High-Intensity Interval Training Increases Cardiac Output and $\dot{V}O_{2max}$

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ABSTRACT

ASTORINO, T. A., R. M. EDMUNDS, A. CLARK, L. KING, R. M. GALLANT, S. NAMM, A. FISCHER, and K. A. WOOD. High-Intensity Interval Training Increases Cardiac Output and $\dot{V}O_{2max}$. *Med. Sci. Sports Exerc.*, Vol. 49, No. 2, pp. 00–00, 2017. Increases in maximal oxygen uptake ($\dot{V}O_{2max}$) frequently occur with high-intensity interval training (HIIT), yet the specific adaptation explaining this result remains elusive. **Purpose**: This study examined changes in $\dot{V}O_{2max}$ and cardiac output (CO) in response to periodized HIIT. **Methods**: Thirty-nine active men and women (mean age and $\dot{V}O_{2max}$ = 22.9 ± 5.4 yr and 39.6 ± 5.6 mL·kg^{-1}·min^{-1}) performed HIIT and 32 men and women (age and $\dot{V}O_{2max}$ = 25.7 ± 4.5 yr and 40.7 ± 5.2 mL·kg^{-1}·min^{-1}) were nonexercising controls (CON). The first 10 sessions of HIIT required eight to ten 60 s bouts of cycling at 90%–110% percent peak power output interspersed with 75 s recovery, followed by randomization to one of three regimes (sprint interval training (SIT), high-volume interval training ($HIIT_{HI}$), or periodized interval training (PER) for the subsequent 10 sessions. Before, midway, and at the end of training, progressive cycling to exhaustion was completed during which $\dot{V}O_{2max}$ and maximal CO were estimated. **Results**: Compared with CON, significant ($P < 0.001$) increases in $\dot{V}O_{2max}$ in HIIT + SIT (39.8 ± 7.3 mL·kg^{-1}·min^{-1} to 43.6 ± 6.1 mL·kg^{-1}·min^{-1}), HIIT + $HIIT_{HI}$ (41.1 ± 4.9 mL·kg^{-1}·min^{-1} to 44.6 ± 7.0 mL·kg^{-1}·min^{-1}), and HIIT + PER (39.5 ± 5.6 mL·kg^{-1}·min^{-1} to 44.1 ± 5.4 mL·kg^{-1}·min^{-1}) occurred which were mediated by significant increases in maximal CO (20.0 ± 3.1 L·min^{-1} to 21.7 ± 3.2 L·min^{-1}, $P = 0.04$). Maximal stroke volume was increased with HIIT ($P = 0.04$), although there was no change in maximal HR ($P = 0.88$) or arteriovenous O_2 difference ($P = 0.36$). **Conclusions**: Increases in $\dot{V}O_{2max}$ exhibited in response to different HIIT regimes are due to improvements in oxygen delivery. **Key Words:** INTERVAL EXERCISE, MAXIMAL OXYGEN UPTAKE, STROKE VOLUME, THORACIC IMPEDANCE, FICK EQUATION, CYCLE ERGOMETRY

In untrained individuals, significant improvements in maximal oxygen uptake ($\dot{V}O_{2max}$) occur in response to 2–12 wk of high-intensity interval training (HIIT) (4,11,25,29). Data from a meta-analysis (5) show a mean increase in $\dot{V}O_{2max}$ equal to 0.51 L·min^{-1} in response to 6–13 wk of HIIT, with higher-volume HIIT eliciting even greater improvements (0.8–0.9 L·min^{-1}) in $\dot{V}O_{2max}$. Compared with moderate-intensity continuous training (MICT), results from some studies (8,37) exhibit similar increases in $\dot{V}O_{2max}$ in response to HIIT, although this is not a universal finding (29). This discrepancy in $\dot{V}O_{2max}$ response to exercise training across studies is likely due to differences in volume and intensity of each HIIT regime as well as the influence of genetics on $\dot{V}O_{2max}$ response to training (7). This increase in $\dot{V}O_{2max}$ is important because it increases exercise tolerance and long-term health status (27). For example, a 3.5-mL·kg^{-1}·min^{-1} increase in $\dot{V}O_{2max}$ is associated with a 13% and 15% lower risk of all-cause mortality and cardiovascular event, respectively (22).

Despite the widely seen increases in $\dot{V}O_{2max}$ demonstrated in response to HIIT, the specific adaptations explaining this outcome remain elusive. The Fick Equation states that oxygen uptake is determined by the product of cardiac output (CO) and arteriovenous oxygen difference (a-vDO_2). In untrained men and women, Daussin et al. (11) reported that maximal CO (CO_{max}) and a-vDO_2 are increased in response to 8 wk of HIIT which led to significant improvements in $\dot{V}O_{2max}$. Similarly, Warburton et al. (37) showed significant increases in CO_{max} and $\dot{V}O_{2max}$ in response to 12 wk of HIIT in untrained men. In contrast, MacPherson et al. (24) demonstrated a 12% increase in $\dot{V}O_{2max}$ in response to 6 wk of treadmill sprint interval training (SIT) despite no increase in CO_{max}, which implies that peripheral adaptations mediate the improvement in $\dot{V}O_{2max}$. In another study (13), resting and/or submaximal measures of stroke volume (SV) and CO assessed with echocardiography were increased in response to HIIT, although no maximal values were determined which does not allow identification of adaptations responsible for the training-induced improvements in $\dot{V}O_{2max}$. Across all

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studies, the participant population, specific characteristics of HIIT, and method of CO determination varied, which may elicit some of the dissimilar findings reported. Overall, changes in CO_{max} in response to HIIT are poorly understood despite significant increases in $\dot{V}O_{2max}$ seen after as little as six to 10 sessions of training (4,13).

Although previous findings show similar and in some cases superior adaptations with HIIT versus MICT, the optimum HIIT regimen is unknown, as in most studies, adaptations to interval training are compared with those from MICT. Only a few studies have directly compared changes in $\dot{V}O_{2max}$ between unique HIIT regimes, and these data are equivocal. In untrained women (4), similar increases in $\dot{V}O_{2max}$ were exhibited between HIIT regimes differing in absolute intensity. Findings from Matsuo et al. (25) in untrained men showed similar increases in $\dot{V}O_{2max}$ between high-volume HIIT and low-volume SIT. In contrast, only high-volume HIIT led to significant increases in $\dot{V}O_{2max}$ in overweight individuals compared with low-volume HIIT or MICT (6).

In each study (4,6,25), the specific duration, intensity, and recovery duration differed across HIIT regimes. However, in no study was the actual structure of HIIT modified *during* the course of training. In the discipline of resistance training, periodization is widely used (35) to minimize onset of plateau and attempt to sustain physiological adaptations. Because increases in $\dot{V}O_{2max}$ seem to level off during prolonged HIIT when only intensity is adjusted (4,30), there seems to be potential for periodized training within the domain of HIIT. In addition, it could be argued that constantly modifying the structure of HIIT may improve the participants' perception of training by providing an ever-changing training stimulus. To our knowledge, no study has incorporated periodized HIIT in an attempt to augment adaptations to training. In the present study, we incorporated 10 sessions of low-volume HIIT to rapidly and significantly enhance $\dot{V}O_{2max}$, as widely reported (4,13), and subsequently randomized participants to 1 of 3 HIIT regimes consisting of an additional 10 sessions to examine if periodization of subsequent training continues to augment $\dot{V}O_{2max}$. Furthermore, CO was assessed throughout training to identify if improved central O_2 delivery is responsible for potential increases in $\dot{V}O_{2max}$ seen in response to HIIT.

METHODS

Participants. In response to word-of-mouth and electronic bulletins posted between January 2014 and February 2016, 192 individuals initially contacted the primary investigator to potentially participate in the study. Of these prospective participants, 77 enrolled in the study, 43 initiated HIIT and 34 nonexercising controls. Physical characteristics of participants who completed all requirements of the study are shown in Table 1. Participant ethnicity included whites (n = 41), Hispanic (n = 24), African-American (n = 2), Asian/Filipino (n = 7), and Middle Eastern (n = 3) and reflected the ethnic composition of the University community. All performed a minimum of 150 min·wk^{-1} of physical activity including resistance training, noncompetitive sport, aerobic exercise, surfing, and so on, which was confirmed with a questionnaire (28). Inclusion criteria included healthy, body mass index less than 30 kg·m^{-2}, weight stable in the last 12 months, nonsmoker, as well as lack of knee pain and medication/supplement use which may modify study outcomes. All women enrolled in the study were eumenorrheic. Written informed consent was obtained from all participants before initiating the study, whose procedures were approved by the university institutional review board and were conducted in accordance with the Declaration of Helsinki (1964). During the study, four withdrew from the experimental group due to lack of time (n = 2), unrelated injury (n = 1), and failure to adhere to inclusion criteria (n = 1), and two controls did not complete the follow-up trial due to cessation of habitual exercise outside of the study.

TABLE 1. Baseline participant characteristics (mean ± SD).

Parameter	HIIT + SIT	HIIT + $HIIT_{HI}$	HIIT + PER	CON
Age (yr)	22.5 ± 5.8	21.9 ± 1.9	23.1 ± 5.5	25.8 ± 5.8*
Gender (n = M/F)	7/7	7/6	5/7	15/17
Height (cm)	172.0 ± 7.8	174.4 ± 9.6	170.9 ± 9.9	172.2 ± 11.3
Mass (kg)	68.5 ± 10.3	69.6 ± 11.4	68.2 ± 7.0	72.2 ± 14.3
$\dot{V}O_{2max}$ (mL·kg^{-1}·min^{-1})	39.6 ± 6.8	41.1 ± 4.9	39.5 ± 5.3	40.5 ± 4.9
PPO (W)	226.9 ± 36.6	240.0 ± 34.4	229.2 ± 51.7	243.0 ± 40.9
PA (h·wk^{-1})	6.7 ± 2.3	5.5 ± 2.1	6.0 ± 2.4	6.2 ± 2.4

PPO from progressive exercise test.
M, male; F, female; PA, habitual physical activity.
*P < 0.05 compared to all HIIT groups.

Experimental design. At baseline, $\dot{V}O_{2max}$ and hemodynamics were measured in all individuals participating in the study. Before this trial and all subsequent assessments, participants completed an overnight fast (12 h) and abstained from medication use for 12 h and physical activity for 48 h. Participants subsequently completed 20 sessions of periodized HIIT with time of day maintained within participants. The posttraining assessment was performed 48–120 h after the final session of HIIT. Participants were advised to maintain their dietary intake and physical activity during the study.

Assessment of body composition. Initially, height and body mass were determined using a balance beam scale with stadiometer (Health-o-Meter; Creative Health Products, Ann Arbor, MI). Subsequently, subcutaneous fat was recorded using a metal skinfold caliper (Lange, Santa Cruz, CA) at the chest, abdominal, and thigh (men) and triceps, suprailiac, and thigh (women) (19,20) in rotational order following standardized procedures (18). If scores differed by more than 10%, a third score was obtained and an average value was calculated from the two scores meeting this criterion. Gender- and ethnicity-specific equations were used to calculate body density and percent body fat.

Assessment of $\dot{V}O_{2max}$. After 5 min of resting gas exchange data were obtained to yield resting values of $\dot{V}O_2$ (3–4 mL·kg^{-1}·min^{-1}), participants initiated progressive cycling on an electrically braked cycle ergometer (Velotron DynaFit Pro, RacerMate, Seattle, WA) consisting of 7 min at

30 (women) or 40 W (men) followed by 20 W increases in power output every 3 min until mean RER was greater than 1.0 for an entire stage, after which power output was increased by 20 $W \cdot min^{-1}$ until voluntary exhaustion (cadence < 50 rpm) and attainment of maximal oxygen uptake ($\dot{V}O_{2max}$) and peak power output (PPO). Maximal oxygen uptake was determined as the mean of the two highest values attained during exercise from any 30-s period. In our laboratory, coefficient of variation and typical error for $\dot{V}O_{2max}$ determined from this progressive protocol are equal to 3.0% and 1.1 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively. Attainment of $\dot{V}O_{2max}$ was confirmed with constant load cycling to exhaustion at 110% PPO performed 10 min after the initial bout (26). During exercise, gas exchange data ($\dot{V}O_2$, ventilation (V_E), carbon dioxide production ($\dot{V}CO_2$), and RER) were obtained every 15 s using a metabolic cart (ParvoMedics TrueOne, Sandy, UT) which was calibrated before exercise according to the manufacturer's recommendation. Gas exchange data were visually inspected, and any values differing by more than two standard deviations from the mean value were removed from subsequent analysis. PPO from this trial was used to set intensities for resultant HIIT. $\dot{V}O_{2max}$ assessment was repeated after 10 and 20 sessions of training, respectively.

Assessment of hemodynamic function. An impedance cardiograph device (Physioflow Enduro, Manatec, Strasbourg, France) was used to evaluate hemodynamic function. This method has been described in detail elsewhere (9,31) and is valid and reliable at rest and during exercise up to $\dot{V}O_{2max}$. This method detects changes in transthoracic impedance during phases of the cardiac cycle to calculate SV, which is multiplied by HR to estimate CO.

Participants entered the laboratory and were required to sit quietly for approximately 5 min. An alcohol swab was used to clean the neck, right chest, trunk at V6, and spine, and then an electrode gel (NuPrep; Weaver and Company, Aurora, CO) was rubbed into these areas, and the skin was further cleaned with a paper towel. Two sets of electrodes (Skintact ECG electrodes; Leonhard Lang GmbH, Innsbruck, Austria), one electrode transmitting and the other sensing, were applied above the supraclavicular fossa at the left base of the neck and at the height of the xiphoid on the spine. Another pair of electrodes (one placed on the right chest and another at V6) was used to monitor the ECG trace. Once applied, these leads were taped to the skin to minimize movement. The participant was seated on the cycle ergometer for 2 min, blood pressure was manually recorded twice at the antecubital space with a stethoscope and sphygmomanometer (Omron Health Care Inc., Vernon Hills, IL), and they were told not to talk and remain motionless. Then, the device was calibrated after a 30-beat procedure using the baseline blood pressure value, which was averaged.

Once calibration was completed, resting values for HR, SV, and CO were obtained after which the warm-up began. During exercise, HR, SV, and CO values were determined every 15 s. Maximal values of HR, SV, and CO were identified as the highest values at any point during exercise. Maximal arteriovenous oxygen difference (a-vDO_2) was calculated as the quotient of $\dot{V}O_{2max}$ ($mL \cdot min^{-1}$)/CO_{max} ($L \cdot min^{-1}$) and expressed in $mL \cdot dL^{-1}$.

Incorporation of HIIT. At least 48 h after baseline testing, all participants initiated 10 sessions of progressive low-volume HIIT on the same electrically braked cycle ergometer consisting of 8–10 min of training time per day (Table 2). T2
Participants were randomized using a Latin Squares design (12) to one of three regimes for the remaining 10 sessions. SIT (HIIT + SIT) consisted of 8–12 "all-out" sprints (4–6 min training duration per day) during which participants were required to pedal maximally. High-volume interval training (HIIT + $HIIT_{HI}$) required repeated 2.5-min bouts of cycling with 60 s recovery, leading to training duration equal to 12.5–17.5 $min \cdot d^{-1}$. Lastly, the periodized regime (HIIT + PER) consisted of 3 sessions of high-volume HIIT, 3 sessions of SIT, and 4 sessions of low-volume HIIT during which training duration varied from 5 to 15 min per session (Table 2).

Before all sessions, a 5-min warm-up at 20% PPO was completed, and active recovery was performed at this work rate between bouts. During the initial 10 sessions, overload was incorporated after every three sessions by incorporating an additional one to two sessions and increasing work rate by 10% PPO. Participants were given additional recovery when requested. All exercise training was supervised, performed in a climate-controlled laboratory (temperature and relative humidity = 19°C–22°C and 30%–50%, respectively), and held at the same time of day (±1 h) within participants. During HIIT, HR was determined using telemetry (Polar Electro, Woodbury, NY) to identify peak (mean score of all values recorded at the end of each bout) and session HR, and strong verbal encouragement was provided. Average work rate (W) which included the HIIT bouts as well as recovery was also recorded using the Velotron software. Training was performed 3 $d \cdot wk^{-1}$ and at least 24 h was provided between sessions,

TABLE 2. Description of 20 sessions of HIIT performed in the study.

Training Regimes	No. Bouts	Bout Duration (s)	Intensity (% PPO)	Recovery (s)	Session Duration (min)
HIIT + SIT					
1–3[a]	8	60	90	75	23.0
4–6	9	60	100	75	25.25
7–10	10	60	110	75	27.5
11–13	8	30	130	120	25.0
14–16	10	30	140	120	30.0
17–20	12	30	150	120	35.0
HIIT + $HIIT_{HI}$					
1–3	8	60	90	75	23.0
4–6	9	60	100	75	25.25
7–10	10	60	110	75	27.5
11–13	5	150	70	60	22.5
14–16	6	150	75	60	26.0
17–20	7	150	80	60	29.5
HIIT + PER					
1–3	8	60	90	75	23.0
4–6	9	60	100	75	25.25
7–10	10	60	110	75	27.5
11–13	6	150	70	60	26.0
14–16	10	30	140	120	30.0
17–20	8	60	100	75	23.0

[a]Specific days of HIIT sessions. $\dot{V}O_{2max}$ was determined after session 10 with PPO from this bout used to set intensity of sessions 11–20.

which were held on Monday/Wednesday/Friday, Monday/Tuesday/Thursday, and Tuesday/Thursday/Friday.

Habitual physical activity. Participants were advised to maintain their habitual physical activity during the study. They were given a training log upon study initiation and were required to denote daily physical activity in this log, which was submitted at study termination.

Data analyses. Data are expressed as mean ± SD and were analyzed using SPSS Version 22.0 (Chicago, IL). The Shapiro-Wilks test was used to assess normality. One-way ANOVA was performed to identify differences in demographic and physiological variables across all groups at baseline. Repeated three-way ANOVA (time = pre versus post, gender, and regimen = HIIT + SIT, HIIT + $HIIT_{HI}$, HIIT + PER, and CON) with repeated-measures was performed to identify differences in $\dot{V}O_{2max}$ and maximal SV, CO, and a-vDO_2 during the study. If a significant F ratio was obtained, Tukey's *post hoc* test was used to identify differences between means. Partial eta-squared (η^2_p) and Cohen's *d* were used as an estimate of effect size. Pearson product moment correlation was used to determine the association between variables. Ninety-five percent confidence intervals (95% CI) are reported for the change in $\dot{V}O_{2max}$ in response to HIIT. At a statistical power equal to 0.80 and expected difference in absolute $\dot{V}O_{2max}$ from pretraining to posttraining equal to 15%, the minimum total sample size was determined to be 20 individuals (14). Using similar parameters other than a difference in $\dot{V}O_{2max}$ response across regimens equal to 5%, a minimum of nine participants per regimen was determined which is similar to a previous study (25). Statistical significance was established as $P < 0.05$.

RESULTS

Training fidelity. Training compliance was equal to 778 of 780 (99.7% of all sessions). To verify the intensity of HIIT (36), average peak HR in response to training is shown in Figure 1. HR increased during training ($P < 0.001$, $\eta^2_p = 0.25$) but there was no time–regimen interaction ($P = 0.41$). HR significantly increased ($P < 0.05$) from sessions 1–3, 4–6, and 7–10, when it peaked at 91.0% ± 2.6% of baseline HR_{max}. HR declined ($P < 0.05$) in sessions 11–13 and was similar during the last seven sessions of training compared with values from sessions 1–10. Average work rate during HIIT increased across time ($P < 0.001$, $\eta^2_p = 0.36$) and a significant time–regimen interaction ($P < 0.001$, $\eta^2_p = 0.43$) was shown. During the first 10 sessions of HIIT, average work rate increased every three sessions of HIIT although it was similar ($P > 0.05$) across regimes (115–130 W). During sessions 11–20, participants in HIIT + $HIIT_{HI}$ were exercising at significantly higher ($P < 0.05$) average session power outputs (127–143 W equal to 52.9%–59.6% PPO) versus HIIT + SIT (107–112 W equal to 47.3%–49.6% PPO) and HIIT + PER (109–124 W equal to 47.6%–54.6% PPO). Body mass did not change during the study (data not reported).

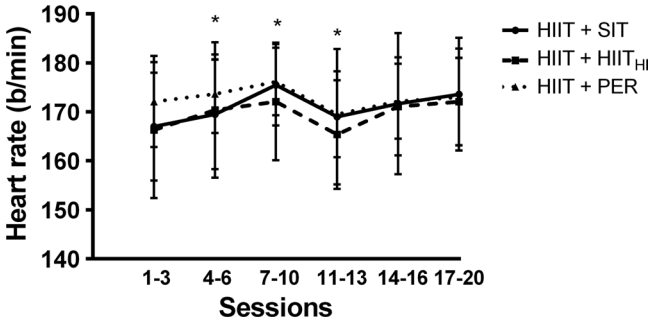

FIGURE 1—Change in HR (mean ± SD) in response to 20 sessions of HIIT. *$P < 0.05$ versus sessions. HIIT is separated into phase 1 (sessions 1–3), phase 2 (sessions 4–6), phase 3 (sessions 7–10), phase 4 (sessions 11–13), phase 5 (sessions 14–16), and phase 6 (sessions 17–20).

Differences in $\dot{V}O_{2max}$, gas exchange variables, and PPO in response to HIIT. At baseline, there were no differences ($P > 0.05$) in any variable between groups, although differences ($P < 0.05$) between men and women occurred in $\dot{V}O_{2max}$, $\dot{V}CO_2$, PPO, and V_E. Relative and absolute $\dot{V}O_{2max}$ showed a significant main effect for time ($P < 0.001$, $\eta^2_p = 0.62$) as well as time–regimen interaction ($P < 0.001$, $\eta^2_p = 0.39$ and $P < 0.001$, $P < 0.001$, $\eta^2_p = 0.51$), as increases in $\dot{V}O_{2max}$ were shown in all HIIT regimes but not in CON (Table 3). Individual data for change in absolute $\dot{V}O_{2max}$ across all HIIT regimes are shown in Figure 2.

From baseline to posttraining, mean percent change in $\dot{V}O_{2max}$ was equal to 10.9 ± 7.6 (95% CI, 6.5%–15.3%), 8.9% ± 6.1% (95% CI, 5.2%–12.6%), and 12.3% ± 6.0% (95% CI, 8.3%–16.3%) for HIIT + SIT, HIIT + $HIIT_{HI}$, and HIIT + PER, respectively, which were all higher ($P < 0.001$) than the change in CON (−0.4% ± 6.6%; 95% CI, −2.3% to 2.0%). $\dot{V}O_{2max}$ determined from the verification test was also increased ($P < 0.001$) in response to HIIT + SIT (39.3 ± 5.5 $mL \cdot kg^{-1} \cdot min^{-1}$ vs 42.3 ± 6.4 $mL \cdot kg^{-1} \cdot min^{-1}$, + 7.7%), HIIT + $HIIT_{HI}$ (40.2 ± 4.4 $mL \cdot kg^{-1} \cdot min^{-1}$ vs 43.7 ± $mL \cdot kg^{-1} \cdot min^{-1}$, + 9.0%), and HIIT + PER (39.1 ± 4.9 $mL \cdot kg^{-1} \cdot min^{-1}$ vs 42.2 ± 5.2 $mL \cdot kg^{-1} \cdot min^{-1}$, + 8.0%), with no change demonstrated in CON (39.7 ± 5.0 $mL \cdot kg^{-1} \cdot min^{-1}$ vs 39.0 ± 5.4 $mL \cdot kg^{-1} \cdot min^{-1}$).

Post hoc analyses showed that $\dot{V}O_{2max}$ in HIIT + $HIIT_{HI}$ increased ($P < 0.05$) at 10 sessions of training (Cohen's $d = 1.68$) versus baseline after which it was unchanged (Cohen's $d = 0.50$) (Table 3). In contrast, the other regimes showed significant and sustained increases in $\dot{V}O_{2max}$. In the first 10 sessions of HIIT + SIT, $\dot{V}O_{2max}$ increased by 6.1% (Cohen's $d = 1.49$) and by an additional 3.5% in the last 10 sessions of training (Cohen's $d = 0.87$). Similarly, $\dot{V}O_{2max}$ increased ($P < 0.05$) by 6.3% in the initial 10 sessions (Cohen's $d = 1.68$) and subsequently ($P < 0.05$) by 5% (Cohen's $d = 1.31$) in the latter 10 sessions of HIIT + PER.

Significant effects of time ($P < 0.001$) and a time–regimen interaction ($P < 0.001$, $\eta^2_p = 0.35$) were shown for V_E (data not reported). Similar differences across time and a time–regimen interaction were found for $\dot{V}CO_2$ ($P < 0.001$, data not reported). There was no change in RER in response to

TABLE 3. Maximal exercise responses (mean ± SD) for power output, $\dot{V}O_{2max}$, HR, SV, CO, and a-vDO_2 in response to 10 and 20 sessions of HIIT.

Regime	PPO (W)	$\dot{V}O_{2max}$ (mL·kg^{-1}·min^{-1})	$\dot{V}O_{2max}$ (L·min^{-1})	HR (bpm)	SV (mL)	CO (L·min^{-1})	a-vDO_2 (mL·dL^{-1})
HIIT + SIT							
Pre	230 ± 35	39.8 ± 7.3	2.7 ± 0.6	186 ± 9	104 ± 16	19.2 ± 2.6	14.3 ± 2.1
Mid	242 ± 36*	42.2 ± 6.8*	2.9 ± 0.5*	187 ± 9	108 ± 12	20.2 ± 2.1*	14.5 ± 2.0
Post	252 ± 39*,**	43.6 ± 6.1*,**	3.0 ± 0.5*,**	189 ± 7	113 ± 10*,**	21.3 ± 1.7*,**	14.1 ± 1.9
HIIT + $HIIT_{HI}$							
Pre	240 ± 34	41.1 ± 4.9	2.9 ± 0.6	185 ± 9	112 ± 19	20.6 ± 3.0	14.1 ± 1.6
Mid	262 ± 43*	43.8 ± 6.0*	3.1 ± 0.7*	189 ± 9	117 ± 16*	21.8 ± 2.9*	14.4 ± 1.7
Post	265 ± 47*	44.6 ± 7.0*	3.1 ± 0.8*	186 ± 8	120 ± 18*	22.2 ± 3.1*	14.3 ± 2.0
HIIT + PER							
Pre	229 ± 56	39.5 ± 5.6	2.7 ± 0.8	189 ± 8	107 ± 19	20.3 ± 3.7	13.3 ± 2.5
Mid	245 ± 50*	42.0 ± 5.1*	2.8 ± 0.8*	186 ± 11	111 ± 24	21.1 ± 4.7	13.6 ± 2.7
Post	249 ± 53*	44.1 ± 5.4*,**	3.0 ± 0.9*,**	189 ± 7	115 ± 21*	21.8 ± 4.4*	13.8 ± 2.4
CON							
Pre	243 ± 40	40.4 ± 5.1	2.9 ± 0.6	184 ± 10	112 ± 16	20.6 ± 3.0	14.0 ± 2.0
Post	244 ± 41	40.2 ± 5.5	2.9 ± 0.6	184 ± 9	112 ± 15	20.5 ± 2.9	14.0 ± 1.9

*$P < 0.05$ from pretraining value.
**$P < 0.05$ from value after session 10.

HIIT ($P = 0.79$). PPO increased ($P < 0.001$) in all HIIT regimes by 20–25 W with no change seen in CON (Table 3).

Relationship between baseline $\dot{V}O_{2max}$ and change in $\dot{V}O_{2max}$ in response to HIIT. There was a significant inverse association ($r = -0.36$, $P = 0.01$) between baseline $\dot{V}O_{2max}$ and the training-mediated change in $\dot{V}O_{2max}$, suggesting that a low baseline $\dot{V}O_{2max}$ is related to a greater change in $\dot{V}O_{2max}$ with training.

Differences in hemodynamic variables in response to HIIT. At baseline, there were no differences in any variable between groups, although men showed higher ($P < 0.05$) SV, CO, and a-vDO_2 versus women. Data were combined across men and women because there was no time–gender interaction. HR did not change in response to training ($P = 0.88$). Cardiac output increased across time ($P < 0.001$, $\eta^2_p = 0.36$) and a significant time–regimen interaction occurred ($P = 0.002$, $\eta^2_p = 0.25$) in that it increased in response to training and did not change in CON (Table 3). *Post hoc* analyses showed that CO in HIIT + SIT increased ($P < 0.05$) by 5.3% (Cohen's $d = 0.91$) and then an additional 5.4% (Cohen's $d = 1.0$) after 10 and 20 sessions of training. Nevertheless, in HIIT + $HIIT_{HI}$, CO increased by 6% from 10 sessions of training (Cohen's $d = 1.1$) after which it was maintained (Cohen's $d = 0.37$). In HIIT + PER, it was similar after 10 sessions (Cohen's $d = 0.73$) and was higher ($P < 0.05$) after 20 sessions of training (7.5%, Cohen's $d = 1.37$) (Table 3). Compared with baseline, SV increased ($P < 0.05$ between session 20 and preceding values) in HIIT + SIT and increased ($P < 0.05$) after 10 and 20 sessions of training for HIIT + $HIIT_{HI}$ and after 20 sessions for HIIT + PER, respectively. SV did not change in CON. Individual changes in SV and CO are shown in Figure 3. Results showed no difference in a-vDO_2 in response to training ($P = 0.36$) (Table 3).

DISCUSSION

The present study is the first to examine the effects of 6 wk of HIIT characterized by a modification of intensity, duration, and overall structure *during* training on changes in cardiac output and $\dot{V}O_{2max}$ in active men and women. This approach models what exercisers frequently do on a day-to-day basis yet is rarely used in research. In all participants, the initial 10 sessions of HIIT led to similar and significant increases in $\dot{V}O_{2max}$, but the subsequent 10 sessions varying in intensity and volume did not lead to continued increases in $\dot{V}O_{2max}$ in all regimes. Our findings refute previous studies using both SIT and HIIT (21,24) as we show that significant increases in SV and CO, but not a-vDO_2, occur in response to HIIT which are consequent with increases in $\dot{V}O_{2max}$. The overall magnitude of increase in $\dot{V}O_{2max}$ was similar across regimes, although the time course of change differed across groups, and the increase in $\dot{V}O_{2max}$ was inversely related to baseline cardiorespiratory fitness.

Our data showing similar increases in $\dot{V}O_{2max}$ across different HIIT regimes varying in intensity and structure support data from recent studies in habitually active individuals. Similar increases in $\dot{V}O_{2max}$ were demonstrated in men performing 4 wk of repeated 15 or 30 s bouts of SIT (38) as well as in men and women performing six sessions of repeated 30 or 10 s Wingate tests (17). However, in sedentary individuals (age and $\dot{V}O_{2max}$ = 41 yr and 33.9 mL·kg^{-1}·min^{-1}), 6 wk of high-volume HIIT consisting of repeated 4 min bouts led to significantly greater increases in $\dot{V}O_{2max}$ compared with low-volume HIIT (ten 1 min bouts) or MICT (6). These data, however, are opposed by similar increases in $\dot{V}O_{2max}$ found in sedentary, young, nonobese women who completed 12 wk of moderate or more intense HIIT (4) as well as in sedentary men completing HIIT (repeated 3 min efforts at 85%–90% $\dot{V}O_{2max}$) and SIT (repeated 30 s efforts at 120% $\dot{V}O_{2max}$) (25). Discrepancies between studies include the markedly higher training volume used in the Baekkerud et al. (6) study, which has been identified (5) to elicit more substantial increases in $\dot{V}O_{2max}$ in response to HIIT, as well as heterogeneity in $\dot{V}O_{2max}$ response to HIIT (3). This individual response to HIIT (Fig. 2) is also exhibited by 43% of participants showing greater than the mean change in $\dot{V}O_{2max}$ (10.7%) in response to HIIT, 46% showing less than this change, and 10% showing no

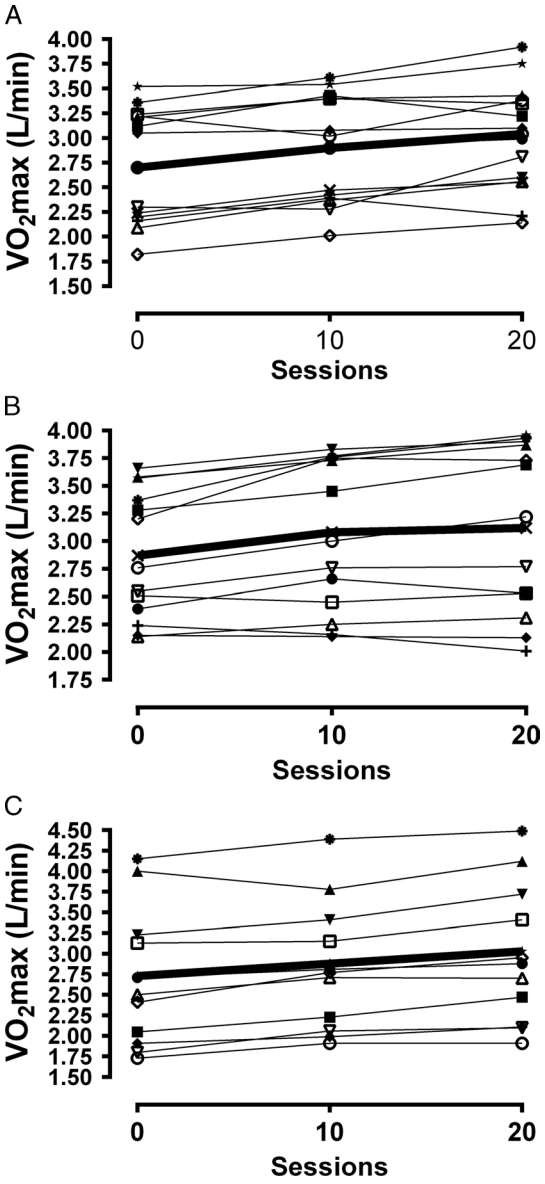

FIGURE 2—Individual changes in $\dot{V}O_{2max}$ ($L \cdot min^{-1}$) in response to 20 sessions of (A) HIIT + SIT, (B) HIIT + $HIIT_{HI}$, and (C) HIIT + PER. 0 = pre-training value, 10 = value recorded after initial 10 training sessions, and 20 = posttraining value. Thin lines are individual participants and thick line is the mean change in $\dot{V}O_{2max}$ across participants within group.

change (less than the typical error for $\dot{V}O_{2max}$). Increases in $\dot{V}O_{2max}$ are inversely associated with baseline $\dot{V}O_{2max}$ after HIIT (3) or SIT (16), although this relationship typically does not occur after prolonged MICT in untrained participants (7,33). Our data also showed that in 20 participants with $\dot{V}O_{2max}$ below 40 $mL \cdot kg^{-1} \cdot min^{-1}$, the mean value of our sample, 55% exhibited greater than average increases in $\dot{V}O_{2max}$ in response to HIIT, which suggests that a portion of the training-induced increase in cardiorespiratory fitness is associated with baseline values.

An intriguing finding of the present study is that an additional 10 sessions of $HIIT_{HI}$ did not further augment $\dot{V}O_{2max}$ compared with SIT or PER (Table 3). This is unexpected considering that this regime is characterized by higher training duration (12.5–17.5 min per session) and mean power output (see Results) which seem to elicit greater increases in $\dot{V}O_{2max}$ (5,6) compared with SIT (4–6 min per session) or PER (5–15 min per session). In contrast, Hazell et al. (17) denoted that it is the peak intensity achieved during SIT that mediates the resultant change in $\dot{V}O_{2max}$ rather than training volume. Similar findings from highly trained cyclists show that completion of repeated 30-s intervals allowing greater time above 90% $\dot{V}O_{2max}$ led to greater increases in $\dot{V}O_{2max}$ compared with longer intervals (5 min) at lower intensity (32). It is plausible that after the initial 10 sessions of training, the novel use of SIT and PER, which dramatically modifies the HIIT regime by including brief efforts at supramaximal intensities, promotes continued improvements in $\dot{V}O_{2max}$ which do not happen when primarily training volume but not intensity was enhanced in $HIIT_{HI}$.

Our findings show a significant increase in maximal CO in response to all HIIT regimes ranging from 1.5 to 2.1 $L \cdot min^{-1}$ (8%–11%) (Table 3). However, the time course of increase in CO varied as it steadily increased in HIIT + SIT, plateaued after 10 sessions of training in HIIT + $HIIT_{HI}$, and did not initially change in HIIT + PER after which it increased. Although speculative, the slightly different SV response in each group may have led to this result. In untrained adults (11) (age and $\dot{V}O_{2max}$ = 47 yr and 26 $mL \cdot kg^{-1} \cdot min^{-1}$), CO_{max} was increased by 12% in response to 24 sessions of HIIT. However, in this study, HIIT consisted of repeated 5-min bouts including 4 min at work rate coincident with lactate threshold and 1 min at 90% PPO, so this regimen was higher volume and lower intensity compared with our HIIT protocols. In untrained men (age and $\dot{V}O_{2max}$ = 30 yr and 38 $mL \cdot kg^{-1} \cdot min^{-1}$) (37), 36 sessions of HIIT (repeated 2 min efforts at 90% $\dot{V}O_{2max}$) led to significant increases in $\dot{V}O_{2max}$ which was associated with increased CO_{max} but not a-vDO_2. In these studies, an increase ($P < 0.05$) in maximal SV led to the increase in CO, supporting our results (Table 3), although maximal HR was also increased in the Daussin et al. (9) study. One likely mechanism explaining this increase in SV is a significant increase in plasma volume (PV) via HIIT, which is seen after only three and six sessions of training (37) and elicits an increase in blood volume (BV) and thus SV. Only six sessions of HIIT at 95%–100% $\dot{V}O_{2max}$ also promoted significant increases in PV (10.8%) leading to an 11% increase in $\dot{V}O_{2max}$ in untrained men (age and $\dot{V}O_{2max}$ = 25 yr and 39 $mL \cdot kg^{-1} \cdot min^{-1}$) (13). In young active adults ($\dot{V}O_{2max}$ = 46.8 $mL \cdot kg^{-1} \cdot min^{-1}$, completing 6 wk of maximal treadmill-based SIT, there was no change in maximal CO and the increase in $\dot{V}O_{2max}$ was due to a greater a-vDO_2 (24). Similarly, six sessions of HIIT at 100% PPO did not change maximal PV, BV, or CO despite a significant 8% increase in $\dot{V}O_{2max}$ (21), which led the authors to conclude that peripheral adaptations increase $\dot{V}O_{2max}$ after short-term HIIT. One characteristic of both studies is that participants' $\dot{V}O_{2max}$ was 15%–20% higher compared with studies in which CO was enhanced with HIIT (11,37). It may

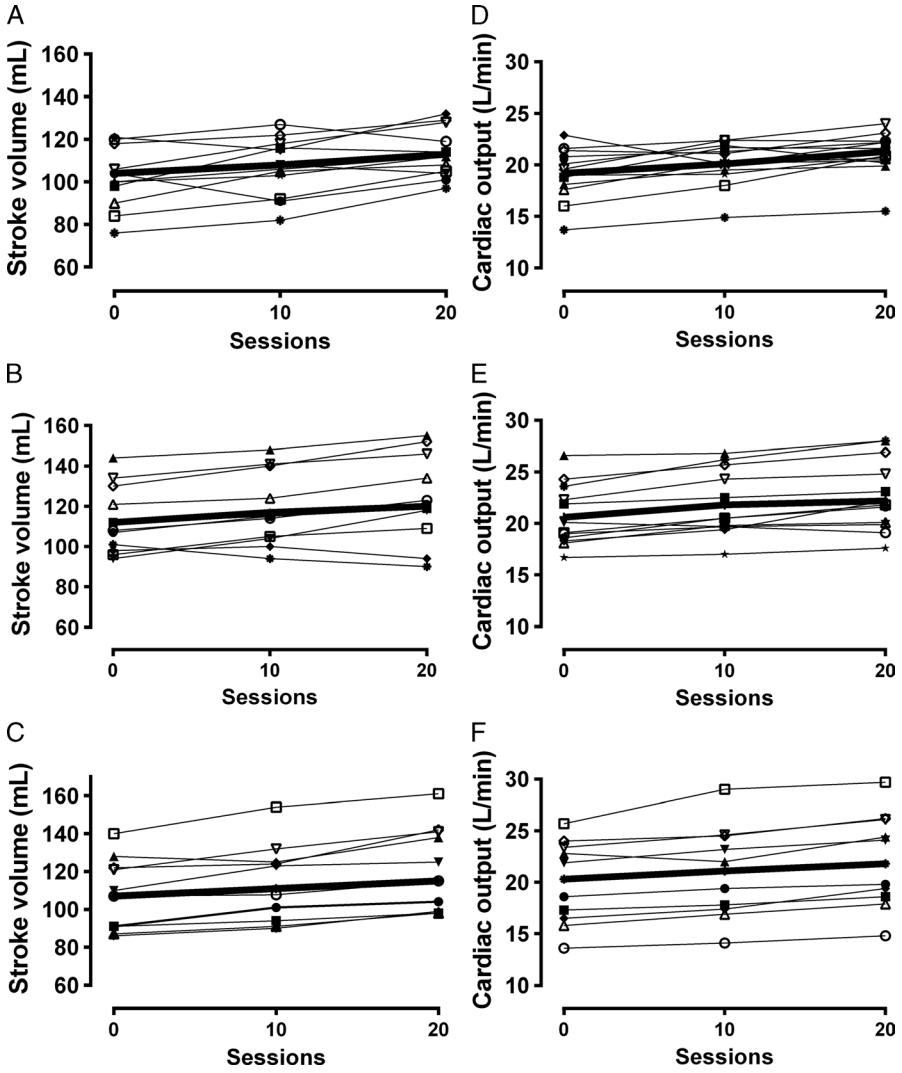

FIGURE 3—Individual changes in SV (mL) in response to 20 sessions of (A) HIIT + SIT, (B) HIIT + $HIIT_{HI}$, and (C) HIIT + PER. 0 = pre-training value, 10 = value recorded after initial 10 training sessions, and 20 = posttraining value. Thin lines are individual participants and thick line is the mean change in SV across participants within group. Individual changes in cardiac output ($L \cdot min^{-1}$) in response to 20 sessions of (D) HIIT + SIT, (E) HIIT + $HIIT_{HI}$, and (F) HIIT + PER. 0 = pretraining value, 10 = value recorded after initial 10 training sessions, and 20 = posttraining value. Individual participants (*thin lines*). Mean change in cardiac output across participants within group (*thick line*).

be that persons with lower baseline $\dot{V}O_{2max}$ are more apt to experience hypervolemia soon after initiation of HIIT leading to rapid increases in BV, SV, and thus CO. In addition, it is plausible that participants with higher $\dot{V}O_{2max}$ such as used in previous studies (21,24) require higher training volumes or duration of HIIT/SIT to significantly increase SV and CO. This discrepancy requires additional study to ascertain if there is a specific $\dot{V}O_{2max}$ value or training dose for which central rather than peripheral adaptations mediate the increased $\dot{V}O_{2max}$ demonstrated in response to HIIT.

In the current study, we used thoracic impedance to estimate SV and CO during progressive exercise to $\dot{V}O_{2max}$. This technique was also used in another study (11), showing significant increases in SV and CO in response to HIIT. Previous data show an intraclass correlation coefficient equal to 0.95 for repeated determinations of maximal CO obtained in habitually active men and women (2), and data from our control participants showed low typical error equal to 0.5 $L \cdot min^{-1}$ for two determinations of CO conducted 6 wk apart. In a previous study (23), this technique was able to detect small differences in CO across different protocols leading to $\dot{V}O_{2max}$.

Our study is strengthened by precise allocation of intensity for all training sessions as well as high compliance to training. In addition, the fidelity of HIIT was demonstrated (Fig. 1) by showing that all regimes elicited intensities approaching 90% HR_{max}. Results from the verification test and maximal HR/RER data suggest that participants did exhibit "true" $\dot{V}O_{2max}$ and that our reported increases in $\dot{V}O_{2max}$ are repeatable and not due to random error. We also recruited both men and women to apply our data to a broader population of active adults. Nevertheless, there is some evidence (15) that women

may respond differently to HIIT versus men, although this is not a universal finding (1). In addition, accelerometry was not used to monitor habitual physical activity completed outside the study, although data from exercise training logs showed no difference in hours per week of physical activity (data not reported). We did not include a group of individuals who completed endurance training, so we cannot state if any of the HIIT regimes elicit superior increases in $\dot{V}O_{2max}$ compared with traditional MICT. Although control participants had similar physical activity, body fat, and $\dot{V}O_{2max}$ versus those who completed HIIT, they were recruited by convenience rather than through random assignment. Assessments of $\dot{V}O_{2max}$ and CO were made 2–5 d after the previous HIIT session, so slight reductions in PV could have occurred (10), leading to small underestimations in CO and consequently $\dot{V}O_{2max}$. As an equal number of participants in each regimen completed their assessments 4–5 d after their final HIIT session, the effect of this discrepancy on our results is likely small.

CONCLUSIONS

Overall, in young individuals with average cardiorespiratory fitness, 20 sessions of periodized HIIT led to significant increases in $\dot{V}O_{2max}$ which were attendant with increases in maximal SV and CO. Data suggest that increases in $\dot{V}O_{2max}$ as a result of HIIT are mediated by improvements in central O_2 delivery rather than peripheral adaptations as frequently reported (8,21,24).

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