Effect of Sprint Interval Exercise on Postexercise Metabolism and Blood Pressure in Adolescents

Stephen F. Burns, Hnin Hnin Oo, and Anh Thanh Thuy Tran

The current study examined the effect of sprint interval exercise on postexercise oxygen consumption, respiratory-exchange ratio (RER), substrate oxidation, and blood pressure in adolescents. Participants were 10 normal-weight healthy youth (7 female), age 15–18 years. After overnight fasts, each participant undertook 2 trials in a random balanced order: (a) two 30-s bouts of sprint interval exercise on a cycle ergometer and (b) rested in the laboratory for an equivalent period. Time-matched measurements of oxygen consumption, RER, and blood pressure were made 90 min into recovery, and substrate oxidation were calculated over the 90 min. Total postexercise oxygen uptake was significantly higher in the exercise than control trial over the 90 min (mean [SD]: control 20.0 [6.0] L, exercise 24.8 [9.8] L; p = .030). After exercise, RER was elevated above control but then fell rapidly and was lower than control 30–60 min postexercise, and fat oxidation was significantly higher in the exercise than control trial 45–60 min postexercise. However, total fat oxidation did not differ between trials (control 4.5 [2.5] g, exercise 5.4 [2.7] g; p = .247). Post hoc tests revealed that systolic blood pressure was significantly lower in the exercise than control at 90 min postexercise (control 104 [10] mm Hg, exercise 99 [10] mm Hg; p < .05). These data indicate that acute sprint interval exercise leads to short-term increases in oxygen uptake and reduced blood pressure in youth. The authors suggest that health outcomes in response to sprint interval training be examined in children.

**Keywords:** postexercise oxygen uptake, fat metabolism, respiratory-exchange ratio, youth, intermittent exercise, vascular health

Exercise is one strategy to prevent overweight and obesity in children and adolescents (Flodmark, Marcus, & Britton, 2006; Kimm et al., 2005). Moreover, increased physical activity and fitness in childhood are associated with decreased cardiovascular and metabolic risk factors, including blood pressure (Andersen et al., 2006; Schmidt, Walkuski, & Stensel, 1998). Cohort studies in the United Kingdom and United States, however, have demonstrated that few children and adolescents achieve the 60 min of moderate to vigorous physical activity per day (Nader, Bradley, Houts, McRitchie, & O’Brien, 2008; Riddoch et al., 2007) recommended by expert panels. Moreover, physical activity, assessed by questionnaire (Kimm et al., 2002) or accelerometry (Nader et al., 2008), has been shown to decline substantially during adolescence. Many children and adolescents (Armstrong, Balding, Gentle, & Kirby, 1990; Armstrong & Welsman, 2006) undertake activity that is intermittent in nature and often lasts less than 5–10 min. Given that so few children meet the current physical activity guidelines, shorter duration exercise, which can improve metabolic and cardiovascular health but which also corresponds more closely to childhood patterns of activity, needs to be examined so that a greater variety of exercise options can be promoted.

Recent studies have demonstrated the benefits of high-intensity sprint interval training in normal-weight and overweight adults (Burgomaster et al., 2008; Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Rakobowchuk, Stuckey, Millar, Gurr, & Macdonald, 2009; Rakobowchuk et al., 2008; Whyte, Gill, & Cathcart, 2010). This type of exercise is characterized by repeated all-out sprint efforts, often performed on a cycle ergometer, of 30 s in duration with recovery periods of 4 min. Only 2–3 weeks of this type of training has been shown to increase peak oxygen uptake and endurance capacity in recreationally trained individuals (Burgomaster et al., 2008; Burgomaster et al., 2005). Moreover, a number of health-related indicators have shown positive responses with sprint interval training in both normal-weight and overweight individuals. One study (Rakobowchuk et al., 2008) compared 6 weeks of sprint interval training with endurance training in healthy young men and women and found that popliteal artery distensibility was improved to the same degree in both groups. Two weeks of sprint interval training in 10 sedentary overweight or obese adults with a mean age of 32 years significantly increased fat oxidation and decreased carbohydrate oxidation, respiratory-exchange ratio (RER), blood pressure, and waist and hip circumference (Whyte et al., 2010). The changes in blood pressure, RER, and substrate oxidation all lasted 24 but not 72 hr postintervention, suggesting some acute response to the last bout of training rather than a chronic effect of sprint interval training. This observation has had some confirmation in two recent adult studies that found that sprint interval exercise acutely reduces peripheral artery stiffness (Rakobowchuk et al., 2009) and blood.

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pressure (Rossow et al., 2010) to a degree similar to that of steady-state aerobic exercise (Rossow et al., 2010).

Although there is good evidence to demonstrate the efficacy of sprint interval exercise training as a regimen to improve adult health, there is little evidence to suggest that the same is true in pediatric populations. One study examined the training responses to 8 weeks of sprint interval running in comparison with a continuous cycle-ergometer program but found no change in peak or submaximal oxygen uptake, minute ventilation, and heart rate after training in prepubertal boys (Williams, Armstrong, & Powell, 2000). Nonetheless, the measured indices are indicators of cardiorespiratory fitness, and there is no evidence of the effect of sprint interval exercise on postexercise changes in metabolism or indicators of health in children. Moreover, performance responses of prepubescent children to sprint interval exercise are known to differ from those of adults and pubescent children (Ratel, Bedu, Hennegrave, Doré, & Duché, 2002). Thus, it seems reasonable to hypothesize that adolescents may benefit from sprint interval exercise in a way similar to that of adults, from a combination of acute responses to the last bout of training and training adaptations. Given that the duration of sprint interval exercise is more consistent with childhood and adolescent activity patterns (Armstrong et al., 1990; Armstrong & Welsman, 2006) than steady-state continuous exercise, the health-related responses to this type of exercise should be examined so that a variety of exercise options can be promoted. The purpose of the current study, therefore, was to investigate the immediate postexercise effects of a bout of sprint interval exercise on oxygen uptake, RER, substrate oxidation, and blood pressure in adolescents. We selected these outcome measures because adult data suggest that the measures respond to sprint interval exercise and are determined by the last bout of exercise performed rather than from longer term training adaptations (Richards et al., 2010; Whyte et al., 2010). We compared sprint interval exercise with a resting control trial to characterize the extent of the metabolic changes that occur with this type of exercise and to ensure that changes observed were a result of the exercise performed rather than because of an extended fasting period or circadian variation.

Research Design and Methods

Participants

All procedures took place at the exercise physiology laboratory of Nanyang Technological University after ethics board approval. All participants and their parents gave written informed assent and consent. Participants consisted of 10 healthy Asian (6 Chinese, 2 Vietnamese, and 2 Asian mixed race) adolescents (7 female) age 15–18 years recruited from two local schools. Participant characteristics are described in Table 1. All participants were considered to have an acceptable body-mass index for their age and sex (5th–90th percentile) according to Singaporean standards (Health Promotion Board of Singapore, 2010). Participants self-identified pubertal development as being in the range of Tanner Stages III–V.

### Anthropometric Measurements

Height was measured to the nearest 0.1 cm using an electronic wall-mounted stadiometer (Seca, Germany). Body mass was recorded to the nearest 0.1 kg using a scale (Mettrler-Toledo Pte. Ltd., Singapore). Waist circumference was measured to the nearest 0.1 cm using a flexible measuring tape. Skinfold thickness was assessed to the nearest 0.2 mm at the triceps and subscapular sites (Tanner & Whitehouse, 1975) using calipers (Holtain Ltd., Crymych, UK).

### Study Design

Participants maintained free-living conditions during the experimental period. None reported engaging in regular exercise of 30 min or more, including school physical education classes, more than four times per week. All participants were asked to remain sedentary the day before each trial apart from activities of daily living. No participants reported fasting for religious reasons, dieting, or following any special diet (including vegetarians) before or during the study.

Each participant undertook two tests on separate occasions after a 10-hr overnight fast: (a) two 30-s bouts of high-intensity sprint interval exercise on a cycle ergometer (Wingate anaerobic test protocol) separated by 4 min of recovery and (b) rested in the laboratory for an equivalent period. We were unable to control for the effect of menstrual cycle in our female participants because of school commitments or an irregular cycle. For this reason the time between the two trials exceeded the ideal 1 month. Two females completed the study with an irregular menstrual cycle. One completed both trials in the follicular phase and I completed both trials in the

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
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<tbody>
<tr>
<td>Sex, male/female</td>
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</tr>
<tr>
<td>Age, years</td>
<td>17.2 (0.7)</td>
</tr>
<tr>
<td>Tanner stage, pubic</td>
<td>III–V</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.63 (0.12)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>54.2 (12.1)</td>
</tr>
<tr>
<td>Body-mass index, kg/m²</td>
<td>20.2 (1.9)</td>
</tr>
<tr>
<td>Body-mass-index percentilea</td>
<td>40 (20)</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>72 (8)</td>
</tr>
<tr>
<td>Triceps skinfold, mm</td>
<td>16.3 (5.2)</td>
</tr>
<tr>
<td>Subscapular skinfold, mm</td>
<td>16.3 (4.2)</td>
</tr>
</tbody>
</table>

*aCalculated from Singapore percentiles.*

Table 1  Physical Characteristics of the Participants, $M$ (SD), $N = 10$
Baseline Measurements

Participants came to the laboratory at 8:30 a.m. by car. They were seated in a chair and a 30-min measurement of pulmonary-gas exchange was made via a respiratory mask attached to an automated metabolic cart (Cosmed Quark b2) to assess oxygen uptake and RER. After 15 and 30 min, blood pressure and pulse rate were measured in duplicate using an automated sphygmomanometer (Omron IW2, Omron Healthcare, Kyoto, Japan). We chose to use an automated system to measure blood pressure to prevent investigator bias in the reading of blood pressure between trials.

Sprint Interval Exercise

All participants were familiarized with the exercise protocol beforehand. They undertook two 30-s bouts of high-intensity sprint interval exercise separated by 4 min of recovery. After some initial investigation in adults, we were concerned about the possible effects of fainting or nausea we had found to accompany four bouts of sprint interval exercise. We chose to conduct only two 30-s sprints after completing pilot work in two adolescents who both tolerated the exercise protocol well. Four minutes of recovery was chosen following similar acute studies on sprint interval exercise in adults (Rakobowchuk et al., 2009; Rossow et al., 2010) and because at least one study examining recovery of power output in children has suggested that adolescents need a similar time as adults to recover from sprint efforts (Ratel et al., 2002). Each participant performed a standardized warm-up for 5 min on a cycle ergometer at 20% of the subsequent sprint load followed by 3 min of stretching. Participants then completed the two sprints on a cycle ergometer (Monark Ergomedic 824E) against a braking force calculated as 0.075 kg/kg body mass. During each sprint they were given verbal encouragement to make an “all out” effort. Peak and mean power output and rate of fatigue, calculated as peak power minus the lowest power divided by the peak power, were determined with the use of a computer interfaced to the cycle ergometer. Between sprints, participants sat passively on the ergometer and were allowed to drink water ad libitum. The respiratory mask was placed on the participant between sprints so oxygen uptake and RER could be recorded immediately at the end of exercise. At the end of the sprint the participant was immediately helped off the ergometer and into a chair. None of the participants reported feeling nauseated or faint in response to the exercise bouts. During the control trial participants rested in the chair for 15 min—the same duration of time as for the exercise session.

Postexercise

Postexercise, participants remained seated for 90 min during which pulmonary-gas exchange was continuously measured via the respiratory mask to assess oxygen uptake and RER. Participants were given two 8-min breaks at 15 min and 60 min during the 90-min period to use the bathroom or drink water ad libitum. Blood pressure was measured in duplicate immediately and 15, 30, 60, and 90 min postexercise.

Calculations

For baseline, oxygen uptake and RER were averaged over the last 15 min of the 30-min measurement for each trial. Postexercise metabolic measurements were averaged every 5 min, with corresponding times for the control trial and time points compared between trials at 5, 10, 15, 30, 45, 60, 75, and 90 min. Total postexercise oxygen consumption was calculated as the area under the oxygen-consumption-versus-time curve for the entire 90-min period using the trapezium rule. Energy derived from carbohydrate and fat oxidation was calculated from established equations (Kuo, Fattor, Henderson, & Brooks, 2005), assuming no protein oxidation, as follows:

\[
\text{Energy from CHO oxidation (kcal/min)} = \left(\frac{\text{Carbohydrate oxidation (g/min)}}{5.05 \text{ kcal/L O}_2}\right) \left(\frac{4 \text{ kcal/g CHO}}{100}\right)
\]

\[
\text{Energy from fat oxidation (kcal/min)} = \left(\frac{\text{Fat oxidation (g/min)}}{4.7 \text{ kcal/L O}_2}\right) \left(\frac{9 \text{ kcal/g fat}}{100}\right)
\]

These equations assume steady-state conditions, which are not necessarily observed after sprint interval exercise. Thus, where RER > 1.0 or < 0.7 it was assumed to represent 100% CHO or fat oxidation, respectively. Total fat and CHO oxidation were calculated as the area under the substrate-oxidation-versus-time curve for the entire 90-min period using the trapezium rule. Systolic and diastolic blood pressure and pulse rate were calculated as the mean of each duplicate measurement.

Statistical Analysis

Statistics were calculated using SPSS 18.0 for Windows. Baseline measurements of oxygen uptake, RER, substrate oxidation, systolic and diastolic blood pressure, and pulse rate were compared between trials using paired t tests. Peak and mean power output and rate of fatigue were compared between trials using paired t tests. Total postexercise oxygen consumption and fat and CHO oxidation were compared between trials using paired t tests. Postexercise values of all variables were compared between trials and over time using a two-way ANOVA (Trial × Time) for repeated measures. Where appropriate,
least-significant-difference pairwise comparison was used to identify differences between trials. In addition, postexercise measurements were compared with baseline values taken in the same trial with a paired t test. Data are presented as $M (SD)$. Significance was set at $p < .05$.

**Results**

**Anthropometric Measurements**

Height ($p = .343$), body mass ($p = .163$), and body-mass index ($p = .127$) were similar between visits.

**Baseline Measurements**

There were no significant baseline differences between trials in oxygen uptake, RER, or substrate oxidation (Table 2). Baseline systolic and diastolic blood pressure and pulse rate were similar between trials after 15 min or 30 min of seated rest (Table 2).

**Exercise**

Mean power output was significantly greater in Sprint 1 than 2 (Sprint 1, 6.1 [1.0] W/kg; Sprint 2, 5.6 [0.8] W/kg; $p = .018$). Peak power output (Sprint 1, 7.5 [1.2] W/kg; Sprint 2, 7.2 [0.9] W/kg; $p = .217$) and rate of fatigue (Sprint 1, 38% [8%]; Sprint 2, 46% [10%]; $p = .073$) did not differ between sprints.

**Postexercise Oxygen Uptake**

Total 90-min postexercise oxygen uptake was significantly higher than in the control trial (Figure 1[A]). Oxygen uptake was significantly elevated for 10 min postexercise, with no differences between trials thereafter (Figure 1[B]). Compared with baseline measurements, oxygen consumption was elevated for 15 min postexercise.

**Postexercise RER and Substrate Oxidation**

Immediately postexercise, RER > 1.0 and remained higher than in the control for 15 min, then fell rapidly and was lower than in the control 30–60 min postexercise (Figure 2[A]). Compared with baseline values, RER was elevated

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**Table 2** Baseline Measures in the Control and Exercise Trials, $M (SD), N = 10$

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Exercise</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen uptake, ml/min</td>
<td>210 (55)</td>
<td>213 (80)</td>
<td>.850</td>
</tr>
<tr>
<td>Respiratory-exchange ratio</td>
<td>0.88 (0.11)</td>
<td>0.90 (0.13)</td>
<td>.789</td>
</tr>
<tr>
<td>Carbohydrate oxidation, g/min</td>
<td>0.14 (0.06)</td>
<td>0.15 (0.09)</td>
<td>.654</td>
</tr>
<tr>
<td>Fat oxidation, g/min</td>
<td>0.05 (0.04)</td>
<td>0.05 (0.04)</td>
<td>.771</td>
</tr>
<tr>
<td>Systolic blood pressure, 15 min, mm Hg</td>
<td>99 (8)</td>
<td>99 (9)</td>
<td>.606</td>
</tr>
<tr>
<td>Diastolic blood pressure, 15 min, mm Hg</td>
<td>61 (6)</td>
<td>61 (8)</td>
<td>.882</td>
</tr>
<tr>
<td>Pulse rate, 15 min, beats/min</td>
<td>67 (6)</td>
<td>69 (7)</td>
<td>.502</td>
</tr>
<tr>
<td>Systolic blood pressure, 30 min, mm Hg</td>
<td>101 (8)</td>
<td>104(9)</td>
<td>.277</td>
</tr>
<tr>
<td>Diastolic blood pressure, 30 min, mm Hg</td>
<td>65 (6)</td>
<td>67 (10)</td>
<td>.333</td>
</tr>
<tr>
<td>Pulse rate, 30 min, beats/min</td>
<td>68 (5)</td>
<td>67 (7)</td>
<td>.786</td>
</tr>
</tbody>
</table>

*Note. Comparisons made with a paired t test.*
for 10 min postexercise and lower 30–60 min postexercise. There was a higher percentage CHO oxidation (main effect of time \( p < .001 \), Trial \( \times \) Time interaction \( p < .001 \)) and lower percentage fat oxidation (main effect of time \( p < .001 \), Trial \( \times \) Time interaction \( p < .001 \)) for the first 15 min postexercise and the reverse pattern 30–60 min postexercise. Energy from CHO oxidation was significantly higher for the first 15 min postexercise than in control and lower 45–60 min postexercise, with the opposite true for fat oxidation (Figure 2[B]). Similarly, compared with baseline values, fat oxidation was reduced for 15 min postexercise and increased 45–60 min postexercise. Total CHO oxidation over the 90 min was significantly higher after exercise (control 14.3 [5.2] g, exercise 18.2 [8.4] g; \( p = .014 \)), but total fat oxidation did not differ between trials (control 4.5 [2.5] g, exercise 5.4 [2.7] g; \( p = .247 \)).

### Postexercise Blood Pressure and Pulse Rate

Figure 3 shows postexercise blood pressure and pulse rate. Systolic blood pressure was elevated immediately postexercise compared with control but was lower 90 min after exercise (Figure 3[A]). The same results were seen when postexercise systolic pressure was compared with baseline values. Diastolic blood pressure did not differ between trials (Figure 3[B]) but was lower than baseline values immediately postexercise. Postexercise pulse rate was higher than that in control for the entire 90 min (Figure 3[C]).
Discussion

The current study is the first to examine the responses of postexercise metabolism and blood pressure to sprint interval exercise in adolescents. We observed significant elevations in oxygen uptake immediately postexercise and a decreased RER and fat oxidation 30–60 min postexercise, although absolute fat oxidation did not differ over the 90-min observation period. Although the effects of exercise were short-lived, it should be noted that participants completed a total of only 60 s of sprint exercise. In addition, and in agreement with adult data (Rossow et al., 2010), systolic blood pressure was significantly decreased 90 min postexercise. Because this type of exercise may correspond more closely with current patterns of childhood activity (Armstrong et al., 1990; Armstrong & Welsman, 2006), our data, although preliminary, indicate that this type of exercise should be considered as an exercise model for further research on health outcomes in children.

It has long been known that submaximal exercise intensity is the major determinant of the duration and magnitude of postexercise oxygen consumption (Bahr & Sejersted, 1991; Gore & Withers, 1990a, 1990b). To our knowledge, however, only two studies (Bahr, Gronnerød, & Sejersted, 1992; Laforgia, Withers, Shipp, & Gore, 1997), both in adults, have examined the effect of supramaximal exercise on postexercise oxygen uptake. The first found that cycling at 108% of maximal oxygen consumption, interspersed with 3-min rest periods, significantly elevated oxygen consumption for 30 min after one 2-min sprint, for 60 min after two sprints, and for 4 hr after three sprints (Bahr et al., 1992). The second study found that twenty 1-min intervals at 105% of maximum oxygen consumption interspersed with 2-min rest periods significantly elevated oxygen consumption for 8 hr postexercise (Laforgia et al., 1997). The increased postexercise oxygen consumption with sprint exercise may result from a number of metabolic processes that are affected by single or multiple sprints (Bahr et al., 1992; Bogdanis, Nevill, Boobis, & Lakomy, 1996; Gaitanos, Williams, Boobis, & Brooks, 1993). These processes include replenishment of oxygen stores in blood and muscle; resynthesis of ATP and creatine phosphate; increased body temperature; elevated catecholamines, which stimulate triglyceride–fatty-acid cycling; increased ventilation; and the conversion of lactate to glucose or glycogen (Bahr et al., 1992; Bahr & Sejersted, 1991). The current study is the first to examine the responses of high-intensity 30-s sprint interval exercise on postexercise oxygen uptake in a pediatric population. Our data showed that the elevation in oxygen consumption was short-lived, lasting ~10–15 min postexercise. Although we did not measure any indicators of metabolic stress in our participants, we have no reason to suspect they did not produce a maximal effort, and power-output data from the sprints are similar to those reported by others for this age group (Armstrong, Welsman, & Chia, 2001). Thus, it seems likely that the limited duration and number of sprints used were insufficient to raise oxygen consumption for any length of time. These data, along with previous work with adults (Bahr et al., 1992), suggest a dose response whereby a greater number of sprints is required to raise postexercise metabolism for an extended period of time.

Steady-state aerobic exercise can reduce postexercise RER and increase fat oxidation in adults (Kuo et al., 2005), but studies are divided as to whether supramaximal exercise increases postexercise fat oxidation. One to three 2-min bouts of supramaximal exercise did not produce any significant drop in RER lasting >60 min postexercise (Bahr et al., 1992). Conversely, twenty 1-min intervals at 105% of maximum oxygen uptake suppressed RER for 8 hr (Laforgia et al., 1997). In that study, however, a 5.8-MJ standardized lunch was provided 3 hr postexercise, so the effects of postprandial digestion may have influenced fat oxidation (Laforgia et al., 1997). More recently, in a 2-week study, sprint interval training in 10 sedentary overweight and obese adults significantly increased fat oxidation and decreased RER measured 24 hr after the intervention (Whyte et al., 2010). Thus, there may be differences between overweight and normal-weight participants or the effects of acute and training responses to sprint interval exercise on RER and lipid oxidation. In response to sprint interval exercise in the current study, we observed a suppressed RER and an increase in fat oxidation 30–60 min postexercise. The suppressed RER is indicative of carbon dioxide retention after the sprint exercise to replenish the bicarbonate used to buffer lactic acid (Laforgia et al., 1997), so we acknowledge shortcomings in calculating fat oxidation when participants had not necessarily achieved a steady-state condition. Certainly, in the current study, total fat oxidation did not differ between trials over the 90-min measurement period. Moreover, any increased fat oxidation was short-lived, and it is uncertain whether it would be sustained with daily exercise training. Pediatric studies examining 24-hr fat oxidation and changes in body composition and mass in response to sprint interval training are needed.

The finding that systolic blood pressure was reduced 90 min postexercise concurs with recent studies in adults. Systolic blood pressure was reduced by an average of 6 mm Hg 24 hr after the last of six sessions of sprint interval training conducted over 2 weeks in overweight adults age 18–40 years (Whyte et al., 2010). No reduction was observed 72 hr after the final exercise bout, suggesting that changes in blood pressure with sprint interval exercise are short-lived. Another study compared four 30-s bouts of sprint interval exercise, interspersed by 4 min recovery, with 60 min of steady-state cycling at 60% of heart-rate reserve on aortic and brachial blood pressure in 25 young, healthy, endurance-trained adults (Rossow et al., 2010). Both types of exercise produced significant reductions in aortic and brachial systolic blood pressure at 60 min, but not 30 min, postexercise, although it should be noted that there was no control condition for comparison. This is similar, however, to our data in that the effects of sprint interval exercise on systolic blood pressure were delayed postexercise. Moreover, in both studies, participants’ blood pressure was healthy, and collectively such data demonstrate that, similar to steady-state aerobic
exercise (Miyashita, Burns, & Stensel, 2008; Rossow et al., 2010), sprint interval exercise may reduce blood pressure even in individuals considered to have optimal blood pressure. Whether reductions in blood pressure benefit such individuals cannot be determined from the available evidence. Nonetheless, the effectiveness of sprint interval exercise as a model to improve vascular health should be considered.

There are several limitations of the current work. We included both male and female participants, so we cannot discount possible sex differences or influence of menstrual-cycle phase on the results. Nonetheless, one recent study showed that the hemodynamic and cardiovascular responses to sprint interval exercise do not differ between men and women (Rossow et al., 2010). Moreover, although no studies have examined sex differences in RER or substrate oxidation after sprint interval exercise, at least one well-controlled study found no difference in postexercise RER or substrate oxidation between males and females after steady-state exercise at 45% and 65% of maximum oxygen uptake (Kuo et al., 2005). Second, we used a protocol in which subjects were fasted throughout.

We used a protocol in which subjects were fasted throughout. Few children, or even adults, are likely to complete such exercise in the postabsorptive state, and most individuals would consume food within 1–2 hr postexercise. Thus, our study lacks ecological validity, and the effects of sprint exercise on postprandial metabolism need studying. Because we were concerned about adverse responses to the exercise bout, participants performed only two sprints in the current study. Thus, the physiological changes that occurred in response to the exercise in the current study may have been limited; evidence-based data from adult studies (Rossow et al., 2010; Whyte et al., 2010) better support substantial physiological changes with the use of four sprints. Finally, our participants did not complete a continuous steady-state exercise trial to provide a comparison with the sprint interval exercise. The aim of the current study was to provide an initial examination of the acute effects of sprint interval exercise on changes in metabolism and blood pressure in youth because this type of exercise may correspond more closely to adolescent activity patterns (Armstrong & Welsman, 2006; Armstrong et al., 1990). We did not aim to determine whether sprint interval exercise is more effective than continuous exercise in improving these outcomes.

Future studies need to determine the optimal exercise type for improving health-related outcomes in youth, because the greater exercise stimulus brings about significant physiological changes in adult studies (Rossow et al., 2010; Whyte et al., 2010). Moreover, given that sprint interval exercise may correspond more closely to current exercise patterns in children, future research should examine the training responses to this type of exercise in both prepubertal and pubertal groups along with assessing its ecological validity outside the laboratory setting.

Acknowledgments

S.B. was responsible for analysis and interpretation of data and overall supervision of the study. All authors contributed to the conception and design of the study, data collection, and review and editing of the manuscript.

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References


