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The effect of high-intensity interval training on hematological variables and lipid profiles in team game athletes

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Abstract

Introduction. High-intensity interval training (HIIT) has gained popularity as a quick and effective way to exercise, and the training consists of a short burst of intense exercise that precedes a period of rest or low intensity exercise. Aim of Study. The present study aimed to investigate the effect of an 8-week HIIT program on the lipid profiles and hematological variables of young players. Material and Methods. A training program was introduced to 40 male players [football (n = 20) and field hockey (n = 20)]. The training set includes 2-min intense sprint interval workout (at 90-95% of HR_{max}, work:rest = 1 : 1) followed by a minute each of active recovery and complete rest continued for 8 weeks with 3 days/week alterations. The lipid profiles, hematological variables, maximum oxygen uptake capacity (VO_{2max}), and anaerobic power (W_{peak}) of the participants were assessed following standard procedures. Results. A significant ($p < 0.001$) decrease was recorded in body fat% (7.6%), white blood cell (9.2%), red blood cell (2.3%), ferritin (21.5%), hemoglobin (2.5%), hematocrit (2.3%), total cholesterol (7.5%), triglycerides (8.7%), total cholesterol/high-density lipoprotein cholesterol (14.2%) and very-low-density lipoprotein cholesterol (6.1%, $p < 0.05$) after the introduction of the HIIT protocol. In turn, body weight (1.1%), body mass index (1.1%), high-density lipoprotein cholesterol (7.9%), platelet (5.9%), mean corpuscular volume (3.5%), platelet-to-leukocyte (16.7%), VO_{2max} (13.6%) and W_{peak} (11.6%) were found to be significantly ($p < 0.001$) increased after the training. Conclusions. The 8-week sprint HIIT protocol resulted in an improved endurance capacity and anaerobic power with an overall improvement in the lipid profiles of young athletes. The HIIT also contributed to a reduction in oxygen-carrying capacity with increased erythrocytic hemolysis.

KEYWORDS: sprint interval training, high-intensity training response, lipid profile measure, blood hematological parameters, endurance capacity.

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Introduction

High-intensity interval training (HIIT) improves both aerobic and anaerobic energy systems over a short period by involving alternating bouts of intensive sprinting exercise with low-intensity recovery periods [18, 29]. HIIT is even defined for longer (30 sec-4 min) intervals with submaximal oxygen consumption levels (approx. 85-90% VO_{2max}) [9]. Whyte et al. [29] reported the impact of HIIT on fat and body weight loss and concluded that HIIT can increase fat oxidation (even up to 18%), especially on post-training days. Besides, Sarkar, et al. [27] reported that low lactate accumulation and reduction in glycogen utilization by improving the efficiency of muscular buffering capacity resulted from high-intensity sprint interval training (HISIT) compared to conventional endurance training.

The lipid profile is an indicator for predicting cardiovascular disease (CVD) development in adults,

with an increase in low-density lipoprotein-cholesterol (LDL-C) and a decrease in high-density lipoprotein cholesterol (HDL-C) serum levels also indicating the onset of coronary heart disease (CHD) in adults [23]. During physical activity, 30-80% of energy comes from fats and muscles use free fatty acids and triglycerides (TG), either circulating or stored, for energy production [23]. Earlier Musa et al. [24] reported that the 8-week HIIT protocol significantly elevated the HDL-C and reduced the total cholesterol (TC) of healthy adults.

Exercise has a pronounced physiologic impact on hematological variables depending on the type, intensity, and duration of the exercise [6]. During HIIT, an increasing number of erythrocytes was reported, which may compensate for the elevated demand for oxygen [13]. Earlier studies reported that endurance training likely has an impact on blood volume changes mainly due to an exercise-induced plasma volume (PV) expansion, which results in lower hemoglobin (Hb) and hematocrit (HCT) levels in athletes. Leukocytes and circulating platelet (PLT) counts have been found to increase during the post-exercise phase in healthy adults [8]. Intense exercise can increase the plasma viscosity and erythrocyte rigidity, but can decrease the sedimentation rate [1]. Studies such as e.g. Belviranli et al. [2] and Heidari et al. [15] confirmed HIIT-induced acute alterations in hematological variables of immediate post-exercise conditions, but the 12-24 hr recovery conditions were only reported by Jamurtas et al. [16].

Except for the study of Jastrzebska et al. [17], previous studies of Belviranli et al. [2], Jamurtas et al. [16], etc. only focused on the effect of high-intensity interval exercise (acute/short-term effect) on various indices. Musa et al. [24] reported unpredictable results of high-intensity exercise-induced changes in the lipid profile and hematological variables mainly due to various work:rest intervals. The present study aimed to investigate the effect of an 8-week sprint HIIT protocol on lipid profile variables, and hematological [complete blood count (CBC)] indices as a long-term adaptive response in young Indian male team game athletes.

Material and Methods

Subject Selection

Forty (n = 40) young Indian male football and field hockey players were recruited as subjects of the present study. The subjects were divided into two age-matched groups: (i) the control group (n = 20; mean age = 16.7 ± 1.43 yrs), and (ii) the HIIT group (n = 20; mean age = 16.4 ± 1.34 yrs), respectively. All the participants had a minimum 5 years of formal training experience and they were only included after clinical examination. During the clinical examination prior to the commencement of the study, the athletes were examined for any serious health issues, hereditary diseases, and injury history. All the subjects were residential players and thus they were considered to form a homogeneous group, characterised by similar dietary habits and socioeconomic status. They underwent training in identical environmental/climatic conditions. The study protocol followed the guidelines of the Declaration of Helsinki and a signed informed consent was obtained from each subject. Proper ethical clearance (Ref No. IHEC/AB/P82/2019) was also obtained from the Institutional Human Ethical Committee (IHEC), the Department of Physiology, the University of Calcutta.

Detailed training program

Three hours of HIIT (sprint intervals) were introduced for 3 days/week (alternative days) for 8 weeks. Daily 3-hr HIIT was divided into two sessions [both morning and evening sessions of 1:30 hr each]. Each training session started with a warm-up session and ended with a cool-down session (both sessions consisted of 15-20 min of slow running at an intensity around 50% of HR_{max}). During HIIT the subjects were asked to perform a total 4 sets/sessions (morning or evening) of all-out training, which was divided into 2 phases of 2 sets each, 2 minutes of intense sprint workout (at 90-95% of HR_{max}) followed by an active recovery and complete rest each with a 1-min interval. The whole training workload was maintained at work:rest = 1 : 1. Each interval training set comprised max. 3 repetitions. Sets of repetition were modified by increasing their number throughout the 8 weeks, i.e., 5 sets in the 1^{st}–2^{nd} week, 6 sets in the 3^{rd}–4^{th} week, 7 sets in the 5^{th}–6^{th} week, and 8 sets in the 7^{th}–8^{th} week, respectively.

The control group players continued systematic low-volume physical training, which included low-intensity physical activity (i.e., stretching, jogging, low-intensity running, etc.) for 6 days/week for 2 hours/day. Players of both groups performed game-specific skill training (i.e., dribbling, passing, tackles, movement techniques, etc.) activity on a regular basis.

Anthropometric variables

Physical characteristics, i.e., standing height (cm) and body weight (kg), were measured using a Seca Alpha stadiometer (model 213, Seca Deutschland, Germany) and a Seca Alpha weighing scale (model 770, Seca Deutschland, Germany), respectively. The body mass index (BMI) was calculated by the standard formulae [26].

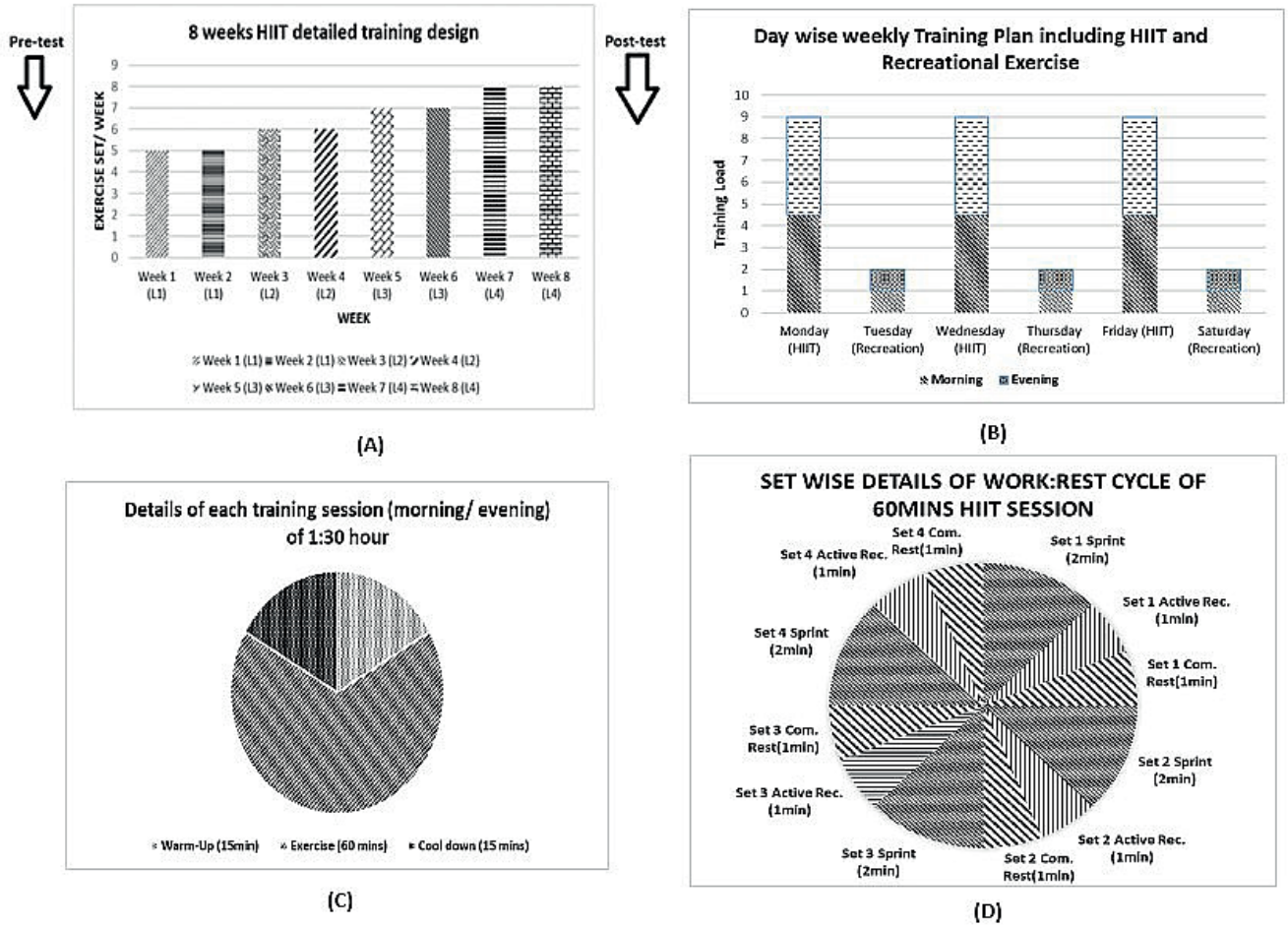

Figure 1. (A) 8-weeks HIIT detailed training design, (B) day-wise weekly training plan including HIIT and recreational exercise, (C) details of each training session (morning/ evening) of 1:30 hour, (D) set-wise details of work:rest cycle of 60-min HIIT session

Body fat percentage (BF%) was calculated by measuring the skinfold thickness at the biceps, triceps, subscapular, and supra-iliac muscles [12].

Blood collection and plasma sample preparation

Blood samples were collected from the antecubital vein into centrifuge tubes for serum preparation (without anticoagulant) between 6:00 AM-8:00 AM in the pre-prandial state (after 10 hrs of fasting) to avoid possible differences due to diurnal variation. Blood samples were centrifuged (REMI centrifuge, R-8C) at 3000 rpm for 15 minutes to ensure complete separation of serum. All laboratory tests were performed at room temperature of 23-25 ºC with 50-60% relative humidity.

Assay for lipid profile

The serum lipid profile included TC, TG, HDL-C, LDL-C, and very-low-density lipoprotein cholesterol (VLDL-C). Complete lipid profile measurements were made using the reagents of Span Diagnostics Ltd., India. The TC and the HDL-C fractions were estimated by the method of Manna and colleagues [22] at 560 nm. TG was estimated at 500 nm on a spectrophotometer and LDL-C was indirectly measured using the following standard procedure. All values of TC, TG, HDL-C, LDL-C, and VLDL-C were expressed in 'mg/ dl' [22].

$$LDL\text{-}C = TC - (HDL\text{-}C + TG/5)$$

Determination of CBC

White blood cell (WBC), PLT, red blood cell (RBC), Hb, HCT, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) were the inclusive parameters under the CBC profile. The CBC was analyzed using a Beckman Coulter ACT 5 DIFF CP analyzer

(Bera, California, US) [11]. MCV was calculated using the formula Ht/RBC × 10. MCH was calculated by Hb/RBC × 10. MCHC was calculated by Hb/Ht × 100 [11].

Physical fitness variables

A breath by breath automated pre-calibrated metabolic gas analyzer (MetaMax 3B, CORTEX Biophysik GmbH, Leipzig, Germany) was used to determine the maximal oxygen consumption (VO_{2max}) by maintaining the following criteria: (i) a plateau in VO_2 (2 ml/kg/min) despite the increasing workload, (ii) respiratory exchange ratio (RER) ≥ 1.1, (iii) > 90% of age-predicted HR_{max} ± 5%, and (iv) voluntary exhaustion [27]. Relative anaerobic peak power output (W_{peak}) was measured by dividing absolute peak anaerobic power (A_{peak}) by body weight, whereas A_{peak} was predicted by using the running-based anaerobic sprint test (RAST). The participants were asked to perform six consecutive sprints at maximal speed over the distance of 35m with a 10-second rest period between each sprint. The timing of each sprint was recorded using a Brower timing gate system [26].

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS) version 18.0 (SPSS Inc., Chicago, Il, USA, 2009) was used for the statistical analysis of the data. All the data were expressed as means ± standard deviation (SD). Shapiro-Wilk's test was conducted to check the normality of distribution. The two-tailed paired sample t-test was carried out to calculate the mean differences between pre- and post-phase variables within the same group. ANOVA was also performed to calculate the mean differences between the post-phase variables of the two different groups. A 95% confidence interval was considered as the level of significance. The linear regression prediction models were tested at the $p < 0.05$ level of significance.

Results

The effect of HIIT intervention on the anthropometric variables is presented in Table 1. Weight and BMI were significantly increased in the post-training phase of both the HIIT ($p < 0.001$) and control ($p < 0.01$) groups when compared with their pre-training data. In turn, fat mass percentage (FM%) of the post-training phase was significantly decreased in the HIIT group ($p < 0.001$) and increased in the control group ($p < 0.001$) in comparison to their pre-training values. On the other hand, body height was found to be significantly ($p < 0.01$) increased in the post-training phase of both the HIIT and control groups in comparison to their pre-training counterparts. The effect of HIIT intervention on lipid profile variables is presented in Table 2. Among the lipid profile variables, TC, TG, VLDL-C, and the TC/HDL-C ratio were found to be significantly ($p < 0.001$) lower in the post-intervention phase of the HIIT group when compared with the baseline data except for VLDL-C at $p < 0.05$. In turn, only HDL-C was found to be significantly ($p < 0.001$) increased after the training intervention in the HIIT group. On the other hand, the lipid profile of the control group players was altered in a statistically significant manner.

The effect of HIIT intervention on hematological variables is presented in Table 3. In the HIIT group, WBC, RBC, ferritin, Hb, and HCT were found to be decreased significantly ($p < 0.001$) in the post-intervention phase in comparison to the PT1 phase data. On the other hand, PLT, MCV, and platelet-to-leukocyte

Table 1. Comparison of anthropometric parameters between pre- and post-HIIT intervention

Parameters	Groups	Pre-training (n = 20)	Post-training (n = 20)	Level of significance	P value	% change	ANOVA
Height (cm)	Control	168.20 ± 5.22	168.24 ± 5.21	–3.559**	0.002	0.02(~)	0.953^{NS} (p = 0.335)
	HIIT	169.65 ± 4.17	169.70 ± 4.16	–2.932**	0.009	0.03(~)	
Weight (kg)	Control	59.19 ± 5.37	60.08 ± 4.73	–3.192**	0.005	1.5(↑)	0.036^{NS} (p = 0.853)
	HIIT	59.13 ± 4.78	59.80 ± 4.74	–10.642***	<0.001	1.1(↑)	
BMI (kg/m^2)	Control	20.92 ± 1.59	21.23 ± 1.46	–3.164**	0.005	1.5(↑)	1.082^{NS} (p = 0.305)
	HIIT	20.54 ± 1.43	20.76 ± 1.40	–10.157***	<0.001	1.1(↑)	
FM%	Control	11.90 ± 2.92	12.24 ± 2.85	–8.643***	<0.001	2.9(↑)	1.269^{NS} (p = 0.267)
	HIIT	12.08 ± 3.04	11.16 ± 3.17	11.942***	<0.001	7.6(↓)	

Note: Values are means ± SD, **p < 0.01, ***p < 0.001, NS – non-significant, HIIT – high-intensity interval training, BMI – body mass index, FM% – fat mass percentage, ANOVA – ANOVA between the post-training phase of HIIT and the control group

Table 2. Comparison of plasma lipid profiles between pre- and post-HIIT intervention

Parameters	Groups	Pre-training (n = 20)	Post-training (n = 20)	Level of significance	P Value	% change	ANOVA
TC (mg/dl)	Control	150.0 ± 30.71	150.5 ± 29.29	–5.185***	<0.001	0.3(↑)	10.079** (p = 0.003)
	HIIT	147.60 ± 22.70	136.50 ± 18.73	4.888***	<0.001	7.5(↓)	
TG (mg/dl)	Control	63.7 ± 9.30	63.9 ± 8.85	–3.018**	0.007	0.3(↑)	8.649** (p = 0.006)
	HIIT	61.15 ± 12.23	55.85 ± 10.34	7.491***	<0.001	8.7(↓)	
HDL-C (mg/dl)	Control	43.1 ± 8.49	43.0 ± 7.73	0.076(NS)	0.940	0.2(↓)	3.812^{NS} (p = 0.058)
	HIIT	44.20 ± 7.42	47.70 ± 7.49	–20.571***	<0.001	7.9(↑)	
LDL-C (mg/dl)	Control	87.3 ± 21.14	87.9 ± 19.64	–2.196*	0.041	0.7(↑)	0.533^{NS} (p = 0.470)
	HIIT	86.20 ± 18.01	84.60 ± 17.09	0.406(NS)	0.689	1.9(↓)	
VLDL-C (mg/dl)	Control	16.5 ± 5.48	16.7 ± 5.03	–2.436*	0.025	1.2(↑)	1.068^{NS} (p = 0.308)
	HIIT	16.80 ± 2.93	15.78 ± 3.25	2.765*	0.012	6.1(↓)	
TC/HDL-C	Control	3.5 ± 0.66	3.6 ± 0.51	–2.526*	0.021	2.9(↑)	36.910*** (p < 0.001)
	HIIT	3.37 ± 0.45	2.89 ± 0.36	9.147***	<0.001	14.2(↓)	

Note: Values are means ± SD, *p < 0.05, **p < 0.01, ***p < 0.001, NS – non-significant, HIIT – high-intensity interval training, TC – total cholesterol, TG – triglycerides, HDL-C – high-density lipoprotein-cholesterol, LDL-C – low-density lipoprotein-cholesterol, VLDL-C – very high-density lipoprotein-cholesterol, ANOVA – ANOVA between the post-training phase of HIIT and the control group

Table 3. Comparison of hematological variables between pre- and post-HIIT intervention

Parameters	Groups	Pre-training (n = 20)	Post-training (n = 20)	Level of significance	P Value	% change	ANOVA
WBC (*10^3/µl)	Control	6.84 ± 1.20	6.71 ± 1.67	0.326(NS)	0.748	1.9(↓)	2.083^{NS} (p = 0.157)
	HIIT	6.63 ± 1.45	6.02 ± 1.34	13.038***	<0.001	9.2(↓)	
RBC (*10^6/µl)	Control	5.42 ± 0.49	5.35 ± 0.50	1.000(NS)	0.330	1.3(↓)	3.371^{NS} (p = 0.074)
	HIIT	5.21 ± 0.27	5.09 ± 0.30	10.450***	<0.001	2.3(↓)	
PLT (*10^3/µl)	Control	247.40 ± 58.93	244.60 ± 57.32	0.836(NS)	0.413	1.1(↓)	0.029^{NS} (p = 0.866)
	HIIT	228.20 ± 52.37	241.65 ± 52.15	–12.543***	<0.001	5.9(↑)	
Ferritin (ng/ml)	Control	61.10 ± 21.72	59.78 ± 21.87	0.719(NS)	0.481	2.2(↓)	3.572^{NS} (p = 0.066)
	HIIT	60.60 ± 20.37	47.58 ± 18.99	23.936***	<0.001	21.5(↓)	
Hb (gm/dl)	Control	14.76 ± 0.93	14.59 ± 0.72	1.398(NS)	0.178	1.2(↓)	1.940^{NS} (p = 0.172)
	HIIT	14.66 ± 0.68	14.29 ± 0.61	9.797***	<0.001	2.5(↓)	
HCT	Control	43.86 ± 2.53	43.37 ± 2.86	0.573(NS)	0.574	1.1(↓)	1.083^{NS} (p = 0.305)
	HIIT	43.45 ± 3.07	42.44 ± 2.81	5.992***	<0.001	2.3(↓)	
MCV (fl)	Control	82.43 ± 6.33	82.65 ± 4.98	–0.162(NS)	0.873	0.3(↑)	3.639^{NS} (p = 0.064)
	HIIT	82.45 ± 4.13	85.38 ± 4.03	–36.254***	<0.001	3.5(↑)	
MCH (pg)	Control	27.74 ± 2.63	28.25 ± 2.00	–0.842(NS)	0.410	1.8(↑)	0.091^{NS} (p = 0.764)
	HIIT	28.57 ± 1.30	28.43 ± 1.76	0.344(NS)	0.734	0.5(↓)	
MCHC (gm/dl)	Control	33.65 ± 1.09	34.19 ± 1.08	–1.499(NS)	0.150	1.6(↑)	2.513^{NS} (p = 0.121)
	HIIT	33.79 ± 0.95	33.72 ± 0.74	0.260(NS)	0.798	0.2(↓)	
PLR	Control	36.83 ± 9.22	37.71 ± 9.79	–0.478(NS)	0.638	2.4(↑)	1.265^{NS} (p = 0.268)
	HIIT	34.97 ± 7.17	40.80 ± 7.37	–21.029***	<0.001	16.7(↑)	

Note: Values are means ± SD, ***p < 0.001, NS – non-significant, HIIT – high-intensity interval training, WBC – white blood cell, RBC – red blood cell, PLT – platet, Hb – hemoglobin, HCT – hematocrit, MCV – mean corpuscular volume, MCH – mean corpuscular hemoglobin, MCHC – mean corpuscular hemoglobin concentration, PLR – platelet-to-leukocyte ratio, ANOVA – ANOVA between the post-training phase of HIIT and the control group

Table 4. Comparison of endurance capacity and anaerobic power output between pre- and post-HIIT intervention

Parameters	Groups	Pre-training (n = 20)	Post-training (n = 20)	Level of significance	P Value	% change	ANOVA
VO_{2max} (ml/kg/min)	Control	50.34 ± 3.66	50.95 ± 3.51	–5.719***	<0.001	1.2(~)	31.007*** (p < 0.001)
	HIIT	51.23 ± 4.78	58.19 ± 4.64	–31.830***	<0.001	13.6(↑)	
W_{peak} (watt/kg)	Control	7.51 ± 1.05	7.44 ± 0.95	1.313(NS)	0.205	0.9(↓)	9.578** (p = 0.004)
	HIIT	7.60 ± 1.35	8.48 ± 1.35	–16.474***	<0.001	11.6(↑)	

Note: Values are means ± SD, *p < 0.05, **p < 0.01, ***p < 0.001, NS – non-significant, HIIT – high-intensity interval training, VO_{2max} – maximum oxygen consumption, W_{peak} – relative anaerobic peak power output, ANOVA – ANOVA between the post-training phase of HIIT and the control group

Table 5. Pearson's product-moment correlation coefficient of selected lipid profile and hematological variables with endurance capacity and anaerobic power output

Variables	VO_{2max}	W_{peak}
Cholesterol	–0.126	–0.322*
HDL	0.330*	–0.005
TC/HDL ratio	–0.448**	–0.291
WBC	–0.144	0.064
RBC	–0.402*	–0.171
PLT	–0.164	–0.086
Ferritin	–0.388*	–0.485**
Hb	–0.212	0.214
HCT	–0.267	0.112
PLR	–0.067	–0.126

Note: *p < 0.05, **p < 0.01, NS – non-significant, VO_{2max} – maximum oxygen consumption, W_{peak} – relative anaerobic peak power, TC – total cholesterol, HDL – high-density lipoprotein, WBC – white blood cell, RBC – red blood cell, PLT – platet, Hb – hemoglobin, HCT – hematocrit, PLR – platelet-to-leukocyte ratio

Table 6. Prediction of regression coefficient based on the linear regression model of young players

Variables	VO_{2max}					W_{peak}				
	R^2	Adj R^2	β	t value	Sig.	R^2	Adj R^2	β	t value	Sig.
	0.333	0.211				0.420	0.315			
TG			0.028	0.164	0.871			–0.102	–0.638	0.528
TC/HDL ratio			–0.354	–2.349	0.025			–0.221	–1.571	0.126
RBC			–0.223	–0.963	0.343			0.093	0.430	0.670
Ferritin			–0.183	–1.065	0.295			–0.580	–3.626	0.001
Hb			–0.156	–0.865	0.393			0.228	1.355	0.185
HCT			0.049	0.226	0.822			0.199	0.983	0.333

Note: Adj R^2 – adjusted R^2, VO_{2max} – maximum oxygen consumption, W_{peak} – relative anaerobic peak power, TG – triglycerides, TC/HDL ratio – total cholesterol to high-density lipoprotein ratio, Hb – hemoglobin, HCT – hematocrit

(PLR) ratios were found to be increased significantly ($p < 0.001$) in the post-intervention phase of the HIIT group when compared with the baseline data. Only the MCH and MCHC variables of the HIIT group were altered in a statistically insignificant manner. Among the control group data, all the hematological variables of CBC were found to be altered in a statistically non-significant manner.

The effect of HIIT intervention on some selected physical fitness parameters is presented in Table 4 VO_{2max} and W_{peak} were found to be significantly increased in the post-training HIIT group in comparison to pre-training HIIT ($p < 0.001$) and post-training control ($p < 0.001$ for VO_{2max}; $p < 0.01$ for W_{peak}) counterparts. In turn, the changes in VO_{2max} and W_{peak} were statistically non-significant when comparing the post-training with pre-training periods of the control counterparts.

Table 5 presents Pearson's correlation coefficient between the selected lipid profile and hematological parameters with physical fitness variables (i.e., VO_{2max}, and W_{peak}). VO_{2max} was found to be negatively and significantly correlated with the TC/HDL ratio, RBC, and ferritin. In turn, VO_{2max} was only positively and significantly correlated with the HDL level. On the other hand, W_{peak} was found to be negatively and significantly correlated with cholesterol and ferritin levels.

Table 6 presents the prediction regression model for VO_{2max} and W_{peak}. The first regression prediction model represents $F = 2.741$, $Sig = 0.028$ for the dependent variable – VO_{2max} (predictors: TG, HDL/cholesterol ratio, RBC, ferritin, Hb, and HCT). In turn, the second model represents $F = 3.989$, $Sig = 0.004$ for the dependent variable – W_{peak} (predictors: TG, HDL/cholesterol ratio, RBC, ferritin, Hb, and HCT).

Discussion

The present study investigated the adaptive training effect of 8-week sprint HIIT on the lipid profile and hematological parameters in trained male athletes. The results of the present study showed an overall improvement in lipid profile variables with a reduction in body weight. The study also showed a reduction in the oxygen-carrying ability of blood cells, which may be the effect of HIIT.

In the present study, HIIT intervention was suggested to improve body composition by significantly ($p < 0.001$) reducing BF% (7.6%) and increasing body weight (1.1%) and BMI (1.1%). This observation is in agreement with the study report of Musa et al. [24]. High-intensity training generally leads to a reduction in body weight and fat% by a suggested increase in the lipolysis rate (fat oxidation) induced by an increase in certain hormonal profiles (i.e., catecholamine) controling the β-adrenergic receptors in the adipose tissue [19]. Skeletal muscle fat oxidation is a highly regulated process and may be limited by several long-chain fatty acid membrane transporters, among which fatty acid-binding protein ($FABP_{pm}$) and fatty acid translocase CD36 are the most important [3]. Previously high-intensity exercise/ HIIT was suggested to increase the lipolysis rate/fat oxidation triggered by an increase in certain hormonal profiles, i.e., catecholamine, which controls the β-adrenergic receptors in the adipose tissue and increased $FABP_{pm}$ content in skeletal muscle [28]. On the other hand, the increase in skeletal muscle fat oxidation likely results from several adaptations, including an increase in mitochondrial volume [7], and altering several regulatory steps; adipose tissue lipolysis of TG to fatty acids, transport of fatty acid into the cell, intramuscular lipolysis of TG to fatty acids and ultimately fatty acid transport into the mitochondria [3]. In the present study, TC, TG, TC/HDL-C, and VLDL-C ($p < 0.05$) were found to be decreased significantly ($p < 0.001$) by 7.4%, 9.2%, 14.7%, and 5.9%, respectively, after the 8-week HIIT program. In turn, only HDL-C was found to be increased significantly ($p < 0.001$) by 7.9% after the same exercise protocol. The present study corroborates the research data of Musa et al. [24] and Ouerghi et al. [25]. Musa et al. [24] reported an 18% increase in HDL-C and an 18% reduction in the atherogenic index (TC/HDL-C ratio) followed by an 8-week HIIT (work:rest = 1 : 1) training with 4-5 min interval length. Finally, the longer interval training and/or a higher work:rest ratio were found to be more efficient in raising HDL-C and lowering the TG response. The HIIT-induced increase in HDL-C and reduction in the atherogenic index could potentially lead to a reduction in heart disease risk by 18-27%. It has been hypothesized that the risk of CHD can even be reduced by 53% with every unit drop in the TC/HDL-C ratio [20]. The study of Manna et al. [22] showed that the aerobic part of high-intensity training felicitates beneficial changes in HDL-C compared to low-intensity exercises. Furthermore, Ouerghi et al. [25] reported that training can induce significant changes in the lipid profile both in athletes and in sedentary untrained subjects not only for high-intensity training, but also for continuous or intermittent aerobic training. Such discrepancies among previous findings may be due to variations in several factors i.e., training intensity, training type, the timing of blood draw, the subject's ethnicity, study methodology, etc. [25].

In their study Jamurtas et al. [16] reported a significant increase in MCV, MCHC, WBC, and lymphocyte counts among all the hematological variables in immediate post-exercise conditions. However, the altered hematological profile will get back to its resting level after 3-6 hrs of the recovery phase [2]. In turn, Halson et al. [14] reported a consistent change in some hematological parameters at even resting-state conditions. In the present study, a decrease in WBC, RBC, ferritin, Hb, and HCT and an increase in PLT, MCV, and PLR counts were observed even after 8 weeks of the HIIT protocol, which corroborates the findings of Jastrzebska et al. [17] and Chou et al. [5]. The HISIT-induced decrease in RBC (2.3%), Hb (2.5%), and HCT (2.4%) might be due to the exercise-induced PV expansion and increased hemolysis of RBC, which confers the consequence of exhaustive exercise-induced muscular fatigue [8, 30]. The strenuous exercise-induced RBC hemolysis may be caused by oxidative stress, and/or physical trauma in circulation, and the damaged erythrocyte membrane can lead to hemolysis due to the reduced cellular deforming capability and increased membrane rigidity [5]. The presently studied decrease in HCT (2.4%) may indicate the mechanism of blood dilution and movements of transmission fluids inside the blood vessels, which helps to compensate for the immediate effect of exercise-induced blood volume loss and to restore cellular homeostasis [14]. Similarly, Jastrzebska et al. [17] and Wilkinson et al. [30] found a 25-30% reduction in ferritin levels after 6-8 weeks of HIIT, indicating latent iron deficiency which may be explained by the dilution effect of blood volume expansion. However, a reduction in serum ferritin was reciprocally related to the TIBC, which maintains the storage of iron in the blood by improving the iron-binding capacity [30]. The presently studied non-significant increase in PLT count (5.9%) may be due to the variation in releasing fresh platelets from the spleen and bone marrow and/or may be due to the increased secretion of epinephrine during exercise [14]. Further, an increase in MCV count (3.6%) in the present study may reflect the increasing number of erythrocytes deformed (structural damage) by high-intensity exercise resulting in hemolysis [11].

The present study showed a significant ($p < 0.001$) rise in VO_{2max} (13.6%), and W_{peak} (11.6%), thus corroborating the study of MacPherson et al. [21] where VO_{2max} and W_{peak} improvements were reported for the HIIT protocols varying in durations, training sets, work:rest cycle gaps, athletic groups, and genders. Significantly increased VO_{2max} was mainly due to improved mitochondrial enzyme activities, i.e., citrate synthase [7], cytochrome c oxidase (COX), COX subunits II, IV protein content [10], and β–hydroxy acyl-CoA dehydrogenase [7, 4]. In turn, MacPherson et al. [21] and Gibala and McGee [10] demonstrated that post-training enhancements in stroke volume and maximal cardiac output (Q_{max}) led to a 15-35% improvement in VO_{2max} following HIIT. Considerably, the improved W_{peak} corresponds to an improved anaerobic capacity index, which might be due to the improved lactate tolerance and developed sprinting ability within the anaerobic zone with a higher glycolytic activity level [27]. Similarly, Sarkar et al. [27] showed that the HIT response comprises increased glycolytic enzymatic activities (e.g., hexokinase, glycogen phosphorylase, phosphofructokinase), increased muscle buffering capacity, and ionic adaptations including increased $Na^+ – K^+$ –ATPase content and function as the adaptive response to compensate for the increased energy demand during an anaerobic HIIT. In other studies, Burgomaster et al. [4] and Sarkar et al. [27] reported an improved anaerobic capacity that may be due to muscular adaptations [reduced phosphocreatine degradation, enhanced glycogen content] along with an increased type IIA ratio and a reduced IIB ratio, which has been repeatedly demonstrated in response to low-volume high-intensity sprint interval training.

In this study Pearson's product-moment correlation for VO_{2max} not only showed the oxygen-carrying dependency (a direct significant relation), but also revealed a novel finding indicating lipid profile variance for training adaptations in VO_{2max}. Furthermore, in the correlation study ferritin was identified to be the most definite variable for predicting and varying physical fitness levels (both VO_{2max}, and W_{peak}) as the individual capacity of training adaptation. In the present study, two separate linear regression models were drawn from phase II data to predict endurance capacity (VO_{2max}) and relative anaerobic power (W_{peak}). Here, TG, the HDL/cholesterol ratio, RBC, ferritin, Hb, and HCT were able to significantly predict the linear regression model for VO_{2max} with $t = 4.762$ (significance at $p < 0.001$) and F value = 2.741 (significance at $p = 0.028$). However, those variables were not able to predict the linear regression model for W_{peak} with $t = 0.038$ (significance at $p = 0.970$) and F value = 3.989 (significance at $p = 0.004$).

Strengths and limitations of the study

This is one of the pioneering studies to assess the effect of sprint HIIT on the blood hematological parameters and lipid profile among the young athlete population of the Indian subcontinent. The study results will not only help athletes and coaches to establish guidelines,

but also help to maintain the balance between increasing thetraining load and blood hematological balance within the physiologic fatigue limit to reach optimal performance. The present study was concerned only with a specific type of intense sprint interval training (2 min intense sprinting at 90-95% of HR_{max} with work:rest = 1 : 1) as HIIT, which is a limitation of the study, as there are many more types of HIIT available, which may modify training-induced alterations in the hematological profile. On the other hand, the study included only male players as the subjects because of their easy availability, so it can add another limitation to the study. Another limitation of the study is the ethnicity of the study sample, which may only be found in south-east Asia.

Conclusions

The present study suggested an 8-week sprint HIIT protocol as an effective and time-efficient strategy to significantly induce the enhancement in fat oxidation [Fat% (↓7.6%)] with an improved lipid profile [↑7.9% HDL-C and ↓14.2% atherogenic index (TC/HDL-C)] along with enhanced endurance capacity (↑13.6%) and anaerobic power output [W_{peak}(↑11.6%)]. However, the present findings also indicate that the HIIT protocol may limit the oxygen-carrying capacity of blood cells with an increased risk of erythrocytic hemolysis (↓2.3% RBC) supported by a significant decrease in ferritin (21.5%), Hb (2.5%) and HCT (2.3%) values. The present training mode is also likely to have some damaging effect on the immune system, as it significantly decreases the WBC (9.2%) count with increased PLT (5.9%) and PLR (16.7%) values after HIIT intervention. So, the present high-intensity training intervention may be observed to improve performance variables for endurance capacity, anaerobic power, and explosive strength with enhanced fat oxidation and better lipid profiling, but in terms of inducing the hemolytic prone condition. The present observations may also help athletes and coaches in planning/formulating a systematic HIIT program under impairing exercising intensity with proper work:rest intervals, which might not induce any damage and help to maintain the athletes' physiologic harmony under the fatigue limit. However, the present study results may also be helpful for future research in the sprint HIIT program based on the limitations and expected outcomes.

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Conflict of Interest

The authors declare no conflict of interest.

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