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ORIGINAL ARTICLE

Exercise training-induced visceral fat loss in obese women: The role of training intensity and modality

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Visceral fat loss in response to four-cycle ergometer training regimens with explicit differences in exercise intensity and modality was compared. Fifty-nine obese young women (body fat percentage ≥ 30%) were randomized to a 12-week intervention consisting of either all-out sprint interval training ($SIT_{all\text{-}out}$, n = 11); supramaximal SIT (SIT_{120}, 120% $\dot{V}O_{2peak}$, n = 12); high-intensity interval training ($HIIT_{90}$, 90% $\dot{V}O_{2peak}$, n = 12), moderate-intensity continuous training (MICT, 60% $\dot{V}O_{2peak}$, n = 11), or no training (CON, n = 13). The total work done per training session in SIT_{120}, $HIIT_{90}$, and MICT was confined to 200 kJ, while it was deliberately lower in $SIT_{all\text{-}out}$. The abdominal visceral fat area (AVFA) was measured through computed tomography scans. The whole-body and regional fat mass were assessed through dual-energy X-ray absorptiometry. Pre-, post-, and 3-hour post-exercise serum growth hormone (GH), and epinephrine (EPI) were measured during selected training sessions. Following the intervention, similar reductions in whole-body and regional fat mass were found in all intervention groups, while the reductions in AVFA resulting from $SIT_{all\text{-}out}$, SIT_{120}, and $HIIT_{90}$ (>15 cm^2) were greater in comparison with MICT (<3.5 cm^2, $P < .05$). The AVFA reductions among the SITs and HIIT groups were similar, and it was concomitant with the similar exercise-induced releases of serum GH and EPI. CON variables were unchanged. These findings suggest that visceral fat loss induced by interval training at or above 90% $\dot{V}O_{2peak}$ appeared unresponsive to the change in training intensity. Nonetheless, $SIT_{all\text{-}out}$ is still the most time-efficient strategy among the four exercise-training regimes for controlling visceral obesity.

KEYWORDS
abdominal fat, continuous training, interval training, lipolytic hormones, obesity

1 | INTRODUCTION

It is recognized that visceral adiposity carries greater risk of developing obesity-related complications, including type 2 diabetes and cardiovascular disease, than peripheral obesity,[1] and has been shown as a strong predictor of mortality in obese women.[2] The elimination of excessive visceral fat has been associated with attenuation of metabolic syndrome including reductions in insulin resistance markers and fasting plasma glucose, and that were more noticeable compared with those resulting from the reduction in subcutaneous fat.[3,4]

Increasing evidence suggests that lifestyle modifications with exercise intervention are a predominant non-pharmacological strategy for attenuating excessive visceral fat deposition and related complications in people with obesity.[5,6] This is mainly because the extent of lipolysis in visceral adipose tissues in response to the increased metabolic demand is greater in comparison with that of subcutaneous fat,[7] partly attributable to the higher activity of lipolytic hormones and lower activity of anti-lipolytic counterparts in the visceral adipocytes.[8] Based on the assumption that lipolytic hormones and their activities increase with exercise intensity,[9,10] the regional differences in the abdominal fat metabolism have led to the postulation that an intervention composed of high-intensity exercise would facilitate the reduction of visceral fat.[11] In recent studies, the reduction in visceral fat in obese young women following 12-week high-intensity interval training (HIIT, repeated 4-minute cycling at 90% $\dot{V}O_{2peak}$) was comparable to that resulting from work-equivalent prolonged moderate-intensity continuous training (MICT, cycling at 60% $\dot{V}O_{2peak}$), yet the HIIT training time was apparently less than that of the MICT.[12] Such time-efficiency advantage in reducing visceral fat with HIIT was further optimized when the exercise was performed at the intensity of all-out supramaximal level—sprint interval training (SIT), concomitant with a lessened training volume.[13]

Exercise intensity appears to be of the essence in reducing visceral fat with HIIT. However, the direct relationship between the increase in exercise intensity and the acute increase in the release of lipolytic hormones of catecholamines and growth hormone shown in submaximal exercise[9,14] has not been reported in obese individuals during supramaximal exercise. Moreover, the positive relationship between the lipolytic actions of catecholamines and exercise intensity that was observed in moderate exercise has been reported to be vague in high-intensity exercise.[15] Furthermore, the translation of the high rate of lipolysis into augmented fat oxidation is discordant during high-intensity exercise.[16] It is not known if the advocate of "the higher, the better" in manipulating the exercise intensity of interval training regimen is favorable to visceral fat reduction. In this context, the purpose of this study was to examine the specific adaptations to the mechanical work-equivalent cycle ergometer training regimens with explicit differences in exercise intensity (ranging from moderate to all-out) and modality (continuous *vs* interval) for visceral fat loss. It was hypothesized that the magnitude of the reductions in visceral fat and in other body composition parameters would be various among the different training regimens, with the time efficiency of the training-induced visceral fat loss depends upon the training intensity.

2 | METHODS

2.1 | Participants

An a priori, two-tailed power calculation at an alpha of .05 and a power of 80%, carried out based on the previous findings of the adaptations in fat mass to 12-week HIIT (effect size of 0.45) in obese young females,[12] suggested that a minimum of total 50 participants, 10 for each group, were required in this study. Given the dropout rate of ~20% that appeared in previous HIIT studies,[12,13] the sample size was inflated to 15 participants per group.[17] Seventy-five eligible female students were recruited from a university for this study, with the following inclusion criteria: (a) age 18-23 years; (b) body fat percentage ≥ 30% (measured by DEXA); (c) constant body weight (±2 kg) in the past 3 months; (d) no regular physical activity except attending physical education class twice per week; and (e) no history of metabolic, hormonal, orthopedic, or cardiovascular diseases, and no current use of prescribed medication including oral contraception. Following an explanation of the purpose and constraints of the study, the participants provided written informed consent. All participants underwent initial assessment and randomization. The Ethical Committee of Hebei Normal University for the Use of Human and Animal Subjects in Research provided ethical approval of the study (no. 2019SC21). The experiments in the present study were performed in accordance with the Helsinki Declaration. During the intervention, 16 participants did not complete the program for reasons unrelated to the study (Figure 1). Table 1 is the physical characteristics of the participants.

2.2 | Study design

Figure 2 is the overview of the study design. In detail, all participants were invited to record their daily food intake and physical activities for monitoring the habitual energy intake and expenditures, respectively, starting from the 3 weeks prior to the intervention till the end of it. Within 1 week prior to the intervention, the whole-body and regional fat mass, abdominal visceral and subcutaneous fat areas, blood metabolic variables, and $\dot{V}O_{2peak}$ were measured. Participants with matching body fat percentages were randomly assigned to either all-out SIT ($SIT_{all\text{-}out}$), supramaximal SIT (SIT_{120}), submaximal HIIT ($HIIT_{90}$), MICT, or no training (CON) on a cycle ergometer for 44 sessions in 12 weeks. The work rate prescribed in $SIT_{all\text{-}out}$, SIT_{120}, $HIIT_{90}$, and MICT was corresponding to the exercise intensity of all-out effort, 120%, 90%, and 60% pre-intervention $\dot{V}O_{2peak}$, respectively. The total work done per training session in SIT_{120}, $HIIT_{90}$, and MICT was confined to 200 kJ, while it was designated to be lower in $SIT_{all\text{-}out}$, intent upon demonstrating the time efficiency of the regimen in fat control. By the

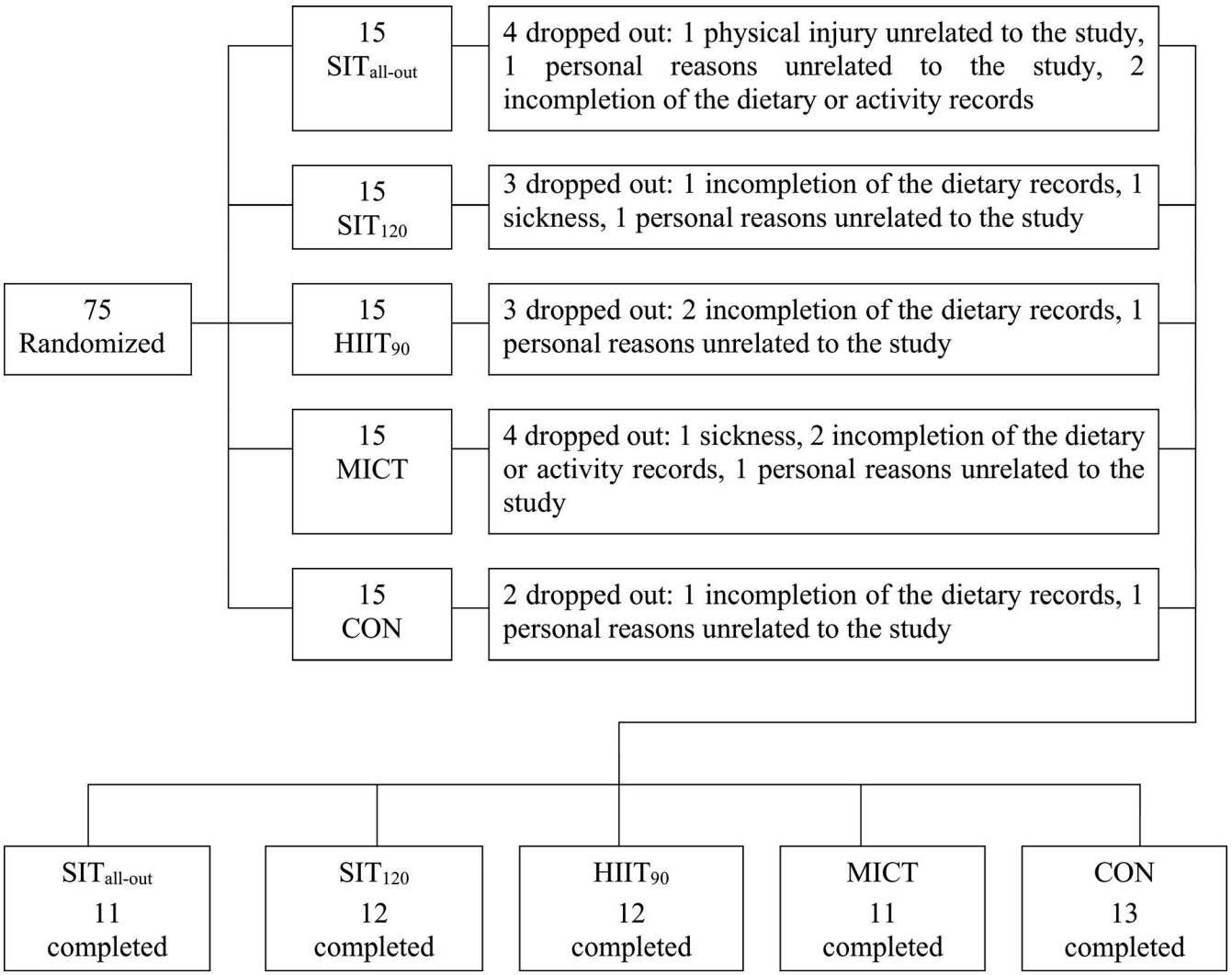

FIGURE 1 Distribution of study participants. CON, control group; $HIIT_{90}$, high-intensity interval training group (90% $\dot{V}O_{2peak}$); MICT, moderate-intensity continuous training group; SIT_{120}, supramaximal sprint interval training group (120% $\dot{V}O_{2peak}$); $SIT_{all\text{-}out}$, all-out sprint interval training group

end of the 4th and 8th weeks, the $\dot{V}O_{2peak}$ test was repeated for necessary adjustment of the work rate corresponding to the preset training intensity in subsequent weeks. In the 1st, 20th, and 44th training sessions, selected serum lipolytic hormones, blood lactate, exercise heart rate (HR), and total energy expenditure (TEE) derived from the exercise $\dot{V}O_2$ were measured and compared across the 4 intervention groups. At most 3 days subsequent to the intervention, pre-intervention tests were repeated. The alterations in the fat and metabolic variables following interventions were subsequently compared among the intervention and control groups.

2.3 | Estimations of daily energy intake and expenditure for physical activities

The daily energy intake of participants was estimated based on 24-hour dietary recall. A questionnaire designed according to the guidelines of the Sports Nutrition Centre of the National Research Institute of Sports Medicine (NRISM) in China for caloric intake estimation was used to capture the foods and beverages consumed in the past day from midnight to midnight. Participants provided the dietary information, including type of food, portion size, and preparation method under the guidance of a dietician. The corresponding energy intake was analyzed using the NRISM dietary and nutritional analysis system (version 3.1). Dietary advice was provided whenever violation of maintenance of daily caloric intake was detected.

The daily physical activities, apart from those sedentary activities and the cycle ergometer training, were assessed based on 24-hour activity recall. The physical activities performed in the past day from midnight to midnight were recorded by using a structured, self-reported instrument that provided details about the type and intensity of physical activities, such as brisk walking and leisure

TABLE 1 Physical characteristics of the participants and the changes in their $\dot{V}O_{2peak}$ during the 12-wk intervention

	$SIT_{all-out}$ (n = 11)	SIT_{120} (n = 12)	$HIIT_{90}$ (n = 12)	MICT (n = 11)	CON (n = 13)
Age (y)	20.9 ± 1.7	19.7 ± 1.3	19.7 ± 1.1	21.0 ± 2.4	21.2 ± 2.2
Height (cm)	161.4 ± 6.0	163.3 ± 2.9	159.3 ± 5.4	160.5 ± 6.3	160.7 ± 6.4
Body mass index (kg/m^2)	25.6 ± 2.4	26.1 ± 3.2	26.0 ± 2.9	25.1 ± 3.0	25.2 ± 1.8
$\dot{V}O_{2peak}$ (mL/kg/min)					
Pre-intervention	26.7 ± 3.4	26.4 ± 4.1	28.7 ± 2.3	28.9 ± 2.6	28.9 ± 3.8
4th week	30.2 ± 2.2[a]	30.4 ± 2.9[a]	32.7 ± 1.9[a]	32.4 ± 3.0[a]	–
8th week	30.9 ± 3.1[a]	32.1 ± 3.8[a,b]	34.9 ± 1.5[a,b]	34.5 ± 3.7[a,b]	–
Post-intervention	33.0 ± 2.8[a,b,c,d]	35.3 ± 5.4[a,b,c,d]	37.7 ± 2.6[a,b,c,d]	34.2 ± 3.8[a,b,d]	29.6 ± 2.3

Note: Values represent mean ± SD.

[a]Significantly different from Pre-intervention at the P level < .05.

[b]Significantly different from 4th week at the P level < .05.

[c]Significantly different from 8th week at the P level < .05.

[d]Significantly different from corresponding CON value at the P < .05 level.

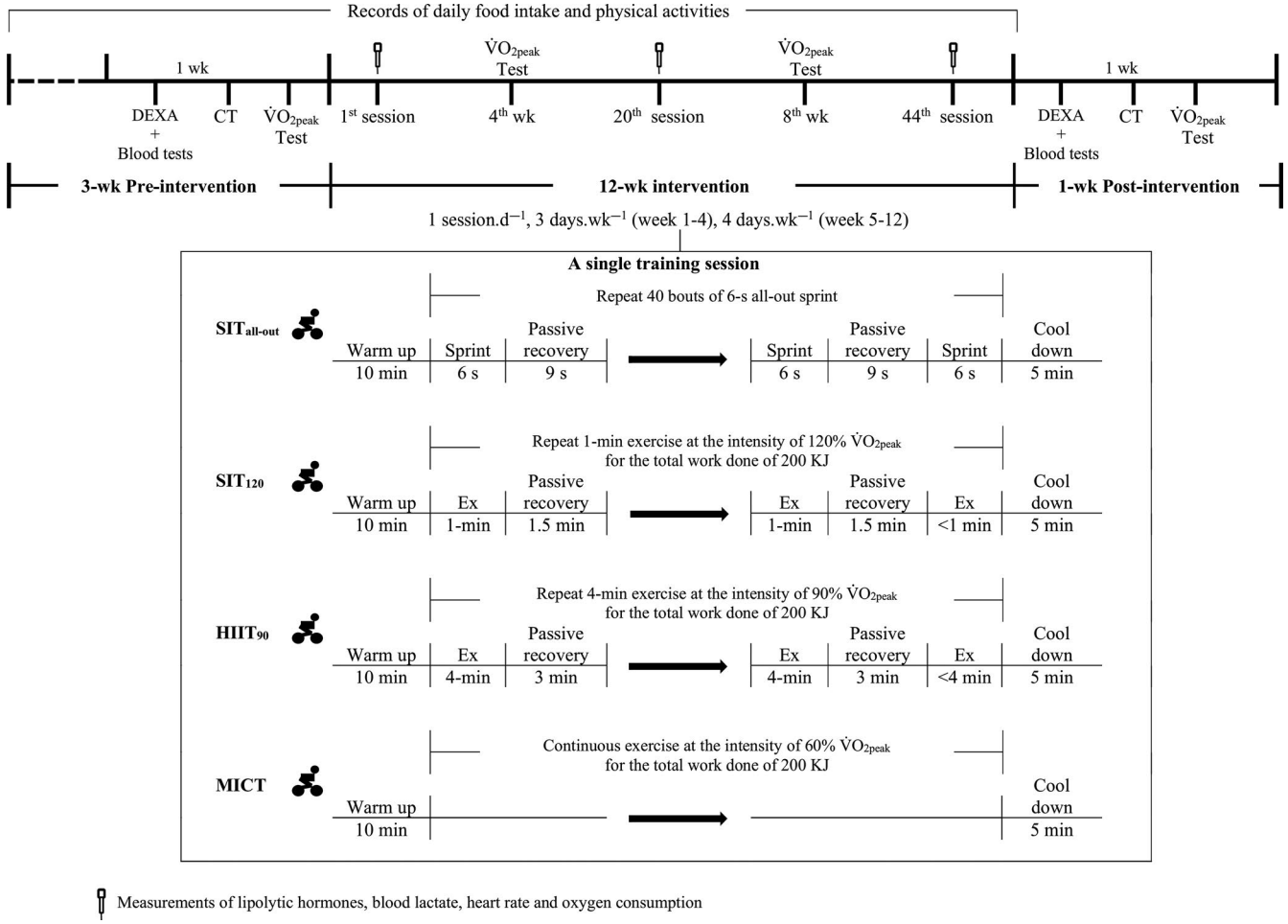

FIGURE 2 The overview of the study design

cycling. The time for each selected activity was recorded in durations of 5 minutes or more. The activity data were then converted to estimates of energy expenditure using established MET codes from the Compendium of physical activity.[18]

2.4 | Measurements of body composition and cardiometabolic parameters

The measurements of body fat mass and blood metabolic variables were carried out on the same day, while the visceral

fat area and $\dot{V}O_{2peak}$ were measured in the subsequent 2 days. The pre- and post-intervention tests were of the same order and were performed in avoidance of the menses phases of the participants. On the days of body composition and blood tests, the participants reported to the laboratory at 8:00 AM after a minimum 8-hour fast and refraining from strenuous exercise for 48 hours. Body mass and body fat percentage, as well as the fat mass of the whole-body, trunk, android, and gynoid regions, were measured through dual-energy X-ray absorptiometry (DEXA, Discovery Wi, Hologic Inc). Regional demarcations were adjusted by a trained technologist according to the guidelines adopted previously.[19] Briefly, the trunk region included the area from the bottom of the neckline to the top of the pelvis, excluding the arms; the android region measured from the cut of the pelvic region to 20% of the distance between the pelvic cut and the bottom of the neckline, excluding the arms; the gynoid region was below the android region and had a height equal to 2 times that of the android region, with the pelvic cut as the upper demarcation. For the assessment of the cross-sectional abdominal visceral (AVFA) and subcutaneous (ASFA) fat areas, a computed tomography (CT) scanner (Somatom Definition Flash; Siemens), with a consistent acquisition protocol set at 120 kVp and 150 mA, was used. During assessment, the participants laid in the supine position with their arms stretched above their heads; a 2-seconds, 5-mm scan was obtained from the umbilicus level (approximately the L4-L5 intervertebral space). The AVFA and ASFA were evaluated using the built-in volume calculation software of the CT scanner. For each scan, the number of voxels in the entire data set, with CT numbers between −190 and −30 HU, was plotted for visceral and subcutaneous fat. The AVFA and ASFA measured by the single-slice scan have been shown to be highly correlated ($r \geq .85$) with the corresponding volumetric reconstructions at the umbilicus in females.[20] The technicians responsible for the DEXA and CT measurements and the analyses were the same pre- and post-intervention and unaware of the participants and intervention groups. The intra-observer CV for measuring fat variables with DEXA and CT were ≤2.3% and ≤5.9%, respectively.

During the blood tests, blood samples were collected with participants in a seated position. A total amount of 5 mL venous blood were drawn from the antecubital vein using venipuncture. Blood glucose (GLC), triglyceride (TG), and total cholesterol (TC) concentrations were assessed immediately using an enzymatic assay (Jiancheng Biotech). The resting blood sample was separated at 2000 *g* for 10 minutes, aliquoted, and stored at −80°C for serum insulin (INS) analysis with ELISA (RayBiotech). The intra- and inter-assay CV for the variables were ≤9.8% and ≤9.3%, respectively.

The $\dot{V}O_{2peak}$ and HR_{peak} of participants were determined using a graded cycling exercise protocol starting at 60 W with a pedal frequency of 60 rpm; power output was increased by 30 W every 3 minutes until volitional exhaustion. $\dot{V}O_2$ and HR during the test were measured using the Quark-PFT equipment (COSMED) and Polar HR monitor (H6, Finland), respectively. The $\dot{V}O_{2peak}$ and HR_{peak} were the highest 30-second average values.

2.5 | Interventions

The $SIT_{all\text{-}out}$ protocol was composed of 40 bouts of 6-s all-out sprint on a Monark Wingate cycle ergometer (894E), interspersed with 9-second passive recovery intervals in a single session.[13] The cycling load in the first training session was set at 1 kp and would be increased by an increment of 0.5 kp whenever the participants were able to complete the 40 all-out exercise bouts at a given load in 1 session without undue fatigue. The average of the work rate that was recorded immediately before the end of each sprint, of the 40 sprints; and the total work done, referring to the sum of the product of the work rate and the exercise time of 6 seconds of the 40 sprints, were calculated for each training session.

In SIT_{120}, $HIIT_{90}$, and MICT, participants accomplished a total work done of 200 kJ on an electronically braked cycle ergometer (Monark 839E) at a pedal frequency of 60 rpm in each training session with varied work rate and exercise bout duration among the 3 groups. During the SIT_{120} session, participants repeated 1-minute exercise bouts at the work rate corresponding to 120% $\dot{V}O_{2peak}$, interspersed with 1.5-minute passive recovery intervals. In $HIIT_{90}$ session, the work rate corresponding to 90% $\dot{V}O_{2peak}$ was set, and the durations of exercise bouts and passive recovery intervals were 4 and 3 minutes, respectively. In the MICT session, participants performed continuous exercise at the work rate corresponding to 60% $\dot{V}O_{2peak}$. The work rate corresponding to the designated training intensity of different interventions was determined based on the linear regression related steady-state $\dot{V}O_2$ to power output that was developed in the pre-intervention $\dot{V}O_{2peak}$ test. Associated total number of exercise bouts in SIT_{120} and $HIIT_{90}$ [200 kJ work done/(work rate × exercise bout duration)], and total exercise time in MICT (200 kJ work done/work rate) for accomplishing the work done of 200 kJ per session were calculated. Subsequent to the $\dot{V}O_{2peak}$ test in the 4th and 8th weeks, the work rate of the participants was readjusted according to the reassessed $\dot{V}O_{2peak}$.

The warm-up and cool down exercises in training sessions were standardized in all groups. Water replacement ad libitum was voluntarily taken throughout the session. All groups exercised for 1 session/d, 3 d/wk in the first 4 weeks, and were increased to 4 d/wk in subsequent 8 weeks. The training adherence was calculated as the number of training sessions completed in compliance with the targeted intensity and duration, relative to the total number of training sessions prescribed. In the 1st, 20th, and 44th training sessions, pre-, immediate post-, and 3-hour post-exercise serum growth

hormone (GH), and epinephrine (EPI), as well as pre- and post-exercise blood lactate (LA) levels were analyzed. Blood sample collection and processing were the same as mentioned above. Serum GH was analyzed by ELISA (RayBiotech); a similar method was also used in the EPI analysis (Cusabio). The intra- and inter-assay CV for the serum hormones were ≤8.1%, and ≤4.7%, respectively. Blood LA was measured using a Sirius lactate analyzer (h/p/cosmos Germany). The HR, measured by the same Polar HR monitor, immediately after every 5 of the 40 sprints in $SIT_{all\text{-}out}$, and after each of the exercise bouts in SIT_{120}, and $HIIT_{90}$, was averaged. MICT HR was recorded immediately at the end of exercise. The continuous measurement of exercise $\dot{V}O_2$ and $\dot{V}CO_2$ using the same Cosmed equipment was utilized to predict the TEE via the Weir equation.[21]

2.6 | Statistical analysis

A priori, power calculation for determining the sample size was performed using the G-Power software (University of Trier, Trier, Germany). The Shapiro-Wilk normality test revealed that data for all variables were normally distributed. Two-way ANOVA with repeated measures was computed to assess the differences in body fat variables, blood variables, daily energy intake, and energy expenditure for physical activities between time points and across groups. Post hoc analyses for ANOVA, using the Bonferroni test for identifying simple main effects, were performed when a significant interaction was detected. Partial eta squared ($\eta\rho^2$) was used as effect size to measure of the main and interaction effects, which was considered small when < 0.06, and large when > 0.14.[22] Within-group effect size was revealed by calculating Cohen's *d*. Values of $d = 0.2$, 0.5, and 0.8 indicate small, medium, and large effect sizes.[23] Statistical significance was set at $P < .05$, and values are reported as means ± SD.

3 | RESULTS

3.1 | Participants

Table 1 is the physical characteristics of the participants of each group. The age, height, and pre-intervention $\dot{V}O_{2peak}$ were not different across the 5 groups ($P > .05$).

3.2 | Estimated daily energy intake and expenditure for physical activities

Table 2 is the averages of the estimated daily energy intake, and energy expenditure for physical activities apart from the sedentary activities and cycle ergometer training during the

TABLE 2 Estimated daily energy intake and estimated daily energy expenditures for physical activities apart from the sedentary activities and cycle ergometer training during the 3-wk pre-intervention (Pre) and 12-wk (12-wk) periods in $SIT_{all\text{-}out}$, SIT_{120}, $HIIT_{90}$, MICT, and CON groups

	$SIT_{all\text{-}out}$ (n = 11)		SIT_{120} (n = 12)		$HIIT_{90}$ (n = 12)		MICT (n = 11)		CON (n = 13)	
	Pre	12-wk	Pre	12-wk	Pre	12-wk	Pre	12-wk	Pre	12-wk
Estimated daily energy intake (kJ/d)	7926 ± 1354	7871 ± 1570	8161 ± 1188	8124 ± 1281	7768 ± 1334	7747 ± 1621	7891 ± 612	8034 ± 509	7997 ± 659	8187 ± 691
Estimated daily energy expenditure for physical activities apart from the sedentary activities and cycle ergometer training (kJ/d)	1741 ± 431	1668 ± 493	1871 ± 1230	1778 ± 1030	1819 ± 683	1959 ± 779	1606 ± 520	1635 ± 380	1714 ± 681	1650 ± 543

Note: Values represent mean ± SD. No significant difference was found within or between groups at the $P > .05$ level.

3-week pre-intervention and 12-week intervention periods. Both of the estimated values between the 2 periods, and across the 5 groups were not significantly different ($P > .05$).

3.3 | Training sessions

The compliance with the exercise intervention in the $SIT_{all-out}$, SIT_{120}, $HIIT_{90}$, and MICT groups was 99 ± 2%, 94 ± 4%, 98 ± 3%, and 99 ± 1%, respectively. No adverse events were reported during testing or training in either group.

Table 3 is the average values of the work done, work rate, and duration of training sessions in every 4 weeks of the intervention. During the intervention, the work done increased progressively in $SIT_{all-out}$ and was correspondingly less than that of the other 3 groups ($P < .05$). The exercise intensity revealed by the work rate increased progressively in all groups ($P < .05$). Among the 4 groups, the greatest work rate was in $SIT_{all-out}$, with a sequential decrease from SIT_{120}, $HIIT_{90}$ to MICT ($P < .05$). Session duration decreased progressively in all groups during the intervention ($P < .05$), except for $SIT_{all-out}$. Among the 4 groups, the MICT session duration was the longest while $SIT_{all-out}$ was the shortest ($P < .05$). The session duration of $HIIT_{90}$ and SIT_{120} was similar.

The average HR and TEE of the selected training sessions were compared among the 4 intervention groups. In the $SIT_{all-out}$, SIT_{120}, and $HIIT_{90}$, the HR (172.1 ± 7.4, 164.7 ± 7.6, 171.6 ± 8.5 b/min, $P > .05$), and the $\%HR_{max}$ (89.2 ± 2.7%, 86.6 ± 4.3%, 89.9 ± 3.5%, $P > .05$) were similar and were higher than that of MICT (136.7 ± 7.9 b/min, 70.8 ± 3.4%, $P < .05$). The TEE in MICT (1537 ± 110 kJ) was significantly greater than that of $HIIT_{90}$, SIT_{120}, and $SIT_{all-out}$ ($P < .05$). The TEE in $HIIT_{90}$ and SIT_{120} (1188 ± 27, 1190 ± 50 kJ, $P > .05$) were similar and were greater than that of $SIT_{all-out}$ (398.6 ± 34.7 kJ, $P < .05$). $\dot{V}O_{2peak}$ increased progressively in every 4 weeks in all intervention groups (Table 1, $P < .05$) and were not different among the groups. In CON, post-intervention $\dot{V}O_{2peak}$ did not change significantly and was lower than the corresponding values of the 4 intervention groups ($P < .05$).

3.4 | Fat and metabolic variables

3.4.1 | Whole-body and regional fat

The pre- and post-intervention body composition variables of the 5 groups, as well as the repeated measures ANOVA results, are shown in Table 4. The baseline body mass and body fat variables did not differ significantly among the 5 groups ($P > .05$). After the 12-week intervention, significant reductions in body mass, body fat percentage, and whole-body and regional fat mass variables were observed in $SIT_{all-out}$, SIT_{120}, and $HIIT_{90}$ ($P < .05$), and the alterations of each variable among the 3 groups were not different ($P > .05$). In MICT, similar reductions were only found in the body fat percentage and the fat mass of the whole-body, trunk, and android regions ($P < .05$), but not in the body mass and the gynoid fat mass ($P > .05$). No variable was changed in CON ($P > .05$).

3.4.2 | Abdominal visceral and subcutaneous fat

Following the 12-week intervention, the combined ASFA and AVFA were reduced in all intervention groups ($P < .05$), and the alterations were similar among all intervention groups. Nevertheless, the reductions in AVFA in the $SIT_{all-out}$, SIT_{120}, and $HIIT_{90}$ groups, which were of similar magnitude, were greater than that in the MICT group ($P < .05$, Figure 3). For the ASFA, a significant reduction was only found in the $SIT_{all-out}$ and MICT ($P < .05$), but not in the SIT_{120}, and $HIIT_{90}$ groups ($P > .05$). CON had no change in all variables ($P > .05$).

3.4.3 | Metabolic variables

Table 4 also shows the pre- and post-intervention resting values of selected metabolic variables and the corresponding repeated measures ANOVA results. After the intervention, fasting blood glucose decreased significantly in $SIT_{all-out}$, SIT_{120}, and $HIIT_{90}$ ($P < .05$), but not in MICT ($P > .05$). A significant reduction in serum INS was found in SIT_{120} and $HIIT_{90}$ ($P < .05$), but not in other groups ($P > .05$). The changes in the blood lipid profile were minor, and reductions in TG and TC were only found in $HIIT_{90}$ and $SIT_{all-out}$, respectively ($P < .05$). No variable was changed in CON ($P > .05$).

3.5 | Lipolytic hormones and blood lactate

The pre-, post-, and 3-hour post-exercise serum GH and EPI levels of the 4 interventions, which are the average values of the 1st, 20th, and 44th training sessions, are shown in Figure 4. Serum GH increased during exercise and returned to baseline 3-hour post-exercise in similar magnitude in all groups ($P < .05$). Serum EPI increased during exercise and returned to baseline 3-hour post-exercise in all groups ($P < .05$). However, the levels of serum EPI at the 3 time points in $SIT_{all-out}$ were significantly higher than the corresponding values of MICT ($P < .05$), while no difference was found among the interval training groups. Higher post-exercise serum EPI levels were also found in SIT_{120} and $HIIT_{90}$ compared with those of MICT ($P < .05$). For the average

TABLE 3 Work done, work rate, and duration of training sessions in every 4 wk during the 12-wk intervention in $SIT_{all\text{-}out}$, SIT_{120}, $HIIT_{90}$, and MICT groups

	$SIT_{all\text{-}out}$ (n = 11)			SIT_{120} (n = 12)		
	wk 1-4	wk 5-8	wk 9-12	wk 1-4	wk 5-8	wk 9-12
Work done (kJ)	47.1 ± 3.3	56.0[a] ± 3.4	63.6[a] ± 5.9	200	200	200
Work rate (W)	196.1 ± 13.7	233.3[a] ± 14.0	264.9[a] ± 24.4	158.6[b,c,d] ± 17.3	191.1[a,b,c,d] ± 18.4	199.6[a,b,c,d] ± 25.7
Session duration (min)	10	10	10	49.7[b,c,d] ± 4.4	42.6[a,b,d] ± 4.1	40.8[a,b,d] ± 4.4

Note: Values represent mean ± SD. The work done values of $SIT_{all\text{-}out}$ are less than the corresponding values of SIT_{120}, $HIIT_{90}$, and MICT at the $P < .05$ level.

[a]Significantly different from wk 1-4 at the P level < .05.

[b]Significantly different from corresponding $SIT_{all\text{-}out}$ at the $P < .05$ level.

[c]Significantly different from corresponding $HIIT_{90}$ at the $P < .05$ level.

[d]Significantly different from corresponding MICT at the $P < .05$ level.

TABLE 4 Pre- and post-intervention levels and changes in body fat variables [body mass, % body fat, fat mass of whole-body, android FM, gynoid FM, trunk FM, abdominal visceral and subcutaneous fat areas], and metabolic variables [fasting blood glucose, triglyceride, total cholesterol, and serum insulin] in $SIT_{all\text{-}out}$, SIT_{120}, $HIIT_{90}$, MICT, and CON groups

	$SIT_{all\text{-}out}$ (n = 11)		SIT_{120} (n = 12)		$HIIT_{90}$ (n = 12)	
	Pre	Post	Pre	Post	Pre	Post
Body mass (kg)	66.6 ± 7.2	63.6 ± 6.3[a]	69.6 ± 9.5	67.5 ± 8.9[a]	65.8 ± 7.4	62.7 ± 6.9[b]
	−3.0 ± 1.8 (−4.2, −1.8)[c]; 0.44		−2.2 ± 2.3 (−3.7, −0.7)[c]; 0.23		−3.1 ± 3.8 (−5.5, −0.7)[c]; 0.43	
% Body fat (%)	44.1 ± 4.1	42.1 ± 4.4[a]	43.4 ± 4.8	40.5 ± 4.6[a]	44.6 ± 5.0	41.5 ± 4.8[a]
	−2.1 ± 1.4 (−3.0, 1.1)[c]; 0.47		−2.9 ± 1.5 (−3.8, −1.9)[c]; 0.62		−3.1 ± 2.3 (−4.6, −1.7)[c]; 0.63	
Whole-body FM (kg)	28.4 ± 5.3	25.8 ± 4.7[a]	29.3 ± 6.5	26.6 ± 5.8[a]	28.5 ± 6.3	25.3 ± 5.6[a]
	−2.6 ± 1.6 (−3.7, −1.5)[c]; 0.52		−2.8 ± 1.7 (−3.9, −1.7)[c]; 0.44		−3.3 ± 3.3 (−5.3, −1.2)[c]; 0.54	
Android FM (kg)	2.2 ± 0.4	2.0 ± 0.4[a]	2.4 ± 0.6	2.1 ± 0.5[a]	2.3 ± 0.5	2.0 ± 0.5[a]
	−0.2 ± 0.2 (−0.3, −0.1)[c]; 0.50		−0.2 ± 0.2 (−0.4, −0.1)[c]; 0.54		−0.3 ± 0.3 (−0.5, −0.2)[c]; 0.60	
Gynoid FM (kg)	5.4 ± 0.9	4.9 ± 0.7[a]	5.5 ± 1.2	5.1 ± 1.1[a]	5.5 ± 1.1	5.0 ± 1.0[b]
	−0.5 ± 0.3 (−0.7, −0.2)[c]; 0.62		−0.4 ± 0.4 (−0.6, −0.1)[c]; 0.35		−0.5 ± 0.6 (−0.9, −0.1)[c]; 0.48	
Trunk FM (kg)	15.1 ± 2.6	13.7 ± 2.7[a]	15.3 ± 3.4	13.8 ± 2.9[a]	15.2 ± 3.4	13.2 ± 3.1[a]
	−1.4 ± 0.8 (−2.0, 0.9)[c]; 0.53		−1.6 ± 1.3 (−2.4, −0.8)[c]; 0.47		−1.9 ± 1.3 (−2.8, −1.1)[c]; 0.61	
AVFA (cm^2)	79.3 ± 24.9	59.9 ± 24.5[a]	75.0 ± 29.1	57.1 ± 18.9[a]	78.2 ± 24.2	62.9 ± 16.6[a]
	−19.3 ± 12.7 (−27.9, −10.8)[c]; 0.79		−17.9 ± 18.9 (−29.9, −5.8)[c]; 0.73		−15.3 ± 14.1 (−24.3, −6.3)[c]; 0.74	
ASFA (cm^2)	220.9 ± 46.1	201.3 ± 54.1[b]	238.6 ± 72.1	232.0 ± 57.8	234.7 ± 44.8	213.4 ± 52.7
	−19.6 ± 28.8 (−39.0, −0.3); 0.39		−6.6 ± 26.2 (−23, 10.1); 0.10		−21.3 ± 35.7 (−43.9, 1.4); 0.44	
AVFA + ASFA (cm^2)	300.2 ± 55.0	261.2 ± 60.6[a]	313.6 ± 88.6	289.1 ± 66.9[b]	313.0 ± 60.5	276.3 ± 63.9[b]
	−39.0 ± 35.6 (−62.9, −15.0)[c]; 0.67		−24.4 ± 36.0 (−47.3, −1.5); 0.31		−36.7 ± 45.3 (−65.5, −7.9)[d]; 0.59	
Glucose (mmol/L)	3.9 ± 0.3	3.3 ± 0.6[b]	4.0 ± 0.6	3.4 ± 0.4[b]	3.9 ± 0.4	3.3 ± 0.6[a]
	−0.6 ± 0.7 (−1.1, −0.1)[d]; 1.26		−0.6 ± 0.5 (−0.9, −0.3)[c]; 1.18		−0.6 ± 0.5 (−1.0, −0.3)[c]; 1.18	
Insulin (μIU/mL)	19.9 ± 2.6	19.9 ± 2.1	19.4 ± 2.2	16.8 ± 1.9[a]	20.3 ± 3.1	18.7 ± 2.4[b]
	−0.1 ± 2.5 (−1.7, 1.7); 0.00		−2.6 ± 2.2 (−4.0, −1.2); 1.26		−1.6 ± 2.5 (−3.2, −0.1); 0.58	
Triglyceride (mmol/L)	1.9 ± 0.8	1.5 ± 0.2	2.1 ± 1.6	1.5 ± 0.3	1.7 ± 0.3	1.5 ± 0.3[b]
	−0.4 ± 0.9 (−1.0, 0.2); 0.69		−0.6 ± 1.5 (−1.6, 0.3); 0.52		−0.2 ± 0.3 (−0.8, −0.1); 0.67	
Total cholesterol (mmol/L)	5.1 ± 1.5	4.1 ± 1.0[b]	5.0 ± 1.4	4.5 ± 1.1	5.5 ± 1.1	4.9 ± 1.1
	−1.1 ± 1.2 (−1.9, −0.2)[d]; 0.78		−0.5 ± 0.8 (−1.0, 0.0); 0.40		−0.5 ± 1.1 (−1.2, 0.2); 0.55	

Note: Values represent means ± SD [mean change (95% confidence interval); Cohen's *d*].

Abbreviations: ASFA, Abdominal Subcutaneous Fat Area; AVFA, Abdominal Visceral Fat Area; FM, Fat Mass.

[a]Significantly different from corresponding Pre value at the $P < .01$ level.

[b]Significantly different from corresponding Pre value at the $P < .05$ level.

[c]Significantly different from corresponding CON value at the $P < .01$ level.

[d]Significantly different from corresponding CON value at the $P < .05$ level.

$HIIT_{90}$ (n = 12)			MICT (n = 11)		
wk 1-4	wk 5-8	wk 9-12	wk 1-4	wk 5-8	wk 9-12
200	200	200	200	200	200
117.3[b,d] ± 13.0	132.1[a,b,d] ± 13.3	145.1[a,b,d] ± 14.8	56.1[b] ± 8.9	61.5[a,b] ± 8.0	66.1[a,b] ± 9.3
42.6[b,d] ± 5.7	41.4[b,d] ± 6.3	37.6[a,b,d] ± 3.9	60.9[b] ± 10.3	55.3[a,b] ± 7.7	51.6[a,b] ± 7.5

MICT (n = 11)		CON (n = 13)		Two-way ANOVA *P* value ($\eta\rho^2$) (group, time, interaction)
Pre	Post	Pre	Post	
64.6 ± 8.7	64.4 ± 8.3	65.3 ± 7.5	65.9 ± 6.8	0.661 (0.04), <.000 (0.32), <0.000 (0.31)
−0.2 ± 1.3 (−1.1, 0.7); 0.02		0.7 ± 1.9 (−0.5, 1.8); −0.08		
44.1 ± 4.5	43.4 ± 4.1[b]	43.5 ± 4.0	43.8 ± 4.0	0.861 (0.02), <.000 (0.57), <0.000 (0.45)
−0.7 ± 0.9 (−1.3, −0.1)[d]; 0.16		0.3 ± 1.2 (−0.4, 1.0); −0.07		
27.7 ± 6.2	27.1 ± 5.7[b]	27.2 ± 3.7	27.8 ± 4.2	0.991 (0.01), <.000 (0.47), <0.000 (0.40)
−0.6 ± 0.7 (−1.0, −0.1)[c]; 0.10		0.6 ± 1.1 (−0.1, 1.3); −0.15		
2.2 ± 0.7	2.1 ± 0.6[b]	2.3 ± 0.5	2.3 ± 0.6	0.918 (0.02), <.000 (0.45), 0.001 (0.28)
−0.1, ± 0.2 (−0.2 ± 0); 0.15		0 ± 0.1 (−0.1, 0.1); 0.00		
5.3 ± 0.9	5.3 ± 0.9	5.1 ± 0.7	5.3 ± 0.7	0.995 (0.00), <.000 (0.32), <0.000 (0.36)
0 ± 0.2 (−0.1, 0.1); 0.00		0.1 ± 0.2 (0, 0.3); −0.29		
14.4 ± 3.8	13.8 ± 3.5[a]	14.5 ± 2.5	14.8 ± 2.9	0.993 (0.00), <.000 (0.54), <0.000 (0.43)
−0.6 ± 0.5 (−0.9, −0.2)[c]; 0.16		0.3 ± 0.7 (−0.1, 0.8); −0.11		
70.7 ± 29.6	67.2 ± 28.2	74.5 ± 18.3	73.9 ± 19.2	0.930 (0.02), <.000 (0.43), 0.002 (0.26)
−3.5 ± 13.1 (−12.3, 5.3); 0.12		−0.7 ± 7.5 (−5.2, 3.9); 0.03		
254.5 ± 65.3	222.7 ± 58.7[a]	248.2 ± 59.1	248.1 ± 57.0	0.543 (0.01), <.000 (0.24), 0.082 (0.14)
−31.8 ± 26.8 (−49.8, −13.8)[c]; 0.51		−0.1 ± 27.8 (−16.9, 16.7); 0.00		
325.2 ± 87.9	289.9 ± 84.6[a]	322.7 ± 74.6	322.0 ± 68.7	0.675 (0.04), <.000 (0.38), 0.063 (0.15)
−35.3 ± 32.4 (−57.0, −13.5)[d]; 0.41		−0.8 ± 29.7 (−18.7, 17.2); 0.01		
4.0 ± 1.0	3.9 ± 0.7	3.5 ± 0.5	3.7 ± 0.5	0.174 (0.11), .001 (0.18), 0.065 (0.15)
−0.1 ± 1.4 (−1.0, 0.8); 0.12		0.1 ± 0.6 (−0.2, 0.5); −0.40		
22.1 ± 9.1	21.7 ± 8.0	20.5 ± 1.9	18.8 ± 3.2	0.100 (0.13), .094 (0.05), 0.812 (0.03)
−0.4 ± 12.3 (−9.2, 8.0); 0.05		−1.7 ± 3.0 (−3.5, 0.1); 0.65		
1.9 ± 0.4	1.9 ± 0.4	1.9 ± 0.4	2.0 ± 0.8	0.401 (0.07), .043 (0.07), 0.200 (0.10)
0.0 ± 0.5 (−0.3, 0.3); 0.00		0.1 ± 0.2 (−0.2, 0.4); −0.16		
4.7 ± 1.2	4.5 ± 0.9	5.0 ± 1.1	5.2 ± 1.5	0.507 (0.06), .004 (0.14), 0.149 (0.12)
−0.3 ± 1.1 (−1.0, 0.5); 0.19		0.1 ± 1.3 (−0.6, 0.9); −0.15		

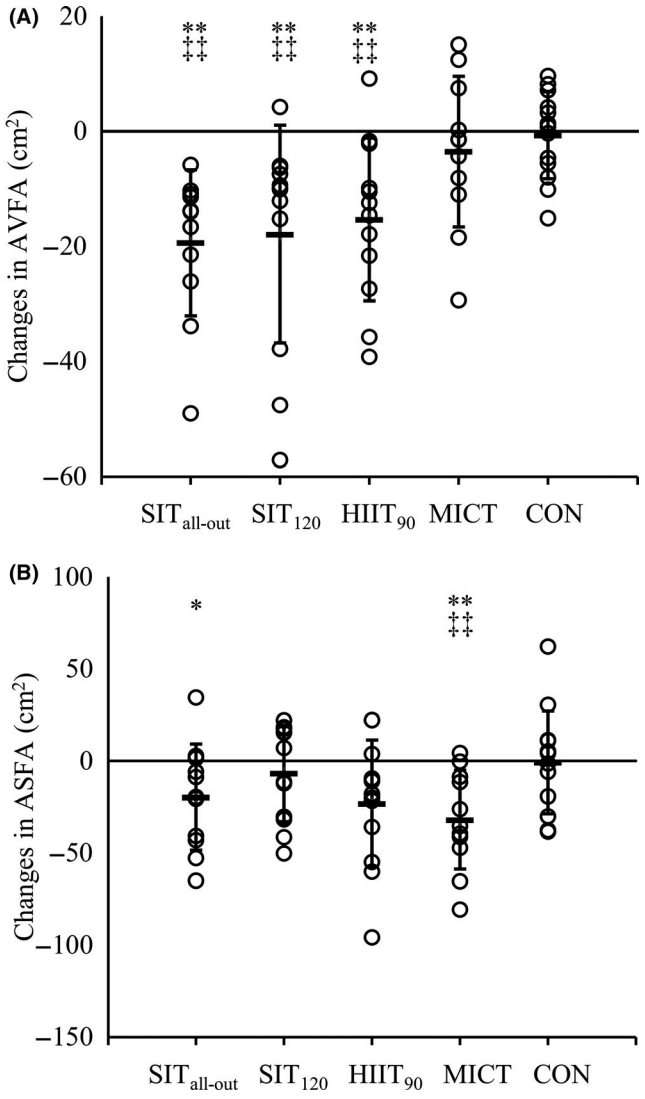

FIGURE 3 Changes in (A) AVFA and (B) ASFA of participants post-intervention in $SIT_{all\text{-}out}$, SIT_{120}, $HIIT_{90,}$ MICT, and CON groups. $^{*}P < .05$, $^{**}P < .01$, significant change post-intervention. $^{‡‡}P < .01$, significantly different from CON

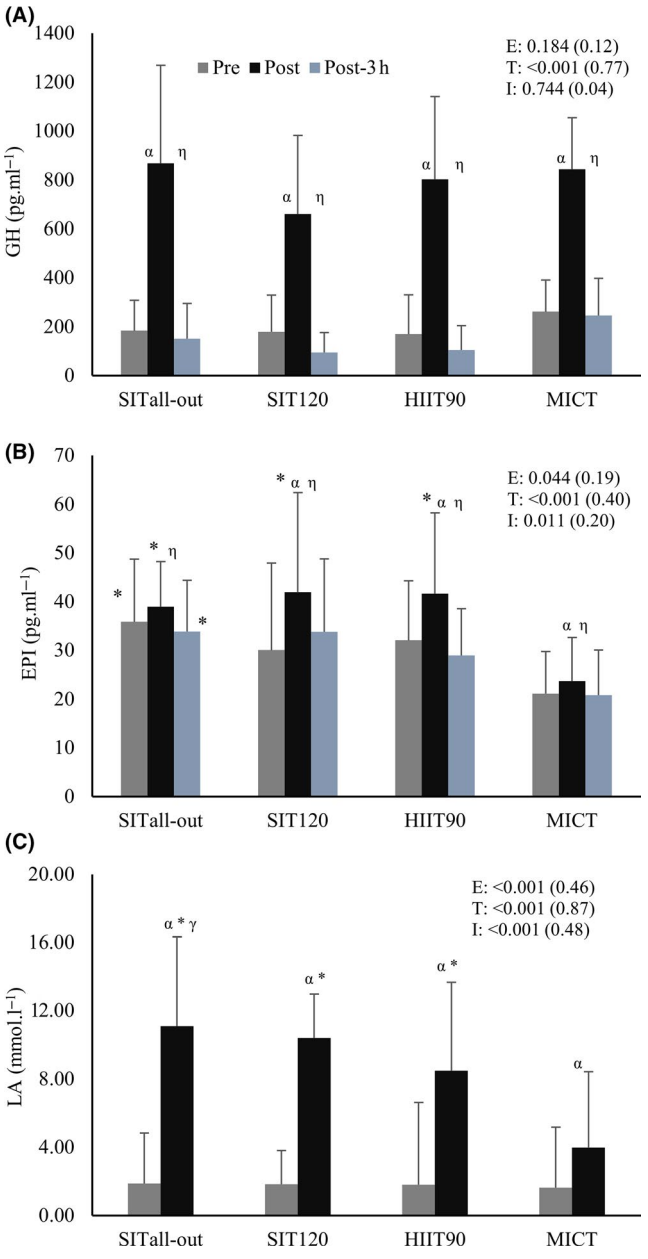

FIGURE 4 Pre- (Pre), post- (Post), and 3-h post-exercise (Post-3h) serum (A) GH, and (B) EPI, and (C) pre- and post-exercise blood LA levels of the $SIT_{all\text{-}out}$, SIT_{120}, $HIIT_{90}$, and MICT groups. E, exercise effect ($\eta\rho^2$); T, time effects ($\eta\rho^2$); I, interaction effects ($\eta\rho^2$). $^{*}P < .05$, significantly different from corresponding MICT value. $^{\gamma}P < .05$, significantly different from corresponding $HIIT_{90}$ value. $^{\alpha}P < .05$, significantly different from corresponding Pre value. $^{\eta}P < .05$, significantly different from corresponding post-3 h value

blood LA of the selected training sessions, a significant increase was observed post-exercise in all groups (Figure 4). The post-exercise value of MICT was significantly lower than those of $SIT_{all\text{-}out}$, SIT_{120}, and $HIIT_{90}$ ($P < .05$) while the $SIT_{all\text{-}out}$ and SIT_{120} values were similar; the $SIT_{all\text{-}out}$ was significantly higher than that of $HIIT_{90}$ ($P < .05$).

4 | DISCUSSION

This study compared the exercise training-induced fat reductions resulting from the 12-week $SIT_{all\text{-}out}$, SIT_{120}, and $HIIT_{90}$, and MICT regimes with training intensity ranging from all-out to moderate, concomitant with lightened work done of 200 kJ or below per session. Essentially, it was found that the visceral fat loss following $SIT_{all\text{-}out}$, SIT_{120}, and $HIIT_{90}$ was greater in comparison with MICT. Moreover, interval training at intensities above 90% $\dot{V}O_{2peak}$ did not lead to further improvements in the visceral fat response to 12 weeks of exercise training. Although the current findings show less support to the advocate of "the higher, the better"

in manipulating the exercise intensity of HIIT regimen to reduce visceral fat, the $SIT_{all\text{-}out}$ protocol characterized by all-out effort and brief training sessions is still the most time-efficient strategy among the 3 interval training regimes for combatting central obesity.

Following the 12-week intervention, the alterations in body fat as well as those selected cardiometabolic variables including blood glucose and $\dot{V}O_{2peak}$ (Tables 1, 4) in the $SIT_{all\text{-}out}$, SIT_{120}, and $HIIT_{90}$ groups were of a similar magnitude, and the reductions in selected variables including AVFA were greater than that subsequent to MICT. Although the TEE per session, which was derived from training session $\dot{V}O_2$, was greater in MICT among the 4 training regimens, the increase in post-exercise resting EE following SITs and HIIT might have been greater than that of MICT as the magnitude of the post-exercise EE increase is dependent upon the preceding exercise intensity.[24] Indeed, the greater elevation of post-exercise EE was deemed as one of potential contributors to the promising effects of HIIT and SIT interventions on fat loss.[25] It has been shown that a single session of SIT could induce an elevation of daily EE of >225 kcal,[26] markedly greater than that mediated by the MICT counterparts.[25] The elevated post-exercise EE following vigorous exercise, which is likely a consequence of the restoration of physiological equilibrium from the exercise-mediated metabolic perturbations, appears to be relied on fat oxidation.[27] Recently, a higher rate of 2-hour post-exercise fat oxidation after a single session of SIT in comparison correspondingly with that of MICT (0.12 vs 0.05 g/min) was reported.[28] Apart from the possible greater elevations of post-exercise EE and associated fat oxidation, high-intensity exercise-induced hydrocarbon source redistribution might have also potentially contributed to the greater reductions in AVFA observed in SITs and HIIT relative to that in MICT.[29,30] Indirect evidences have suggested that following exercise active muscles may transiently increase competition with adipose tissues, including abdominal fat depots, for circulating postprandial hydrocarbons from multiple sources (fat, carbohydrate, and protein) for tissue reconstruction,[31,32] and the extent of competition appears to be dependent upon exercise intensity.[29,30] The resultant greater partitioning of postprandial hydrocarbon-based nutrients into the active muscles may create apparent negative energy balance in the abdominal fat cells and promote the fat reduction.[29,30] Besides, it has been reported that high-intensity interval exercise may resulted in suppressed appetite.[33] Although marked reduction in estimated daily energy intake during the 12-week intervention, in comparison with that of pre-intervention, was not observed in the participants, the possible contribution of the SITs- and HIIT-induced suppressed appetite and associated reduction in food intake to the fat loss should not be neglected.

In line with our previous findings,[13] the $SIT_{all\text{-}out}$, which composed of brief all-out training sessions, induced a similar AVFA reduction in comparison with that of the SIT_{120}, and $HIIT_{90}$. The similar fat reduction has been presumed to be associated with the greater release of lipolytic hormones resulting from the higher exercise intensity despite the specific hormone-induced lipolysis may not totally translate into fatty acid oxidation.[15] However, the time course of the release of serum lipolytic hormones of GH and EPI from pre-exercise to 3-hour post-exercise during the training sessions among the $SIT_{all\text{-}out}$, SIT_{120}, and $HIIT_{90}$ were not different (Figure 4). The present findings appear to be in contrast to the previous notion that lipolytic hormones, mainly the catecholamines and growth hormones, increase with exercise intensity.[9,14] Nevertheless, such a dose-response relationship previously reported was demonstrated in non-obese subjects exercising at an intensity below their $\dot{V}O_{2peak}$. According to the current findings, the release of the lipolytic hormones during the interval exercise of the 2 SITs and $HIIT_{90}$ is not likely to be definitely proportional to the exercise intensity when it is near or beyond the $\dot{V}O_{2peak}$. It was further noted that the difference in blood LA among the 2 SITs and $HIIT_{90}$ groups in response to heavy exercise were minimal, suggesting that the possible mitigation of catecholamine-induced lipolysis and the inhibition of the hormone-sensitive lipase,[34,35] resulting from high-level blood LA accumulation, might have occurred similarly among the 3 groups. Based on the present findings, it is reasonable to postulate that visceral fat loss reaches a plateau when the training intensity is beyond a cutoff corresponding to 90% $\dot{V}O_{2peak}$. HIIT at exercise intensities above this cutoff results in similar lipolysis from visceral fat storage, regardless of further increases in exercise intensity, and is likely driven by lipolytic hormones.

In contrast to MICT, the dose-response effect of HIIT on visceral fat reduction is vague. It has been shown that extra HIIT sets induced no additional visceral fat loss resulting from the lowest dose.[36] This was further supported by our previous studies when 2 groups of age-matched, obese female subjects participating in two 12-week HIIT groups with identical exercise mode (cycling) and intensity (90% $\dot{V}O_{2peak}$), but varied work done (300 vs 400 kJ) and exercise duration (~34 vs ~46 minutes), resulted in similar AVFA reductions (−9.1 vs −9.7 cm^2). The absence of the dose response implied that the particular low work done in $SIT_{all\text{-}out}$ was not likely to hinder the effects of the training regimen on the specific fat loss. Interestingly, in comparison with the visceral fat loss resulting from the above-mentioned previous prolonged HIIT protocols,[13] the AVFA reduction induced by the current $HIIT_{90}$ with less work done (200 kJ) was relatively greater (−15.3 vs −9.7 cm^2). The greater AVFA reductions, in comparison with those resulting from the previous $SIT_{all\text{-}out}$ protocol,[13] were also found when the 80 sprint repetitions were trimmed down

to 40 sprint repetitions in the current $SIT_{all\text{-}out}$ (−19.3 vs −6.3 cm^2). The present data could not clearly explain the underlying mechanism for the advantaged reduction in visceral fat with relatively brief $SIT_{all\text{-}out}$ and HIIT regimens. Potentially, this endeavor might harness the benefits of the lower blood LA accumulation and the associated less adverse effect on lipolysis in response to the abbreviated interval training maneuver.[37] Nonetheless, the interpretation of the effectiveness of the brief regimens on visceral fat reduction must be considered with caution, as the factor of the inter-individual variability in the exercise training-induced visceral fat loss, which was partly attributed to the gene polymorphisms,[38] has not been ruled out in this case. Although comparable visceral fat loss was found among the 3 interval training regimens, the brief $SIT_{all\text{-}out}$ protocol with session duration more than fourfold lesser than that of the other training regimens (Table 3) is currently still considered as the most time-efficient lifestyle intervention strategy for controlling central obesity-related complications. We have shown that extra work done in addition to the current interval training regimens is not likely to lead to significant improvements in visceral fat loss in obese participants.[12,13] However, the minimum work done of the interval training regimens that would induce apparent visceral fat loss is not clear. It is worth examining further the time-efficient advantage of the 3 interval training regimens in visceral fat loss by accomplishing less work done per training session in future studies. This could facilitate the ascertainment of the most tolerable and time-efficient interval training regimen to obese people for controlling visceral obesity.

In the present study, there are some limitations deserve discussion. Firstly, the absence of plasma volume correction in hormone measurements might have probably interfered with the results of the comparison of the exercise-induced increase in the lipolytic hormones among the time points and across interventions. Nonetheless, the interference was deemed minor as little exercise-induced changes in plasma albumin and hematocrit (data not shown) have been observed in obese women participating in similar exercises with voluntary water replacement. Another possible limitation to this study is that the time course of the release of serum lipolytic hormones of GH and EPI in response to exercise is only based on the blood samples collected pre-, post-, and 3-hour post-exercise. We cannot rule out the existence of potential differences in the hormones response at time points others than those three. A protocol that includes measurements at additional time points during the training session may improve the accuracy of the time course of the exercise-induced GH and EPI releases. Further, the present study is lack of the data of hormone-sensitive lipase (HSL). As only a given molar concentration of lipolytic hormone would interact with target adipocytes, the measurement of HSL in exercises may gain further insight into the differences in the mobilization of free fatty acids from adipocytes and associated adipose metabolism among interventions. However, the HSL measurement from adipose tissue requires biopsies related investigations using animal model is recommended. Finally, although the use of the 24-hour dietary and activity recall in the current study might have reduced the error associated with a long-term recall, the high respondent burden might have diminished the motivation of participants to provide accurate dietary and activity information for daily energy intake and expenditure estimations. Appropriate method of dietary and activity recalls with balance in between the long-term recall error and the high respondent burden would be more practical for obtaining accurate estimated daily energy intake and expenditure.

In conclusion, the 12-week mechanical work-equivalent MICT, SIT_{120}, and $HIIT_{90}$ regimens, as well as the $SIT_{all\text{-}out}$ regimen, could induce significant reductions in whole-body and regional fat mass, and cardiometabolic adaptations in obese young females. However, the reductions in AVFA resulting from $SIT_{all\text{-}out}$, SIT_{120}, and $HIIT_{90}$ were greater in comparison to MICT. Moreover, despite the exercise intensity was distinctive among the SITs and HIIT groups, the AVFA reduction induced by the 3 interval training regimes was similar and could partly be attributed to the similar magnitude of the release of lipolytic hormones of GH and EPI. Such findings may imply that a similar reduction in visceral fat may result when the exercise intensity of the HIIT is beyond a cutoff corresponding to ~90% $\dot{V}O_{2peak}$.

5 | PERSPECTIVE

This study demonstrated that the visceral fat loss induced by the interval training regimens composed of exercise at or above 90% $\dot{V}O_{2peak}$ were not responsive to the change in training intensity. The findings suggest that the HIIT regimen at relative low intensity (~90% $\dot{V}O_{2peak}$) may be an alternative for people who have had difficulties in performing all-out interval exercise to achieve significant visceral fat loss and cardiometabolic adaptations. However, the current study did not investigate the minimal exercise intensity and training volume of the HIIT that could induce comparable health-promoting effects resulting from $SIT_{all\text{-}out}$. We suggest future randomized studies investigating the optimal time-efficient HIIT regimen by adoptions of lighter training intensity and lesser work done per session. Further, since evidence is limited on the feasibility of the SIT and HIIT in special populations such as individuals with increased risk for exercise-related complications, comprehensive studies for establishing the most beneficial HIIT regimen that is optimal and sustainable for different populations are warranted in this regard.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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