Eur J Appl Physiol (2008) 104:541–548
DOI 10.1007/s00421-008-0804-9

ORIGINAL ARTICLE

Effect of low-dose endurance training on heart rate variability at rest and during an incremental maximal exercise test

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Accepted: 9 June 2008 / Published online: 28 June 2008

Abstract We evaluated the effects of low-dose endurance training on autonomic HR control. We assessed the heart rate variability (HRV) of 11 untrained male subjects (36.8 ± 7.2 years) at rest and during an incremental maximal aerobic exercise test prior to a 7-week preparatory period and prior to and following a 14-week endurance training period, including a low to high intensity exercise session twice a week. Total (0.04–1.2 Hz), low (0.04–0.15 Hz) and high (0.15–1.2 Hz) frequency power of HRV were computed by short-time Fourier transform. The preparatory period induced no change in aerobic power or HRV. The endurance training period increased peak aerobic power by 12% ($P < 0.001$), decreased the HR ($P < 0.01$) and increased all HRV indices ($P < 0.05$–0.01) at absolute submaximal exercise intensities, but not at rest. In conclusion, low-dose endurance training enhanced vagal control during exercise, but did not alter resting vagal HR control.

Keywords Endurance training · Autonomic nervous system · Parasympathetic nervous system · Heart rate variability · Time-frequency analysis

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Introduction

Epidemiological studies provide evidence that recreational physical activity promotes and maintains health and that regular endurance training provides additional health benefits, including a reduction of cardiovascular risk factors and mortality (Bucksch and Schlicht 2006; Haskell et al. 2007). Endurance training induces central adaptations, including an increase in maximal cardiac output, stroke volume, diastolic filling and left ventricular hypertrophy as well as peripheral adaptations in skeletal muscle (Blomqvist and Saltin 1983). These adaptations are produced by a complex set of structural, metabolic, humoral and neural changes, including modifications of autonomic nervous system function.

One possible pathway by which endurance training benefits cardiovascular health is through causing a shift in autonomic HR control towards increased vagal dominance. The relative bradycardia observed in highly trained endurance athletes may result from an increase in vagal tone and/or a decrease in sympathetic tone or, alternatively, from a decrease in intrinsic HR (Katona et al. 1982; Scheuer and Tipton 1977). Longitudinally, the dose-response relation between a training stimulus (i.e., the intensity, duration and frequency of training) and autonomic cardiac adaptation has remained uncertain. Furthermore, there is limited information about the minimal amount of endurance training required to provide a sufficient training stimulus to induce health benefits by improving autonomic balance and physical fitness (Bucksch and Schlicht 2006).

Spectral analysis of heart rate variability (HRV) provides a non-invasive measure of autonomic HR control (Akselrod et al. 1981). Two frequency components are usually examined: high frequency power (HFP, 0.15–0.40 Hz at rest and 0.15–1.2 Hz during exercise), reflecting vagal effects on the heart, and low frequency power (LFP, 0.04–0.40 Hz),

reflecting combined vagal and sympathetic effects on the heart (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology 1996). Until recently, HRV analysis has been restricted to settings of a steady-state HR, and, therefore, the majority of HRV studies have obtained information about autonomic HR control only at rest. However, a recently developed time-frequency analysis method, short-time Fourier transform (STFT), can be applied to non steady-state HR settings and it provides a promising tool for estimating transient changes in autonomic HR control (Kaikkonen et al. 2007; Martinmäki et al. 2006b; Martinmäki and Rusko 2008; Pichon et al. 2004).

According to recent review articles, most cross-sectional studies have concluded that endurance training induces a decrease in HR and an increase in HRV at rest (e.g., Achten and Jeukendrup 2003; Aubert et al. 2003). Results from longitudinal studies on young and middle aged subjects have been less conclusive. Some studies have found a decrease in resting HR combined with an increase in HFP after endurance training (Carter et al. 2003; Leicht et al. 2003b; Mourot et al. 2004; Tulppo et al. 2003). Others have found no change in either HFP or LFP after endurance training (Leicht et al. 2003a; Loimaala et al. 2000). Only a few studies have examined the effects of endurance training on HRV during exercise. Endurance training has been reported to induce an increase in both LFP and HFP components (Leicht et al. 2003b) or in only HFP or LFP (Carter et al. 2003; Leicht et al. 2003a) during constant-load submaximal exercise. One study has also examined HRV at work loads determined in relation to maximal work capacity and reported similar pre- and post-training HRV values at the same relative exercise intensity (Leicht et al. 2003b).

The purpose of the present study was to evaluate the effects of endurance training on autonomic HR control at rest and during exercise at different intensities in previously untrained male subjects. HRV was computed with the STFT method at rest as well as during and after an incremental maximal exercise test prior to a 7-week preparatory period and prior to and following a low-dose 14-week endurance training period. The endurance training period included low to high intensity exercise twice a week in order to clarify whether the low-dose endurance training would have any beneficial effects on autonomic HR control.

Methods

Experimental design

Twelve untrained healthy males volunteered for this study. The data of one subject was excluded because he did not complete all exercise training sessions. The characteristics of the remaining 11 subjects are presented in Table 1. Some subjects had been previously involved in various recreational physical activities, but none of the subjects had any background in regular endurance training or competitive sports. During the last 2 years, the subjects were not involved in exercise training of any kind. The subjects completed prescreening questionnaires and medical examinations to confirm their healthy status. All subjects were normotensive, non-smokers, and were not taking any medication or drugs that would alter cardiovascular control. They were familiarized with the testing equipment and procedures used in the study and provided informed consent prior to participation. They had the right to withdraw from the study at any time. The study was conducted according to the declaration of Helsinki and was approved by the Ethics Committee of the University of Jyväskylä, Finland.

This study consisted of a 7-week preparatory period followed by a 14-week endurance training period. HRV measurements were carried out prior to the preparatory period (Control, C) as well as prior to (Pre-Training, Pre-T) and following (Post-Training, Post-T) the endurance training period. HRV was assessed in supine, sitting and standing postures, at different exercise intensities during an incremental maximal exercise test and during a subsequent recovery period.

Procedures

The subjects were asked to refrain from any physical exertion starting 2 days before the testing days. During the test days, the subjects were instructed to avoid consumption of alcohol and caffeinated beverages. The tests were carried out in a quiet laboratory room (23–24°C) at the same time each day. Height, weight, percentage of body fat (Durnin and Womersley 1974) and resting blood pressure were measured before each test. The test session started with the measurements at rest. The subjects rested quietly for 5 min in a supine, sitting and standing posture. This was followed by the standardized incremental maximal exercise test in an upright seated position on an electronically braked cycle ergometer (Ergoline Ergometrics 800S) and the controlled

Table 1 Baseline characteristics of the subjects

	C
Age (year)	36.8 (7.2)
Height (cm)	181 (7.9)
Weight (kg)	79.8 (13.0)
Body fat (%)	17.6 (3.6)
SBP (mm Hg)	131 (14)
DBP (mm Hg)	72 (16)

Values are means (SD), systolic (SBP) and diastolic blood pressure (DBP) were measured in a supine position

post-exercise recovery of 15 min in a supine position. During the maximal exercise test, the initial workload of 75 W was increased in a ramp fashion, by 25 W every 2 min until exhaustion. Seat and handlebar heights and pedaling frequency (60 rev min^{-1}) were kept constant during all testing sessions.

ECG R-to-R-peak intervals (RRI) were continuously recorded throughout the testing session using a RR-recorder (Polar Electro Ltd., Kempele, Finland), with a sampling frequency of 1,000 Hz from the ECG signal, providing an accuracy of 1 ms for each RRI. During the incremental maximal exercise test, respiratory frequency, tidal volume, and fractional gas concentrations of expired O_2 and CO_2 were continuously recorded breath-by-breath (V_{max} 229, Sensormedics, Palo Alto, CA, USA). Prior to each test, the gas analyzer was calibrated using ambient air (20.9% O_2 and 0.04% CO_2) and calibration gas (15.87 and 4.17%). The calibration of the flow-meter of the analyzer was performed with a 3-L syringe. Fingertip venous blood samples for determining blood lactate concentration (BLa, Eppendorf EBIO 6666, Eppendorf, Hamburg, Germany) were taken at rest and after each workload. A plateau of oxygen consumption, respiratory exchange ratio exceeding 1.10, and HR and BLa approximating the age-predicted maximum were employed as criteria for the attainment of peak power output (W_{peak}) and oxygen uptake (VO_{2peak}) (Taylor et al. 1955). W_{peak} was assessed as the mean power during the last 2 min of the exercise test. VO_{2peak} and the peak value of ventilation (VE_{peak}) and HR (HR_{peak}) were determined at W_{peak}. Aerobic and anaerobic thresholds were determined as described in detail previously (Aunola and Rusko 1984) and oxygen uptake (VO_{2AnT}), ventilation (VE_{AnT}) and HR (HR_{AnT}) corresponding to the anaerobic threshold were computed.

Venous blood noradrenaline and adrenaline concentrations, providing blunt estimates of sympathetic nervous system activity (Christensen and Galbo 1983), were determined prior to the testing session, at the end of the maximal exercise test and after the 15-min recovery. A blood sample of 10 ml was taken from the antecubital vein and centrifuged (3,600 rpm) at +4°C. A plasma sample was aliquoted for noradrenaline and adrenaline measurements and stored at −80°C until assayed. Plasma catecholamine concentrations were determined by using high-pressure liquid chromatography with an electrochemical detector (^{TM}ESA Coulochem II detector, Model 5011 Analytical Cell, ESA, Inc., Chelmsford, MA, USA.) at the Kuopio University Hospital.

RRI-signal processing and HRV computations were performed using the MATLAB 7 programme (The MathWorks, Inc., 2007). All analyses were performed from the RRI series free from ectopic beats and technical artifacts. The original RRI series were resampled at a rate of 5 Hz by using linear interpolation to obtain equidistantly sampled time series. In order to remove low frequency trends and variances below and above interest, a polynomial filter and a digital FIR band-pass filter were used (see e.g., Oppenheim and Schafer 1999). Since the RRI time series were not stationary during the exercise and recovery, conventional spectral analysis was not suitable for analyzing HRV. Therefore, the STFT method was used to compute a time-frequency decomposition of the RRI time series (see Cottin et al. 2006; Oppenheim and Schafer 1999). Briefly, the method calculated consecutive power spectra of short sections of the signal: a section of 256 samples was multiplied by the Hanning window function and the fast Fourier transform of their product was taken. The window was then shifted one sample ahead and the same calculations were performed again. This process was repeated until the whole RRI time series—including all postures, the incremental maximal exercise test and the recovery period—was covered. Integrals of the power spectral density curve within the following frequency boundaries were calculated as a function of time: low frequency power (LFP, 0.04–0.15 Hz), high frequency power (HFP, 0.15–1.2 Hz) and total power (TP, 0.04–1.2 Hz). LFP, HFP and TP values as well as HR during the different phases of the testing session were calculated as follows: (1) mean of 3 min at the end of each posture, (2) mean of 1 min at the end of each workload during the incremental maximal exercise test, and (3) mean of 3 min from recovery minute 4–6 and 12–14.

Training

During the 7-week preparatory period, the subjects participated in supervised low intensity training twice a week. They became familiarized with regular training and the laboratory testing procedures. The preparatory period included 30-min exercise sessions below the intensity level of the aerobic threshold (43 ± 4% of maximal power) twice a week. One exercise session was performed on a cycle ergometer under supervision by the researcher. Another exercise session was unsupervised and to increase subject retention rate, it included cycling, running and Nordic walking.

The 14-week endurance training period was designed to improve aerobic power and consisted of low and high intensity training twice a week. Again, one exercise session was performed on a cycle ergometer under supervision and another exercise session was unsupervised and included the above-mentioned training modes. During the first 7 weeks of the actual endurance training period, the supervised exercise session lasted for 45 min and included four stages: a 15-min warm-up below aerobic threshold (45 ± 5% of maximal power), 10 min between the aerobic and anaerobic threshold (70 ± 5% of maximal power), 5 min above the anaerobic threshold and a 15-min cool-down below the aerobic threshold. The unsupervised exercise session lasted for

60 min below the aerobic threshold. During the remaining 7 weeks of the endurance training period, the supervised exercise session lasted for 75 min and included the following stages: a warm-up below the aerobic threshold, 2 × 10 min between the aerobic and anaerobic thresholds, 2 × 5 min above the anaerobic threshold and a cool-down below the aerobic threshold. The unsupervised exercise session lasted for 60–90 min below the aerobic threshold. The subjects used a HR monitor during the training sessions in order to maintain the intensity of exercise at the required level. The subjects were not involved in other physical activities than those in the study's training programme.

Statistical analysis

All values are expressed as means and standard deviations. To meet the assumptions of parametric statistical analysis, a natural log transformation of HRV values was used. A standard of 1 was added to the HRV values before calculating the natural logarithm so that the values would not be negative, e.g., $y = \ln(1 + x)$. Depending on the distributional characteristics of the variable, a paired samples *t* test or Wilcoxon signed rank test was used to evaluate whether the preparatory training (C vs. Pre-T) or the endurance training period (Pre-T vs. Post-T) affected the variables describing the subjects baseline physical characteristics or aerobic power. The corresponding comparisons were performed for HRV, hormones and BLa obtained at rest or during the recovery period. Repeated measures analysis of variance with a repeated contrast was used to assess the effects of 'endurance training' and 'exercise intensity' on HRV, respiratory frequency, tidal volume and BLa during the maximal exercise test. This analysis was performed separately for the absolute submaximal exercise intensity (5 work loads, 75–175 W) and for the relative exercise intensity in relation to W_{peak} (7 work loads, 40–100% of W_{peak}). Differences were considered significant when $P < 0.05$.

Results

There were no significant differences in any of the parameters of aerobic power between C and Pre-T. HR, HRV indices, plasma catecholamine concentrations, blood pressure, weight or body fat did not differ statistically between C and Pre-T.

Endurance training

Aerobic power

The endurance training period induced a small increase in aerobic power as expressed by a greater VO_{2peak}, VO_{2AnT}, W_{peak} and W_{AnT} at Post-T than at Pre-T (Table 2). No significant change from Pre-T to Post-T was observed in HR_{peak} [189 (11) vs. 187 (12) bpm] or HR_{AnT} [160 (3) vs. 159 (13) bpm]. Respiratory frequency and tidal volume at the same absolute submaximal exercise intensity levels were not significantly altered after the endurance training period. The endurance training period had no effect on body weight, body fat or resting blood pressure.

Table 2 The parameters of aerobic capacity prior to and after the endurance training period

	Pre-T	Post-T
W_{peak} (W)	254 (29)	285 (30)***
VO_{2peak} (l min^{-1})	2.9 (0.4)	3.1(0.4)***
VO_{2peak} (ml min^{-1} kg^{-1})	37 (4)	39 (4)***
VE_{peak} (L min^{-1}) BTPS	142.0 (22.2)	151.7 (19.3) *
W_{AnT} (W)	179 (23)	203 (24)***
VO_{2AnT} (l min^{-1})	2.1 (0.3)	2.4 (0.2)**
VO_{2AnT} (ml min^{-1} kg^{-1})	27 (4)	30 (4)**
VE_{AnT} (L min^{-1}) BTPS	54.6 (10.4)	60.1 (9.4)*

BTPS Body temperature pressure saturated; *W* power output; VO_2 oxygen uptake; *VE* ventilation; *peak* at the peak level; *AnT* the level corresponding to the anaerobic threshold

Significantly different from Pre-T at *$P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; values are means (SD)

HR and heart rate variability

Rest Table 3 shows the Pre-T and Post-T values of HR and HRV observed in the supine, sitting and standing posture. Neither HR nor HRV were different between Pre-T and Post-T.

Absolute submaximal exercise intensity Figure 1 shows the Pre-T and Post-T values for HR and HRV as a function of absolute submaximal exercise intensity. A main effect for endurance training and for absolute exercise intensity was observed for HR and all HRV indices (Fig. 1). At the same absolute submaximal exercise intensity levels, HR was lower and all HRV indices were higher at Post-T than Pre-T.

Relative exercise intensity Figure 2 shows the Pre-T and Post-T values of HR and HRV as a function of relative exercise intensity. The Post-T values for HR and all HRV indices were similar to Pre-T values at all relative exercise intensity levels. A main effect for relative exercise intensity was found for HR and all HRV indices ($P < 0.001$). HR increased in response to each increment in relative exercise intensity. HFP and LFP decreased in response to an increment in exercise intensity up to the intensity level of 70 and 90% of W_{peak}, respectively. A decrease in TP occurred up to the intensity level of 80% of W_{peak}. At higher exercise

Table 3 Heart rate (HR) and heart rate variability observed in the supine, sitting and standing posture prior to and after the endurance training

	Pre-T	Post-T
HR (bpm)		
Supine	61 (5)	61 (8)
Sitting	68 (5)	67 (9)
Standing	80 (7)	78 (10)
HFP [ln(ms^2)]		
Supine	6.80 (1.02)	6.90 (0.94)
Sitting	6.25 (0.83)	6.53 (1.00)
Standing	5.57 (0.78)	5.75 (0.75)
LFP [ln(ms^2)]		
Supine	6.77 (0.61)	6.77 (0.79)
Sitting	6.96 (0.68)	7.15 (0.66)
Standing	7.09 (0.65)	7.13 (0.71)
TP [ln(ms^2)]		
Supine	7.62 (0.73)	7.69 (0.77)
Sitting	7.47 (0.66)	7.75 (0.62)
Standing	7.38 (0.63)	7.44 (0.64)

Data of one subject was excluded because of a technical artefact. Values are means (SD)

HFP high frequency power, *LFP* low frequency power, *TP* total power

intensity levels, the HRV indices remained unchanged despite the increments in exercise intensity.

Recovery In spite of the higher VO_{2peak} and W_{peak} achieved after the endurance training period, there was no difference in the post-exercise recovery values for HR and HRV between Pre-T and Post-T. At the end of the recovery period, HR remained increased and all HRV indices remained decreased when compared to their resting values ($P < 0.001$) at both Pre-T and Post-T.

Blood lactate and plasma catecholamine concentrations

BLa at rest did not differ between Pre-T and Post-T. BLa at the same absolute exercise intensity levels was significantly lower at Post-T than Pre-T, whereas BLa at the same relative exercise intensity levels was not significantly different between Pre-T and Post-T. The maximal value for BLa increased from 14.0 (2.1) mmol L^{-1} at Pre-T to 15.2 (2.2) mmol L^{-1} at Post-T ($P < 0.01$). Table 4 shows plasma noradrenaline and adrenaline concentrations during the testing sessions. Plasma noradrenaline concentration at rest was higher at Pre-T than Post-T. No difference was observed in plasma adrenaline concentration at rest between Pre-T and Post-T. There was no significant difference between the Pre-T and Post-T values of either plasma noradrenaline or adrenaline concentration after the maximal exercise test.

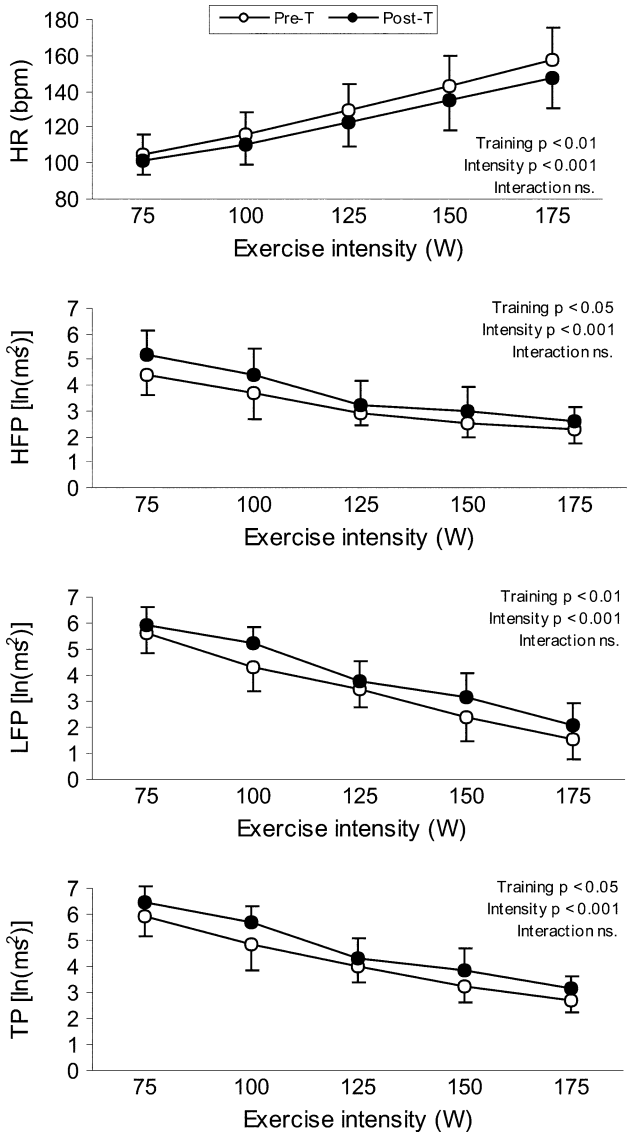

Fig. 1 Heart rate (*HR*) and heart rate variability as a function of submaximal exercise intensity prior to and after the endurance training. *HFP* high frequency power; *LFP* low frequency power; *TP* total power

Discussion

The present study included the 7-week preparatory period and the low-dose 14-week endurance training period. The subjects did not have any background in regular endurance training, and therefore, were taken to represent a reference for evaluation of the effects of low-dose endurance training on autonomic HR control. The preparatory period induced no change in any parameters of aerobic power. The endurance training period resulted in a mean increase of 12% in W_{peak} and of 8% in VO_{2peak} as well as an increase in anaerobic threshold. Thus, the endurance training programme consisting of low to high intensity training twice a week

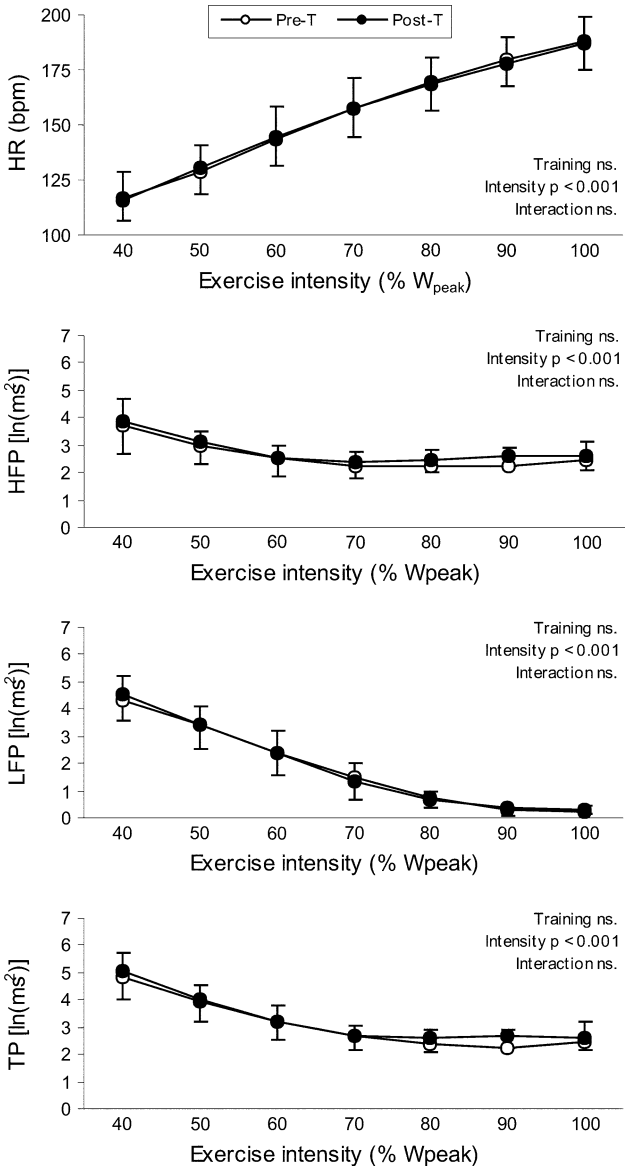

Fig. 2 Heart rate (*HR*) and heart rate variability as a function of relative exercise intensity prior to and after the endurance training. *HFP* high frequency power; *LFP* low frequency power; *TP* total power

was effective in inducing a small but significant increase in aerobic power.

We assessed HRV, and particularly HFP, in order to determine whether the low-dose endurance training period could induce an increase in vagal HR control. The HRV indices at rest showed only a tendency to increase after the low-dose endurance training period. This lack of training-induced changes in HRV is in line with results obtained after endurance training, including three to four bouts per week at moderate intensity (Leicht et al. 2003a; Loimaala et al. 2000). In contrast, more intense training programmes have demonstrated a decrease in HR and an increase in HFP at rest (Carter et al. 2003; Leicht et al. 2003b; Mourot et al. 2004). In these studies, the endurance training programme has included 3–5 weekly exercise sessions at high intensity (>70% of maximal HR) and the duration of the programme has ranged from 6 to 12 weeks. In addition or alternatively to the increase in vagal HR control, endurance training may result in decreased sympathetic HR control and/or lowered intrinsic HR (Katona et al. 1982; Lewis et al. 1980). Therefore, we determined the plasma catecholamine concentration and found a training-induced decrease in plasma noradrenaline concentration at rest. However, even though the plasma noradrenaline concentration decreased slightly after the present training period, the contribution of this decrement to HR at rest was negligible (Table 3).

Table 4 The plasma noradrenaline and adrenaline concentrations prior to and after the endurance training

	Pre-T	Post-T
Adrenaline (nmol L)		
Rest	0.18 (0.05)	0.19 (0.07)
Rec_0	2.52 (1.62)	2.68 (1.46)
Rec_{15}	0.42 (0.31)	0.53 (0.48)
Noradrenaline (nmol L)		
Rest	2.01 (0.63)	1.55 (0.36)*
Rec_0	27.35 (6.66)	24.95 (5.18)
Rec_{15}	5.45 (1.76)	5.55 (1.36)

Rec_0 at the end of the maximal exercise test, Rec_{15} after the 15-min recovery period.

Significantly different from Pre-T at $^*P < 0.05$. Values are means (SD)

During exercise, the training period induced a decrease in HR and an increase in all HRV indices, including HFP, at the same submaximal exercise intensity levels. These findings are in accordance with a longitudinal study by Leicht et al. (2003b) and a cross-sectional study by Tulppo et al. (1998). In contrast to our results, some studies have found no change in HFP at submaximal exercise loads after endurance training, despite decreased submaximal HR (Carter et al. 2003; Leicht et al. 2003a). We measured respiration during the incremental maximal exercise test in order to clarify whether the endurance training programme induced a decrease in respiratory frequency and/or tidal volume at the same exercise intensity levels. Respiration was not altered by the low-dose endurance training programme. These findings suggest that the present increase in HRV at the same absolute submaximal exercise intensities after the endurance training period resulted from the changes in autonomic HR control and was not mediated by training-induced alterations in respiration.

HR and HRV were similar at the same relative exercise intensity prior to and following the low-dose endurance training period. This supports that the relationship between autonomic HR control and relative exercise intensity is not

altered by endurance training (Ekblom et al. 1973). The increase in HR as a function of increased metabolic demand was accompanied by the decrease in HFP to the exercise intensity of 70% peak power, corresponding to an average HR of about 157 bpm. This could reflect a progressive decrease in vagal HR control that occurs mainly at the low exercise intensity levels. A complete vagal blockade produces a HR of about 120–130 bpm (Blomqvist and Saltin 1983; Martinmäki et al. 2006a), and thus, any increase above this level must be mediated by other factors, such as sympathetic and/or intrinsic mechanisms. We further observed that HFP was detectable at each exercise load to the maximal work load in all subjects, even if vagal HR control is negligible at the high exercise intensity levels. In fact, in some subjects HFP even increased at the highest exercise loads. This supports the observations that respiratory frequency and tidal volume can modify HFP during exercise, especially at the intensity levels above the anaerobic threshold (Bernardi et al.1990; Cottin et al. 2006).

There are no data in the literature on the effects of endurance training on HRV during recovery from maximal exercise. In the present study, even if the subjects reached a higher maximal work load after the endurance training, the recovery of their HR and HRV was not affected when compared to the pre-training values.

Taken together, our findings showed that the low-dose endurance training programme induced changes in autonomic HR control during exercise. The training-induced lower HR was accompanied by greater HFP at the submaximal exercise intensity levels. This supports that the lower HR resulted from increased vagal HR control during submaximal exercise following endurance training. However, we cannot exclude that the training-induced lowering in HR during exercise may have been partly due to a decrease in sympathetic HR control. Plasma levels of adrenaline and noradrenaline are lower at any given submaximal exercise intensity level after endurance training (Christensen and Galbo 1983). The present lactate data suggests that there may have been a lower need for metaboreflex-induced sympathetic activation at the same submaximal exercise intensity after the endurance training. There was a decrease in resting plasma noradrenaline concentration with no change in resting HR or HRV after the endurance training. This suggests that sympathetic control at rest decreased slightly, while vagal HR control at rest remained unchanged.

Limitations

Although the present subjects served as their own controls and a preparatory period was included, the number of subjects and the lack of a control group can be seen as limitations. All the subjects became familiarized with low intensity training and the testing procedures during the preparatory period. Thus, the measurements performed after the preparatory period constituted a stable reference level against which the effect of the endurance training could be evaluated. Another limitation is that the present data were collected in conditions with spontaneous respiration. We did not control respiration because it could have disturbed the autonomic modulation during exercise and in the recovery. However, as mentioned, we measured respiratory frequency and tidal volume and found no changes in respiration in response to the low-dose endurance training programme.

In conclusion, we studied the effects of the 14-week endurance training period on autonomic HR control at rest and during exercise at different intensities in untrained male subjects by STFT derived HRV indices. Low to high intensity training twice a week resulted in lower HR and higher HRV at the same absolute submaximal exercise intensity levels but not at rest. This indicates that the training stimulus was sufficient to induce increased vagal HR control during submaximal exercise, but was insufficient to alter resting vagal HR control in the healthy subjects. Thus, vagal HR control may be more responsive to endurance training during exercise than at rest.

Acknowledgments This study was funded by grants from the Ministry of Education, Finland, TEKES-National Technology Agency of Finland, Emil Aaltonen's Foundation, Finland and Finnish Cultural Foundation.

Conflict of interest statement This study was partly funded by grants from Suunto Ltd., Finland, and Firstbeat Technologies Ltd., Finland. Heikki Rusko is currently a stockowner of Firstbeat Technologies Ltd., Finland.

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