Ibrahim Musa, J Biomed & App Sci FUD (2024) 3:2
www.jobas.fud.edu.ng
ISSN: 2814-3221 (print), ISSN: 2955-0874 (online)

JOBASFUD
Journal of Biomedical and Applied Sciences

RESEACH ARTICLE

THE EFFECT OF 90-MINUTE FOOTBALL MATCH AND TRAINING STATUS ON HEPATORENAL FUNCTIONS IN ADULT HEALTHY MALES

Ibrahim Musa[1], Mohammed Abdul-Aziz Mabrouk[2], Yusuf Tanko[3], Abdulrazaq Bidemi Nafiu[4]

1 Department of Human Physiology, Prince Abubakar Audu university, Anyigba,
2 Department of Human Physiology, Bayero University, Kano,
3 Department of Human Physiology, Ahmadu Bello University, Zaria, 4 Department of Physiology, Federal University, Dutse.

Abstract

Background: While the health benefits of regular muscular exercise are well-documented through numerous epidemiological studies, the precise dose-response relationship remains inadequately understood. This study was designed to assess the acute impact of a soccer match on the hepatorenal functions of male football players. **Methods:** Using a purposeful sampling technique, a total of 20 adult male football players and twenty age-matched sedentary males were recruited for this study. Twenty male football players were recruited for this study. Blood samples (3 ml) were collected at rest and immediately following a 90-minute competitive soccer match to evaluate hepatorenal function parameters, including total bilirubin (TBIL), albumin (ALB), alanine aminotransferase (ALT), aspartate aminotransferase (AST), AST/ALT ratio, creatinine, urea, and various electrolytes. Standard clinical methods were employed to measure these parameters. **Results:** At baseline (pre-match), the footballers exhibited significantly higher serum levels of TBIL, ALB, AST/ALT ratio, and creatinine compared to sedentary control group (TBIL: 11.35 ± 2.6 vs. 5.05 ± 1.7 mmol/l, p = 0.060; ALB: 48.75 ± 1.02 vs. 44.31 ± 0.7 g/l, p = 0.001; AST/ALT: 0.98 ± 0.06 vs. 0.53 ± 0.07, p = 0.0001; creatinine: 70.05 ± 2.3 vs. 64.46 ± 5.1 µmol/l, p = 0.003; K+: 5.07 ± 0.2 vs. 4.6 ± 0.1 mmol/l, p = 0.043). Conversely, their pre-match serum ALT and chloride levels were significantly lower than those of the sedentary group (ALT: 27.70 ± 2.1 vs. 38.78 ± 3.1 IU/L, p = 0.006; Cl-: 97.65 ± 2.9 vs. 104.69 ± 0.8 mmol/l, p = 0.01). No significant differences were observed between the two groups in serum alkaline phosphatase (ALP), conjugated bilirubin (CBIL), total protein (TP), bicarbonate (HCO3-), sodium (Na+), and urea levels at baseline. Following the soccer match, significant increases were observed in the footballers' serum urea and creatinine levels compared to their pre-match values (urea: 3.7 ± 0.2 vs. 2.9 ± 0.1 mmol/l, p = 0.012; creatinine: 81.9 ± 3.0 vs. 70.0 ± 2.3 µmol/l, p = 0.003). Additionally, post-match serum AST, AST/ALT ratio, and K+ levels were significantly reduced compared to pre-match levels (AST: 18.85 ± 1.5 vs. 26.85 ± 2.1 IU/L, p = 0.002; AST/ALT: 0.81 ± 0.05 vs. 0.98 ± 0.06, p = 0.007; K+: 4.2 ± 0.1 vs. 5.07 ± 0.2 mmol/l, p = 0.009). A similar reduction in serum ALT was observed in the sedentary group post-match (ALT: 31.84 ± 2.0 vs. 38.78 ± 3.1 IU/L, p = 0.029). Other parameters showed no significant differences between pre- and post-match levels. Comparatively, the post-match analysis revealed that the footballers had significantly higher serum levels of TP, ALB, AST/ALT ratio, urea, and creatinine (p < 0.05) than the sedentary group (TP: 82.70 ± 1.1 vs. 73.26 ± 3.7 g/l, p = 0.02; ALB: 49.85 ± 1.0 vs. 40.89 ± 1.8 g/l, p = 0.0001; AST/ALT: 0.81 ± 0.05 vs. 0.53 ± 0.07, p = 0.006; urea: 3.77 ± 0.2 vs. 2.56 ± 0.1 mmol/l, p = 0.0001; creatinine: 81.90 ± 3.0 vs. 55.63 ± 4.8 µmol/l, p = 0.0001). However, their post-match serum ALT and Na+ levels were significantly lower than those of the sedentary group (ALT: 22.40 ± 1.2 vs. 31.84 ± 2.0 IU/L, p = 0.001; Na+: 137.15 ± 3.0 vs. 141.78 ± 0.7 mmol/l, p = 0.01). Moreover, the percentage change in serum ALP, TBIL, CBIL, and Cl- from pre- to post-match was significantly higher in footballers compared to the sedentary (ALP: 50.65 ± 9.4 vs. 26.57 ± 4.8 IU/L, p = 0.031; TBIL: 12.15 ± 2.3 vs. 5.89 ± 1.8 mmol/l, p = 0.043; CBIL: 3.9 ± 0.9 vs. 1.2 ± 0.6 mmol/l, p = 0.027; Cl-: 10.45 ± 2.7 vs. 3.52 ± 0.7 mmol/l, p = 0.025). Conversely, the percentage change in TP and creatinine levels was significantly lower in footballers compared to the sedentary group (TP: 7.95 ± 0.8 vs. 10.57 ± 3.0 g/l, p = 0.001; creatinine: 14.15 ± 2.9 vs. 25.63 ± 4.01 µmol/l, p = 0.025). **Conclusions**: The findings of this study indicate that acute exposure to a soccer match and regular training does not have a deleterious effect on hepatorenal functions in male football players. Despite the exercise-induced fluctuations in hepatorenal blood flow, the liver and kidneys appear capable of maintaining their metabolic functions within homeostatic limits.

Keywords: soccer, football, biochemical, liver, enzymes, kidney, sedentary, trained

Introduction

Exercise-induced change in the body system (Bangsbo *et al.*, 2006) has been given considerable attention to understanding the fundamental science behind it and its implications on general body functions. Changes in appearance established through epigenetic alterations are usually noticed after some years of training (Allender *et*

* Corresponding Author: Ibrahim Musa,
Tel: +2347031570218
E-mail: ibrophs@yahoo.com

al., 2006). These changes form the basis for training and optimal sports performance (Parsons and Betz, 2001) as well as the state of health of the participants (Bonci *et al.,* 2008). Physical activity and exercise training programs strongly influence health, fitness and quality of life (Marquez *et al.* 2020). In the fields of clinical medicine and physiology, the benefits of exercise are apparent. These benefits include enhanced insulin resistance, improved lipid profile, decreased inflammation and oxidative stress, improved organ functions, and prevention of complications associated with or without weight gain (Johnson and George, 2010). Exercise training delays chronic degenerative diseases (Booth et al., 2012), ageing (Distefano *et al.,* 2018), osteoporosis, atherosclerosis, sarcopenia (Booth *et al.,* 2012), diabetes (Syeda *et al.,* 2023) and Non-Alcoholic Fatty Liver Disease (NAFLD) pathophysiology (Carneros *et al.,* 2020). Whether a differential response exists due to the exercise type, duration and/or intensity is debatable but has been reported by some studies (Tanasescu *et al.,* 2002; Lee *et al.,* 2003; Kyu *et al.,* 2016). Each sport type and duration make various demands on the structural and functional cells to mitigate athletes' body physiology (Gottlieb *et al.,* 2021). Several studies have shown that aerobic exercise training improves measurements of cardiovascular performance in health as expressed by an increase in peak volume of oxygen (VO_2) that the body consumes during exercise or at rest (Fujimoto *et al.,* 2010) and an improvement of other parameters of functional and prognostic relevance such as the ventilatory efficiency during exercise (VE/VCO_2) slope, the ventilator aerobic threshold, and the Heart Rate Recovery (HRR), a parameter expressing the sympatho-vagal balance (Giallauria *et al.,* 2005; Fujimoto *et al.,* 2010). Indeed, Soccer is considered to be the most popular sporting event in the world (Shephard, 1999). However, scholarly information on the effects of a soccer match on the hepatorenal functions in professional players and untrained remains unavailable. Therefore, this study aimed to investigate the impact of a 90-minute football match and training status on hepatorenal functions. Examination of the hepatorenal functions responses to acute exercise in professional players and untrained males may help to pinpoint the mechanisms by which soccer training can reduce the risk of hepatorenal diseases, inform the optimization of soccer training programmes and assist with the identification of training- and non-training-related alterations in hepatorenal functions based on the type of sports and the duration of play in years.

Materials and Methods

Subjects

A sample of twenty adult male football players and twenty sedentary males of similar age range of 18-25 years were recruited for this observational study using a purposeful sampling technique. Approval for the study protocol was granted by the Ethics Committee of Ahmadu Bello University Teaching Hospital (ABUTH/HREC/TRG/36) in accordance with the Declaration of Helsinki. The twenty male footballers were selected from the football club of the University amateur league except for the goalkeepers. The 20 sedentary selected had not been engaged in regular exercise programs (less than 2 times per week) during the previous 6 months. These subjects were questioned about their performance and their health problems. Physical parameters such as age, height and body weight were recorded. Informed consent was obtained from all the subjects. None of the subjects was taking drugs or medications. Volunteers were excluded if they had cardiovascular, metabolic, pulmonary or limiting osteoarticular diseases, obesity (body mass index > 30 kg/m2), if they were smokers, or if they used any medication that could influence the cardiovascular responses investigated here-in

Experimental Design

On the morning of an experiment, subjects reported to the field between 09:00 and 011:00 A.M. after a 12–14-h overnight fast. After a rest period of 10 minutes, and immediately after the match, a venous blood sample (3 mL) was collected into plain tubes from a forearm vein with minimal stasis. The blood was allowed to clot at room temperature and was then centrifuged at 4000 rpm for 10 minutes in the Departmental Laboratory. The serum was then collected and stored at -20°C until biochemical analyses.

Acute Exercise Study

The study involved a 90-minute football match as described by (Rahnama *et al.,* 2009). The match consists of 90 minutes: 45 minutes, 15 minutes rest, then 45 minutes each between the playing teams. It was organized for the trained and the untrained group on separate days. The athletic group played as two separate teams on day one, while the non-athletic group played as two separate teams on day two of the test. The protocols were performed between 09:00 and 011:00 A.M to standardize the circadian influence with temperature between 23 °C and 24 °C and humidity between 60% and 70%.

Determination of liver function parameters

The determination of serum; albumin (ALB) and total protein (TP) for liver synthetic functionality, aspartate amino transferase (AST) and alanine amino transferase (ALT) for cellular integrity of the liver, Alkaline phosphatase (ALP) with total bilirubin (TBIL) and conjugate bilirubin (CBIL) for condition linked to the biliary tract integrity were all performed in a laboratory automated analyser (Selectra XL Vital Scientific, Spankeren, NL) as adopted by Ouédraogo *et al.,* (2013). Briefly, the laboratory automated analyzer was put on and allowed to warm for 5 minutes followed by blanking of the machine (0 reading calibration). Serum samples were placed into the sample rotors of the machine. The specific test reagents were also placed in the respective reagent rotor and the cuvettes were also fixed into the respective cuvettes rotor. Then, the appropriate tests to be performed were selected or highlighted using the computer software interface of the machine. The machine was started and it automatically pipettes the sample from the test tubes, mixed it with the appropriate reagents in the cuvettes and followed by incubation for 3 minutes. The absorbance of the standard and samples against the reagent blank were displayed on the screen of the automated analyzer and read.

Determination of renal function parameters

Urea and creatinine for renal functionality were measured using spectrophotometric methods and electrolyte levels were analyzed using standard methods as described below. The entire tests were carried out in the Department of Chemical Pathology, Ahmadu Bello University Teaching Hospital Zaria, Nigeria. Serum urea was measured according to the method described by Wybenga *et al.,* (1971) based on the reaction of urea and hot acidic Diacetylmonoxime in the presence of Thiosemicarbazide to produce a rose-purple coloured complex which was determined colourimetrically on a spectrophotometer at 520 nm within 10 min. The concentration in mmol/l was then calculated from the equation; Urea (mmol/l) = Test/Standard x concentration of standard (10). Serum creatinine was measured according to the methods described by Sirois (1995) based on the reaction of creatinine and alkaline picrate reagent to form an orange-red colour which is measured in a spectrophotometer at 520 nm. The concentration in mmol/l was then calculated from the equation; Creatinine (mmol/l) = Test/standard x concentration of standard (530).

Determination of serum electrolytes

Serum sodium, potassium, chloride and bicarbonate concentrations were measured according to Lorenz *et al.,* (2003) using an Audicom automated machine: model AC9000 Electrolyte Analyzer (Audicom Medical Technology, Jena, Germany).

Statistical analysis

The data generated were expressed as mean ± standard error of the mean (X±SEM). For statistical analysis, SPSS software (version 20.0) was used; the students- t-test was used to compare values between the two groups. A comparison was considered statistically significant if the P value was < 0.05.

Results

There was no significant difference ($p > 0.05$) in age and body weight between the athletic group and the non-athletic group. However, the height of the athletic group was significantly higher ($p < 0.05$) compared to the non-athletic group. Notably, the body mass index among the athletic group reduced significantly ($p < 0.05$) compared to the non-athletic group (Table 1).

Table I: Physical characteristics of the study population (n=39).

Characteristics	Non-athletes	Athletes
Age (Years)	20.00±0.4	20.60±0.5
Height (meters)	1.70±0.0	1.76±0.0*
Weight (Kg)	58.60±1.0	60.25±1.3
Body mass index (Kg/m^2)	20.19±0.3	19.34±0.2*

Values are mean ± SEM. * indicates significant difference ($p < 0.05$) athletes vs. non-athletes.

The mean physiological variables of the study population are presented (Table 2). There was a significant increase ($p < 0.05$) in the mean VO_2 max among the athletic group compared to the non-athletic group. Similarly, the mean Heart rate showed a significant reduction ($p < 0.05$) among the athletic group compared to the non-athletic group. In addition, the mean diastolic blood pressure (DBP) was significantly higher ($p < 0.05$) among the athletic group compared to the non-athletic group. However, there was no significant difference ($p > 0.05$) in the mean systolic blood pressure (SBP), pulse pressure (PP), and mean arterial pressure (MAP) levels between the athletic group and the non-athletic group.

Table 2: Physiological variables of the study population (n =39).

Physiological Variables	Non-athletes	Athletes
VO_{2max} (ml/Kg/min)	50.26±0.3	58.39±0.3*
Heart rate (b/min)	80.40±3.6	65.40±2.1*
Systolic blood pressure (mmHg)	128.0±2.3	128.05±2.1
Diastolic blood pressure (mmHg)	69.05±1.7	74.15±1.7*
Pulse pressure (mmHg)	58.95±2.5	53.90±1.7
Mean arterial blood pressure (mmHg)	88.30±1.5	91.85±1.6

Values are mean ± SEM. * indicates significant difference ($p < 0.05$) athletes vs. non-athletes.

The mean hepatorenal function markers of the study population are presented (Table 3-8). The results of this study showed that the footballers' pre-match serum TBIL, ALB, AST/ALT Ratio, and Creatinine were significantly higher than those of the untrained (TBIL: 11.35 ± 2.6 vs. 5.05 ± 1.7 mmol/l, p = 0.060; ALB: 48.75 ± 1.02 vs. 44.31 ± 0.7 g/l, p = 0.001; AST/ALT:0.98 ± 0.06 vs. 0.53 ± 0.07, p = 0.0001; creatinine:70.05 ± 2.3 vs. 64.46 ± 5.1 µmol/l, p = 0.003; K: 5.07 ± 0.2 vs. 4.6 ± 0.1 mmol/l, p = 0.043), whereas the footballers' pre match serum ALT and chloride ions was significantly lower than those of the untrained (ALT: 27.70 ± 2.1 vs. 38.78 ± 3.1 IU/L p = 0.006; Chloride: 97.65 ± 2.9 vs. 104.69 ± 0.8 mmol/l, p = 0.01). Moreover, there were no significant differences in the pre-match serum ALP, CBIL, TP, Bicarbonate, Na+ and Urea between the footballers and those of the untrained. In addition, the footballers' serum urea and creatinine were significantly higher immediately after the match above the pre-match level (Urea: 3.7± 0.2 vs. 2.9 ± 0.1 mmol/l, p = 0.012; Creatinine: 81.9 ± 3.0 vs. 70.0 ± 2.3 µmol/l, p = 0.003), whereas the footballers' serum AST, AST/ALT Ratio and K+ were significantly lower immediately after the match below the pre match level (AST: 18.85 ± 1.5 vs. 26.85 ± 2.1 IU/L, p = 0.002; AST/ALT: 0.81 ± 0.05 vs. 0.98 ± 0.06, p = 0.007; K: 4.2± 0.1 vs. 5.07 ± 0.2 mmol/l, p = 0.009). Similarly, the untrained' serum ALT was significantly lower immediately after the match below the pre-match level (ALT: 31.84 ± 2.0 vs. 38.78 ± 3.1 IU/L, p = 0.029). However, all other markers do not show significant differences between the pre-and post-match levels. While the footballers' post- match serum TP, ALB, AST/ALT, urea and creatinine were significantly higher (p< 0.05) as compared with the untrained (TP: 82.70±1.1 vs 73.26±3.7 g/l, p = 0.02; ALB: 49.85±1.0 vs 40.89±1.8g/l, p = 0.0001; AST/ALT: 0.81 ± 0.05 vs. 0.53 ± 0.07, p = 0.006; Urea: 3.77±0.2 vs 2.56±0.1 mmol/l, p = 0.0001; Creatinine: 81.90±3.0 vs 55.63±4.8 µmol/l, p = 0.0001), their post- match serum ALT and sodium was significantly lower (p< 0.05) as compared to the untrained group (ALT: 22.40±1.2 vs 31.84±2.0 IU/L, P = 0.001; Na+: 137.15±3.0 vs 141.78±0.7 mmol/l, p = 0.01). The footballers' % change between the pre-and post- match serum ALP, TBIL, CBIL, and Cl- was significantly higher as compared with the untrained (ALP: 50.65± 9.4 vs. 26.57 ± 4.8 IU/L, p = 0.031; TBIL: 12.15± 2.3 vs. 5.89 ± 1.8 mmol/l, p = 0.043; CBIL: 3.9± 0.9 vs. 1.2 ± 0.6 mmol/l, p = 0.027; Cl-: 10.45± 2.7 vs. 3.52 ± 0.7 mmol/l, p = 0.025). On the other hand, their % change between pre-and post-match serum TP and creatinine was significantly lower as compared with the untrained (TP: 7.95 ± 0.8 vs. 10.57 ± 3.0 g/l, p = 0.001; Creatinine: 14.15 ± 2.9 vs. 25.63 ± 4.01 µmol/l, p = 0.025).

Discussions

Football exercise has been regarded as moderate-intensity to high-intensity exercise (Ekun *et al.*, 2017). These are characterized by 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 enzyme parameters analyzed in this work are related to energy production and reflect hepatocyte integrity. AST and ALT are enzymes that catalyze the transfer of amino groups from aspartate and alanine to ketoglutaric acid to generate oxaloacetic and pyruvic acids that are involved in energy production under oxygen presence in mitochondria. However, elevated ALT has been used as a proxy for hepatocellular injury (Lin *et al.*, 2010) because it is more specific for hepatic damage than AST. The AST/ALT ratio is also considered a surrogate marker for hepatocyte necrosis and inflammation, with values >1.0 indicating hepatocyte damage (Anderson *et al.*, 2000). Therefore, using the AST/ALT as a true marker of hepatic damage during exercise may be equivocal (Pettersson *et al.*, 2008). In this study, all participants showed normal AST, ALT and alkaline phosphatase levels before and after the match. Nevertheless, the footballers' pre-match serum AST/ALT Ratio, was significantly higher than those of the untrained, whereas the footballers' pre-match serum ALT was significantly lower than those of the untrained. In addition, the footballers' serum AST, and AST/ALT Ratio were significantly lower immediately after the match below the pre-match level. Similarly, the untrained' serum ALT was significantly lower immediately after the match below the pre-match level. However, the footballers' post-match serum AST/ALT was significantly higher (p< 0.05) as compared with the untrained, likewise, their post-match serum ALT was significantly lower (p< 0.05) compared with the untrained group.

The findings from this study agreed partly with the previous study of Yazgaldi *et al.*, 2014, who investigated the levels of liver enzymes in acute aerobic exercise in sedentary women and observed that AST and ALP levels increased significantly but there was no significant change in ALT enzyme levels. However, in our study, a non-significant decrease (p<0.05) in ALT activities after a football exercise was observed.

Table 3: Pre- and post-match Match Liver Function Markers of the Participants

Parameters Exercise		Non-athletics	%Change	Athletics	%Change
AST (IU/L)					
	Pre	21.26±3.2	13.9	26.85±2.1	29
	Post	18.3±3.2		18.85 ± 1.5*	
ALT (IU/L)					
	Pre	38.78±3.1	17.8	27.70±2.1	19.1
	Post	31.84±2.0*		22.40 ± 1.7	
ALP (IU/L)					
	Pre	92.26±9.1	2.4	93.60±11.9	18.1
	post	94.52±11.8		76.65 ± 10.6	
TBIL (mmol/L)					
	Pre	5.05 ± 1.7	49.9	11.35 ± 2.6	35
	Post	7.57 ± 1.8		7.3 ± 2.5	
CBIL (mmol/L)					
	Pre	1.68 ± 0.7	25	3.5± 1.0	20
	Post	2.10 ± 0.8		2.80 ± 1.0	
TP (g/L)					
	pre	79.63 ±1.4	7.9	82.80 ± 1.4	0.1
	Post	73.26 ±3.7		82.70 ± 1.1	
ALB (g/L)					
	Pre	44.31 ± 0.7	7.7	48.75 ± 1.0	2.2
	post	40.89 ±1.8		49.85 ± 1.0	
AST/ALT					
	pre	0.53 ± 0.07	0	0.98 ± 0.06	17.3
	Post	0.53 ± 0.07		0.81 ± 0.07*	

Table 4: Comparison in the Levels of Pre-Match Liver Function Markers of the Participants

Variables	Non-athlete	Athlete	Normal range values
AST (IU/L)	21.26±3.2	26.85±2.1	5-22
ALT (IU/L)	38.78±3.1	27.70±2.1*	16-40
ALP (IU/L)	92.26±9.1	93.60±11.9	21-92
TBIL (mmol/L)	5.05 ± 1.7	11.35 ± 2.6*	4-17
CBIL (mmol/L)	1.68 ± 0.7	3.5± 1.0	4-17
TP (g/L)	79.63 ±1.4	82.80 ± 1.4	60-82
ALB (g/L)	44.31 ± 0.7	48.75 ± 1.02*	30-52
AST/ALT	0.53 ± 0.07	0.98 ± 0.06*	<1

Values are mean ± SEM (n=20). AST; aspartate aminotransferase, ALT; alanine aminotransferase, ALP; alkaline phosphatase, TBIL; total bilirubin, CBIL; conjugated bilirubin; total protein; ALB; albumin. * Statistically significant ($p< 0.05$) athletes vs. non-athletes and ns Not statistically significant ($p> 0.05$) athletes vs. non-athletes.

Table 5: Comparison in the Levels of Post-Match Liver Function Markers of the Participants

Variables	Non-athlete	Athlete	Normal range values
AST (IU/L)	18.31±3.2	18.85±1.5 ns	5-22
ALT (IU/L)	31.84±2.0	22.40±1.2*	16-40
ALP (IU/L)	94.52±11.8	76.65±10.6ns	21-92
TBIL (mmol/L)	7.57±1.8	7.30±2.5ns	4-17
CBIL (mmol/L)	2.10±0.8	2.80±1.0ns	4-17
TP (g/L)	73.26±3.7	82.70±1.1*	60-82
ALB (g/L)	40.89±1.8	49.85±1.0*	30-52
AST/ALT	0.53 ± 0.07	0.98 ± 0.06*	<1

Values are mean ± SEM (n=20). AST; aspartate aminotransferase, ALT; Alanine aminotransferase, ALP; alkaline phosphatase, TBIL; total bilirubin, CBIL; conjugated bilirubin, TP; total protein; ALB; albumin.* Statistically significant ($p< 0.05$) athletes vs. non-athletes and ns Not statistically significant ($p> 0.05$) athletes vs. non-athletes.

Table 6: Comparison in the Levels of Pre- and Post-Match Renal Function Markers of the Participants

Parameters	Exercise	Non-Athletic	%Change	Athletic	%Change
Urea (mmol/L)	Pre	2.75 ± 0.1	6.9	2.90±0.1	30
	Post	2.56±0.1		3.77 ± 0.2*	
Creatinine (µmol/L)	Pre	65.15±4.3	14.6	70.05±2.3	16.9
	Post	55.63±4.8		81.90 ± 3.0*	
Sodium (mmol/L)	Pre	142.73±0.9	0.6	139.05±1.2	1.3
	Post	141.78±0.7		137.15 ± 3.0	
Potassium (mmol/L)	Pre	4.58 ± 0.2	9.6	5.07 ± 0.2	15.9
	Post	4.14 ± 0.2		4.26 ± 0.1*	
Chloride (mmol/L)	Pre	104.31 ± 0.7	0.02	98.90 ± 1.6	1.2
	Post	105.00 ± 0.8		97.65 ± 2.9	
Bicarbonate (mmol/L)	Pre	25.78 ± 0.4	3.2	25.55± 0.8	2.5
	Post	24.94 ± 0.5		24.90 ± 0.5	

Table 7: Comparison in the Levels of Pre-Match Renal Function Markers of the Participants

Variables	Non-athletic	Athletic	Normal range values
Urea (mmol/L)	2.75 ± 0.1	2.90±0.1	2.5-6.5
Creatinine (µmol/L)	65.15±4.3	70.05±2.3*	9-126
Sodium (mmol/L)	142.73±0.9	139.05±1.2	136-145
Potassium (mmol/L)	4.58 ± 0.2	5.07 ± 0.2	3.6-5.2
Chloride (mmol/L)	104.31 ± 0.7	98.90 ± 1.6*	94-108
Bicarbonate (mmol/L)	25.78 ± 0.4	25.55± 0.8	24-32

Values are mean ± SEM (n=20). * Statistically significant (p< 0.05) athletes vs. non-athletes and ^{ns} Not statistically significant (p> 0.05) athletes vs. non-athletes.

Table 8: Comparison in the Levels of Post-Match Renal Function Markers of the Participants.

Variables	Non-athletic	Athletic	Normal range values
Urea (mmol/L)	2.56±0.1	3.77±0.2*	2.5-6.5
Creatinine (µmol/L)	55.63±4.8	81.90±3.0*	9-126
Sodium (mmol/L)	141.78±0.7	137.15±3.0^{ns}	136-145
Potassium (mmol/L)	4.14±0.2	4.26±0.1^{ns}	3.6-5.2
Chloride (mmol/L)	105.00±0.8	97.65±2.9*	94-108
Bicarbonate (mmol/L)	24.94±0.5	24.90±0.5 ^{ns}	24-32

Values are mean ± SEM (n=20). * Statistically significant (p< 0.05) athletes vs. non-athletes and ^{ns} Not statistically significant (p> 0.05) athletes vs. non-athletes.

The possible explanation for this is sparse at this moment; however, this could explain the critical metabolic activities the liver is involved in during exercise, which may involve this enzyme since ALT is largely found in the liver with much lower concentrations than in the skeletal muscles. Thus, based on tissue distribution, ALT seems to be one of the most specific markers of liver injury (Dufour Robert 2006); thus, a significant decrease in ALT in this study could suggest that soccer as a form of exercise may not pose any danger to liver function. Moreover, our study further agreed with a similar study by Ghorbani and Gaeini, (2013), which evaluated the effects of high-intensity training on liver enzyme levels in soccer players and observed a significant increase in plasma concentration of AST and ALP whereas there was no significant increase in plasma concentration of ALT. Also, ALP as a hydrolase (Nayudu and de Meis 1989) contributes to the hydrolysis of metabolites such as fats across cell membranes to produce energy; hence, the footballers' higher serum ALP % change in the pre-and post-match could aid gluconeogenesis and lipid peroxidation during football activity (Burger-Mendonca et al., 2008). The AST/ALT ratio is also considered a surrogate marker for hepatocyte necrosis and inflammation, with values >1.0 indicating hepatocyte damage (Anderson *et al.,* 2000). Therefore, using the AST/ALT as a true marker of hepatic damage during exercise may be equivocal (Pettersson *et al.,* 2008). In this study, the AST/ALT ratio was < 1.0 before and immediately after the exercise in all the participants, indicating hepatocyte and liver parenchyma structural integrity preservation. Therefore, even in the presence of intense exercise-induced hepatic blood flow, the liver can maintain its metabolic functions (Nielsen *et al.,* 2002).

There was a significant increase ($p<0.05$) in hepatic function biochemical parameters studied (TP, ALB, TBIL, CBIL). However, these elevated values are within the normal physiological range. This observation agrees with the previous studies of Crespo *et al.,* (1995) where it was reported that the pre-albumin levels were higher in athletes than in controls. The post-exercise result also agrees with the findings of Gravina *et al.,* (2011). The increase in the serum albumin level increased the H_2O-binding capacity of the blood (Convertino *et al.,* 1980) due to training-induced plasma hypervolemia. In addition, Wu *et al.,* (2004) also reported a significant increase in the serum total bilirubin and conjugate bilirubin concentrations immediately after 24 h ultra-marathon exercise. The increased level of TBIL observed in this study can be linked to increasing hemoxygenase-1 activity (Loprinzi and Abbott, 2014), which is the enzyme responsible for the conversion of biliverdin to bilirubin. There was a significant increase ($p<0.05$) in renal function biochemical parameters studied (urea, creatinine, Na^+ and K^+) after football exercise when compared with the pre-exercise values across the footballers. The footballers' pre-match serum Creatinine was significantly higher than those of the untrained. In addition, the footballers' serum urea and creatinine were significantly higher with decreased K^+ immediately after the match when compared with the pre-match level. However, the footballers' post-match serum sodium was significantly lower ($p< 0.05$) as compared to the untrained group. This observation agrees with the previous studies of Jacobs *et al.,* (2009) and Weinberger *and Abu-Hasan* (2009) where it was reported that after moderate-intensity and high-intensity exercise, there is a decrease in urine volume and a marked reduction in renal plasma flow and filtration rate leading to the increase in urea and creatinine values observed after exercise. Since football exercise is regarded as a moderate-intensity to high-intensity exercise, it is possible that there was probably a decreased urine volume as a result of a marked reduction in renal plasma flow as well as a reduction in glomerular filtration rate during football activities, hence an increase in post-football match urea and creatinine values. A previous study reported a marked decrease in serum sodium concentration after exercise (Montain *et al.,* 2006). Notably, the cause of hyponatremia in athletes remains controversial. The mechanisms involved in exercise-induced hyponatremia are still being investigated. However, the reduction observed could be due to increased perspiration and interleukin 6-induced antidiuretic hormone release (Kratz *et al.,* 2002). The intensity of the exercise protocol adopted in this study could limit the potassium efflux from the muscles since potassium is said to be released from the muscle during exercise in direct relation to the exercise intensity (Darrin *et al.,* 2005).

Conclusions

The acute football match and its regular training have no deleterious effect on the Hepatorenal functions. Despite its intense exercise-induced hepatorenal blood flow, the liver and the kidneys can maintain their metabolic functions within the homeostatic border.

Acknowledgment

The cooperation and understanding of the participants were highly acknowledged.

Conflict of Interest

The authors have no conflicts of interest to declare.

Funding Support: None

Author Contributions

IM: Conceptualization, Plan experiment, Investigation, Writing- original draft, ABN: Writing - review & editing, MAM: Conceptualization, Plan experiment, Writing - review & editing. YT: Conceptualization, Plan experiment, Writing - review & editing.

Article History:

Received: 05th August 2024.

Accepted: 26th August 2024.

Published online: 11th May 2025.

References

Allender, S., Cowburn, G., & Foster, C. (2006). Understanding participation in sport and physical activity among children and adults: A review of qualitative studies. *Health Education Research, 21*(6), 826-835.

Anderson, F. H., Zeng, L., Rock, N. R., & Yoshida, E. M. (2000). An assessment of the clinical utility of serum ALT and AST in chronic hepatitis C. *Hepatology Research, 18*, 63-71.

Bangsbo, J., Mohr, M., & Krustrup, P. (2006). Physical and metabolic demands of training and match-play in the elite football player. *Journal of Sports Sciences, 24*(7), 665-674.

Booth, F. W., Roberts, C. K., & Laye, M. J. (2012). Lack of exercise is a major cause of chronic diseases. *Comprehensive Physiology*, *2*(2), 1143–1211. https://doi.org/10.1002/cphy.c110025

Bonci, C. M., Bonci, L. J., Granger, L. R., Johnson, C. L., Malina, R. M., Milne, L. W., Ryan, R. R., & Vanderbunt, E. M. (2008). National Athletic Trainers' Association position statement: Preventing, detecting, and managing disordered eating in athletes. *Journal of Athletic Training, 43*(1), 80-108.

Burger-Mendonça, M., Bielavsky, M., & Barbosa, F. C. (2008). Liver overload in Brazilian triathletes after the half-Ironman competition is related to muscle fatigue. *Annals of Hepatology, 7*, 245-248.

Carneros, D., López-Lluch, G., & Bustos, M. (2020). Physiopathology of Lifestyle Interventions in Non-Alcoholic Fatty Liver Disease (NAFLD). *Nutrients*, *12*(11), 3472. https://doi.org/10.3390/nu12113472

Convertino, V. A., Brock, P. J., Keil, L. C., Bernauer, E. M., & Greenleaf, J. E. (1980). Exercise training-induced hypervolemia: Role of plasma albumin, renin, and vasopressin. *Journal of Applied Physiology, 48*(4), 665-669.

Crespo, R., Relea, P., Lozano, D., Macarro-Sanchez, M., Usabiaga, J., Villa, L. F., & Rico, H. (1995). Biochemical markers of nutrition in elite marathon runners. *Journal of Sports Medicine and Physical Fitness, 35*(4), 268-272.

Darrin, S. N., Jens-Jung, J., Bangsbo, J., & Juel, C. (2005). Metabolic alkalosis reduces exercise-induced acidosis and potassium accumulation in human skeletal muscle interstitium. *Journal of Physiology, 566*, 481-489.

Distefano, G., & Goodpaster, B. H. (2018). Effects of Exercise and Aging on Skeletal Muscle. *Cold Spring Harbor perspectives in medicine*, *8*(3), a029785. https://doi.org/10.1101/cshperspect.a029785

Dufour, R. D. (2006). Liver diseases. In C. A. Burtis, E. R. Ashwood, D. E. Bruns (Eds.), *Tietz textbook of clinical chemistry and molecular diagnostics* (pp. 1777-1847). Elsevier.

Ekun, O. A., Emiabata, A. F., Abiodun, O. C., et al. (2017). Effects of football sporting activity on renal and liver functions among young undergraduate students of a Nigerian tertiary institution. *BMJ Open Sport & Exercise Medicine, 3*, e000223. https://doi.org/10.1136/bmjsem-2017-000223

Fujimoto, N., Prasad, A., Hastings, J. L., Arbab-Zadeh, A., Bhella, P. S., Shibata, S., et al. (2010). Cardiovascular effects of 1 year of progressive and vigorous exercise in individuals older than 65 years of age. *Circulation, 122*, 1797-1805.

Ghorbani, P., & Gaeini, A. A. (2013). The effect of one bout high-intensity interval training on liver enzyme levels in elite soccer players. *Journal of Basic and Applied Scientific Research, 5*, 1191-1194.

Giallauria, F., DelForno, D., Pilerci, F., DeLorenzo, A., Manakos, A., & Lucci, R. (2005). Improvement of heart rate recovery after exercise training in older people. *Journal of the American Geriatrics Society, 53*, 2037-2038.

Gottlieb, R., Shalom, A., & Calleja-Gonzalez, J. (2021). Physiology of Basketball - Field Tests. Review Article. *Journal of human kinetics*, *77*, 159–167. https://doi.org/10.2478/hukin-2021-0018

Gravina, L., Ruiz, F., Lekue, J. A., Irazusta, J., & Gil, S. M. (2011). Metabolic impact of a soccer match on female players. *Journal of Sports Sciences, 29*(12), 1345-1352.

Jacobs, D. R., Sluik, D., Rokling-Andersen, M. H., et al. (2009). Association of 1-year changes in diet pattern with cardiovascular disease risk factors and adipokines: Results from the 1-year randomized Oslo Diet and Exercise Study. *American Journal of Clinical Nutrition, 89*, 509-517.

Johnson, N. A., & George, J. (2010). Fitness versus fatness: Moving beyond weight loss in nonalcoholic fatty liver disease. *Hepatology, 52*(1), 370-381.

Kratz, A., Lewandrowski, K. B., Siegel, A. J., Chun, K. Y., Flood, J. G., Van Cott, E. M., & Lee-Lewandrowski, E. (2002). Effect of marathon running on hematologic and biochemical laboratory parameters, including cardiac markers. *American Journal of Clinical Pathology, 118*, 856-863.

Kyu, H. H., Bachman, V. F., Alexander, L. T., Mumford, J. E., Afshin, A., Estep, K., Veerman, J. L., Delwiche, K., Iannarone, M. L., Moyer, M. L., Cercy, K., Vos, T., Murray, C. J., & Forouzanfar, M. H. (2016). Physical activity and risk of breast cancer, colon cancer, diabetes, ischemic heart disease, and ischemic stroke events: Systematic review and dose-response meta-analysis for the Global Burden of Disease Study. *BMJ, 354*, i3857.

Lee, I. M., Sesso, H. D., Oguma, Y., & Paffenbarger, R. S., Jr. (2003). Relative intensity of physical activity and risk of coronary heart disease. *Circulation, 107*, 1110-1116.

Lin, C. Y., Lin, L. Y., Chiang, C. K., Wang, W. J., Su, Y. N., & Hung, K. Y. (2010). Investigation of the associations between low-dose serum perfluorinated chemicals and liver enzymes in US adults. *American Journal of Gastroenterology, 105*, 1354-1363.

Loprinzi, P. D., & Abbott, K. (2014). Physical activity and total serum bilirubin levels among insulin-sensitive and insulin-resistant U.S. adults. *Journal of Diabetes and Metabolic Disorders, 13*(1), 47.

Lorenz, J. M., Kleinman, L. I., Kaplan, L. A., Pesce, A. J., & Kazmierczak, S. C. (2003). Physiology and pathology of body water and electrolytes. In *Clinical Chemistry: Theory, Analysis, Correlation* (4th ed.).

Marquez, D. X., Aguiñaga, S., Vásquez, P. M., Conroy, D. E., Erickson, K. I., Hillman, C., Stillman, C. M., Ballard, R. M., Sheppard, B. B., Petruzzello, S. J., King, A. C., & Powell, K. E. (2020). A systematic review of physical activity and quality of life and well-being. *Translational Behavioral Medicine, 10*(5), 1098-1109. https://doi.org/10.1093/tbm/ibz198

Montain, S. J., Cheuvront, S. N., & Sawka, M. N. (2006). Exercise-associated hyponatremia: Quantitative analysis to understand the etiology. *British Journal of Sports Medicine, 40*, 98-106.

Nayudu, R. V., & de Meis, L. (1989). Energy transduction at the catalytic site of enzymes: Hydrolysis of phosphodiester bonds and synthesis of pyrophosphate by alkaline phosphatase. *FEBS Letters, 255*, 163-166.

Nielsen, H. B., Clemmensen, J. O., Skak, C., Ott, P., & Secher, N. H. (2002). Attenuated hepatosplanchnic uptake of lactate during intense exercise in humans. *Journal of Applied Physiology, 92*, 1677-1683.

Ouédraogo, M., Zerbo, P., Konaté, K., Barro, N., & Laya, L. S. (2013). Effect of long-term use of Sidarhombifolia L. extract on hemato-biochemical parameters of experimental animals. *British Journal of Pharmacology and Toxicology, 4*(1), 18-24.

Parsons, E. M., & Betz, N. E. (2001). The relationship of participation in sports and physical activity to body objectification, instrumentality, and locus of control among young women. *Psychology of Women Quarterly, 25*(3), 209-222

Pettersson, J., Hindorf, U., Persson, P., Bengtsson, T., Malmqvist, U., Werkström, V., & Ekelund, M. (2008). Muscular exercise can cause highly pathological liver function tests in healthy men. *British Journal of Clinical Pharmacology, 65*, 253-259.

Rahnama, N., Younesian, A., Mohammadion, M., & Bambaeichi, E. (2009). A 90-minute soccer match decreases triglyceride and low-density lipoprotein but not high-density lipoprotein and cholesterol levels. *Journal of Research in Medical Sciences, 14*(6), 335-341.

Shephard, R. J. (1999). Biology and medicine of soccer: An update. *Journal of Sports Sciences, 17*(10), 757-786.Sirois, M. (1995). Blood biochemistry. In *Veterinary Clinical Pathology Laboratory Procedures* (pp. 144-167).

Syeda, U. S. A., Battillo, D., Visaria, A., & Malin, S. K. (2023). The importance of exercise for glycemic control in type 2 diabetes. *American Journal of Medicine Open, 9*, 100031.

Mosby. Tanasescu, M., Leitzmann, M. F., Rimm, E. B., Willett, W. C., Stampfer, M. J., & Hu, F. B. (2002). Exercise type and intensity in relation to coronary heart disease in men. *JAMA, 288*, 1994-2000.

Weinberger, M., & Abu-Hasan, M. (2009). Perceptions and pathophysiology of dyspnea and exercise intolerance. *Pediatric Clinics of North America, 56*, 33-48.

Wu, H. J., Chen, K. T., Shee, B. W., Chang, H. C., Huang, Y. J., & Yang, R. S. (2004). Effects of 24 h ultra-marathon on biochemical and hematological parameters. *World journal of gastroenterology, 10*(18), 2711–2714. https://doi.org/10.3748/wjg.v10.i18.2711

Wybenga, R., Di Giorgio, J., & Pileggi, V. J. (1971). Manual and automated methods for urea nitrogen measurement in whole serum. *Clinical Chemistry, 17*, 891-895.

Yazgaldi, N., Abuzar, M., et al. (2014). Response of liver enzymes to acute aerobic exercise in sedentary human subjects. *New York Science Journal, 7*, 89-92.