	Trial 1	Trial 2
UT	21.3±1.3(5)	29.5±1.0(5)
TI	25.4±0.5(5)*	38.1±2.8(7)*
TII		38.3±1.8(7)*
TIII	25.5±1.1(5)*	38.3±1.0(6)*

Values are means ± SE with number of animals in parenthesis.

* Significant difference ($P < 05$) between trained and untrained.

Table 4. The Effect of Training at Three Different Intensities on the Time to Exhaustion at 27 Meters Per Minute at 8% Grade

Groups	Time to Exhaustion in Minutes		
	All Date	Run at 22°C	Run at 32°C
TI	148±16(8)	185±16(4)	111±3(4)
TII	157±21(8)	205±20(4)	110±11(4)
TIII	176±26(8)	241±20(4)	112±8(4)

Values are means ± SE with number of animals in parenthesis.

* Significant difference ($P < .05$) between trained groups. No such difference was observed in this experiment.

The Effect of Length of Time on the Training Schedule and Length of Time of Detraining on the Activity of Succinate Dehydrogenase in Muscle Homogenates:

The data in Table 5 shows that the adaptation of increased succinate dehydrogenase activity had already become evident after only two weeks on the training schedule, producing an increase of approximately 10% in succinate oxidation. Data was also taken at four weeks, however, these data are not presented because technical problems caused erroneous results to be obtained. At six weeks of training, there was an increase of approximately 30% in succinate oxidation confirming the results of the previously mentioned experiments. During the period of detraining, there was a gradual reduction in the adaptation, however, it continued to be evident at three weeks of detraining as an increase in succinate dehydrogenase activity of approximately 10%.

The Effect of Exercise Training on Fatty Acid Oxidation in Muscle Homogenates:

The data on muscle fatty acid oxidation in Table 6 confirms that unsaturated fatty acids are oxidized at a greater rate than are saturated fatty acids (13) within a single chain length. Training increased the rate of oxidation of all four fatty acids used in this study, and the order of the oxidation rates was the same in trained as in untrained; oleate > palmitate > linoleate > stearate.

Table 5. The Effect of Length of Time on the Training Schedule and Length of Time of Detraining on the Activity of Succinate Dehydrogenase in Muscle Homogenates

Period	Nanomoles of Ferricyanide Reduced/Min/Mg Protein	
	UT	T III
2 weeks	19.4∓0.8(7)	22.1∓1.1(7)*
4 weeks		
6 weeks	30.4∓1.1(8)	39.7∓1.4(7)*
1 week detraining	31.7∓1.4(6)	36.3∓1.6(6)*
2 weeks detraining	34.1∓1.2(6)	37.0∓1.4(6)NS
3 weeks detraining	33.6∓0.6(8)	37.2∓1.1(9)

Values are means ∓ SE with number of animals in parenthesis.

*Significant difference ($P < .05$) between trained and untrained.

NS No significant difference between trained and untrained.

Table 6. The Effect of Exercise Training on Fatty Acid Oxidation in Muscle Homogenates

Fatty Acid	Nanomoles of Fatty Acid Oxidized/Min/G Tissue	
	UT	T III
Palmitic	20.9±1.0(8)	27.5±2.4(7)*
Stearic	13.2±1.5(8)	23.3±2.5(8)*
Oleic	22.5±2.6(8)	35.4±3.2(6)*
Linoleic	16.7±2.0(8)	25.5±1.9(8)*

Values are means ± SE with number of animals in parenthesis.

* Significant difference ($P < .05$) between trained and untrained.

DISCUSSION

The results of this study confirm the report of other investigators (8,16) that training increases the activity of the citric acid cycle enzyme, succinate dehydrogenase. However, varying the intensity of the exercise training did not significantly alter the run time to exhaustion nor the adaptation of succinate dehydrogenase to training (Tables 2 and 3). These findings are compatible with the findings of Cotton (28) who demonstrated that when muscular endurance is expressed in terms of net oxygen consumption during a sustained elbow flexion in humans, that the type of training beyond 25% maximum strength causes changes more in strength than in endurance. Likewise it is not unusual to find that this enzyme of aerobic respiration follows the same pattern as that of net oxygen consumption. A similar conclusion was drawn by Clarke and Stull (29) who found that a 5-minute bout of elbow flexion of two groups trained on programs of different intensities produced significant gains in initial and final strength and in total work, but the relative work done (fatigue curves) was not changed by training of different intensities.

Hern and Wainio (30) did not observe an adaptation in muscle oxidation capacity when they swam rats for only 30 minutes a day. Fitts *et al.* (31) found that rats trained on a training schedules which were designed to vary the duration of the daily exercise while holding intensity (running speed) constant for all groups resulted in an adaptation in muscle respiratory capacity such that the adaptation was proportional to both daily run time and endurance. It therefore appears that there is a threshold level of exercise below which no adaptation

in muscle respiratory capacity occurs and above which increasing the intensity of the exercise training does not further enhance the muscle oxidative capacity nor greatly influence the endurance of the animal. Likewise the level of the adaptation of muscle oxidative capacity appears to be closely related to the endurance of the animal, with the duration of exercise sessions playing a major role in the stimulation of this adaptation.

One of the purposes of this study was to investigate the rate at which the adaptation of succinate dehydrogenase occurs in the normal training schedule (TIII) used by this lab to study exercise physiology and to investigate how rapidly the adaptation disappears after the animals have been removed from the training schedule (detrained). The results of this study show that the adaptation is evident after only two weeks of training, producing approximately a 10% increase in succinate dehydrogenase activity. The adaptation then gradually increased until, at six weeks, an increase of approximately 30% was noted. During the period of detraining, the adaptation does not quickly disappear, but rather continued to be present for three weeks (the duration of this study) at which time there remained an approximate 10% increase in succinate dehydrogenase activity.

Measurements of respiratory quotients and of the rate of conversion of ^{14}C-labeled fatty acids to $^{14}CO_2$ have shown that physically trained individuals oxidize more fat and less carbohydrate than untrained individuals during submaximal exercise (10,11,32,33,34). This shift in the carbon source for the citric acid cycle likely plays an important role in the slower rate of glycogen depletion observed in trained animals, probably mediated by the control mechanisms that regulate carbohydrate catabolism, i.e. higher rates of fat oxidation inhibit glycolysis and

and pyruvate oxidation (35,36,37), and could postpone the fatigue associated with the depletion of muscle glycogen.

Another purpose of this study was to acertain if the oxidation of all fatty acids, both the previously reported saturated fatty acids and unsaturated fatty acids, are enhanced by training.

The data in this study confirms that unsaturated fatty acids are oxidized at a more rapid rate than are saturated fatty acids of the same chain length. The data also demonstrates that training increases capacity of muscle homogenates to oxidize both saturated and unsaturated fatty acids and that no alteration in the preferential oxidation of these fatty acids takes place as a result of exercise training, i.e. oleate > linoleate > palmitate > stearate.

Dohm et al (5) have shown that no alteration in the relative composition of muscle triglycerides occurs as a result of exercise training; however, there is a reduction of certain fatty acids (mono-unsaturated fatty acids) in the adipose tissue of trained animals. This is apparently the result of their preferential oxidation and/or mobilization during exercise. It therefore appears that no further adaptation in the preferential oxidation nor of the storage of fats occurs which would further enhance the capacity for ATP synthesis as a result of training.

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