

Liver overload in Brazilian triathletes after half-ironman competition is related muscle fatigue

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Abstract

Triathlon competition is dependent on the athletes' ability to perform each discipline at optimal time, without excessive fatigue influencing the next one. Objectives: Determine the effects of a long distance triathlon on biochemistry parameters related to liver function. **Design and methods:** Blood samples from six athletes were collected before (T = 0) and immediately after the triathlon competition (T = 1). AST, ALT and alkaline phosphatase (ALP) values were assessed. **Results:** Significant changes after triathlon competition were found for AST and ALP and no significant changes were found for ALT over time. **Conclusions:** A series of metabolical alterations, mainly related to energy production and also to muscle and skeletal adaptations occurs during and after strenuous exercise. The altered status of those metabolical changes cannot directly reflect the intensity of any possible muscular or hepatic damage or overload and elevated AST/ALT ratio is better associated to skeletal muscle lesion during competition.

Key words: Liver enzymes, aspartate aminotransferase, alanine aminotransferase, triathlon, half-ironman.

Introduction

Physical inactivity is a recognized risk factor in the development of obesity, diabetes, and cardiovascular disease, and the health benefits associated with regular moderate-intensity exercise are incontrovertible. Physical training decreases the incidence of hypertension and results in favorable changes in the blood lipid profile.¹

Triathlon is a unique endurance sport that comprises a sequential swim, swim-to cycle transition, cycle, cycle-to-run transition, and run.² The half-Ironman triathlon

race consists of 1.9 km of swimming, 90 km of cycling and 21 km of running. Such ultraendurance exercise imposes strenuous physical load on the bodies of athletes, characterized by significant energy demands,³ muscle damage,⁴ thermal stress associated to dehydration,⁵ and oxidative stress.⁶

Aspartate aminotransferase (AST, also known as glutamate oxaloacetate transaminase or GOT), alanine aminotransferase (ALT, also known as glutamate pyruvate transaminase or GPT), closely reflect the altered function and even necrosis of hepatocytes in liver injury, and some studies have found that exercise causes changes in the levels of these enzymes.⁷ Fewer data are available in relation to changes in liver function after half-ironman triathlons.

Several biochemical alterations normally occur during and after exercise practice. Haematological disturbances after exercise, such as hyponatremia, haemolysis and/or iron deficiency, are well documented and thought to be of concern for optimal endurance performance, but of little concern from a health perspective.⁸ The aim of this study was to determine the effects of a long distance triathlon on biochemistry parameters related to liver injury.

Materials and methods

Subjects

Six well-trained male amateurs' triathletes who completed the 2004 half-ironman triathlon in Cabo-Frio, Brazil participated in the study. The characteristics of the athletes are shown in *Table I*. The athletes had all completed a minimum of one prior half-ironman triathlon distance event in less than 7 h. Each subject provided written informed consent, and the investigation was approved by the medical research ethics committee of The Estácio de Sá University. Athletes were allowed to eat and drink *ad libitum* during their race and no particular guidance was given to them as to what quantities or types of fluids and fuels they should consume.

Race conditions

The event consisted of a 1.9 km swim, followed by a 90 km cycle, and completed with a 21 km half-marathon

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run. Environmental conditions ranged from 18-23 °C (18 ± 2.3 °C) according local airport. Performance times for the swim, cycle, and run phases, and the total time are shown in *Table II*.

Blood sampling

Blood samples (~15 mL) were obtained from the antecubital vein on morning day of the competition after dietary restriction between 6-7 am, and immediately before the race. Post race blood samples were taken within 5 min of athletes finishing the race, with the athlete lying supine in the medical tent. All blood samples were stored at ambient temperature (20 °C), until arrival in the laboratory and then centrifuged during 10 min with relative centrifugal force of 1,200G (2,600 rpm). Serum samples had been stored 4 °C for posterior analyze the same in next day.

Blood markers

Alanine aminotransferase (ALT) aspartate aminotransferase (AST) and alkaline phosphatase (ALP) were measured as markers of liver damage. All markers were measured by commercially available assays (*Invitro* Diagnostics, Brazil) using an automatic analyzer BTS-370 Plus (BioSystems, Barcelona, Spain).

Statistical analysis

Data were tested for normality using the Shapiro-Wilk test, with all data sets found to be normally distributed

except ALT pre (0.001). Values are presented as means ± standard deviation. The one sample Wilcoxon signed rank test was used to check for significant changes of pre and post race values. Pearson’s product-moment correlation coefficients were used to examine potential relationships between of finishing time with liver damage markers. Significance was set at the 0.05 level of confidence, and statistical analyses were performed on SPSS (version 14.0) software.

Results

Significant changes after triathlon completion were found for AST and ALP and no significant change was found for ALT over time (Individual values are present in *Table III*). ALT-Pre = 17.9 ± 8.98 U/I; ALT-Post = 23.33 ± 11.22 U/I (P = 0.116); AST-Pre = 18.47 ± 3.50 U/I; AST-Post = 37.50 ± 8.41 U/I (P = 0.028); ALP-Pre = 52.17 ± 20.34 U/I/L; ALP-Post = 104.33 ± 31.61 U/I/L (P = 0.046). Were not found significant correlation between all biochemistry parameters and finishing time (*Table IV*).

Some athletes present individual values out of normality according Souba et al.⁹ Volunteer number 2 present AST above normality values for AST before the race, volunteers 1, 3, 4 present ALP values beneath normality after the race, and volunteer number 5 present ALP above before the race. AST/ALT ratios were above 1 for all athletes after competition and 3 of them presented this ratio altered before the competition.

Table I. Characteristics of subjects.

Age (years)	27.33 ± 5.78
Weight (kg)	72.17 ± 9.82
Length (cm)	177.50 ± 9.65
IMC(kg/m ²)	22.84 ± 1.98

Data are mean ± SD (N=6).

Table II. Performance in the half-ironman triathlon race. Mean (SEM) values of six athletes are shown.

Swim time	0:23:13 ± 0:28:04
Cycle time	2:51:15 ± 0:03:12
Run time	1:43:50 ± 0:05:57
Total race time	4:58:19 ± 0:22:21

Data are mean ± SD (N=6).

Table III. Individual values biochemical parameters (values expressed U/I).

	ALT Pre	ALT Post	AST Pre	AST Post	ALP Pre	ALP Post	Relation AST/ALT Pre	Relation AST/ALT Post
1	35.9	36	22.4	46	36	98	0.62	1.28
2	17.6	20	14.9	47	80	99	0.85	2.35
3	13.0	10	18.7	30	36	97	1.44	3.00
4	14.0	38	13.6	39	32	105	0.97	1.03
5	14.0	20	20.4	26	59	162	1.46	1.30
6	12.9	16	20.8	37	70	65	1.61	2.31
M ± SD	17.90 ± 8.98	23.33 ± 11.22	18.47 ± 3.50	37.50 ± 8.41	52.17 ± 20.34	104.33 ± 31.61	1.16 ± 0.40	1.88 ± 0.78
P	0.116• •		0.028#		0.046#		0.75• •	

Data are M ± SD (N = 6). ALT - alanine aminotransferase; AST - aspartate aminotransferase; ALP - alkaline phosphatase; Pre, before the race; Post, after the race; • not significant different from before race; # significantly different from before race (P < 0.05)

Table IV. Pearson's product-moment correlation coefficients relationships between of finishing time with liver damage markers.

		AST Post	ALP Post	Total race time
ALT Post	PC	0.509	0.080	0.405
	Sig	0.303	0.881	0.426
AST Post	PC		-0.521	0.274
	Sig		0.289	0.599
ALP Post	PC			0.051
	Sig			0.923

ALT - alanine aminotransferase; AST - aspartate aminotransferase; ALP - alkaline phosphatase; PC - Pearson's product-moment correlation coefficients; Sig - Significance ($P < 0.05$)

Discussion

Triathlon and half-ironman competitions are examples of endurance sports. These are characterized by an intense aerobic energy production due to the extension of the competition, leading to increased function of these physiological systems and increasing risk of damage.

The hepatic enzymes analyzed in this work are related to energy production and reflects hepatocyte integrity.

AST and ALT are enzymes that catalyze the transfer of amino groups from aspartate and alanine to ketoglutaric acid to the to generate oxaloacetic and piruvic acids that are involved in energy production under oxygen presence in mitochondria.

Both enzymes are localized in liver at high concentrations AST is also present in heart, skeletal muscle kidneys, brain and red blood cells. ALT has low concentrations in skeletal muscle, thus, an increase in this enzyme is an indication of liver damage or overload.¹⁰

In this study, all athletes showed normal AST, ALT and alkaline phosphatase levels before and after competition (levels under 45U/L for AST; 50 U/L for ALT and between 38 and 126 U/L for alkaline phosphatase).⁹

Interestingly, AST/ALT ratio was above 1, before and after competition, which could indicate some extent of liver damage or overload.

In the literature there is some discussion on the diagnostic importance of the AST/ALT ratio. Alterations in this parameter are related to hepatic damage, which can be a result of acetaminophen chronic use, alcohol intoxication, hepatitis.¹¹⁻¹⁵ The magnitude of the AST/ALT ration alteration alone can not serve as a concluding laboratory result for liver or muscle damage, as both enzymes changes their concentrations accordingly to patient's clinical history, age, sex, physical activity level and diet.^{16,17}

Observing our data, there was a significative alteration on AST levels after the competition, considering that elevated AST levels are related to a series of clinical disorders such as hepatitis, cirrhosis, anemia, muscular dystrophies, cardiac muscle failure and skeletal muscle damage for example, the elevated ratio was a result of this enzyme alteration due to the skeletal muscle lesion that occurred during competition.

As cited above, AST is present in various organs, including skeletal muscle and is mostly mitochondrial, so, its alteration reflects destruction of cells, liberating this enzyme in systemic circulation, leading to elevated concentrations in venous blood.¹⁸

During strenuous exercise, such as half-ironman competitions, skeletal muscle function is extremely requested, for strength, potency and obviously, for energy production to maintain this functions.

During aerobic energy production AST and ALT catabolizes amino acids, permitting them to enter the citric acid cycle thus generating ATP to sustain not only muscle contraction but all physiologic adaptations as increased heart and respiratory rates and thermoregulation among others.

Thus, considering AST and ALT concentrations in athletes blood before and after endurance competitions corroborates previous works.

Exercise temporally increases the activities of AST, ALT and CPK (creatine phosphokinase), and physical fitness has no direct relation on resting serum enzymes levels, exception made for AST and CPK which can be altered by exercise duration.¹⁹

In 1999, Margaritis et al²⁰ observed significant changes after triathlon competition, that caused muscle damage and serum enzymes activities and concluded that muscle enzyme release alone cannot be used to predict the magnitude of muscle function impairment.

Smith et al²¹ conducted a similar study in marathon runners and concluded that biochemical and hematological tests can show «abnormal» results, indicating that several physiological alterations occur during strenuous exercise and can be quantified using routinely laboratory tests.

Another important finding in our study is a significant alteration in alkaline phosphatase serum levels. This enzyme transports metabolites across cell membranes including lipids for oxidative energy production. Liver and bone diseases are the most common causes of pathological elevation of ALP as for these tissues shows.²² Again, ALP increase taken alone could not be related to any pathological alteration in liver or bone in athletes, for the reasons described above regarding energy production via oxidative pathways and use of long-term energy production metabolites, as aminoacids and lipids, which occur during long-term physical activity. Thus an elevation in ALP after the competition reflects liver increased activity for gluconeogenesis, lipid peroxidation and probably increased bone turnover induced by the duration and intensity of the competition. This altered parameter is also related to an antioxidant capacity induced by exercise as a adaptative response to minimize damage by free radicals produced during long-term energy production using aminoacids or lipids.^{23,24}

In conclusion, our results demonstrate, as other authors have done before, that physical activities, at a com-

petition level induces a series of metabolically alterations, mainly related to energy production and also to muscle and skeletal adaptations during training and pre-competition preparation that remain as 'default' in athletes reflecting those physiological adaptations. The altered status of those metabolical changes, specially after strenuous exercise cannot reflect the intensity of any possible muscular or hepatic damage or overload, other parameters, such as hematological and even subjective indicators as pain perception must be considered.

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