

Heart rate variability in prediction of individual adaptation to endurance training in recreational endurance runners

V. Vesterinen¹, K. Häkkinen², E. Hynynen¹, J. Mikkola¹, L. Hokka¹, A. Nummela¹

¹KIHU-Research Institute for Olympic Sports, Jyväskylä, Finland, ²Department of Biology of Physical Activity, University of Jyväskylä, Jyväskylä, Finland

Corresponding author: Ville Vesterinen, MSc, KIHU-Research Institute for Olympic Sports, Rautpohjankatu 6, 40700 Jyväskylä, Finland. Tel: +358 40 543 1273, Fax: +358 20 781 1501, E-mail: ville.vesterinen@kihu.fi

Accepted for publication 1 June 2011

The aim of this study was to investigate whether nocturnal heart rate variability (HRV) can be used to predict changes in endurance performance during 28 weeks of endurance training. The training was divided into 14 weeks of basic training (BTP) and 14 weeks of intensive training periods (ITP). Endurance performance characteristics, nocturnal HRV, and serum hormone concentrations were measured before and after both training periods in 28 recreational endurance runners. During the study peak treadmill running speed (V_{peak}) improved by $7.5 \pm 4.5\%$. No changes were observed in HRV indices after BTP, but after ITP, these

indices increased significantly (HFP: 1.9%, $P = 0.026$; TP: 1.7%, $P = 0.007$). Significant correlations were observed between the change of V_{peak} and HRV indices (TP: $r = 0.75$, $P < 0.001$; HFP: $r = 0.71$, $P < 0.001$; LFP: $r = 0.69$, $P = 0.01$) at baseline during ITP. In order to lead to significant changes in HRV among recreational endurance runners, it seems that moderate- and high-intensity training are needed. This study showed that recreational endurance runners with a high HRV at baseline improved their endurance running performance after ITP more than runners with low baseline HRV.

Regular aerobic endurance training and good maximal aerobic performance are widely accepted as factors that reduce all-cause mortality and improve a number of health outcomes (Kesaniemi et al., 2001). Numerous studies have shown that long-term endurance training induces many physiological adaptations leading to improved endurance performance (McArdle et al., 1996; Iwasaki et al., 2003; Purge et al., 2006; Scharhag-Rosenberger et al., 2009). The majority of the published studies have focused on main effects of endurance training and group differences while paying little attention to individual differences in training adaptation. However, it has been shown that individuals may adapt differently after exposure to very similar training loads. Although mean improvements in maximal oxygen uptake ($\text{VO}_{2\text{max}}$) following 6–20 weeks of standardized training have been within 10–15% of baseline values, individual adaptations have been shown to range from negative values to over 40% improvement (Bouchard & Rankinen, 2001; Hautala et al., 2003, 2009; Vollaard et al., 2009; Buchheit et al., 2010).

Physiological mechanisms causing the remarkable heterogeneity in the responsiveness to endurance training remain partly unclear. It has been proposed that many factors, like genetics, age, gender, nutrition,

prior training, fitness level, sleep, rest, and stress can result in great variation in the adaptation to endurance training (Bouchard & Rankinen, 2001; Hedelin et al., 2001; Hautala et al., 2003, 2009; Buchheit et al., 2010; Nummela et al., 2010). However, Bouchard and Rankinen (2001) summarized that age, gender, race, and baseline fitness level together accounted for only 11% of the variance in the adaptation to standardized endurance training. In addition, it has been reported that serum hormone concentrations may be associated with the endurance training adaptation. Endurance training which has led to a negative training adaptation also results in decreased basal levels of serum testosterone concentration (Wheeler et al., 1991; Urhausen et al., 1995; Hoogeveen & Zonderland, 1996; Uusitalo et al., 1998). It has also been suggested that the increase in serum testosterone represents a positive adaptation to the training load (Purge et al., 2006). However, previous studies are partly contradictory and the possible association between hormone concentrations and the endurance training adaptation may be rather complicated.

It has been suggested that cardiovascular autonomic regulation is an important determinant of training adaptation (Hautala et al., 2009). Several studies have shown that endurance training increases

heart rate variability (HRV) (Buchheit et al., 2004; Kiviniemi et al., 2006; Nummela et al., 2010). In addition, increased HRV and high baseline HRV have been observed to be associated with improvement in endurance performance (Boutcher & Stein, 1995; Hedelin et al., 2001; Hautala et al., 2003; Buchheit et al., 2010; Nummela et al., 2010). Hautala et al. (2003) found that high frequency power (HFP) was the most powerful determinant associated with future training adaptation, accounting for 27% of the variance in the adaptation to endurance training.

However, most of the previous studies which have investigated determinants of endurance training adaptation have used relatively short training periods (<8 weeks). Less is known about predictors of the adaptation to long-term endurance training. Loimaala et al. (2000) investigated effects of 5 months of high- and low-intensity endurance training on HRV, but did not find any changes in nocturnal or 24h HRV indices regardless of improvements in endurance performance. Iwasaki et al. (2003) observed that previously sedentary people improved endurance performance over a whole 1-year training period but resting HRV, as measured during a 6-min period in the morning, increased only during the first 3 months. Association between the training adaptation and HRV after prolonged endurance training is partly unclear. In addition, sedentary people have been subjects in the major part of the previous studies but less is known about determinants of the long-term endurance training adaptation among recreationally trained endurance runners. Previous studies have reported that cardiac vagal modulation of HR during exercise and at rest, as determined by HRV, is higher in trained than in sedentary subjects (Buchheit & Gindre, 2006; Hautala et al., 2009; Buchheit et al., 2010). It is also clear that fitness level differs greatly between sedentary people and endurance trained runners. Based on the previous findings, determinants of the adaptation to endurance training may be different among different populations.

The aims of this study were (1) to investigate whether nocturnal HRV can be used to predict changes in endurance performance and (2) to assess baseline determinants which are associated with the training adaptation and can be used to predict the individual training adaptation during long-term endurance training in recreational endurance runners. It was hypothesized that nocturnal HRV at baseline will be correlated to the individual training adaptation.

Methods

Subjects

Twenty-eight male recreational endurance runners were recruited to the study. All subjects (age: 36 ± 6 years, height: 1.79 ± 0.05 m, body mass: 78.1 ± 5.6 kg) participated in a marathon-training project, which prepared them for a marathon run at the end of the project. All subjects were healthy, non-smokers, non-obese (BMI <30 kg/kg), and they did not have any diseases or use regular medication. In addition, resting ECG (Cardiofax ECG-9 320, Tokyo, Japan) was analyzed to ensure they had no cardiac abnormalities, which would have affected the HRV analysis or preclude them from participating in intense endurance training. According to a questionnaire about prior endurance training activity, the subjects had trained primarily running on average 4.4 ± 0.8 times/week during the last 2 months before the study. Most of the subjects had a training background of many years and had already run at least one half or full marathon before they volunteered for this study. One subject dropped out because of a lack of motivation, and two subjects were excluded because of insufficient compliance with the training during the study. Finally, 25 men were included in the study. Subjects were fully informed about the study design, including information on the possible risks and benefits, before signing an informed consent document. The study was approved by the Ethics Committee of the University of Jyväskylä, Finland.

Experimental design and training

The subjects took part in a 28-week training program (Table 1). The training program was divided into a 14-week basic training period (BTP) and a 14-week intense (increased running volume and intensity) training period (ITP). In BTP, the subjects were asked to maintain the same training volume as before the study (3–6 times/week). During BTP, training was

Table 1. Week template of training over 28 weeks of training program

	Basic training period		Intense training period	
	Weeks 1–14	Weeks 15–19	Weeks 20–24	Weeks 25–28
Week periodization (intense weeks : recovery week)	3:1	2:1	2:1	2:1
High-intensity runs	None	None	1 session*, 4–5 km	2 sessions*, 4–5 km
Moderate-intensity runs	None	2 sessions*, 8–10 km	1 session*, 8–10 km	None
Long low-intensity run	1 session, 15–20 km	1 session, 20–25 km	1 session, 20–30 km	1 session, 25–30 km
Basic low-intensity runs	2–5 sessions, 5–15 km	1–4 sessions, 5–15 km	1–4 sessions, 5–15 km	1–4 sessions, 5–15 km
Strength training	1–2 sessions	1 session	1 session	1 session

*Exercises were not performed during recovery weeks.

High-intensity, intensity above anaerobic threshold; Moderate-intensity, intensity between aerobic and anaerobic thresholds; Low-intensity, intensity below aerobic threshold.

performed primarily below the aerobic threshold (avg. 64% V_{peak}), which was individually determined for each subject from the incremental treadmill test (Aunola & Rusko, 1986). The training program of BTP was periodized to cycles of 4 weeks, thus 3 weeks of intense training was followed by an easy training week. Endurance training consisted primarily of running but occasionally included also cycling, nordic walking and/or cross country skiing. In addition, the subjects were asked to complete strength training 1–2 times/week. A training program of the 14-week ITP included higher running training volume (prolonged duration of the training sessions) and intensity compared with the basic training period. The training utilized the 3-week training cycles (2 intense weeks followed a recovery week). During the recovery weeks the subjects were asked to train at low-intensity [below the aerobic threshold, Aunola & Rusko (1986)], but during the first 3 intense weeks the subjects replaced two low-intensity training sessions with moderate-intensity [between the aerobic and anaerobic thresholds (avg. 67–84% V_{peak}); Aunola & Rusko, 1986] training sessions per week. During the next 3 intense weeks the subjects were asked to complete one moderate- and one high-intensity (above anaerobic threshold) training sessions per week beyond low-intensity sessions. During the last 3 intense weeks they replaced two low-intensity training sessions with high-intensity training sessions per week. In addition, the subjects were asked to complete one strength training session per week throughout the intense training period.

The subjects controlled their training intensity by measuring their HR during all exercises using Suunto t6 heart rate monitors and GPS pod speed/distance sensors (Suunto Ltd., Vantaa, Finland). Subjects kept a training diary throughout the study recording training mode, duration of the training session, average HR and running distance. In addition, the subjects rated their perceived exertion (RPE) using the scale from 0 to 10 after each training session (Borg, 1982). HR data was used for determining the times at the three different intensity zones; low (below aerobic threshold), moderate (between aerobic and anaerobic thresholds) and high (above anaerobic threshold) intensities. Training impulse (TRIMP), an index of training load, was calculated by using the following formula (Banister, 1991):

$$\begin{aligned} \text{TRIMP} = & t [\text{exercise duration (min)}] \\ & \times (\text{HR}_{\text{exercise}} - \text{HR}_{\text{rest}}) / (\text{HR}_{\text{max}} - \text{HR}_{\text{rest}}) \\ & \times (0.64 \times e^{[1.92 \times (\text{HR}_{\text{exercise}} - \text{HR}_{\text{rest}})]} \\ & / (\text{HR}_{\text{max}} - \text{HR}_{\text{rest}})) \end{aligned}$$

Anthropometry and resting blood samples

All measurements were performed before and after both training periods (at weeks 0, 14 and 28). In addition to height; body mass and body composition were measured using bioimpedance (In body 720 body composition analyzer, Biospace Co. Ltd., Seoul, South Korea). The measurements were performed after 10 h of fasting in the morning between 7:30 and 8:30 hours. After the bioimpedance measurements, venous blood samples (10 mL) were collected into serum tubes (Venosafe, Terumo Medical Co., Leuven, Belgium) for the determination of basal serum testosterone and cortisol concentrations. The whole blood was centrifuged at 2500 g (Megafuge 1.0R, Heraeus, Germany) for 10 min after which serum was removed and stored at -80°C until analysis. The concentrations of testosterone and cortisol were determined by using a chemical luminescence techniques (Immunitite 1000, DPC Diagnostics Corporation, Los Angeles, California, USA) and hormone-specific immunoassay kits (Siemens, New York, New York, USA). The sensitivity of testosterone

and cortisol assays was 0.05 and 5.5 nmol/L, respectively. The intra-assay coefficients of variation for testosterone and cortisol were 3.9% and 4.6%, respectively. All the assays were carried out according to instructions of the manufactures. All samples of the test subject were analyzed in the same assay for each hormone.

Incremental treadmill test

The initial velocity was 8 km/h and was increased by 1 km/h every third minute until exhaustion. The incline was kept at 0.5° during the whole test. HR was recorded continuously using a heart rate monitor (Suunto t6, Suunto Ltd.). Oxygen consumption was measured breath-by-breath throughout the test using a portable gas analyzer (Oxycon Mobile[®], Jaeger, Hoechberg, Germany). After each 3-min stage, the treadmill was stopped for about 15–20 s for fingertip blood samples (20 μL) and blood lactate (La) analysis. Blood lactate was determined using Biosen S_line Lab+lactate analyzer (EKF Diagnostic, Magdeburg, Germany). The highest 60-s VO_2 value during the treadmill test was considered as maximal oxygen uptake ($\text{VO}_{2\text{max}}$). The maximal endurance performance was determined as the peak treadmill running speed (V_{peak}) when the subject became exhausted. If the subject could not complete the whole 3 min of the last velocity, V_{peak} was calculated as follows: speed of the last completed stage (km/h) + (running time (s) of the speed at exhaustion $- 30$ s / 180 $- 30$ s) \times 1 km/h. In the present study a change of V_{peak} , which has been shown to be closely related to maximal endurance performance (Noakes et al., 1990), was used as the main variable for describing the adaptation to endurance training during the training periods. Aerobic (AerT) and anaerobic (AnT) thresholds were determined using La, ventilation, VO_2 and VCO_2 (production of carbon dioxide) (Aunola & Rusko, 1986). The running economy (RE) was determined as the average VO_2 from the last minute at the velocity of 10 km/h.

Nocturnal HRV

Nocturnal R–R interval (RRI) recordings were taken during three consecutive nights before and after both training periods with Suunto Memory Belt (Suunto Ltd.) having a sampling frequency of 1000 Hz. Nocturnal RRI data were recorded after a light training day according to TRIMP. RRI recordings were started before going to bed to sleep and stopped after waking up in the morning. The first 30 min after going to bed was excluded and the succeeding 4 h were accepted for the analysis. RRI data was analyzed using the Firstbeat Health software (version 3.0.1.0, Firstbeat Technologies Ltd., Jyväskylä, Finland). RRIs were checked and edited by an artifact detection filter of the Firstbeat Health software and subsequently verified by visual inspection to exclude all falsely detected, missed, and premature heart beats (Saalasti, 2003). The consecutive artifact corrected RRI data were then re-sampled at the rate of 5 Hz by using linear interpolation to obtain equidistantly sampled time series. From the resampled data, the software calculated HRV indices second-by-second using the short-time Fourier Transform method. For a given segment of data, a time window (Hanning) with a length of 256 samples was applied, fast Fourier transform was calculated and a power spectrum was obtained. The window was then shifted one sample to another and the same process was repeated. The following HRV indices were analyzed with time and frequency domain methods: average HR, standard deviation of RRI (SDNN), root mean square of differences between adjacent R–R intervals (RMSSD), low frequency power (LFP; 0.04–0.15 Hz), high frequency power (HFP; >0.15 –0.40 Hz),

total power (TP = LFP+HFP; 0.04–0.40 Hz). The results are provided as averages of two nights, since there were so many erroneous RRI recordings that it was not possible to use averages of three nights on all subjects.

Statistical analysis

Values are expressed as mean \pm standard deviation (SD) and 95% confidence interval (CI) for mean. The Gaussian distribution of the data was assessed with the Shapiro–Wilk goodness-of-fit test. Ln-transformation was used with the nocturnal HRV variables, in order to meet the assumptions of parametric statistical analysis. Repeated-measures analysis of variance (ANOVA) was used for statistical testing, followed by Bonferroni as a *post hoc* test. Pearson's product moment correlation coefficient was used to determine the relationships between the baseline characteristics and the training adaptation. Correlations between HRV and the training adaptation were adjusted by age using partial correlations due to effects of age on baseline HRV. The data were analyzed using SPSS software (PASW Statistics 18.0; SPSS Inc., Chicago, Illinois). Statistical significance was accepted as $P < 0.05$.

Results

Training volume variables (h/week, times/week) did not differ between the two training periods (Table 2). TRIMP ($P = 0.027$), running volume ($P < 0.001$) and training intensity variables [average HR ($P < 0.001$), %HR_{max} ($P < 0.001$), percentage at moderate ($P < 0.001$) and high intensities ($P = 0.004$)] were greater in ITP compared with BTP.

Body mass at weeks 0, 14 and 28 were 78.1 ± 5.6 , 77.5 ± 5.5 and 76.5 ± 5.7 kg. Body mass after ITP was significantly smaller compared with the baseline level ($P < 0.001$) and after BTP ($P = 0.009$). Body fat% was lower after BTP ($16.7 \pm 5.1\%$, $P = 0.011$) and ITP ($16.3 \pm 5.4\%$, $P = 0.012$) compared with the baseline value (17.7 ± 5.1). There were no differences in basal levels of serum testosterone at weeks 0, 14

and 28 (16.2 ± 3.4 , 17.1 ± 4.2 , 16.5 ± 4.0 nmol/L and cortisol (442 ± 79 , 437 ± 119 , 438 ± 82 nmol/L, respectively).

All subjects successfully completed a marathon ($n = 22$) or half-marathon ($n = 3$) as the main performance goal of the training program. Mean marathon time improved by 8.2% compared with the previous personal best (241 ± 23 vs 221 ± 24 min, $P < 0.001$). V_{peak} improved by $7.5 \pm 4.5\%$ ($P < 0.001$, min–max: -3.7 – 13.2%) and $\text{VO}_{2\text{max}}$ by $5.1 \pm 6.2\%$ ($P < 0.001$, min–max: -3.9 – 20.2%) during the 28 weeks of training (Table 3). Velocities at anaerobic and aerobic thresholds increased by $12.4 \pm 6.5\%$ ($P < 0.001$) and $15.5 \pm 8.4\%$ ($P < 0.001$), respectively. The individual heterogeneity of training adaptation during both training periods is presented in Fig. 1. The mean increase in V_{peak} was $4.1 \pm 3.1\%$ ($P < 0.001$) during BTP and $3.3 \pm 3.6\%$ ($P < 0.001$) during ITP. The improvement did not differ between the training periods. In addition, velocities at AerT increased by $9.1 \pm 7.3\%$ ($P < 0.001$) in BTP and $5.9 \pm 5.3\%$ ($P < 0.001$) in ITP and AnT $8.2 \pm 6.4\%$ ($P < 0.001$), $4.0 \pm 4.5\%$ ($P < 0.001$), respectively. RE improved ($3 \pm 5\%$, $P = 0.002$) only during ITP.

No changes were observed in nocturnal HR and HRV indices during BTP (Table 4). After the 28-week training resting HR ($P = 0.037$) decreased and SDNN ($P = 0.013$) and RMSSD ($P = 0.001$) increased compared with the baseline level. In addition, HFP ($P = 0.026$) and TP ($P = 0.007$) increased significantly during ITP.

Age did not correlate with the training adaptation (the change in V_{peak}) in either BTP ($r = -0.11$) or ITP ($r = 0.16$). In addition, the previous training activity ($r = -0.11$, $r = 0.13$) and the baseline endurance performance ($r = -0.06$, $r = 0.02$), as well as any training volume or intensity variables did not correlate with the training adaptation in either periods. A good correlation was observed between the change in V_{peak} during ITP and HRV indices at the baseline measurement (Fig. 2, Table 5). The strongest relationship ($r = 0.75$, $P < 0.001$) was between the change in V_{peak} and TP at baseline [Fig. 2(b)]. No significant correlations between these parameters were found during BTP [Fig. 2(a)]. However, a weak trend was observed between the baseline testosterone level and the training adaptation ($r = 0.41$, $P = 0.085$). The change of RE did not correlate significantly to any baseline characteristics in either periods.

Discussion

The main findings of the present study showed that nocturnal HRV at baseline was associated with the endurance training adaptation in ITP, not in BTP (Table 5). The present results thus suggest that

Table 2. Training data of the subjects in the training periods are means \pm SD (95% CI)

	Basic training period	Intense training period
Training volume		
h/week	5.8 ± 1.8 (5.1–6.6)	5.5 ± 1.7 (4.8–6.2)
times/week	4.6 ± 0.9 (4.2–5.0)	4.2 ± 0.9 (3.8–4.5)
TRIMP (a week)	379 ± 113 (333–426)	$421 \pm 119^*$ (372–470)
Running km (a week)	26.4 ± 12.2 (21–31)	$39.9 \pm 14.6^{***}$ (34–46)
Average heart rate (bpm)	127 ± 7 (124–130)	$133 \pm 9^{***}$ (130–137)
Average heart rate (%HR _{max})	68 ± 3 (67–69)	$72 \pm 4^{***}$ (70–73)
RPE (0–10+)	4.6 ± 1.3 (4.1–5.2)	4.8 ± 1.3 (4.2–5.4)
HR below AerT in running (%)	86 ± 12 (81–91)	$73 \pm 14^{***}$ (67–79)
HR between AerT and AnT in running (%)	13 ± 12 (8–18)	$24 \pm 14^{***}$ (18–30)
HR above AnT in running (%)	1 ± 1 (0–2)	$2 \pm 3^{**}$ (1–4)

Significant difference between the periods:

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

TRIMP, training impulse; RPE, rate of perceived exertion; HR, heart rate; AerT, aerobic threshold; AnT, anaerobic threshold.

Table 3. Performance parameters of the incremental treadmill test are means \pm SD (95% CI)

	Baseline	Week 14	Week 28
VO _{2max} (mL/kg/min)	49 \pm 4(48–51)	51 \pm 4** (50–53)	52 \pm 5** (50–54)
V _{peak} (km/h)	14.7 \pm 1.0 (14.3–15.2)	15.3 \pm 1.1*** (14.9–15.8)	15.8 \pm 1.2***,### (15.3–16.3)
vAnT (km/h)	12.0 \pm 1.2 (11.5–12.5)	12.9 \pm 1.1*** (12.5–13.4)	13.4 \pm 1.0***,### (13.0–13.8)
VAerT (km/h)	9.4 \pm 0.9 (9.0–9.8)	10.2 \pm 1.0*** (9.8–10.6)	10.8 \pm 0.9***,### (10.4–11.2)
RE (mL/kg/km)	221 \pm 13 (215–226)	219 \pm 16 (213–225)	208 \pm 13***,## (203–214)

Significant difference from baseline:

** $P < 0.01$, *** $P < 0.001$.

Significant difference from week 14:

$P < 0.01$, ### $P < 0.001$.

VO_{2max}, maximal oxygen consumption; V_{peak}, peak treadmill running speed; vAnT, velocity at anaerobic threshold; vAerT, velocity at aerobic threshold; RE, running economy.

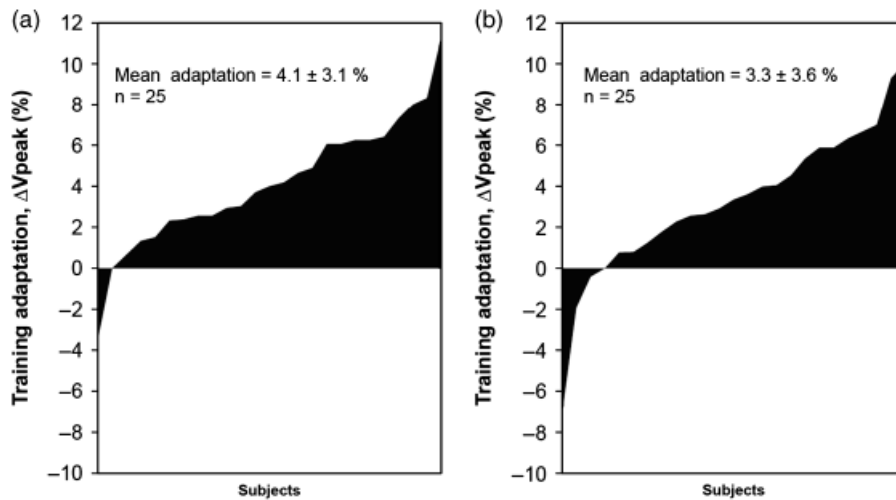


Fig. 1. Heterogeneity of training adaptations (ΔV_{peak}) during the basic training period (a) and the intense training period (b) in recreational endurance runners.

Table 4. Nocturnal HR and HRV values are means \pm SD (95% CI)

	Baseline	Week 14	Week 28
HR (bpm)	51.1 \pm 4.4 (49.0–53.3)	49.9 \pm 6.1 (47.0–52.9)	48.9 \pm 5.5* (46.3–51.6)
SDNN (ms)	134 \pm 20 (124–143)	140 \pm 28 (127–154)	152 \pm 31* (137–167)
RMSSD (ms)	84 \pm 25 (73–96)	93 \pm 36 (76–111)	109 \pm 41** (90–129)
LFP (ln ms ²)	8.36 \pm 0.47 (8.14–8.59)	8.29 \pm 0.45 (8.08–8.51)	8.42 \pm 0.40 (8.23–8.62)
HFP (ln ms ²)	8.02 \pm 0.64 (7.72–8.33)	8.06 \pm 0.68 (7.73–8.39)	8.21 \pm 0.67# (7.89–8.54)
TP (ln ms ²)	9.01 \pm 0.50 (8.77–9.25)	8.99 \pm 0.53 (8.74–9.25)	9.14 \pm 0.49## (8.91–9.38)

Significant difference from baseline:

* $P < 0.05$, ** $P < 0.01$.

Significant difference from week 14:

$P < 0.05$, ## $P < 0.01$.

HR, heart rate; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; LFP, low-frequency power; HFP, high-frequency power; TP, total power.

moderate- and high-intensity training is needed for significant changes in vagal activity of cardiovascular autonomic regulation to occur among recreational endurance runners. In addition, the present results suggest that progressively increased training load led to the prolonged endurance training adaptation during 28 weeks of endurance training.

Individual adaptation to training

In the present study, recreational endurance runners trained the first 14 weeks at low-intensity followed by 14 weeks of a combination of low-, moderate- (11 sessions) and high-intensity (seven sessions) training. All endurance performance characteristics improved

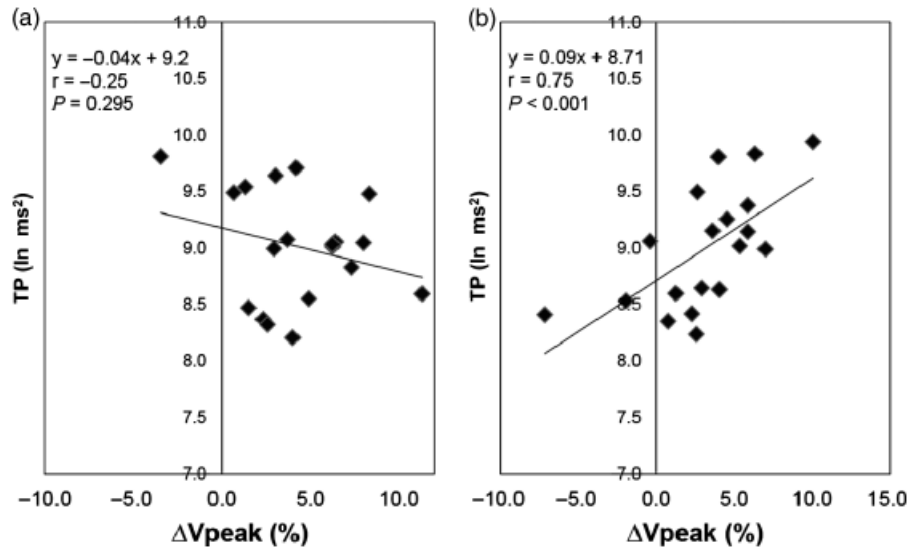


Fig. 2. Correlation between the interindividual training adaptation (ΔV_{peak}) and baseline heart rate variability (TP, total power) after adjustment on age in the basic training period (a) and the intense training period (b).

Table 5. Correlations between endurance training adaptation (ΔV_{peak}) and baseline hormones and heart rate variability

	Basic training period	Intense training period
Cortisol (nmol/L)	0.01	-0.18
Testosterone (nmol/L)	0.32	0.27
HR (bpm)	-0.21	-0.44
SDNN (ms)	-0.02	0.48*
RMSSD (ms)	-0.11	0.57*
LFP (ln ms ²)	-0.17	0.69**
HFP (ln ms ²)	-0.29	0.71***
TP (ln ms ²)	-0.25	0.75***

Significant correlation:

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

Correlations to HRV indices were adjusted for age.

V_{peak} , peak treadmill running speed; HR, heart rate; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; LFP, low-frequency power; HFP, high-frequency power; TP, total power.

continuously throughout the whole 28 weeks of training, except RE which improved only during ITP. Mean improvements were 5–8% in peak treadmill running speed ($VO_{2\text{max}}$, V_{peak}) and 12–15% at submaximal velocities (v_{AnT} , v_{AerT}) including large individual variation in the training adaptation. Three subjects (12%) could not improve their endurance performance during the study. This possibly explains why the mean improvement in $VO_{2\text{max}}$ was slightly smaller compared with previous long-term studies (Loimaala et al., 2000; Iwasaki et al., 2003; Scharhag-Rosenberger et al., 2009). In the study of Loimaala et al. (2000) an 11% improvement was observed in the low-intensity group (4–6 times/week at 55% of HR_{max}) and 15% improvement in the high-intensity group (4–6 times/week at

75% of HR_{max}) during 5 months of endurance training among previously untrained. Iwasaki et al. (2003) found that $VO_{2\text{max}}$ increased 16% after 6 months and 20% after 1 year of progressive endurance training among sedentary subjects. It is rational that sedentary subjects in the previous studies (Loimaala et al., 2000; Iwasaki et al., 2003) improved more than recreational endurance runners in the present study because of remarkably lower baseline endurance performance level and minor training background. Scharhag-Rosenberger et al. (2009, 2010) reported a 10% increase in V_{peak} and unchanged $VO_{2\text{max}}$ in four of the 18 untrained individuals (22%) after a 12 months endurance training period when training load remained constant (3 times/week, 45 min/session at 60% heart rate reserve) during whole 50 weeks of training. The authors concluded that beginners in recreational endurance exercise are advised to increase their training stimulus after 6 months of training to maintain the effectiveness of training (Scharhag-Rosenberger et al., 2009, 2010). The present results support the previous finding that progressively increased training load and intensity is beneficial for the continuous endurance training adaptation during long-term training.

HRV and endurance training adaptation

The association between HRV indices and the endurance training adaptation has been widely observed (Boutcher & Stein, 1995; Aubert et al., 2003; Hautala et al., 2003; Buchheit & Gindre, 2006; Buchheit et al., 2010; Nummela et al., 2010). However, most of the previous training studies have been relatively short (<8 weeks) and intensity of training have been mainly limited to moderate or vigorous

intensity. We observed a significant correlation between the baseline HRV indices (LFP, HFP, TP) and the endurance training adaptation only during ITP. TP showed to have the strongest relationship with a change in V_{peak} ($r = 0.75$, $P < 0.001$), which accounts for 56% of the variance in the adaptation to endurance training. Our finding is in line with Hautala et al. (2003) and Boutcher & Stein (1995) who showed that high resting HRV at baseline was associated with good adaptation to 8-week endurance training period among sedentary males. Based on the present findings, the same association appeared among recreational endurance runners but only when training included moderate- and high-intensity training. In previous long-term (> 5 months) endurance training studies, improvements in endurance performance were found but no associations between HRV indices and the adaptation (Loimaala et al., 2000; Iwasaki et al., 2003). However, Hedelin et al. (2001) found that subjects who increased their $\text{VO}_{2\text{max}}$ during the 7-month training period showed higher HFP and TP values throughout the study compared with those who showed reduced $\text{VO}_{2\text{max}}$ in regional and national level cross country skiers and canoeists. Manzi et al. (2009) found a curvilinear dose-response relationship between individualized training load and HRV indices. It was observed that an increase in normalized LFP at peak exercise training could predict improvement in recreational athletes, which the authors interpreted to reflect enhanced sympathetic modulation (Manzi et al., 2009). On the other hand, the opposite relationship (the subjects with the lower vagal modulation improved more, $r = 0.82$) has also been reported during an 8-week endurance training period in moderately trained runners (Buchheit et al., 2010). Buchheit et al. (2010) concluded that the association is more likely to be related to the interdependence of cardiac autonomic control and aerobic performance than to an individual trainability component *per se* which has been expressed by Hautala et al. (2003). However, the findings of the present study do not support the conclusion of Buchheit et al. (2010), because endurance performance at baseline was not associated with the endurance training adaptation. The differences between the studies might be partly explained by different HRV recording methods. In the study of Buchheit et al. (2010) HRV indices were calculated during a 5-min rest period immediately after awakening in the mornings, whereas in the study of Manzi et al. (2009) HRV recordings were performed over a 10-min rest period in the afternoon. In the present study HRV was analyzed over the 4-h period during nights. Hautala et al. (2003) reported that baseline HFP during nighttime was the most powerful HRV index associated with the future training adaptation com-

pared with HRV during daytime or 24-h recording. As suggested previously (e.g. Pichot et al. 2000) the nighttime reflects a more standardized condition, and the results are less influenced by the subject's behavioral pattern. Based on these findings, it seems that high nocturnal HRV at the baseline is related to the positive adaptation to intensive endurance training. On the other hand, low HRV may reflect limitations in the capacity to improve the cardiorespiratory fitness, as suggested previously by Hautala et al. (2003). However, the mechanisms underlying the association between the baseline vagal activity and the training adaptation remain unclear and should be clarified in future studies.

It has been widely reported that endurance training increases HRV indices (Buchheit et al., 2004; Kiviniemi et al., 2006; Nummela et al., 2010). However, decrements have also been observed in HRV indices during very intensive training with insufficient recovery (Pichot et al., 2000; Portier et al., 2001; Iellamo et al., 2002; Manzi et al., 2009). Pichot et al. (2000) observed that a decrease in nocturnal HRV was followed by a significant increase during the easy training week in middle-distance runners. Effects of long-term endurance training on HRV are partly unclear. Loimaala et al. (2000) did not find any changes in HRV, measured over 24-h period, during 5 months of either high- or low-intensity endurance training, and Iwasaki et al. (2003) found increases in SDNN and LFP during the first 6 months but no changes in HRV during the last 6 months. The authors concluded that more prolonged and intense training does not necessarily lead to greater enhancement of the changes in LFP and HFP. The authors expressed an explanation that after 12 months of intense training, subjects may have been slightly overtrained which could explain unchanged HRV during the last 6 months of training (Iwasaki et al., 2003). Contrary to the study of Iwasaki et al. (2003), we observed unchanged HRV during the first 14 weeks of low-intensity training and significantly increased HRV indices (except in LFP) during ITP. However, it has to be taken into consideration that different HRV recording methods were used in the present study (nocturnal 4-h recording) compared with the study of Iwasaki et al. (2003) (6 min paced breathing recording in the mornings). The present findings suggest that moderate- and high-intensity training is needed for significant changes in markers of vagally mediated regulation of the cardiovascular system to occur among recreational endurance runners. It seems that low-intensity training had no effect on the homeostasis of cardiovascular autonomic function during nocturnal rest. On the other hand, endurance performance improved also during BTP although training frequency (4.6 ± 0.9 times/week) did not change compared with preceding

training (4.4 ± 0.8 times/week) of the study. The observation that peak treadmill running speed improved without changes in nocturnal HRV during BTP may be partly explained by regular strength training which may have enhanced the function of the neuromuscular system, and by a learning effect between the first two running tests on treadmill. However, this protocol does not provide a comprehensive explanation for this observation.

Baseline characteristics in prediction of training adaptation

Age has been proposed to be one of the most powerful predictors of the training adaptation (Bouchard & Rankinen, 2001; Hautala et al., 2003). Bouchard & Rankinen (2001) observed that age accounted for 4% and Hautala et al. (2003) for 16% of the endurance training adaptation. Contrary to these previous studies (Bouchard & Rankinen, 2001; Hautala et al., 2003), age was not associated with the training adaptation, contributing only 1.1% to the adaptation in BTP and 2.6% in ITP. The longer duration of the present study compared with the studies of Bouchard & Rankinen (2001) (20 weeks) and Hautala et al. (2003) (8 weeks) may partly explain a smaller contribution of age. In addition, the relatively narrow range of age (20–45 years) in this study may explain that observation. On the other hand, the range of age has also been limited in the studies of Bouchard & Rankinen (2001) (17–29 years) and Hautala et al. (2003) (23–52 years). We also observed that the baseline endurance performance was not significantly associated with the training adaptation which is in agreement with Hautala et al. (2003) and Bouchard & Rankinen (2001). In addition, the previous training activity was not associated with the improvement in endurance performance. This might be explained by the homogenous group of subjects according to their training background, and the individualized training program used in this study.

Association between hormone concentrations and endurance training adaptation

It has been widely reported that endurance training decreases testosterone concentration, especially in the case of overtraining (Wheeler et al., 1991; Urhausen et al., 1995; Hoogeveen & Zonderland, 1996; Uusitalo et al., 1998). However, Purge et al. (2006) and Grandys et al. (2009) have found increases in testosterone and cortisol concentrations in elite male rowers during a 24-week training (Purge et al., 2006), and in physically active men during a 5-week low-intensity endurance training period (Grandys et al., 2009). Purge et al. (2006) concluded that the increase in testosterone represents a positive adaptation to the training load. In addition, an

increase in testosterone concentration was observed during an 18–20-month training period in previously untrained males and females preparing for a marathon (Keizer et al., 1989). On the other hand, Hoogeveen & Zonderland (1996) found that a decrease in testosterone levels did not lead to a decrease in endurance performance among professional cyclists. In the present study, positive adaptation to training was found in both training periods but no changes in basal levels of testosterone and cortisol hormones were found. It is possible that training status and fitness level of the subjects may partly explain contradictory observations about effects of endurance training on the hormonal levels. In the present study, a trend was observed in association between the training adaptation and the baseline testosterone level in BTP but not in ITP. Based on that observation, it seems that high baseline testosterone level might be beneficial for the endurance training adaptation. The explanation for that might be related to a stimulatory effect of testosterone on erythropoiesis (Shahidi, 2001). On the other hand, the low level of blood testosterone may reflect limited trainability. However, Uusitalo et al. (1998) observed marked individual differences in hormonal changes during a heavy endurance training period and concluded that individual hormonal profiles are needed to follow-up training effects. However, in the present study basal serum testosterone and cortisol concentrations were determined only three times which did not provide reliable protocol to follow-up the training adaptation. Future studies are needed to show whether acute hormonal responses to standardized exercise sessions extend information about the importance of testosterone and cortisol concentrations for improvement in the endurance adaptation.

Perspectives

The current study shows that a 28-week program consisting of a combination of low-, moderate- and high-intensity training and a progressively increased training load led to improved endurance running performance in recreational endurance runners. While there is a general improvement, large variation exists in each individuals adaptation to training, supporting the results observed in previous studies (Bouchard & Rankinen, 2001; Hautala et al., 2003, 2009; Vollaard et al., 2009; Buchheit et al., 2010). Mechanisms resulting in remarkable variation in the responsiveness to endurance training remain partly unclear. It has been suggested that cardiovascular autonomic regulation is an important determinant of training adaptation (Hautala et al., 2009). However, most of the previous studies have used relatively short training periods and subjects have

been sedentary people. The findings of this study support the hypothesis that cardiovascular autonomic regulation, as measured by HRV, is potentially an important tool for monitoring how individuals adapt to training programs. As high HRV at baseline was associated with good training adaptation after high-intensity training, low HRV seems to indicate poor training adaptation possibly caused by a state of fatigue. This is in line with the research by Lamberts et al. (2010a, b) and suggests that HRV at baseline can potentially be a useful method to prescribe training and monitor fatigue. The findings of this study also support this hypothesis among trained individuals during prolonged training as it shows that vagal activity of nocturnal cardiovascular autonomic regulation increases and high HRV at baseline is associated with improvements in endurance performance when training is

intensive. It is possible that low HRV can predict an inability to cope with the training load and the accumulation of fatigue. It seems that nocturnal HRV may serve a useful method for predicting individual adaptation to prolonged endurance training.

Key words: endurance training, endurance performance, predicting training adaptation, autonomic nervous system.

Acknowledgements

Funding of the study was provided by the Finnish Funding Agency for Technology and Innovation (TEKES), KIHU – Research Institute for Olympic Sport and the Department of Biology of Physical Activity. The authors wish to thank the participating subjects for their collaboration.

References

- Aubert AE, Seps B, Beckers F. Heart rate variability in athletes. *Sports Med* 2003; 33: 889–919.
- Aunola S, Rusko H. Aerobic and anaerobic thresholds determined from venous lactate or from ventilation and gas exchange in relation to muscle fiber composition. *Int J Sports Med* 1986; 7: 161–166.
- Banister EW. Modelling elite athletic performance. In: MacDougall JD, Wenger HA, Green HJ, eds. *Physiological testing of the high-performance athlete*. Champaign, IL: Human Kinetics Publishers Ltd., 1991: 403–424.
- Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982; 14: 377–381.
- Bouchard C, Rankinen T. Individual differences in response to regular physical activity. *Med Sci Sports Exerc* 2001; 33: 446–451.
- Boutcher SH, Stein P. Association between heart rate variability and training response in sedentary middle-aged men. *Eur J Appl Physiol Occup Physiol* 1995; 70: 75–80.
- Buchheit M, Chivot A, Parouty J, Mercier D, Al Haddad H, Laursen PB, Ahmaidi S. Monitoring endurance running performance using cardiac parasympathetic function. *Eur J Appl Physiol* 2010; 108: 1153–1167.
- Buchheit M, Gindre C. Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. *Am J Physiol Heart Circ Physiol* 2006; 291: 451–458.
- Buchheit M, Simon C, Piquard F, Ehrhart J, Brandenberger G. Effects of increased training load on vagal-related indexes of heart rate variability: a novel sleep approach. *Am J Physiol Heart Circ Physiol* 2004; 287: H2813–H2818.
- Grandys M, Majerczak J, Duda K, Zapart-Bukowska J, Kulpa J, Zoladz JA. Endurance training of moderate intensity increases testosterone concentration in young, healthy men. *Int J Sports Med* 2009; 30: 489–495.
- Hautala AJ, Kiviniemi AM, Tulppo MP. Individual responses to aerobic exercise: the role of the autonomic nervous system. *Neurosci Biobehav Rev* 2009; 33: 107–115.
- Hautala AJ, Makikallio TH, Kiviniemi A, Laukkanen RT, Nissila S, Huikuri HV, Tulppo MP. Cardiovascular autonomic function correlates with the response to aerobic training in healthy sedentary subjects. *Am J Physiol Heart Circ Physiol* 2003; 285: H1747–H1752.
- Hedelin R, Bjerle P, Henriksson-Larsen K. Heart rate variability in athletes: relationship with central and peripheral performance. *Med Sci Sports Exerc* 2001; 33: 1394–1398.
- Hoogeveen AR, Zonderland ML. Relationships between testosterone, cortisol and performance in professional cyclists. *Int J Sports Med* 1996; 17: 423–428.
- Iellamo F, Legramante JM, Pigozzi F, Spataro A, Norbiato G, Lucini D, Pagani M. Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. *Circulation* 2002; 105: 2719–2724.
- Iwasaki K, Zhang R, Zuckerman JH, Levine BD. Dose–response relationship of the cardiovascular adaptation to endurance training in healthy adults: how much training for what benefit? *J Appl Physiol* 2003; 95: 1575–1583.
- Keizer H, Janssen GM, Menheere P, Kranenburg G. Changes in basal plasma testosterone, cortisol, and dehydroepiandrosterone sulfate in previously untrained males and females preparing for a marathon. *Int J Sports Med* 1989; 10(Suppl. 3): S139–S145.
- Kesaniemi YK, Danforth E, Jensen MD, Kopelman PG, Lefebvre P, Reeder BA. Dose–response issues concerning physical activity and health: an evidence-based symposium. *Med Sci Sports Exerc* 2001; 33: S351–S358.
- Kiviniemi AM, Hautala AJ, Makikallio TH, Seppanen T, Huikuri HV, Tulppo MP. Cardiac vagal outflow after aerobic training by analysis of high-frequency oscillation of the R–R interval. *Eur J Appl Physiol* 2006; 96: 686–692.
- Lamberts RP, Rietjens GJ, Tjink HH, Noakes TD, Lambert MI. Measuring submaximal performance parameters to monitor fatigue and predict cycling performance: a case study of a world-class cyclo-cross cyclist. *Eur J Appl Physiol* 2010a; 108: 183–190.
- Lamberts RP, Swart J, Capostagno B, Noakes TD, Lambert MI. Heart rate recovery as a guide to monitor fatigue and predict changes in performance parameters. *Scand J Med Sci Sports* 2010b; 20: 449–457.
- Loimaala A, Huikuri H, Oja P, Pasanen M, Vuori I. Controlled 5-Mo aerobic training improves heart rate but not heart rate variability or baroreflex sensitivity. *J Appl Physiol* 2000; 89: 1825–1829.

- Manzi V, Castagna C, Padua E, Lombardo M, D'Ottavio S, Massaro M, Volterrani M, Iellamo F. Dose-response relationship of autonomic nervous system responses to individualized training impulse in marathon runners. *Am J Physiol Heart Circ Physiol* 2009; 296: H1733–H1740.
- McArdle WD, Katch FI, Katch V.L. Exercise physiology. Energy, nutrition, and human performance. Baltimore: Williams & Wilkins, 1996: 393–415.
- Noakes TD, Myburgh KH, Schall R. Peak treadmill running velocity during the VO_{2max} test predicts running performance. *J Sports Sci* 1990; 8: 35–45.
- Nummela A, Hynynen E, Kaikkonen P, Rusko H. Endurance performance and nocturnal HRV indices. *Int J Sports Med* 2010; 31: 154–159.
- Pichot V, Roche F, Gaspoz JM, Enjolras F, Antoniadis A, Minini P, Costes F, Busso T, Lacour JR, Barthelemy J.C. Relation between heart rate variability and training load in middle-distance runners. *Med Sci Sports Exerc* 2000; 32: 1729–1736.
- Portier H, Louisy F, Laude D, Berthelot M, Guezennec CY. Intense endurance training on heart rate and blood pressure variability in runners. *Med Sci Sports Exerc* 2001; 33: 1120–1125.
- Purge P, Jurimae J, Jurimae T. Hormonal and psychological adaptation in elite male rowers during prolonged training. *J Sports Sci* 2006; 24: 1075–1082.
- Saalasti S. Neural networks for heart rate time series analysis. Ph.D thesis. Department of Mathematical Information Technology, University of Jyväskylä, Finland, Jyväskylä Studies in Computing 33, Jyväskylä, 2003
- Scharhag-Rosenberger F, Meyer T, Walitzek S, Kindermann W. Time course of changes in endurance capacity: a 1-yr training study. *Med Sci Sports Exerc* 2009; 41: 1130–1137.
- Scharhag-Rosenberger F, Walitzek S, Kindermann W, Meyer T. Differences in adaptations to 1 year of aerobic endurance training: individual patterns of nonresponse. *Scand J Med Sci Sports* 2010, doi: 10.1111/j.1600-0838.2010.01139.x.
- Shahidi NT. A review of the chemistry, biological action, and clinical applications of anabolic-androgenic steroids. *Clin Ther* 2001; 23: 1355–1390.
- Urhausen A, Gabriel H, Kindermann W. Blood hormones as markers of training stress and overtraining. *Sports Med* 1995; 20: 251–276.
- Uusitalo AL, Huttunen P, Hanin Y, Uusitalo AJ, Rusko HK. Hormonal responses to endurance training and overtraining in female athletes. *Clin J Sport Med* 1998; 8: 178–186.
- Vollaard NB, Constantin-Teodosiu D, Fredriksson K, Rooyackers O, Jansson E, Greenhaff PL, Timmons JA, Sundberg CJ. Systematic analysis of adaptations in aerobic capacity and submaximal energy metabolism provides a unique insight into determinants of human aerobic performance. *J Appl Physiol* 2009; 106: 1479–1486.
- Wheeler GD, Singh M, Pierce WD, Epling WF, Cumming DC. Endurance training decreases serum testosterone levels in men without change in luteinizing hormone pulsatile release. *J Clin Endocrinol Metab* 1991; 72: 422–425.