New Researches of Management in Sport and Health

Journal homepage: nrmsh.damghan.iau.ir

ORIGINAL ARTICLE

Comparing the Effects of Long-term Continuous and Interval Training on the Serum Levels of Non-alcoholic Fatty Liver Risk Factors in Elite Runners

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KEYWORDS

Fatty liver;
Lipid profiles;
Running;
Training

ABSTRACT

In non-alcoholic fatty liver disease, serum levels of liver enzymes and lipid profiles were increased. One of the ways to deal with the risk factors of non-alcoholic fatty liver is to do physical activity and exercise. This semi-experimental study was conducted with the aim of comparing the effects of long-term continuous and interval training on non-alcoholic fatty liver risk factors in elite runners. Thirty elite runners with average height (176.5±0.05), weight (66.7±5.5) and age (20.2±0.00) were selected, and based on the specialized training were divided into two equal groups of continuous and interval training. Non-alcoholic fatty liver risk factors were measured with special laboratory techniques after standard fasting at the medical diagnosis laboratory. The non-parametric Mann-Whitney U test was used at the significance level of 0.05 to check the hypotheses. Despite high maximum oxygen consumption in both training groups, the levels of HDL_c, TG, LP $_{(a)}$ and HDL/LDL and HDL/TC ratio were lower in the interval training group than in the continuous training group, while the concentration of LDL_c, TC and BMI in the interval training group was higher than the continuous training group. Interval and continuous training increased liver enzymes. This increase was greater in the interval training group, which indicates more liver damage after performing this type of training. Therefore, performing interval and continuous training improves aerobic capacity and adjusts lipid profile and BMI.

Introduction

blood supply to the liver tissue is disturbed, this organ is damaged and enzymes enter the blood (Bentley *et al*., 2002). Physical activity, especially if it is intense and prolonged, has a significant effect on the activity of these enzymes (Bonekamp *et al*., 2008; Wu *et al*., 2004).

High intensity exercising probably causes damage to Liver cells as metabolically complex cells contain high amounts of enzymes (Toshio *et al*., 2002). Aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) are the most important indicators of liver health (Aragon *et al*., 2010). Under normal conditions, these enzymes are inside liver cells; but when the

[*]**Corresponding author**: Email address: ardeshir.zafari@gmail.com
Received: 16 August 2023; Received in revised form: 22 September 2023; Accepted: 17 October 2023

enzymes, but after twelve weeks of interval training, the levels of these variables were lower than continuous training. The results of the research of Mirdar *et al*. (2010) indicated that the levels of AST and ALT increased linearly with the increase in the number of sessions. Mir *et al*. (2012) suggested that eight weeks of aerobic exercise decreased liver enzymes. Zelber-Sagi *et al*. (2011) found that exercise reduced liver enzymes in the serum of patients with fatty liver. In fact, even a small increase in the amount of physical activity can lead to the improvement of liver enzymes. Hallsworth *et al*. (2011) suggested that eight weeks of resistance training caused a thirteen percent decrease in liver fat without any change in body weight as well as in the liver enzyme alanine aminotransferase. Christian *et al*. (2011) studied the relationship between the intensity of physical activity and the amount of liver enzymes. They did not find a relationship between moderate activities and improvement of liver enzymes. Devries *et al*. (2008) stated that performing moderate intensity endurance activity had no effect on liver enzymes. The results of Suzuki *et al*. (2005) showed that the plasma activity of ALT and AST increased two days after the competition, but the plasma ALP activity immediately after the event was lower than before it. In addition, the plasma activity of ALT and AST was lower after the triathlon compared to the marathon. The researchers suggested that probably the duration of the exercise has been effective on these variables (Gholami *et al*, 2017; Sadeghi *et al*. 2016).

Some evidence suggests that doing regular exercise and/or having good cardiorespiratory fitness can have beneficial effects on liver function. Regarding the adaptation effects of training on liver enzymes caused by endurance training, we can refer to Toshio *et al*.'s study (2002) which revealed that following four weeks of endurance training at a speed of twenty-five m m^{-1} with a zero-degree incline on the treadmill, the plasma levels of AST and ALT remained unchanged. The changes of these enzymes are affected by the intensity and type of exercise as well as the levels of liver cells and release of enzymes into the bloodstream (Mir *et al*., 2012). One of the complications of overtraining is acute liver damage, which increases liver enzymes (Kilian *et al*., 2007; Lawlar *et al*., 2005). The activity of liver enzymes changes under the influence of the intensity, duration, type and mode of exercise training (Cinar *et al*., 2006). During exercise, liver enzymes increase in the blood. Liver enzyme activity increased in people who walked on a treadmill for just five minutes (Suzuki *et al*., 2005). Therefore, changing the concentration of these enzymes can be due to liver damage (Burger *et al*., 2008; Christian *et al*., 2011).

The results of some studies indicate the increase of liver enzymes due to exercise and long-term competitions such as marathons and triathlons (Zelber-Sagi *et al*., 2011). Kinoshita *et al*. (2003) in a research aimed at determining the effect of high intensity training on human liver enzymes, showed a significant increase in the daily amount of AST and ALT. Carrying out high intensity physical training, especially strength training, increases AST up to three times and ALT up to fifty percent, and performing physical training at ordinary level is associated with significant lower levels of AST and ALT compared to inactive people. Regarding the effect of anaerobic training on the levels of liver enzymes, we can refer to the research of Zafrani (2004), in which the rapid changes of serum enzymes and metabolic concentrations after 100 m swimming were studied, and the serum levels of AST and ALT were increased.

In examining the effects of acute physical training, Harris *et al*. (2001) found an increase in AST and ALT levels in rats, six hours after the end of the training by running on a treadmill at high intensity (80% of maximum oxygen consumption) until exhaustion. Barzegarzadeh Zarandi *et al*. (2012) measured the changes of some liver enzymes and lipid profile after six and twelve weeks of regular continuous and interval aerobic training in old rats. Implementation of continuous and interval training increased the serum levels of AST, ALT and ALP

time and manner of performing the activity. Each type of training somehow affects the risk factors of non-alcoholic fatty liver, and previous studies indicate the effectiveness of both continuous and interval training, however there are contradictions on the effects of these types of training so that we cannot comment decisively about the effectiveness and superiority of one mode over another one (Rostami *et al*., 2012).

The researchers in their research using both methods have achieved different results, which has led to the question of what changes do the serum levels of the risk factors of non-alcoholic fatty liver make after doing interval and continuous training? Considering the conflicting results of previous studies and the lack of comparison between the effects of continuous and interval training on the risk factors of non-alcoholic fatty liver disease, the present study tries to study the effects of long-term continuous and interval training on the risk factors of non-alcoholic fatty liver disease and to answer the question whether there is a difference between the serum levels of non-alcoholic fatty liver risk factors after performing continuous and interval training.

Materials and Methods

The current semi-experimental and applied research was conducted with the aim of comparing the effects of long-term continuous (1500, 3000 and 5000 m sprints) and interval training (100, 200, 400, 800 m sprints and performing triple, long and hurdle jumps) on non-alcoholic fatty liver risk factors in elite runners participating in national championships in the field and in the laboratory. Thirty available qualified runners were selected as the statistical sample and were purposefully divided into equal groups, including interval training and continuous training. After 9-12 hours of fasting, 5^{cc} of blood was taken from the right antecubital vein using coded venojects while resting in the supine position. The serum was centrifuged for ten minutes at 3000 rpm and the variables were measured. The serum concentrations of aspartate aminotransferase and alanine

individuals' physical fitness.

One of the important factors affecting aerobic capacity is body composition, which includes the percentage of water, muscle, bone, and fat in the body; in other words, it is the percentage and weight of fat and lean mass which can have positive or negative effects on aerobic capacity (Ossanloo *et al*., 2012; Zahedmanesh *et al*., 2013). Chatterjee *et al.* (2006), Shete *et al.* (2014) and Mondal *et al.* (2017) showed a significant relationship between aerobic capacity and body weight and body composition in inactive subjects, while Ozcelik (2004) showed no relationship between aerobic capacity and body composition. On the other hand, other researchers showed a relationship between body fat mass and lipid profile (Ossanloo *et al.*, 2012; Shazia *et al*., 2015). In addition, improper lipid profile is one of the factors of reducing aerobic capacity. Sports success has a direct relationship with low body fat, and there is an inverse correlation between fat percentage and the performance of exercise (Zafari, 2012). During low-intensity training, a greater amount of fat is used as fuel, and with the increase in training intensity, the use of fat as fuel decreases and use of carbohydrate increases (Leahy *et al*., 2013).

A lot of research has been done on the appropriate intensity and mode of training to improve aerobic capacity, body composition, lipid profile and liver enzymes, including the use of two types of interval and continuous training (Kannin *et al*., 2005; Gholami *et al*., 2012; Whyte *et al*., 2010). Han *et al*. (2019) measured the effect of eight weeks of basic military exercises on the lipid profile of people with different body composition (normal, overweight and obese). With the increase in aerobic capacity, the levels of low-density lipoprotein, triglyceride and other serum lipids decreased. Various training modes have been used to adjust the serum levels of risk factors for non-alcoholic fatty liver disease, which include continuous and interval training. Continuous and interval training are different in terms of the type and nature of the energy systems involved in the activity, as well as the

coefficient of 0.98. All assays in the medical diagnosis laboratory were performed by experienced personnel using Hitachi and Biorex Fars devices and laboratory diagnostic kits of Pars Peyvand Company (Iran) according to the present technical and safety standard guidelines. The maximum oxygen consumption was determined by the Bruce test in the exercise physiology laboratory of the Faculty of Physical Education.

Results

In Table 1, the values of non-alcoholic fatty liver risk factors are reported. The results of the Kolmogorov-Smirnov test showed that the distribution of the variables was non-normal. Therefore, the Mann-Whitney U non-parametric statistical test was used for intergroup comparisons of the research variables. The results of Table 2 showed that in comparing the continuous and interval training groups, there was a significant difference in the values of HDL_c (U=93.00, N_1=N_2=15, P = 0.001), LDL_c (U=85.00 , N_1=N_2=15, P = 0.001), TG (U=93.00, N_1=N_2=15, P = 0.001), TC (U=81.00, N_1=N_2=15, P = 0.001), HDL/LDL ratio (U=63.00, N_1=N_2=15, P =0.040), TC/HDL ratio (U=65.00, N_1=N_2=15, P = 0.044) , LP(a) (U=104.00, N_1=N_2=15, P = 0.001), AST (U=96.00, N_1=N_2=15, P = 0.001), ALT (U=74.00, N_1=N_2=15, P = 0.001), ALP (U=54.00, N_1=N_2=15, P = 0.015) and BMI (U=64.00, N_1=N_2=15, P = 0.044).

aminotransferase enzymes were measured fully automatically using the Biorex enzyme kit of England, human model (provided by Elixir Azma Company) by the IFCC method and in terms of units per liter. The serum concentrations of alkaline phosphatase enzyme were measured fully automatically using the Biorex enzyme kit of England, human model (provided by Elixir Azma Company) by the DGKC method and in terms of units per liter. Serum triglyceride was measured by Enzyme-Colorimetric End Point method and laboratory kit with a diagnostic range of 15-100 and a sensitivity of 1 mg dl^{-1} and a correlation coefficient of 0.999. Serum total cholesterol was measured by Enzyme-Colorimetric End Point method and a laboratory kit with a diagnostic range of 5-400 and a sensitivity of 29 mg dl^{-1} and a correlation coefficient of 0.994. Low-density lipoprotein cholesterol was measured by a spectrophotometric direct method using a laboratory kit with a diagnostic range of 5-250, a sensitivity of 2 mg dl^{-1} and a correlation coefficient of 0.992. High-density lipoprotein cholesterol was measured by a spectrophotometric direct method using a laboratory kit with a diagnostic range of 5-130, a sensitivity of 2 mg dl^{-1} and a correlation coefficient of 0.990. Serum lipoprotein (a) was measured by immunoturbidimetric method and using a laboratory kit with a diagnostic range of 1-140 and a sensitivity of 5 mg dl^{-1} and a correlation

Table 1. Mean and SD of the serum levels of non-alcoholic fatty liver risk factors.

Variables	Groups	M±SD	K_S test results
BMI	Interval	22.08±1.00	0.200
(Kg m^2)	Continuous	20.00±1.00	0.200
Vo_{2max}	Interval	46.00±8.00	0.200
(ml kg^{-1} min^{-1})	Continuous	54.00±8.00	0.200
HDL_c	Interval	42.06±10.00	0.001
(mg dl^{-1})	Continuous	45.00±7.00	0.001
LDL_c	Interval	101.06±42.00	0.001
(mg dl^{-1})	Continuous	83.00±0.23	0.001
TG	Interval	99.00±46.00	0.001
(mg.dl^{-1})	Continuous	85.00±29.00	0.001
TC	Interval	165.00±53.00	0.029
(mg dl^{-1})	Continuous	151.00±29.00	0.025
HDL/ LDL	Interval	2.00±0.02	0.001
	Continuous	1.00±0.10	0.001
HDL/ TC	Interval	4.00±0.03	0.001
	Continuous	3.00±0.01	0.001
LP(a)	Interval	19.00±2.00	0.001
(mg dl^{-1})	Continuous	13.00±1.20	0.001
AST	Interval	32.00±8.00	0.005
(U L^{-1})	Continuous	31.00±15.01	0.002
ALT	Interval	31.00±15.01	0.025
(U L^{-1})	Continuous	25.00±13.00	0.005
ALP	Interval	198.00±40.00	0.002
(U L^{-1})	Continuous	280.00±121.00	0.025

Table 2. The results of Man-Whitney U test in intergroup comparison.

Variables	Mann-Whitney U	Z-score	P value
HDL_c (mg dl^{-1})	93.00	0	0.001
LDL_c mg dl^{-1})	85.00	-1.00	0.001
TG (mg dl^{-1})	93.00	0	0.001
TC (mg.dl^{-1})	81.00	-1.00	0.001
HDL/ LDL	63.00	-2.054	0.040
HDL /TC	65.00	-2.017	0.044
LP(a) (mg dl^{-1})	104.00	0	0.001
BMI (Kg m^2)	64.00	-2.012	0.044
AST (U L^{-1})	96.00	0	0.001
ALT (U L^{-1})	74.00	-1.00	0.001
ALP (U L^{-1})	54.00	-2.00	0.015

Discussion

Performing exercise training with any intensity and duration is associated with a decrease in total and relative amounts of TC, LDL_c, TG and an increase in HDL_c. This paper points to the role of the peripheral tissues and the liver, which basically allow the existing mechanisms during short-term or long-

term continuous or interval training to cause favorable changes in the lipid profile and body composition by altering the activity of enzymes. The amount of plasma fats has an inverse relationship with physical fitness and a positive correlation with body mass index and body fat percentage. The decrease in the concentration of plasma lipids after long-term training is attributed to the decrease in the circadian rhythm of lipids and hormonal changes caused by training.

The production of adenosine triphosphate during speed interval training mainly relies on the use of creatine phosphate, glucose and glycogen consumption. As a result, these exercises reduce the amounts of serum lipids by emptying glycogen, increasing glucose uptake in the presence of lactate, intensifying acidosis and increasing catecholamine (Eftakhari *et al*., 2015). During long-term and continuous activities, the body uses fatty acids as fuel, and the activation of lipolysis from adipose tissue increases the concentration of free fatty acids in the plasma; at the same time that insulin decreases, glucagon increases, which both hormones cause increased activity of ketogenesis during exercise, which in turn, lead to an increase in liver load and changes in cholesterol precursors (Gholami *et al*., 2012). In addition, fatty tissues have multiple capillaries and autonomous nerves, so all their metabolic actions are controlled by hormonal and nervous factors.

One of the important reasons for increasing lipolysis is the stimulation of beta-adrenergic receptors of fatty tissue, so that physical activity stimulates the sympathetic nervous system and the release of catecholamines. Lipolysis occurs when the aforementioned hormones are placed on specific receptors of fat cells. It should be noted that the use of fats as energy sources changes with the intensity of training and continuous activities stimulate further lipolysis compared to interval activities (Gholami *et al*., 2012; Gholami *et al*, 2017; Sadeghi *et al*. 2016).

In addition, the role of exercise training on the metabolism of fat tissue has been well clarified. In studies that only had exercise intervention, high-intensity and not moderate-intensity exercise increased adipose tissue lipolysis (Di Pietro *et al*., 2006). A bout of exercise increases the levels of catecholamines and the levels of secretion of catecholamines are directly related to the intensity of exercise (McLean *et al*., 2015).

Regular physical activity has been introduced as a factor for improving quality of life, increasing aerobic capacity, improving body composition, and preventing weight gain, as well as preventing and treating cardiovascular diseases and metabolic disorders and fatty liver; in this vein, research has shown that regular continuous endurance activity increases HDL_c and decreases LDL_c (Zafari, 2012).

It seems that the reduction of triglycerides and the increase of HDL_c are the effects of long-term continuous training. Although studies have focused on the beneficial effects experienced by endurance athletes, strength, resistance, and interval training also have protective benefits and can help improve or maintain blood lipid status and body composition (Aragon, 2010; Zahedamesh *et al*., 2013). Therefore, it seems that continuous running training has better effects on lipid profile changes than interval running training. During exercise, the liver, as the primary organ of metabolism, increases the production and recall of glucose into the blood. Also, considering its role, it requires activation of specific chemical pathways for amino acid and fat metabolism, which increase during muscle work. Proportionate to the intensity of exercise, the glucose produced in the liver also increases linearly.

During light to moderate exercise, glucose output increases two to three times and during high intensity exercise, it increases as seven to ten times as the resting values. The amount of hepatic glucose output depends on the liver glycogen content, which varies in individuals with their fasting state as well as the food consumed before exercise and training. Glucose production during exercise is mainly due to the

breakdown of liver glycogen (glycogenolysis), and only a small part of the produced glucose (10 to 20%) is obtained through gluconeogenesis. With the prolongation of exercise activity (several hours), gluconeogenesis increases so that its share in the total glucose produced by the liver reaches 50%. This increase occurs in parallel with the decrease in liver glycogen reserves and the increase in the reserves of gluconeogenesis precursors in the liver. During light and long-term exercise, the feedback signals of the active muscles (which are carried out through nerves and the blood flow) create a stimulus for glucose production to maintain glucose in the blood at a normal level. An increase in blood glucose directly inhibits glucose production during exercise, while a decrease in blood glucose indirectly increases glucose production in the liver through anti-regulatory hormones such as glucagon. On the contrary, during high intensity exercise and at the beginning of exercise, central mechanisms, along with central motor activity, cause hormonal responses to become more pronounced (e.g., an increase in plasma epinephrine) and glucose demand to increase. This increase in recall is greater than peripheral glucose uptake, which results in an increase in blood glucose during high intensity exercise. Reducing plasma levels of insulin is important in increasing liver glucose production during exercise in humans and other species. Nevertheless, this decrease in plasma levels of insulin does not fully justify increased hepatic output of glucose, which does not occur during light exercise. The increase in plasma glucagon is important in some species, but in humans, the glucose produced by the liver increases even with small changes in glucagon. During high intensity exercise, epinephrine plays a minor role in increasing hepatic glucose, but at the end of long-term exercise, its role increases slightly with increased release of muscle gluconeogenesis precursors and liver reserves.

Hepatic sympathetic innervation does not play a role in increasing hepatic glucose output due to exercise in humans and other species, and growth hormone and cortisol also play a minor role in increasing glucose output due to exercise. During exercise, hormonal mechanisms explain only part of the stimulated glucose production in humans. Therefore, other unknown factors may play a role in increasing hepatic glucose production due to exercise. During exercise, the uptake of amino acids in the liver increases to match the large supply of muscle and intestinal proteolysis. In addition, during gluconeogenesis activity, the production of urea and possibly acute phase proteins during exercise are enhanced. During exercise, sympathetic nerve activity calls visceral fat reserves, which probably indicates an increase in free fatty acids and glycerol, which are released from the intestine and taken from the liver. This increases not only gluconeogenesis, but also ketogenesis during physical activity (Eftakhari *et al*., 2015).

The most sensitive liver diagnostic enzymes are aminotransferases (AST and ALT). These enzymes are normally present in the cytosol and mitochondria of liver cells, and when the liver is damaged, these enzymes enter the blood. An increase in the levels of these enzymes in the blood is a sign of liver damage. In addition to the liver, these enzymes are also present in small amounts in the kidneys, skeletal muscles and heart, and when these tissues are damaged, these enzymes are found in the blood. Alkaline phosphatase plays a role in the transfer of fats from the intestine and bone calcification, and it transports intermembrane metabolites. This liver enzyme is also present in the wall of liver bile cells and its levels increase during liver damage. Aminotransferases have little activity in normal serum, and as a result of endurance training and competitions, short-term and high intensity, eccentric training and even sports in which body weight is not tolerated the levels of these enzymes increase. Aspartate aminotransferase levels increase twelve hours after the start of exercise; on the second day, they reach the highest levels and return to the normal levels in four or five days. On the other hand, the levels of alanine aminotransferase increase four to six hours after the start of exercise; on the

second day, they reach the highest levels, up to twelve times more than the normal levels, and return to the normal levels on the third day (Mirdar *et al*., 2010). Although the serum activity of both AST and ALT enzymes increases whenever the integrity of liver cells is affected by diseases, alanine aminotransferase is a more specific enzyme for the liver (Burtis *et al*., 2011). Laboratory methods affect the results as well. The half-life and conditions of storage and measurement of enzymes are different from each other, and the lack of attention and sufficient accuracy can change the results.

In the current study, the serum concentrations of these liver enzymes were higher in interval training than in continuous training, which indicates high levels of stress and intensity of this type of training. Muscle contractions caused by high intensity eccentric activities lead to a greater increase in the serum levels of the above-mentioned enzymes in comparison with low intensity activities. Therefore, the increase in the serum levels of the liver enzymes aspartate aminotransferase and plasma alanine aminotransferase and alkaline phosphatase can indicate cell leakage and indicate damage to the structure and dysfunction of the liver cell membrane. The increase in the amount of liver enzymes aspartate aminotransferase and plasma alanine aminotransferase after long-term activity can be due to the fact that high-intensity and heavy training reduce the blood flow of the liver and kidneys to five and three percent, respectively, and cause liver damage and increased secretion of these enzymes into the bloodstream (Eftakhari *et al*., 2015).

The results of previous study on adult rats showed that following three sessions of running on a negative slope, a significant increase in the levels of aspartate aminotransferase and alanine aminotransferase enzymes is observed. Suzuki *et al*. (2005) showed that regular exercise significantly reduces serum alanine aminotransferase. Another result of this research was that the amount of alanine aminotransferase enzyme was higher in the interval training group than in the continuous training group. Alanine aminotransferase has a closer relationship with insulin resistance than other liver enzymes.

The cellular mechanism of the secretion of this enzyme during physical activity is still unknown, but most of the time, its cause is considered to be the structural changes that occur in the muscle tissue following high intensity activity. Other factors that can be effective in reducing alanine aminotransferase in beginners are changes in fitness levels and especially maximum oxygen consumption and anthropometric indicators.

There are several hypotheses such as hypoxia, heat stress and hemolysis, cell damage caused by performing activities due to mechanical processes or changes in membrane permeability after performing physical activities to justify the increase in liver enzymes activity. In recent decades, some studies have focused on the role of oxidative stress in tissue damage caused by exercise and on the role of antioxidant factors in the initiation of adaptive processes.

The existing data shows that regular exercise training reduces the capacity of some tissues to release reactive oxygen species and causes the adaptation of antioxidant mechanisms that may eventually play a role in limiting the oxidative damage caused by exercise.

It has been reported that regular exercise training can increase the antioxidant defense capacity and protect active skeletal muscle against cellular stress, while it has little effect on the hepatic antioxidant enzyme system.

The variability of the response of different tissues to resistance training and regular training depends on the previous exposure of these tissues to oxidative stress or on the internal oxidative state of those tissues. The antioxidant function of the liver is lower compared to muscles. One of the suggested mechanisms to improve liver efficiency is to increase muscle oxidative capacity.

The research subjects' maximum oxygen consumption was high, and this itself is a sign of improved

oxidative capacity. Therefore, a possible explanation for the variability of liver enzymes after interval training is the increase in muscle oxidative capacity, which can consume intracellular fat as fuel during exercise and so improve liver efficiency. Training improves insulin resistance in adipose tissue, which further reduces the delivery of free fatty acids to the liver and, in turn, increases mitochondrial biogenesis, thereby improving beta-oxidation.

The type of training also has different effects on the secretory and metabolic systems. Training increases the activity of the sympathetic system (epinephrine and norepinephrine) and growth hormone; each of these hormones, in turn, activates lipolysis and leads to a decrease in body fat mass.

Long-term and endurance activities whose energy production is aerobic have an effect on the activity of alanine aminotransferase and aspartate aminotransferase enzymes; because in order to continue these types of activities, there is a greater need to produce energy through an aerobic system.

Alanine aminotransferase and aspartate aminotransferase enzymes are among the enzymes involved in the liver metabolism. Because the liver plays a greater role in endurance activities than other activities, the probability of damage in the liver cell membrane in long-term and endurance activities is high. If the training is of a heavy resistance type, most of the energy required is provided through anaerobic, and since liver cells, especially liver enzymes, are not involved in energy production, their damage is less.

Another point that should be noted is that despite the fact that the levels of aspartate aminotransferase and alanine aminotransferase liver enzymes did not differ significantly after six weeks of continuous and interval training, the levels of aspartate aminotransferase and alanine aminotransferase liver enzymes after twelve weeks of interval training were significantly less than the continuous training group; it is probably because the rest intervals between the training sessions in the interval group caused less stress in the subjects compared to the subjects in the continuous group, who underwent the same activity continuously.

On the other hand, it is likely that less stress in the interval group subjects causes a further increase in the levels of liver enzymes aspartate aminotransferase and alanine aminotransferase. Given the factors increasing the levels of hepatic enzymes, perhaps the inclusion of rest intervals between training sessions may have caused the levels of liver enzymes in the interval group to be lower than the group that did continuous training.

It seems that the discrepancy between different studies can be related to the methodology and data collection procedure. Unlike the present study, which investigated liver enzymes through serum sampling, some researchers have measured the levels of liver enzymes through tissue sampling. Studies show that alanine aminotransferase enzyme is present in the liver, kidneys and to a lesser extent in the heart and muscle, alternatively aspartate aminotransferase enzyme is present in the liver, heart, muscle, kidneys, brain, pancreas, lungs, and white blood cells, hence these enzymes are not specific to the liver. Therefore, the increase of these enzymes in serum or plasma cannot specifically indicate liver damage.

Given the high levels of maximum oxygen consumption and the adaptations resulting from the past training in the subjects of this study, it appears that the body prefers to supply the needed energy during rest and sub-maximal training from lipid sources, which reduces the lipid profile and liver enzymes as risk factors for non-alcoholic fatty liver disease. Enhanced activity of the lipoprotein lipase enzyme is among the reasons for changes in the serum levels of triglycerides and lipid profiles and liver enzymes, and it seems that interval and continuous exercise training will increase the activity of this enzyme.

In addition, changes in lipid profiles and liver enzymes are related to changes in body fat mass. Considering the high aerobic capacity of both training groups, probably both types of training protocols have

provided the necessary stimulation for the lipolysis process, as a result of which, a decrease in body fat mass and lipid profile and liver enzymes has been yielded.

Acknowledgments

The authors would like to thank all the participants and in the present study.

Conflict of Interests

The authors have no any conflict of interests.

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