Sport Sciences for Health (2019) 15:157–165
https://doi.org/10.1007/s11332-018-0505-7

ORIGINAL ARTICLE

Shorter recovery time following high-intensity interval training induced higher body fat loss among overweight women

Seyed Javad Mirghani[1] · Mehdi Seydyousefi[2] · Satu Pekkala[3] · Shohreh Sharifian[4] · Gheysar Beyshami[5]

Received: 2 July 2018 / Accepted: 21 September 2018 / Published online: 9 October 2018

Abstract

Background High-intensity interval training (HIIT) has been widely employed as an exercise protocol to reduce fat, and also in addition, interval recovery periods are the most important factors which can have great impact on weight loss. Therefore, the aim of this study was the evaluation of different recovery time between HIIT program on metabolic responses and weight loss in overweight women.

Methods Twenty-four overweight (BMI 29.5 ± 3.5 kg/m^2) volunteer women were randomly assigned into three groups ($n = 8$/group) to study the effects of different recovery times: [group 1; HIIT with 60/60 activity–rest ratio (s), group 2; HIIT with 60/30 activity–rest ratio (s), and group 3 was set as a control]. The participants performed 3 times per week and 4 bouts/session (80% of Heart Rate Reserve). The exercise program gradually increased to 10 bouts/session.

Results The most important findings of this study were a change in the body fat percentage (BF %) in the between group comparison: group1 (40.5 ± 0.9), group 2 (41.2 ± 0.7) and group 3 (41.1 ± 1.1). Compared to the control group the 60/30 s HIIT resulted in a significant decrease in BF % ($P = 0.002$). However, no other significant differences in the body composition were found. Either there were no significant differences between the groups in T4, T3 and TSH, cortisol, HGH, FBS, blood insulin, insulin resistance insulin sensitivity or fatness-associated hormones.

Conclusions In conclusion, considerable decrease in BF % in the 60/30 s. rest interval group indicates that 30 s recovery period in HIIT may reduce fat % more efficiently than 60 s.

Keywords Metabolic indices · Weight loss · Interval recovery time · Overweight women · HIIT

Introduction

Obesity is a physiological condition that may act as a potential risk factor for metabolic syndrome, type-II diabetes, cardiovascular disease, cancer and other diseases [1]. Therefore, obesity demands effective strategies to reduce weight and further the risk for metabolic syndrome. It has been demonstrated that physical activity is an effective neurologic-hormonal and metabolic stimulus to mobilize stored fat to produce energy, and therefore, it is an important way to combat obesity [2]. Recent findings indicate that especially high-intensity interval training (HIIT) is an effective exercise protocol that reduces over-weightiness in obese individuals [3–5] and has been shown to cause physiological adaptations similar to endurance training program, such as endocrinologic adaptations [6, 7]. Regardless of weight loss in response to exercise programs, the programs result in changes of hormones involved in fat metabolism [8]. For instance, Wahl et al. [9] showed that HIIT results in cortisol secretion after 10 min and during the recovery period. It also seems that compared to continuous exercise, HIIT causes positive changes that are beneficial to blood glucose control and insulin sensitivity [10, 11] as well as reduction of insulin resistance [12]. It may be that factors such as the interval recovery periods between the stages of activities are involved in fat metabolism and weight reduction in addition to the duration and intensity of exercise program [13, 14]. However, the effects of recovery periods have been scarcely studied, but it has been demonstrated that acute exercise results in an increase of many circulating metabolism-involved hormones [15]. These hormones include catecholamine, corticosteroid, growth hormone, thyroid hormones and gonadotropins (androgen, estrogen), among others, that regulate lipid metabolism [16]. There is

✉ Seyed Javad Mirghani
seyedgavadmirghani@yahoo.com

Extended author information available on the last page of the article

a relationship between thyroid hormones and cortisol after exercise; the immediate response of cortisol has a negative and significant correlation with the delayed secretion of TSH and T3 after 24 h. Besides, thyroid hormones decrease up to 24 h after this exhausting activity while cortisol response has an inverse relationship with the reduction of thyroid hormones. In fact, the cortisol secretion can cause thyroid hormone secretion and function [17]. In addition, Bracken et al. [18] reported that performing high-intensity exercise on treadmill with 30/60 s activity–rest intervals increased the level of catecholamine, one of the hormones involved in fat metabolism. Nevill et al. [19] also found that 30 s of high-speed running resulted in ten times higher increase of GH levels compared to the resting state up to 1 h. Related to body composition, Airin and et al. [20] showed that decreasing the rest interval period to 8/12 s caused more body fat % in addition to improvement of anthropometry index in overweight women. Other studies [21] have reported that longer period of recovery (2 min) compared to shorter ones (30 s) resulted in significant fat reduction after 15 weeks of training, while Lousier et al. demonstrated that aerobic metabolism during the recovery period of intensive exercise plays a significant role in rebuilding creatine phosphate [22]. While these studies are promising, the results seem somewhat contradictory. According to the existing studies on the importance of recovery time in HIIT, the recovery time during high-intensity interval training can lead to a change in the metabolic status and a reduction in body fat. Therefore, we seeked to answer the question of whether HIIT training with different recovery period can change some of the metabolic factors that ultimately lead to weight loss and other obesity-related body composition variables in overweight women?

Materials and methods

Participants

The study was approved by the ethics committee of Sport Sciences Research Institute of Iran. 24 overweight–obese women with the average age of 35.3 ± 3.8 years participated in the study. The height, weight and BMI of the participants were 159 ± 14 cm, 74.6 ± 9.9, and 29.5 ± 3.5 kg, respectively. They were randomly assigned in three groups. The characteristics of the participants are presented in Table 1. For assessing the activity level of the study subject's physical activity questionnaire (RPAQ) Harriss and Atkinson [23] was used. Based on these data, it was determined who had 3–4 days of recreational activities such as gardening, bicycling volleyball, jogging or yoga. No differences between the groups were detected in the physical activity levels at baseline (data not shown).

In addition, health history and nutritional supplements such as the use of creatine, amino acids or vitamins and medical history regarding the medical treatments including surgery, medication or any other information that may interfere with the results of this study within the last 6 months was recorded. They were also asked if they were involved in any other research protocol within the last 2 months [23]. The participants completed the informed consent form before participation in the exercise protocol. In addition, the participants completed a health history questionnaire to reveal whether they had any history of surgery or medical treatment. Finally, after determining inclusion (90% of options) and exclusion criteria (10% of options), any individual who showed two risk factors (Includes in health history questionnaire, nutritional supplement, medical treatments) were excluded from the study. To reduce the influence of previous food consumption on the substrate response during exercise, the subjects were instructed to maintain their normal diet throughout the study.

Body composition and blood pressure

For the purpose of assessing BMI, Asimed analog scale with the discrimination index of 0.1 kg and Asimed height meter with the discrimination index of 1 mm was employed. The waist and hip size was measured in standing position using a flexible tape (cm) to determine the waist–hip ratio (WHR). Body composition was measured by skinfold caliper (Cescorf Brazil) from the right side of the body for seven points [24]. To determine body fat percent, Siri equation was employed [24, 25]. These measures were taken once before

Table 1 general Characteristics of the three groups in two stages of assessment

Variables groups	*N*	Age (year)	Height (cm)	Weight (kg)	BMI (kg/m^2)	Rest heart rate at the start	Rest heart rate at the end
Experimental group 1 (60/60)	8	5.3 ± 33.77	3.60 ± 158.15	12.15 ± 83.54	3.80 ± 32.18	5.54 ± 79.80	3.49 ± 78.62
Experimental group 2 (60/30)	8	5.3 ± 32.94	4.50 ± 159.14	5.16 ± 70.84	3.34 ± 28.11	4.02 ± 77.40	5.12 ± 76.48
Control group	8	5.3 ± 35.11	3.45 ± 160.13	2.61 ± 70.11	1.16 ± 28.15	4.37 ± 75.80	3.45 ± 76.09

BMI Body Mass Index

the start of the protocol and again 1 day after the termination of the exercise program. Blood pressure was measured in sitting position with Mercury manometer 24 h before training and 24 h after 4 weeks training.

Exercise protocol

The exercise protocol was performed under the supervision of 2 researchers and included 12 sessions of HIIT for 4 consecutive weeks (Saturday, Monday, and Wednesday). The exercise protocol included three homogenize groups ($n = 8$). 4 bouts with 1 min HIIT and 1 min of recovery between each bout (group1 = 60/60 activity–rest ratio); and also 4 bouts with 1 min HIIT and 30 s recovery (group 2 = 60/30 activity–rest ratio) between each bout which increased gradually to 10 bouts × 1 min activity and control group (group 3). Overload was increased by two bouts in each week [26].

Exercise intensity based on the 80% average Heart Rate Reserve (HRR) of each person, which was determined using the Karvonen equation [27]. Speed of treadmill based on 80% HRR, heart rate during rest and activity was monitored by Polar heart rate monitors; Polar Electro Inc (T 31 made in Finland).

To control the treadmill to woman perform the active recovery period, another treadmill was adjusted and used according to the individual HRR. Due to the adaptation caused by exercise, intensity was calculated in each session by Karvonen equation for 80% of HRR, in both experimental groups. Prior to the start of the exercise per session, the participant took part in 3 min of warm-up at 6 k/h and after the termination of the exercise protocol, they took part in 2 min of cool down exercise on treadmill.

Biochemical analyses

Following 12 h of fasting, 10 cc of left brachial venous blood was collected in two different stages 24 h before and 24 h after the end of protocol at 8:00 a.m. Then plasma and serum were separated by centrifugation for 10 min at 4000 rpm and was stored at − 20 °C until analyses. By employing a questionnaire, it was ensured that regular menstruation cycle of 28–32 days was present in all the participants and accordingly blood sampling was conducted at a follicle stage in menstruation cycle.

T3, T4 and TSH were measured by ELFA kit (Biomerievx, France) with the same sensitivity (0.05). CV values for thyroid hormones were 4.6, 4.4 and 2.8%, respectively.

In addition, cortisol, growth hormone (GH) and insulin were measured by chemiluminescence kit (Zimmense, Germany, Monobind, USA, and Zimmense, Germany, respectively) and CV for those hormones were 6.1, 0.118 and 3%, respectively. Finally, glucose level was measured with a kit (Pars Azmon Co., Iran) with the least measurable sensitivity of 5 mg/dL and CV of 1.28%. The level of glucose and insulin was calculated by AUC method and trapezoid law.

Insulin sensitivity was calculated using the Insulin Sensitivity Index (ISI), as described by Matsuda and De Fronzo [28]. This calculation uses the fasting plasma glucose in mg dL (− 1) and plasma insulin in mU L (− 1) and the average plasma glucose and insulin values over the 30, 60, 90 and 120 min from an OGTT, i.e., $10{,}000/\sqrt{[(\text{fasting glucose} \times \text{fasting insulin}) \times (\text{mean glucose during OGTT} \times \text{mean insulin during OGTT})]}$. In addition, Homeostasis Model Assessment estimated insulin resistance (HOMA-IR) was calculated using fasting insulin mU L (− 1) × fasting glucose mmol L (− 1)/22.5 [29]. CV for cholesterol, triglycerides, HDL-C, LDL-C were 0.62, 2.8 and 0.73%, respectively (by kit Pars Azmon Co kit., Iran).

Statistical analysis

All statistical analyses were carried out using SPSS version 16.0. The normality of data was confirmed by Shapiro–Wilk test. The difference between the pretest–posttest of all the measured variables was used to test the hypothesis. One-way analysis of variance (ANOVA) was employed to compare the results and Tukey post hoc test was used to compare the means if a significant difference was found. Paired and independent *t* tests were used to investigate the changes in the variables as a result of the effect of recovery time separately in each group and to compare two types of recovery in the training groups. The correlation of cortisol hormone with thyroid hormones was tested by Pearson correlation coefficient. All the hypotheses were examined at the alpha level set to 0.05.

Results

Physical characteristics

The results indicated that none of the exercise modes caused significant changes in weight [$F^{(2,18)} = 0.238$; $P = 1.55$], BMI [$F^{(2,18)} = 0.564$; $P = 0.590$], WHR [$F^{(2,18)} = 0.143$; $P = 2.25$], fat % [$F^{(2,18)} = 0.019$; $P = 1.98$], or systolic [$F^{(2,18)} = 0.517$; $P = 0.657$] and diastolic blood pressure [$F^{(2,18)} = 0.502$; $P = 0.716$] in the experimental groups. However, compared to control group HIIT 60/30 resulted in a significant decrease in fat % ($P < 0.05$; Table 1).

Blood lipids

There were no significant differences in the serum level of cholesterol [$F^{(2,18)} = 0.52$; $P = 0.599$], triglycerides [$F^{(2,18)} = 0.63$; $P = 0.541$], HDL/LDL [$F^{(2,18)} = 0.450$;

$P=0.644$] or HDL-C [$F^{(2,18)}=0.820$; $P=0.201$] between the groups (Table 2).

Thyroid, cortisol and growth hormones

No significant differences were found in the levels of thyroid hormones T3 [$F^{(2,18)}=0.81$; $P=0.46$], T4 [$F^{(2,18)}=0.69$; $P=0.51$], TSH [$F^{(2,18)}=0.14$; $P=0.866$] between the two types of training or compared to the control group. In addition, none of the exercise modes caused significant changes in the level of blood cortisol [$F^{(2,18)}=1.12$; $P=0.345$; Fig. 1] or growth hormone [$F^{(2,18)}=10.13$; $P=0.344$; Fig. 2].

Correlation between cortisol and thyroid hormones

The result of Pearson correlation coefficient in posttest state showed that there was no significant correlation between the T3 and T4 ($r=0.592$, $P=0.161$), T3 and TSH ($r=-0.752$, $P=0.062$), T3 and cortisol ($r=-0.743$, $P=0.056$) in the 60/60 activity–rest ratio group. In addition, there was no significant correlation between the T4 and TSH ($r=-0.370$, $P=0.414$), between T4 and Cortisol ($r=-0.014$, $P=0.977$), cortisol and TSH ($r=-0.590$, $P=0.163$). Moreover, there was no significant correlation between T3 and T4 ($r=0.272$, $P=0.555$), T3 and TSH ($r=0.233$, $P=0.616$), T3, cortisol ($r=-0.706$, $P=0.076$) in the 60/30 activity–rest ratio group.

In addition, no significant correlation was found between T4 and TSH ($r=1$–0.184, $P=0.693$), T4 and cortisol ($r=-0.073$, $P=0.877$), cortisol and TSH ($r=-0.047$, $P=0.920$) in the control group. Moreover, in the control group, there was no significant correlation between T3 and T4 ($r=-0.551$, $P=0.20$), T3 and TSH ($r=0.203$, $P=0.662$), T3 and cortisol ($r=-0.048$, $P=0.919$), T4 and TSH ($r=-0.0407$, $P=0.364$), T4 and cortisol ($F=-0.055$, $P=0.906$), cortisol and TSH ($r=-0.340$, $P=0.455$).

Fasting blood glucose and blood insulin

None of the exercise modes caused significant changes in fasting blood sugar [$F^{(2,18)}=0.371$; $P=0.695$], insulin [$F^{(2,18)}=0.332$; $P=0.772$], insulin resistance (HOMA-IR) [$F^{(2,18)}=0.104$; $P=0.902$] or insulin sensitivity index (ISI) [$F^{(2,18)}=0.223$; $P=0.802$; Table 3].

Table 2 Anthropometric and lipid profiles of the three groups in two stages of assessment

variables	Group N=8	Pretest	Post test	P value of three groups	P value for each group	Mean Difference of between two types of training	Significant difference Between two types of training
HDL-C (µg/dL)	60.60	46.28±11.04	46.28±9.7	0.820	0.212		0.627
	60.30	46.00±11.04	47.57±10.8		0.539	-0.071	
	control	44.28±8.3	44.00±5.8				
HDL/LDL	60.60	0.424±0.11	0.450±0.11	0.644	0.132		
	60.30	0.483±0.15	0.525±0.16		0.188	-0.016	0.626
	control	0.495±0.19	0.506±0.17				
Cholesterol (µg/dL)	60.60	177.7±18.2	173.0±14.8	0.599	0.304		
	60.30	167.2±33.5	169.7±36.2		0.727	-7.285	0.391
	control	162.2±22.6	167.5±43.2				
Triglycerides (µg/dL)	60.60	103.4±50.5	113.2±47.3	0.541	0.190		
	60.30	106±46.6	137.1±92.08		0.197	-21.285	0.375
	control	109±47.7	146.7±90.03				
Fat %	60.60	40.90±0.81	40.44±0.91	4.98	0.031		
	60.30	41.82±0.73	41.17±0.71		0.002	0.199	0.347
	control	41.17±1.14	41.06±1.10				
Waist/hip ratio	60.60	0.872±0.10	0.877±1.10	2.25	0.289		
	60.30	0.897±0.05	0.811±0.06		0.188	-0.008	0.380
	control	0.818±0.06	0.814±0.07				
Weight (kg)	60.60	83.54±12.15	82.38±12.13	1.55	0.013		
	60.30	70.84±5.16	70.21±5.08		0.007	-0.528	0.185
	control	70.11±2.61	69.65±2.07				
Systolic blood pressure(mmHg)	60.60	120.11±0.65	120.14±0.37	0.658	0.890		
	60.30	110.57±0.60	110.57±0.78		1.00	-2.46	0.432
	control	120.07±0.18	110.71±0.48				
Diastolic blood pressure(mmHg)	60.60	80.35±0.62	80.28±0.75	0.716	0.689		
	60.30	80.14±0.24	80.00±0.57		0.631	0.071	0.832
	control	80.57±0.78	80.14±0.37				
BMI (body mass index, weight/height^2	60.60	32.18±3.80	31.74±3.44	0.590	0.171		
	60.30	28.11±3.34	27.70±3.57		0.075	-0.034	0.921
	control	28.15±1.16	28.02±1.05				

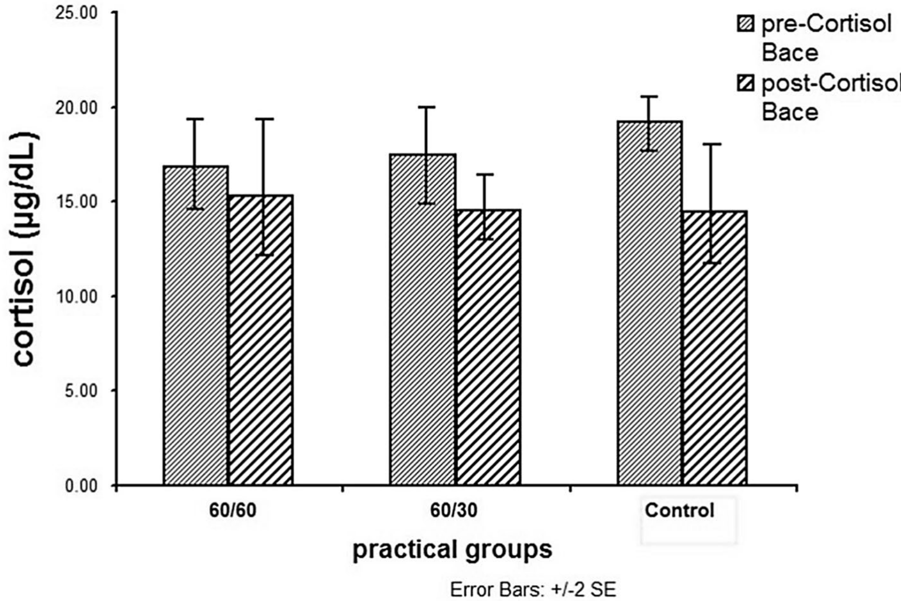

Fig. 1 Pre and posttest means of cortisol in the three groups of activity–rest intervals of 60/60, 30/60 and control with no significant difference ($P = 0.345$). P value for 60/60 group: 0.317, P value for 60/30 group: 0.106, Mean Difference of between two types of training: − 1.425, significant difference Between the training groups: 0.501

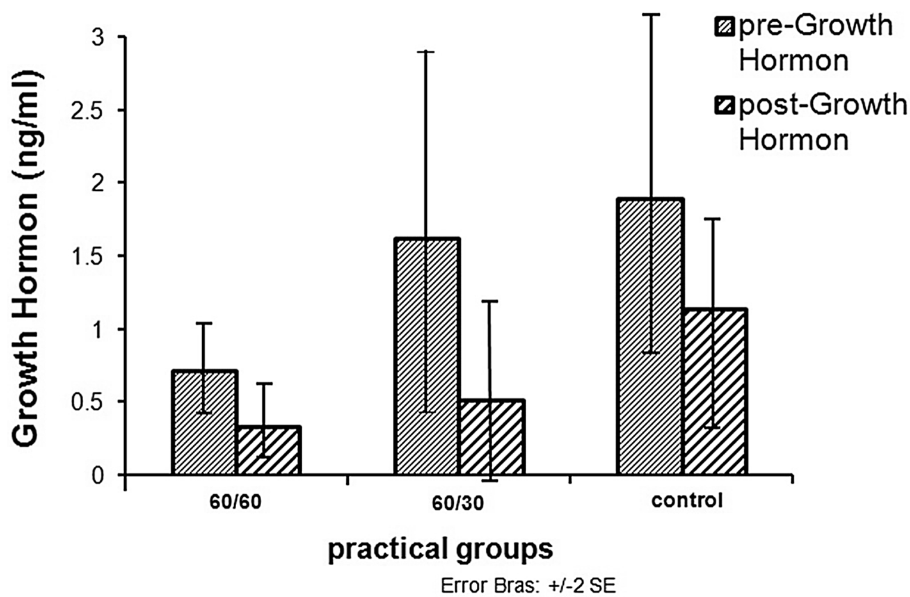

Fig. 2 pre and posttest means of growth hormone in the three groups of activity–rest intervals of 60/60, 30/60 and control with no significant difference ($P = 0.344$). P value for 60/60 group: 0.091, P value for 60/30 group: 0.106, mean difference of between two types of training: 0.714, between the training groups: 0.087

Discussion

The purpose of this research was to examine the effect of 4 weeks of high-intensity interval training program on anthropometric indices that are employed in assessing fatness in overweight women. According to the heart disability of obese people and exercise intolerance related to the age range [30], the probability of skeletal joints injuries due to overweight in women, especially in HIT, increases. Therefore, this study was done in 4 weeks. The results of the present research indicated that besides a decrease in fat % in the 60/30 group, HIIT program had no significant effects on the other body composition variables in overweight women. In agreement with our findings, Keating et al. [31] have also shown that HIIT program in comparison with the long-term endurance program had no significant effects on anthropometrics used to assess fatness in adult men and women. However, conversely, Heydari et al. [32] demonstrated that HIIT programs with shorter activity to rest interval of 8–12 s in young men led to significant changes in body composition. Thus, shorter period of training could contribute to the different findings. The results of the present study showed a significant change in the fat % of the three groups that may be attributed to the 60/30 activity–rest interval compared to

Table 3 hormone and glycemic indices of the three groups in two stages of assessment

variables	Group N=8	pretest	Post test	P value of three groups	P value for each group	Mean Difference of between two types of training	Significant difference Between two types of training
T3 (μg/dL)	60.60	1.56±0.23	1.27±0.23	0.460	0.088		
	60.30	1.40±0.09	1.32±0.32		0.566	-0.218	0.265
	control	1.44±0.21	1.18±0.15				
T4 (ng/dl)	60.60	6.76±0.64	6.36±0.42	0.513	0.285		
	60.30	6.52±0.22	6.42±0.71		0.662	-0.300	0.467
	control	6.68±0.35	-6.69±0.27				
TSH (μIU/ml)	60.60	2.46±1.6	1.91±0.83	0.866	0.473		
	60.30	1.97±1.1	1.75±0.65		0.539	-0.321	0.691
	control	2.81±1.1	2.27±0.81				
Insulin (μU/ml)	60.60	11.21±5.7	10.59±4.8	0.722	0.473		
	60.30	11.59±5.1	10.18±6.4		0.645	0.798	0.79
	control	15.32±4.7	12.14±3.7				
HOMAIR	60.60	53.78±42.7	45.27±26.5	0.902	0.287		
	60.30	56.03±24.4	45.06±23.1		0.457	2.464	0.877
	control	66.05±25.9	50.17±15.4				
Fasting glucose (mg/dL)	60.60	99.57±22.01	93.28±14.6	0.695	0.150		
	60.30	113.57±44.1	110/14±36/7		0.431	-2.857	0.617
	control	95.1±7.94	93.85±9.26				
ISI	60.60	0.337±0.03	0.341±0.02	0.802	0.359		
	60.30	0.326±0.01	0.337±0.01		0.332	-0.006	0.567
	control	0.318±0.01	0.330±0.01				

Data are presented as mean ± SD. $N = 8$. HOMAIR: homeostasis model estimated insulin resistance

ISI Insulin Sensitivity Index

the control group. In agreement, Tjonna et al. [33] found that 12 weeks of aerobic interval training (with rest interval shorter than the training time of 3–4 min decreased significantly subcutaneous fat mass), visceral fat mass and WHR of obese participants. However, in a contrary, Gillen et al. [34] demonstrated that 6 weeks of HIIT program with 60/60 rest–activity interval caused improvement in body composition variables and muscle oxidative capacity of obese women. The importance of the recovery times between HIIIT has been pointed out before [35]. For instance, it has been shown that if the recovery period in high-intensity exercises is decreased, the proportion of glycolysis for providing energy is reduced and as a result the aerobic metabolism increases [22]. This may be one of the contributing mechanisms in HIIT programs that use rest intervals shorter than the activity periods. It is likely that the shorter recovery period compared with the 60/60 s rest–activity group is one of the causes of changes that have not been indicated in previous studies. Trapp et al. [36] reported that participation in 15 weeks of HIIT program with activity–rest period of 8–12 s compared to the steady-state exercise programs led to significant decrease in plasma leptin level and measures of fatness in young women. In addition, Moher et al. [21] also compared prolonged exercise program with 15 weeks of HIIT program of 30 s activity with 2-min rest intervals and concluded that fat mass in sedentary women decreased significantly in longer training time. Despite these findings, there are other results that show greater fat mass reduction in prolonged exercise Romain et al. [37] and most of the indices of fatness versus the HIIT program [38]. These results in regard to different activity–rest intervals suggest that the recovery period is an important variable affecting the changes in physiological indices in an exercise program. It should be noted that many variables including the type of exercise, intensity, duration, body condition during the blood collection, lab methods [39], individual difference [6] and gender [40] may be confounding factors that could lead to the contradictory findings presented in the current literature.

Our further analyses indicated that no significant changes in blood lipid levels occurred in the three groups. Such finding is in agreement with what was reported by Mohr et al. [21] who examined the effects of 15 weeks of HIIT swimming program with activity–rest intervals ratio of 30 s/2 min on lipid profiles and found no effects. In addition, Hydari et al. [32] reported that 12 weeks of HIIT program with activity–rest intervals ratio of 8/12 s did not affect blood lipids. Mannaing et al. [41] claimed that the initial level of blood lipids, being non-obese or the level of fat storage. Blaize et al. [42] may lead to the effectiveness of an exercise program that results in significant changes in blood lipid profiles. Therefore, in our study, the activity level of the participants in 3–4 sessions of leisure activity per month, low sample size and duration of exercise program are limitations of the present study, and may be the reason that the measures of fatness other than fat % did not change significantly.

The results of the present study did not show any significant effect for HIIT program on insulin sensitivity and insulin resistance, of course, decreasing trend has observed in insulin resistance in the experimental group 60/30 activity–rest interval group, which however, was not statistically significant. Boutcher [43] in a review research concluded that HIIT programs considerably decrease insulin resistance and glucose tolerance. On the other hand, Baldwin and Haddad [44] stated that exercise program may decrease the levels of thyroid hormones as a result of a decrease in blood glucose and fatty acid increase, but after the termination of the exercise activity and during the recovery period, the secretion is resumed. Similarly, the level of the hormones changed in our study, but the change was not statistically significant.

Krotkiewski et al. [45] showed that the level of T4 in obese women increased considerably in response to 3 months bicycle ergometer training, but no significant change was seen in T3 and TSH. Huanga et al. [46] did not report any significant change in TSH level as a result of participation in maximum intensity exercise on treadmill but showed a considerable increase in T3 level, Ciloglu et al. [47] showed a significant effect of participation in 3 min of 90% of maximum heart rate on the level of T4 and TSH but reported a decrease in the level of T3. Researchers believe that the hormonal changes may depend on many interfering factors including insufficient sleep [48], emotional pressure and caloric demands [49, 50], and therefore these factors all may disrupt the correct thyroid function [51]. As a result, controlling these nuisance factors increases the validity of findings. In addition, it seems that previous experience of the participants in the physical activity, training variable and resting time for recovery all may have effect on thyroid secretion and cause conflicting findings in different research protocols [51].

Other researchers including Anthony et al. [52] showed a negative correlation between the decrease in free T3 and increase of reverse T3 12 h of following the last training program, whereas the negative correlation between the increase in cortisol and decrease of free T3 in 12 h after the last session of training was present. In this regard, the authors examined the effects of psychological pressure on the subjects and showed that despite the absence of significant change in cortisol levels, negative correlation between cortisol and T3 was found after the training program. Therefore, it is likely that the change in thyroid hormone levels as a result of stress hormones such as cortisol occurs.

On the other hand, acute exercise activity results in an increase of plasma GH probably due to the increase in parasympathetic nervous system [53]. However, the results of our study did not show any significant changes. Pritzlaff et al. [54] claimed that during the recovery period there is a direct association between the GH level and the level of fat oxidation. That is, fat metabolism is increasingly associated with GH release during the recovery [53]. In addition, the level of physical fitness, age and sex are also factors that influence this hormone's release [55]. Increasing lactate is an effective stimulus for GH secretion [56]. In this regard, Gosselin et al. [56] revealed that HIIT with activity–rest periods of 90/30 and 60/90 led to higher lactate levels in comparison with activity–rest periods of 60/60 and 30/30. It could be concluded that increasing lactate levels due to the reduction of recovery time during HIIT increase fat burning-induced GH secretion. It needs to be pointed out that examining the effect of exercise to the recovery time ratio on GH is also important. The effects of recovery time between the exercise bouts on the obesity indices have not been examined and thus it remains unclear which recovery to exercise time ratio in high-intensity workout results in increase of metabolism that triggers fat mobilization.

In conclusion, compared to the control group, the 60/30 s HIIT resulted in a significant decrease in body fat % while no other significant differences in the body composition were found. However, according to the present study and the results of studies mentioned above, 30 s recovery period in HIIT may reduce fat % more efficiently than 60 s.

Acknowledgements The researchers wish to express his appreciation to the women coach, Miss. Nosratabadi, Shahid Mirghani Research Institute and Dr. Hossein Nasehi, the director of Sepid Medical Lab in the city of Aliabad-e-Katoul, in Golestan province, for their sincere contribution to the conduct of this study.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest concerning this article.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study was approved by the ethics committee of Sport Sciences Research Institute of Iran.

Informed consent Informed consent was obtained from all individual participants included in the study.

References

1. Zou C, Shao J (2008) Role of adipocytokines in obesity-associated insulin resistance. J Nutr Biochem 19(5):277–286
2. Coyle EF (2000) Physical activity as a metabolic stressor. Am J Clin Nutr 72(2):512s–520s
3. Chuensiri N, Suksom D, Tanaka H (2018) Effects of high-intensity intermittent training on vascular function in obese preadolescent boys. Child Obes 14(1):41–49
4. Leckey JJ et al (2018) High dietary fat intake increases fat oxidation and reduces skeletal muscle mitochondrial respiration in trained humans. FASEB J 17:fj201700993R

5. Amigo TRR et al (2018) Effectiveness of High-Intensity Interval Training on cardiorespiratory fitness and body composition in preadolescents: a systematic review. Eur J Hum Mov 39:32–47
6. Gibala MJ et al (2012) Physiological adaptations to low-volume, high-intensity interval training in health and disease. J Physiol 590(5):1077–1084
7. Schmitt J et al (2016) A 3-week multimodal intervention involving high-intensity interval training in female cancer survivors: a randomized controlled trial. Physiol Rep 4(3):e12693
8. Hayase H et al (2002) Relation between fat distributions and several plasma adipocytokines after exercise training in premenopausal and postmenopausal women. J Physiol Anthropol Appl Hum Sci 21(2):105–113
9. Wahl P et al (2010) Effect of high-and low-intensity exercise and metabolic acidosis on levels of GH, IGF-I, IGFBP-3 and cortisol. Growth Horm IGF Res 20(5):380–385
10. Metcalfe R, Fawkner S, Vollaard N (2016) No acute effect of reduced-exertion High-Intensity Interval Training (REHIT) on insulin sensitivity. Int J Sports Med 37(05):354–358
11. Adams OP (2013) The impact of brief high-intensity exercise on blood glucose levels. Diabetes Metab Syndr Obes Targets Ther 6:113
12. Jelleyman C et al (2015) The effects of high-intensity interval training on glucose regulation and insulin resistance: a meta-analysis. Obes Rev 16(11):942–961
13. Harmer AR et al (2000) Skeletal muscle metabolic and ionic adaptations during intense exercise following sprint training in humans. J Appl Physiol 89(5):1793–1803
14. MacDougall JD et al (1998) Muscle performance and enzymatic adaptations to sprint interval training. J Appl Physiol 84(6):2138–2142
15. McArdle WD, Katch F, Katch V (1996) Energy expenditure in household, occupational, recreational, and sports activities. Exercise physiology: energy, nutrition and human performance. Williams and Wilkins, Philadelphia, pp 769–781
16. Sone M, Osamura RY (2001) Leptin and the pituitary. Pituitary 4(1–2):15–23
17. Hackney AC, Dobridge JD (2009) Thyroid hormones and the interrelationship of cortisol and prolactin: influence of prolonged, exhaustive exercise. Endokrynologia Polska 60(4):252–257
18. Bracken RM, Linnane DM, Brooks S (2009) Plasma catecholamine and nephrine responses to brief intermittent maximal intensity exercise. Amino Acids 36(2):209–217
19. Nevill M et al (1996) Growth hormone responses to treadmill sprinting in sprint-and endurance-trained athletes. Eur J Appl Physiol Occup Physiol 72(5):460–467
20. Airin S et al (2014) The effects of high-intensity interval training and continuous training on weight loss and body composition in overweight females. In: Proceedings of the international colloquium on sports science, exercise, engineering and technology (ICoSSEET 2014). Springer, Singapore, pp 401–409
21. Mohr M et al (2014) High-intensity intermittent swimming improves cardiovascular health status for women with mild hypertension. BioMed Res Int 2014:728289
22. Linossier M-T et al (1993) Ergometric and metabolic adaptation to a 5-s sprint training programme. Eur J Appl Physiol Occup Physiol 67(5):408–414
23. Harriss D, Atkinson G (2011) Update–ethical standards in sport and exercise science research. Int J Sports Med 32(11):819–821
24. Hayward V, Stolarczyk L (1996) Applied body composition. Human Kinetics, Champaign
25. Jackson AS, Pollock ML, Ward A (1980) Generalized equations for predicting body density of women. Med Sci Sports Exerc 12(3):175–181
26. Rozenek R et al (2016) Acute cardiopulmonary and metabolic responses to high-intensity interval training protocols using 60 s of work and 60 s recovery. J Strength Cond Res 30(11):3014–3023
27. Karvonen MJ (1957) The effects of training on heart rate; a longitudinal study. Ann Med Exp Biol Fenn 35:307–315
28. Matsuda M, DeFronzo RA (1999) Insulin sensitivity indices obtained from oral glucose tolerance testing: comparison with the euglycemic insulin clamp. Diabetes Care 22(9):1462–1470
29. Matthews D et al (1985) Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 28(7):412–419
30. Packer M (2018) Do most obese people with exercise intolerance and a normal ejection fraction have treatable heart failure? Am J Med 131(8):863–864
31. Keating SE et al (2014) Continuous exercise but not high intensity interval training improves fat distribution in overweight adults. J Obes 2014:834865
32. Heydari M, Freund J, Boutcher SH (2012) The effect of high-intensity intermittent exercise on body composition of overweight young males. J Obes 2012:480467
33. Tjønna AE et al (2009) Aerobic interval training reduces cardiovascular risk factors more than a multitreatment approach in overweight adolescents. Clin Sci 116(4):317–326
34. Gillen JB et al (2013) Interval training in the fed or fasted state improves body composition and muscle oxidative capacity in overweight women. Obesity 21(11):2249–2255
35. Mirghani S, Yousefi M (2015) The effect of interval recovery periods during HIIT on liver enzymes and lipid profile in overweight women. Sci Sports 30(3):147–154
36. Trapp EG et al (2008) The effects of high-intensity intermittent exercise training on fat loss and fasting insulin levels of young women. Int J Obes 32(4):684–691
37. Romain A et al (2012) Physical activity targeted at maximal lipid oxidation: a meta-analysis. J nutr metab 2012:285395
38. Nybo L et al (2010) High-intensity training versus traditional exercise interventions for promoting health. Med Sci Sports Exerc 42(10):1951–1958
39. Olson TP et al (2007) Changes in inflammatory biomarkers following one-year of moderate resistance training in overweight women. Int J Obes 31(6):996–1003
40. Smith J et al (2004) Effects of prolonged strenuous exercise (marathon running) on biochemical and haematological markers used in the investigation of patients in the emergency department. Br J Sports Med 38(3):292–294
41. Manning JM et al (1991) Effects of a resistive training program on lipoprotein–lipid levels in obese women. Med Sci Sports Exerc 23(11):1222–1226
42. Blaize AN et al (2014) Body fat has no effect on the maximal fat oxidation rate in young, normal, and overweight women. J Strength Cond Res 28(8):2121–2126
43. Boutcher SH (2010) High-intensity intermittent exercise and fat loss. J Obes 2011:868305
44. Baldwin KM, Haddad F (2001) Invited review: effects of different activity and inactivity paradigms on myosin heavy chain gene expression in striated muscle. J Appl Physiol 90(1):345–357
45. Krotkiewski M et al (1984) The effect of acute and chronic exercise on thyroid hormones in obesity. J Intern Med 216(3):269–275
46. Huang W-S et al (2004) Effect of treadmill exercise on circulating thyroid hormone measurements. Med Princ Pract 13(1):15–19
47. Ciloglu F et al (2005) Exercise intensity and its effects on thyroid hormones. Neuroendocrinol Lett 26(6):830–834
48. Kuetting DL et al (2018) Effects of a 24-hr-shift-related short-term sleep deprivation on cardiac function: a cardiac magnetic resonance-based study. J Sleep Res. https://doi.org/10.1111/jsr.12665
49. Maheu FS et al (2004) Differential effects of adrenergic and corticosteroid hormonal systems on human short-and long-term

declarative memory for emotionally arousing material. Behav Neurosci 118(2):420
50. Barrows K, Snook J (1987) Effect of a high-protein, very-low-calorie diet on resting metabolism, thyroid hormones, and energy expenditure of obese middle-aged women. Am J Clin Nutr 45(2):391–398
51. Viru AA, Viru M (2001) Biochemical monitoring of sport training. Human Kinetics Publishers, Inc., USA
52. Hackney AC et al (2012) Thyroid hormonal responses to intensive interval versus steady-state endurance exercise sessions. Hormones 11(1):54–60
53. Kjaer M et al (1987) Role of motor center activity for hormonal changes and substrate mobilization in humans. Am J Physiol Regul Integr Comp Physiol 253(5):R687–R695
54. Pritzlaff CJ et al (2000) Catecholamine release, growth hormone secretion, and energy expenditure during exercise vs. recovery in men. J Appl Physiol 89(3):937–946
55. Veldhuis JD et al (1995) Differential impact of age, sex steroid hormones, and obesity on basal versus pulsatile growth hormone secretion in men as assessed in an ultrasensitive chemiluminescence assay. J Clin Endocrinol Metab 80(11):3209–3222
56. Gosselin LE et al (2012) Metabolic response of different high-intensity aerobic interval exercise protocols. J Strength Cond Res 26(10):2866–2871

Affiliations

Seyed Javad Mirghani[1] · Mehdi Seydyousefi[2] · Satu Pekkala[3] · Shohreh Sharifian[4] · Gheysar Beyshami[5]

Mehdi Seydyousefi
mehdi.saidyousefi@yahoo.com

Satu Pekkala
satu.p.pekkala@jyu.fi

Shohreh Sharifian
sh_sharifian53@yahoo.com

Gheysar Beyshami
gheysarbeyshami@yahoo.com

[1] Department of Exercise Physiology, Faculty of Physical Education and Sports Science, Islamic Azad University, Central Tehran Branch, Tehran, Iran

[2] Department of Exercise Physiology, Islamic Azad University, Bojnourd Branch, Bojnourd, Iran

[3] Faculty of Sport and Health Sciences, University of Jyvaskyla, PO Box 35 (L226), 40014 Jyvaskyla, Finland

[4] Department of Exercise Physiology, Islamic Azad University, Rasht Branch, Rasht, Iran

[5] Department of Exercise Physiology, Islamic Azad University, East Tehran Branch, Tehran, Iran