Cardiac Autonomic Function and High-Intensity Interval Training in Middle-Age Men


1Department of Exercise and Medical Physiology, Verve Research, Oulu, FINLAND; 2Department of Applied Sciences, London South Bank University, London, UNITED KINGDOM; 3Turku PET Centre, University of Turku and Turku University Hospital, Turku, FINLAND; 4Paavo Nurmi Centre, Turku, FINLAND; 5Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Turku, FINLAND; and 6Institute of Clinical Medicine, University Hospital and University of Oulu, Oulu, FINLAND

ABSTRACT

KIVINIEMI, A. M., M. P. TULPPO, J. J. ESKELENIEN, A. M. SAVOLAINEN, J. KAPANEN, I. H. A. HEINONEN, H. V. HUIKURI, J. C. HANNUKAINEN, and K. K. KALLIOKOSKI. Cardiac Autonomic Function and High-Intensity Interval Training in Middle-Age Men. Med. Sci. Sports Exerc., Vol. 46, No. 10, pp. 1960–1967, 2014. Purpose: The effects of short-term high-intensity interval training (HIT) on cardiac autonomic function are unclear. The present study assessed cardiac autonomic adaptations to short-term HIT in comparison with aerobic endurance training (AET). Methods: Twenty-six healthy middle-age sedentary men were randomized into HIT (n = 13, 4–6 × 30 s of all-out cycling efforts with 4-min recovery) and AET (n = 13, 40–60 min at 60% of peak workload) groups, performing six sessions within 2 wk. The participants underwent a 24-h ECG recording before and after the intervention and, additionally, recorded R-R interval data in supine position (5 min) at home every morning during the intervention. Mean HR and low-frequency (LF) and high-frequency (HF) power of R-R interval oscillation were analyzed from these recordings. Results: Peak oxygen consumption (V\textsubscript{O\textsubscript{2}}peak) increased in both groups (P < 0.001). Compared with AET (n = 11), HIT (n = 13) increased 24-h LF power (P = 0.024), tended to increase 24-h HF power (P = 0.068), and increased daytime HF power (P = 0.038). In home-based measurements, supine HF power decreased on the days after HIT (n = 12) but not AET (n = 9) session. The acute response of HF power to HIT session did not change during the intervention. Conclusions: In conclusion, HIT was more effective short-term strategy to increase R-R interval variability than aerobic training, most probably by inducing larger increases in cardiac vagal activity. The acute autonomic responses to the single HIT session were not modified by short-term training. Key Words: EXERCISE TRAINING, HEART RATE VARIABILITY, VAGAL ACTIVITY, AUTONOMIC NERVOUS SYSTEM

A large body of literature shows the beneficial effects of exercise training on health outcomes in various populations (14). Currently, recommendations for health-enhancing physical activity from the American College of Sports Medicine include ≥150 min of aerobic exercise at moderate intensity or ≥75 min at vigorous intensity weekly and two to three strength-training exercises per week (14). In terms of cardiovascular health, some of the health benefits of this type of aerobic-based exercise training are manifested as improved cardiovascular autonomic regulation. This is indicated by a substantial increase in cardiac vagal activity and a decrease in cardiovascular sympathetic activity (6,25,26,32,43), which have important antiarrhythmic effects (5,27).

Research for the past decade has documented the beneficial effects of various types of high-intensity interval training (HIT) on fitness and health. One of its ultimate forms—short-term high-intensity anaerobic training, including 4–6 × 30 s of all-out efforts—has improved aerobic fitness within 2 wk, similarly to aerobic exercise training (11,15), and
resulted in even better outcomes in glucose metabolism than aerobic endurance training (AET) (4). Therefore, this type of HIT has become very popular training scheme in practice and research. Although, the autonomic adaptations in various types of interval training have been investigated extensively, the effectiveness of all-out HIT scheme, introduced by Burgomaster et al. (11), to improve cardiac autonomic function is unclear. Typically, aerobic exercise training increases cardiac vagal modulation via functional and structural adaptations in cardiovascular system, including improved stroke volume (29). A recent study by MacPherson et al. (30) suggested that adaptations of fitness in response to HIT are mainly peripheral, accompanied by no central cardiovascular adaptations, such as maximal cardiac output. Therefore, limited autonomic adaptations may occur with HIT. However, different types of interval training (work–recovery ratio: 30:60 s, 10:20 s, or 8:12 s for 20–40 min; work: 120% maximal aerobic work rate or 80%–90% of maximal HR) have been effective to increase cardiac vagal activity in young healthy participants (21,37). Nevertheless, although the exercise training with higher metabolic strain is more effective in improving aerobic fitness, it does not alter sympathovagal balance toward vagal predominance as do the training with less metabolic strain (37). Furthermore, Buchheit et al. (9) reported that, among adolescents, short-term interval training within aerobic capacity is a more effective training regimen for increasing cardiac vagal activity than interval training with a larger anaerobic component. However, these training protocols differ saliently from commonly in-

TESTED METHODS

The present study was a part of a larger study titled “The Effects of Short-Term High-Intensity Interval Training on Tissue Glucose and Fat Metabolism in Healthy Subjects and in Patients with Type 2 Diabetes” (NCT01344928), which was conducted at the Turku PET Centre, University of Turku and Turku University Hospital (Turku, Finland). The study was performed according to the Declaration of Helsinki, the protocol was approved by the ethical committee of the Hospital District of Southwest Finland, Turku, Finland (decision 95/180/2010 I228), and all the participants gave their written informed consent.

Subjects. The participants were recruited with newspaper advertisements, through personal contacts, and using electronic and traditional bulletin boards. The candidates were interviewed with a standardized scheme to ascertain their health status. The inclusion criteria were as follows: male sex, age 40–55 yr, body mass index 18.5–30 kg·m⁻², and normal glycemic control verified by an oral glucose tolerance test (3). The exclusion criteria were blood pressure >140/90 mm Hg; any chronic disease, medical defect, or injury interfering with everyday life; a history of eating disorders; a history of asthma; previous use of anabolic steroids, additives, or any other substances; use of tobacco products during past year; use of narcotics; significant use of alcohol; any other condition that in the opinion of the investigator could create a hazard to the participant’s safety, endanger the study procedures, or interfere with the interpretation of the study results; presence of any ferromagnetic objects that would make magnetic resonance imaging contraindicated; current or a history of regular and systematic exercise training; and peak oxygen consumption (VO₂peak) >40 mL·kg⁻¹·min⁻¹ to complement the information about training status. Twenty-eight participants fulfilled these criteria and were randomized into HIT (n = 14) and AET groups (n = 14). This sample size was determined to obtain statistical power of >80% for the expected change in VO₂peak (from 2.8 ± 0.6 to 3.0 ± 0.6 L·min⁻¹, alpha <5%, n >12) (10). During the intervention, one participant from the HIT group dropped out due to training-induced hip pain and one from the AET group due to personal reasons; thus, 13 participants in both groups finalized the intervention.

Training interventions. The training interventions consisted of six supervised exercise sessions within 2 wk, performed in controlled laboratory conditions. The progressive HIT training included 4–6 × 30 s of all-out cycling efforts with 4 min of recovery where the participants were allowed to remain still or do unloaded cycling (Monark Ergomedic 828E; Monark, Vansbro, Sweden). The initial number of bouts was 4, and it increased by one after every other session. The participants were familiarized with the HIT training approximately 1 wk before the intervention (2 × 30-s bouts). Each bout started with 5-s acceleration to maximal cadence without any resistance, followed by a sudden increase of the load (7.5% of whole body weight in kg) and maximal cycling for 30 s. The AET group did traditional aerobic training that included cycling exercises (Tunturi E85; Tunturi Fitness, Almere, The Netherlands) for 40–60 min at moderate intensity (60% of peak workload). The duration of cycling was initially 40 min, and it increased by 10 min after every other session until 60 min was reached during the second week.

Maximal exercise test. The participants performed a maximal exercise test on a bicycle ergometer (Ergoline 800s;
exchange ratio was reported as the mean value per minute. The peak respiratory (Jaeger Oxycon Pro; VIASYS Healthcare) were measured and reported as the mean value per minute. The peak respiratory exchange ratio was ≥1.15, and the peak blood lactate concentration, measured from capillary samples obtained immediately and 1 min after exhaustion (YSI 2300 Stat Plus; YSI Incorporated Life Sciences, Yellow Springs, OH), was ≥8.0 mmol·L⁻¹ for all the tests. A peak HR (RS800CX; Polar Electro Ltd., Kempele, Finland) within 10 beats of the age-appropriate reference value (220 − age) was true in all except one participant in the both groups and in both pre- and posttraining tests. Therefore, the highest 1-min mean value of oxygen consumption was expressed as VO₂peak. Peak workload (Load_peak) was calculated as average workload during the last 2 min of the test (weighted average was used if the final stage was stopped prior completion) and used as a measure of maximal performance.

R-R interval recordings. A 24-h ECG was recorded before the intervention and 3 d after the last exercise with a digital Holter device with the sampling frequency set to 1 kHz (Medilog AR 12 Plus; Schiller AG, Switzerland). Strenuous physical activity was prohibited on the day of measurement and the day before and the participants were asked to keep their daily routines during the recording (e.g., work, sleep, and meals). R-R intervals were extracted from the ECG recordings for further analysis. In addition, the participants recorded R-R intervals with an HR monitor at an accuracy of 1 ms (RS800CX; Polar Electro Ltd., Kempele, Finland) in a supine position (5 min) at home every morning during the intervention. These measurements started immediately after awakening and emptying the urinary bladder. The R-R interval data were stored in the HR monitor and extracted to a computer for further analysis. One participant from the AET group was excluded from all the analyses because of technical problems in both the 24-h and the home-based recordings. In addition, one participant in the HIT group and three in the AET group had insufficient data in the home-based measurements, covering less than two exercises per week. Thus, the final numbers of participants were 13 and 11 in the 24-h analyses and 12 and 9 in the home recordings for the HIT and AET groups, respectively.

HR variability analyses. The R-R interval data were edited based on visual inspection of the tachogram and ECG when available (24-h ECG). All ectopic beats and artifacts were deleted from the data. Mean HR, low-frequency (LF, 0.04−0.15 Hz) and high-frequency (HF, 0.15−0.40 Hz) components of HRV, and their ratio (LF/HF) were analyzed by autoregressive spectral analysis in 1-h segments (42). The analyses were performed for the whole 24-h R-R interval data set, daytime (9:00 a.m.–6:00 p.m.), nighttime (1:00 a.m.–5:00 a.m.) and the final 3-min periods of 5-min supine resting measurements at home. The values of absolute spectral powers and LF/HF ratio were not normally distributed and, thus, were transformed into natural logarithm (ln) before the statistical tests. The 24-h recording was used to assess autonomic adaptations to the training. The acute effects of exercise were assessed on a weekly basis (first and second weeks). Daily HRV values were averaged separately for the preexercise (morning on a training day) and postexercise (morning after a training day) conditions in this approach.

Statistical methods. The training adaptations were assessed by two-way repeated-measures ANOVA with “training” (pre- vs postintervention) as the within-subject factor and “group” (HIT vs AET) as the between-subjects factor. A two-way ANOVA was also used to test the acute effects of the exercise session on HRV separately for the study groups using “acute exercise” (morning before vs after exercise) and “first versus second week” (indicating training adaptations between the first week and the second week) as within-subject factors. The t-test with Bonferroni’s correction was used appropriately as post hoc when significant interaction was observed. Standardized effects sizes (ES) were also calculated with following threshold values: <0.2 trivial, >0.2 small, >0.6 moderate, and >1.2 large (22). The data were analyzed using SPSS software (IBM SPSS Statistics 21; IBM Corp., Armonk, NY). A P value < 0.05 was considered statistically significant.

RESULTS

The intervention increased VO₂peak and Load_peak in the study groups without significant training–group interaction (Table 1). The training responses in VO₂peak and Load_peak were 0.14 ± 0.15 L·min⁻¹ (ES = 0.93, moderate) and 20 ± 16 W (ES = 1.3, large) among the HIT group and 0.09 ± 0.17 L·min⁻¹ (ES = 0.53, small) and 14 ± 11 W (ES = 1.3, large) among the AET group, respectively.

Training adaptations in ambulatory 24-h HR variability. In the 24-h recording measured before and after the intervention, LF power of HRV increased in the HIT group (ES = 1.1, moderate), but not in the AET (P = 0.024 for the training–group interaction; Fig. 1b). Also, the 24-h HF power of HRV tended to increase more in HIT (ES = 0.56, small) than that in AET during the intervention (P = 0.068; Fig. 1c). No significant training effects or training–group interactions were observed in the mean 24-h HR or LF/HF ratio. However, the AET group had a significantly higher mean 24-h HR compared with the HIT group without significant interaction with training (Fig. 1a).

In daytime and nighttime HRV analyses, the training–group interaction was significant only in the daytime HF power and LF/HF ratio (Table 2). The intervention decreased daytime LF/HF ratio in the HIT group (ES = 0.67, moderate; P = 0.032) and tended to increase HF power (ES = 0.56, small; P = 0.067),
Acute effects of exercise on HR variability in daily home-based measurements. Acute exercise effect of HIT session was observed in the supine resting HF power of HRV, which was lower in the mornings after HIT session than before (Fig. 2d). No other significant main effects were observed in any other HR and HRV variables, whereas no such changes occurred in the AET group. The acute exercise effect without significant training-group interaction was observed in the nighttime LF power (Table 2).

**DISCUSSION**

The present study documented that HIT, but not AET, for 2 wk increased the power of LF oscillations in the R-R interval during 24-h ambulatory measurement. HIT also tended to increase the 24-h HF power and significantly increased the daytime HF power compared with AET. Furthermore, HIT also increased the supine HF power in the home-based measurements from the first week to the second week of intervention. These findings suggest that HIT was a more effective short-term strategy to increase HRV than AET, most probably by inducing larger increases in cardiac vagal activity. In addition, the home-based measurements of supine HF power of R-R interval oscillation, in the mornings before and after the exercise, revealed that the acute decrease in cardiac vagal activity after HIT exercise was observed similarly during the first and the second weeks of intervention. This suggests that HIT exercise acutely constituted a substantial stress to the autonomic nervous system, and this acute autonomic response to HIT exercise was not altered within a short-term HIT intervention, despite the cardiac autonomic adaptations observed after the training period of 2 wk.

**Autonomic adaptations to interventions.** Short-term high-intensity anaerobic training for 2 wk increased the power of LF oscillation in R-R interval during 24-h ambulatory measurement, whereas such change was absent with AET. This was accompanied with an apparent tendency for the larger increase in the 24-h HF power and a significant acute autonomic response to HIT exercise was not altered within a short-term HIT intervention, despite the cardiac autonomic adaptations observed after the training period of 2 wk.

**FIGURE 1—Effects of short-term high-intensity interval training (HIT) and aerobic endurance training (AET) on 24-h HR (A) and HR variability. In the 24-h recording, there was a significant training–group interaction, such that the low-frequency (LF, 0.04–0.15 Hz) power of HRV increased during the 2-wk HIT, but not during the 2-wk AET intervention (B). A similar tendency for training–group interaction was observed in high-frequency power (HF, 0.15–0.4 Hz) (C), whereas no significant main effects were observed in the LF/HF ratio (D). *P < 0.001 between pre- and postintervention.**

**TABLE 1. Characteristics and training adaptations in the high-intensity interval training (HIT) and aerobic endurance training (AET) study groups.**

<table>
<thead>
<tr>
<th></th>
<th>HIT, n = 13</th>
<th>AET, n = 13</th>
<th>ANOVA Results (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretraining</td>
<td>Posttraining</td>
<td>Pretraining</td>
</tr>
<tr>
<td>Age, yr</td>
<td>48 ± 5</td>
<td>48 ± 5</td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>180 ± 5</td>
<td>179 ± 4</td>
<td></td>
</tr>
<tr>
<td>Weight, kg</td>
<td>82.5 ± 9.6</td>
<td>81.9 ± 9.5</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>25.6 ± 2.7</td>
<td>25.4 ± 2.8</td>
<td></td>
</tr>
<tr>
<td>Loadpeak, W</td>
<td>225 ± 36</td>
<td>224 ± 29</td>
<td></td>
</tr>
<tr>
<td>Loadpeak, Wkg⁻¹</td>
<td>2.74 ± 0.34</td>
<td>2.70 ± 0.43</td>
<td></td>
</tr>
<tr>
<td>(V'\text{O}_2)peak, L·min⁻¹</td>
<td>2.65 ± 0.41</td>
<td>2.82 ± 0.35</td>
<td></td>
</tr>
<tr>
<td>(V'\text{O}_2)peak, ml·kg⁻¹·min⁻¹</td>
<td>34.7 ± 3.9</td>
<td>33.9 ± 4.6</td>
<td></td>
</tr>
<tr>
<td>HFpeak, bpm</td>
<td>181 ± 12</td>
<td>183 ± 17</td>
<td></td>
</tr>
<tr>
<td>Lapeak, mmol·L⁻¹</td>
<td>11.1 ± 2.0</td>
<td>10.9 ± 2.1</td>
<td></td>
</tr>
<tr>
<td>RERpeak</td>
<td>1.26 ± 0.06</td>
<td>1.23 ± 0.06</td>
<td></td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD.

BMI, body mass index; Load, workload; \(V'\text{O}_2\), oxygen consumption; La, blood lactate concentration; RER, respiratory exchange ratio; peak, the peak value during the exercise test.
increase in daytime HF power of R-R interval oscillation with HIT than AET. As it is known, the HF power of R-R interval oscillation is an established marker for cardiac vagal activity (1,20,36). However, the LF power includes both cardiac sympathetic and vagal effects (36), and a major part of it is determined by vagal modulation (2) and baroreflex (16,33). This suggests that the increase in the power of LF oscillations in the R-R interval is due to the increased vagal and/or baroreflex-mediated modulation of sinoatrial node without major changes in sympathovagal balance, which is supported by the findings of Rakobowchuk et al. (37) with supramaximal interval training with high metabolic strain. Also, the home-based measurements of HRV showed an evident increase in the resting HF power (but not in LF power) from the first week to the second week of intervention in the HIT group. The LF/HF ratio, an estimate—although controversial—of sympathovagal balance (13,31), did not reveal any significant training effects when analyzed during whole 24-h recording. However, daytime LF/HF ratio decreased in the HIT group. Taken together, the present observations are most likely explained by larger increase in vagal or baroreflex-mediated modulation of sinoatrial node with HIT compared with AET. Both the LF and the HF power of R-R interval oscillations are significant predictors of cardiac morbidity of cardiac patients (27). Therefore, HIT might be efficient short-term strategy to improve cardiac autonomic function and have important antiarrhythmic effect (18).

It is important to note that despite significant improvement in aerobic fitness, the present aerobic training regimen did not induce as evident changes in cardiac autonomic regulation as HIT. Several previous studies have documented increased cardiac vagal activity after aerobic training (6,25,26,43), the findings during short-term aerobic training (2 wk), however, being inconsistent (19,28). In addition to the length of the training intervention, a larger training volume may be needed to increase cardiac vagal activity, for example, the present three times per week for 2 wk versus four to six times per week for 2–8 wk (6,25,26,28,43). Nonetheless, HIT seems to be an effective strategy for inducing cardiac autonomic adaptations even in short-term training.

It is noteworthy that differences in training responses of circadian HRV between HIT and AET occurred specifically in the daytime but not nighttime analyses. Longer-term aerobic training has consistent increased both daytime and nighttime HRV (43). Larger increase in the HF power of R-R interval oscillation and larger decrease in LF/HF ratio in the HIT compared with the AET group indicate larger increase in daytime cardiac vagal activity. Although both analyses are similarly reproducible (41), it seems that HIT induces positive adaptations that manifest as lesser vagal withdrawal during daily routines. Whether this is related to autonomic adaptations per se or lesser physiological strain of daily routines because of improved aerobic fitness remains to be established.

The detailed mechanisms for the current observation cannot be confirmed by the present data, but increased blood volume may be the fastest contributor to training-induced increase in cardiac vagal modulation at rest (8), presumably via baroreflex activation, which is an important determinant of LF oscillations in the R-R interval (16,33). However, it has been established that improved aerobic capacity with HIT is mainly due to the improved muscle oxidative capacity rather than changes in cardiac function and blood volume (23,30), and that is why the exact mechanisms for the present findings remains to be established by the future studies.

**Acute autonomic responses to the exercise sessions over the interventions.** Unlike aerobic endurance exercise with moderate intensity, HIT exercise acutely decreased cardiac vagal activity, as documented by the decreased supine HF power of R-R interval oscillations in the home-based measurements on the days after training sessions (Fig. 2). This underscores the well-documented importance of exercise intensity, that is, metabolic strain, in the autonomic recovery from acute physical exercise (7,38–40). Importantly, the acute decrease in cardiac vagal activity by HIT exercise was not modified by training, meaning that the acute decrease in HF power of R-R interval oscillations was observed similarly between the first week and the second week of intervention. Presumably, this lack of adaptations in autonomic recovery from HIT exercise may have been masked by the increased number of all-out bouts during HIT exercise between the first week and the second week and the increased power output in the HIT bouts (e.g., ~10% in the third and fourth bouts) during the training. It has been

**Table 2. Daytime and nighttime mean HR and HR variability in the high-intensity interval training (HIT) and aerobic endurance training (AET) study groups.**

<table>
<thead>
<tr>
<th></th>
<th>HIT, n = 13</th>
<th>AET, n = 11</th>
<th>ANOVA Results (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretraining</td>
<td>Posttraining</td>
<td>Pretraining</td>
</tr>
<tr>
<td>Day, 9:00 a.m.–6:00 p.m.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR, bpm</td>
<td>79 ± 10</td>
<td>77 ± 9</td>
<td>83 ± 9</td>
</tr>
<tr>
<td>LF, ln ms²</td>
<td>7.0 ± 0.6</td>
<td>7.0 ± 0.5</td>
<td>6.6 ± 0.5</td>
</tr>
<tr>
<td>HF, ln ms²</td>
<td>5.2 ± 0.6</td>
<td>5.4 ± 0.6</td>
<td>5.2 ± 0.5</td>
</tr>
<tr>
<td>LF/HF-ratio, ln</td>
<td>1.8 ± 0.3</td>
<td>1.6 ± 0.4*</td>
<td>1.5 ± 0.4</td>
</tr>
<tr>
<td>Night, 1:00 a.m.–5:00 a.m.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR, bpm</td>
<td>56 ± 5</td>
<td>56 ± 5</td>
<td>63 ± 7</td>
</tr>
<tr>
<td>LF, ln ms²</td>
<td>7.3 ± 0.7</td>
<td>7.6 ± 0.5</td>
<td>7.2 ± 0.5</td>
</tr>
<tr>
<td>HF, ln ms²</td>
<td>6.3 ± 0.5</td>
<td>6.5 ± 0.8</td>
<td>5.9 ± 0.8</td>
</tr>
<tr>
<td>LF/HF-ratio, ln</td>
<td>1.0 ± 0.6</td>
<td>1.1 ± 0.7</td>
<td>1.2 ± 0.8</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD. LF, HF and LF/HF-ratio were transformed into natural logarithm (ln).

*P < 0.05 between pre- and postintervention.

LF, low frequency (0.04–0.15 Hz); HF, high frequency (0.15–0.40 Hz).
clearly shown that better aerobic fitness enables faster recovery from aerobic exercise (39), but our results suggest that similar adaptation to all-out exercise is not observed, at least in short term. Longer training period and larger improvement in aerobic fitness might be needed to improve the autonomic recovery from the acute HIT exercise. Buchheit et al. (9) have reported that interval training, although within the aerobic capacity, improves rapid cardiac vagal reactivation immediately after exercise. This type of adaptation may have also occurred in the present study, but we aimed to focus on autonomic recovery on the day after exercise as this approach has been proven to provide a potent tool for periodization of high-intensity training sessions (24,25,35). In terms of cardiac autonomic regulation, our findings suggest that moderate-intensity aerobic exercises could be repeated daily, but more than 1 d is needed to fully recover from heavy HIT exercise.

**Limitations.** Measurements performed at home may not be as highly standardized as those performed in laboratory conditions. For instance, HRV obtained during paced and spontaneous breathing has provided divergent results on cardiac autonomic responses to short-term aerobic training (28). However, this was the most convenient method, without a white-coat effect (17), for the participants to obtain real-life information on cardiac autonomic function, having direct implications in practice (24,25). The 24-h ambulatory recording and home-based measurements did not provide fully consistent results on HRV responses to training interventions. One potential reason may be the better reproducibility of 24-h recording compared with short-term recordings (12). Averaging of home-based measurements for 3 d may have diminished (34) but not completely abolished the problem related to larger day-to-day variation in short-term HRV. The present study is also limited by its relatively small sample size because HRV analyses could not be performed for all participants due to the technical problems. The aerobic training intervention did not fully meet the guidelines for health-enhancing physical activity based on recommendations from the American College of Sports Medicine, especially in terms of frequency of aerobic training. In this regard, the study was however purposefully designed in a way that both training groups had equal numbers of exercise sessions (although AET had a larger total volume). This enabled the present evaluation of autonomic responses to acute exercises, which would not have been possible with a higher frequency of aerobic exercises.

**CONCLUSIONS**

High-intensity interval training (4–6 × 30 s of all-out cycling efforts) was more effective in increasing cardiac vagal activity than aerobic training in short-term. Despite these adaptations in cardiac autonomic function, the acute decrease in cardiac vagal activity after single HIT exercise was observed similarly during the first and second weeks of intervention. Therefore, a short-term HIT intervention did not decrease the acute autonomic disturbances induced by an HIT exercise.

The authors want to thank the personnel of the Turku PET Centre for their excellent assistance.

This study was conducted within the Centre of Excellence in Molecular Imaging in Cardiovascular and Metabolic Research supported by the Academy of Finland, the University of Turku, Turku University Hospital, and Abo Akademi University.
The study was financially supported by the Ministry of Education of the State of Finland, the Academy of Finland (grant nos. 251399 and 256470), and Centre of Excellence funding, the European Foundation for the Study of Diabetes, the Hospital District of Southwest Finland, Orion Foundation, the Finnish Diabetes Foundation, and the Finnish Technology Development Centre (Tekes, Helsinki, Finland).

The authors declare no conflict of interest.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

REFERENCES


