Sports Med (2016) 46:1461–1486
DOI 10.1007/s40279-016-0484-2

SYSTEMATIC REVIEW

Monitoring Athletic Training Status Through Autonomic Heart Rate Regulation: A Systematic Review and Meta-Analysis

Clint R. Bellenger[1] · Joel T. Fuller[1] · Rebecca L. Thomson[1] · Kade Davison[1] · Eileen Y. Robertson[2] · Jonathan D. Buckley[1]

Published online: 18 February 2016

Abstract

Background Autonomic regulation of heart rate (HR) as an indicator of the body's ability to adapt to an exercise stimulus has been evaluated in many studies through HR variability (HRV) and post-exercise HR recovery (HRR). Recently, HR acceleration has also been investigated.

Objective The aim of this systematic literature review and meta-analysis was to evaluate the effect of negative adaptations to endurance training (i.e., a period of overreaching leading to attenuated performance) and positive adaptations (i.e., training leading to improved performance) on autonomic HR regulation in endurance-trained athletes.

Methods We searched Ovid MEDLINE, Embase, CINAHL, SPORTDiscus, PubMed, and Academic Search Premier databases from inception until April 2015. Included articles examined the effects of endurance training leading to increased or decreased exercise performance on four measures of autonomic HR regulation: resting and post-exercise HRV [vagal-related indices of the root-mean-square difference of successive normal *R–R* intervals (RMSSD), high frequency power (HFP) and the standard deviation of instantaneous beat-to-beat *R–R* interval variability (SD1) only], and post-exercise HRR and HR acceleration.

Results Of the 5377 records retrieved, 27 studies were included in the systematic review and 24 studies were included in the meta-analysis. Studies inducing increases in performance showed small increases in resting RMSSD [standardised mean difference (SMD) = 0.58; $P < 0.001$], HFP (SMD = 0.55; $P < 0.001$) and SD1 (SMD = 0.23; $P = 0.16$), and moderate increases in post-exercise RMSSD (SMD = 0.60; $P < 0.001$), HFP (SMD = 0.90; $P < 0.04$), SD1 (SMD = 1.20; $P = 0.04$), and post-exercise HRR (SMD = 0.63; $P = 0.002$). A large increase in HR acceleration (SMD = 1.34) was found in the single study assessing this parameter. Studies inducing decreases in performance showed a small increase in resting RMSSD (SMD = 0.26; $P = 0.01$), but trivial changes in resting HFP (SMD = 0.04; $P = 0.77$) and SD1 (SMD = 0.04; $P = 0.82$). Post-exercise RMSSD (SMD = 0.64; $P = 0.04$) and HFP (SMD = 0.49; $P = 0.18$) were increased, as was HRR (SMD = 0.46; $P < 0.001$), while HR acceleration was decreased (SMD = −0.48; $P < 0.001$).

Conclusions Increases in vagal-related indices of resting and post-exercise HRV, post-exercise HRR, and HR acceleration are evident when positive adaptation to training has occurred, allowing for increases in performance. However, increases in post-exercise HRV and HRR also occur in response to overreaching, demonstrating that additional measures of training tolerance may be required to determine whether training-induced changes in these parameters are related to positive or negative adaptations. Resting HRV is largely unaffected by overreaching, although this may be the result of methodological issues that warrant further investigation. HR acceleration appears to decrease in response to overreaching training, and thus may be a potential indicator of training-induced fatigue.

✉ Clint R. Bellenger
clint.bellenger@mymail.unisa.edu.au

[1] Alliance for Research in Exercise, Nutrition and Activity (ARENA), Sansom Institute for Health Research, University of South Australia, GPO Box 2471, Adelaide, SA 5001, Australia

[2] South Australian Sports Institute, Adelaide, SA, Australia

Key Points

Measures of resting and post-exercise heart rate (HR) variability, post-exercise HR recovery, and HR acceleration increase in response to training, facilitating improvements in athletic performance.

In studies leading to reductions in performance as a result of overload training, HR recovery and HR variability assessed post-exercise also increased, making their interpretation difficult without additional variables allowing for contextualisation of training.

Overload training had little effect on resting HR variability; however, the disagreement between studies may be the result of methodological issues that warrant further investigation.

A small number of studies demonstrated a decrease in HR acceleration following overload training, indicating that it may be a potential indicator of training-induced fatigue.

1 Introduction

Optimizing athletic training through manipulation of training stress and recovery allows for the enhancement of athletic performance at important time points [1–3]. Unfortunately, if the subtle balance between the high training stress required for physiological adaptation and the appropriate recovery from such stress is not adequately maintained, training-induced fatigue may accumulate, leading to attenuated exercise performance (i.e., a state of functional overreaching or, if severe, non-functional overreaching and overtraining) [4]. The establishment of a marker capable of accurately conveying athletic training status that is easily administered and readily incorporated into any training program has become a popular topic in sports and exercise physiology research [1]. Such a marker would allow for recognition of training-induced fatigue or the level of recovery/adaptation achieved, facilitating adjustments in training load to individualize training programs [3].

Several methods to quantify change in autonomic nervous system (ANS) function are proposed to be capable of indicating training status. The ANS functions during and after physical exercise to maintain homeostasis, and repeated exposure to an exercise stress causes physiological adaptation, reducing homeostatic perturbation in response to subsequent stressors [3]. Consequently, examining ANS responsiveness to changes in training load may indicate the body's ability to tolerate or adapt to an exercise stimulus [3, 5]. Since the ANS controls cardiovascular function through sympathetic and parasympathetic modulation [6], and the balance of parasympathetic and sympathetic modulation is altered following changes in training load [7, 8], research has specifically focused on conveying training status through autonomic heart rate (HR) regulation, as it provides a simple and non-invasive measure of ANS function [9]. Two popular measures of autonomic HR regulation are HR variability (HRV) and post-exercise HR recovery (HRR).

HRV utilizes a non-invasive assessment of the variation in time between consecutive heart beats or *R*-to-*R* intervals [10]. The time between consecutive *R*-to-*R* intervals is constantly fluctuating as a result of the interaction between pulmonary ventilation, blood pressure, and cardiac output to maintain blood pressure homeostasis within specific limits [11]. In its simplest form, HRV has been analysed and presented in the time domain; however, more complex assessments include power spectral and non-linear analysis (see Malik et al. [10] for more information on methods of HRV analysis). These analyses of HRV are able to provide researchers with direct information on the parasympathetic contributions (and by extension, inferred information on the sympathetic contributions) to resting and post-exercise modulation of HR.

HRR is the assessment of the rate at which HR decreases following the cessation of exercise [12] and reflects the coordinated interaction between parasympathetic re-activation and sympathetic withdrawal [13, 14]. As such, HRR also has the potential to provide insight into the balance of parasympathetic and sympathetic HR modulation.

One mode of assessing autonomic HR regulation that has not been well investigated is the measurement of HR kinetics at the onset of exercise. Since these kinetics are controlled by the parasympathetic and sympathetic divisions of the ANS [6, 15], training-induced changes in HR kinetics at the onset of exercise may provide the potential to indicate training status. Indeed, cross-sectional research supports this potential [16], with mono-exponential curve fitting showing that the half-time for the increase in HR at the onset of exercise (i.e., the time taken for HR to reach one half of the difference between steady-state HR and pre-exercise HR) was shorter in well-trained athletes than in untrained individuals and was correlated with maximal oxygen consumption in both groups. The faster HR increase in trained individuals may contribute to improved exercise performance through a more rapid increase in oxygen delivery to active muscle, thus reducing peripheral muscle fatigue [17].

More recently, a novel method of examining HR kinetics at the onset of exercise using sigmoidal curve fitting has been investigated [18–20]. Specifically, the first derivative of the sigmoidal HR curve obtained during the transition from rest to steady state during sub-maximal exercise provided a measure of the maximal rate of HR increase (rHRI) at the onset of exercise.

Given the interest in autonomic HR regulation and its potential application in the field of sports and exercise physiology to monitor athletic training status, the aim of this systematic literature review was to determine the effect of training interventions leading to changes in exercise performance (as a measure of training status) on measures of autonomic HR regulation in endurance-trained athletes undergoing endurance-based training. Specifically, this review sought to investigate the effect of negative adaptations to training (i.e., a period of overreaching leading to attenuated performance) on autonomic HR regulation compared with the effects of positive adaptations to training (i.e., training leading to improved performance).

2 Methods

This review followed the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement for improved reporting of systematic reviews [21].

2.1 Literature Search

A literature search was conducted on 15 August 2013, and database update alerts were monitored until April 2015. The databases Ovid MEDLINE, Embase, Cumulative Index to Nursing and Allied Health (CINAHL), SPORTDiscus, PubMed, and Academic Search Premier were searched. Database searches were complemented by pearling of the reference lists of relevant articles. Where available, searches were limited to peer-reviewed, English language studies using human participants only.

Title, abstract, and keyword search fields were searched in each of the aforementioned databases using the following search terms:

> overreaching OR overtraining OR OT OR OTS OR overload OR training OR training status AND heart rate variability OR HRV OR heart rate recovery OR HRR OR rate of heart rate increase OR rHRI

2.2 Eligibility Criteria

To be eligible for inclusion in this review, study participants had to have been previously undertaking some form of endurance training (i.e., they were not previously sedentary), the training intervention utilized needed to induce a change in training status (either a decrease in performance (i.e., a state of overreaching) or an improvement in performance), and maximal exercise performance must have been measured and reported to confirm such a change in training status (measures of maximal oxygen consumption were not considered to be an acceptable proxy for exercise performance) [4, 22, 23]. Studies also had to include all necessary data to calculate effect size (i.e., number of participants, mean, and standard deviation).

2.3 Study Selection

All articles identified in the literature search were exported to a reference management software program (Endnote version X6, Thomson Reuters, 2012), and duplicate references were removed. Eligibility assessment was performed independently by two investigators (CRB and JTF), with disagreement settled by consensus. All records were examined by title and abstract in order to exclude obviously irrelevant records, and full-text articles were then assessed for eligibility using the eligibility criteria. For studies with more than one article based on the same study population, inclusion was limited to the original publication.

Data extraction was performed by the lead author (CRB) and confirmed by a second investigator (JTF). The following information was collected from included studies: publication details, participant characteristics, length of training intervention, details of conditions under which the relevant autonomic parameter was assessed (i.e., length of recording, posture, and intensity of exercise if applicable), and results (measures of maximal exercise performance and autonomic HR regulation as assessed by HRV, HRR, and HR acceleration).

2.4 Risk of Bias Assessment

Risk of bias assessment was performed by the lead author (CRB) and confirmed by a second investigator (JTF) utilizing the Cochrane Collaboration's tool for assessing risk of bias [24]. This tool was used to assess the selection, performance, detection, attrition, and reporting bias of each of the eligible studies identified from the literature search.

2.5 Statistical Analysis

The standardized mean difference (SMD) in autonomic HR regulation and maximal exercise performance before and after the training intervention was calculated by standardizing the mean difference (post-intervention value minus

pre-intervention value) by the pooled between-subject standard deviation at the pre- and post-intervention time points. Effects were quantified as trivial (<0.2), small (0.2–0.6), moderate (0.61–1.2), large (1.21–2.0), and very large (>2.0) [25]. Precision of the effect size estimate was assessed using 95 % confidence intervals (CIs).

Random-effects meta-analysis was performed in Review Manager software (RevMan, version 5.2, Cochrane Collaboration, Oxford, UK) using the inverse-variance method. Data were presented as SMD ± 95 % CI with statistical significance set at $P < 0.05$. Where not reported, the standard error of mean difference and correlations between treatment outcomes were estimated from P values using the equivalent T-statistic. When this was not possible, standard error of the mean difference was estimated according to the methods described by Elbourne et al. [26], using the lowest correlation estimate among other studies. The presence of statistical heterogeneity was determined by the I^2 statistic and Cochran's Q statistic [27].

Separate meta-analyses were carried out on indices of autonomic HR regulation (HRV, HRR, and HR acceleration) in those studies that found decrements in performance following a training intervention and those that found improvements in performance following a training intervention. With regard to HRV, meta-analyses were only carried out on vagal-related indices in accordance with recent recommendations of Buchheit [28] and Plews et al. [29]. Thus, the root-mean-square difference of successive normal *R–R* intervals (RMSSD) in the time domain, high frequency power (HFP) in the power spectral domain, and the standard deviation of instantaneous beat-to-beat *R–R* interval variability (SD1) from non-linear Poincare plots (or their natural logarithms) were extracted and analysed since they represent pure parasympathetic HR modulation [10]. Separate meta-analyses were performed on each of these indices. Researchers have also hypothesized that the posture in which the assessment of resting HRV occurs, in addition to the time of day at which it occurs, can affect the magnitude of training-induced change in resting HRV [28]. As such, sub-group meta-analyses were carried out on combinations of posture and recording time of day in studies assessing resting HRV.

3 Results

After removal of duplicates, the initial search identified 5377 records. A summary of the search, including the number of studies suitable for qualitative synthesis and meta-analysis is shown in Fig. 1. The 27 studies included for review are summarized in Tables 1 and 2 [1, 18, 19, 30–53].

3.1 Reasons for Exclusion

A total of 35 studies for which the full text was reviewed were excluded from qualitative synthesis, as follows: previously sedentary nature of the participants ($n = 5$) [54–58], not using endurance-trained athletes ($n = 2$) [59, 60], inducing a decline in performance that was not due to an overreaching intervention (i.e., athletes were allowed to detrain) ($n = 1$) [61], not including/reporting a valid measure of exercise performance ($n = 7$) [12, 62–67], not assessing an autonomic HR parameter of interest ($n = 11$) [68–78], not assessing a vagal-related index of HRV ($n = 2$) [79, 80], not identifying what HRV index was assessed ($n = 1$) [81], being a case study of an individual ($n = 1$) [82], being a secondary analysis of study data for which the original publication [31, 37, 41, 44] had already been included for analysis ($n = 4$ [83–86], and not inducing a decline in performance during a deliberate overreaching intervention (and thus any associated change in autonomic HR regulation was not due to a change in training status) ($n = 1$) [87].

Three studies were included for qualitative synthesis, but were excluded from meta-analysis as they presented median and interquartile ranges [44], did not present a standard deviation for SMD calculation [43], and presented repeat measures of resting HRV relative to baseline measures (and thus a valid measure of SMD could not be calculated) [42]. These authors were emailed for the required information but did not respond.

Boullosa et al. [30] measured ultra-short-term HRR (0–20 s); however, this work was excluded from qualitative synthesis and meta-analysis as it was not adequately standardized to allow accurate comparison with other studies (i.e., HRR was measured whenever an athlete had up to 20 s of recovery at any point during training without regard for exercise intensity or duration).

3.2 Risk of Bias

Selection bias (random sequence generation and allocation concealment bias) was not assessed in this review, as the nature of the training interventions utilized in the identified studies did not allow for such study rigour. Seven studies [18, 19, 39, 40, 42, 43, 53] utilized a period of controlled baseline training before intensifying training in order to induce a decrease in performance. In each of these studies, the order of training was fixed such that a period of 'light' training occurred before intensified training. Given that training needs to be intensified to induce the reductions in performance required in these studies, the order in which these periods of training occurred could not be randomized.

Performance and detection bias as a result of participant and study personnel blinding to the training intervention

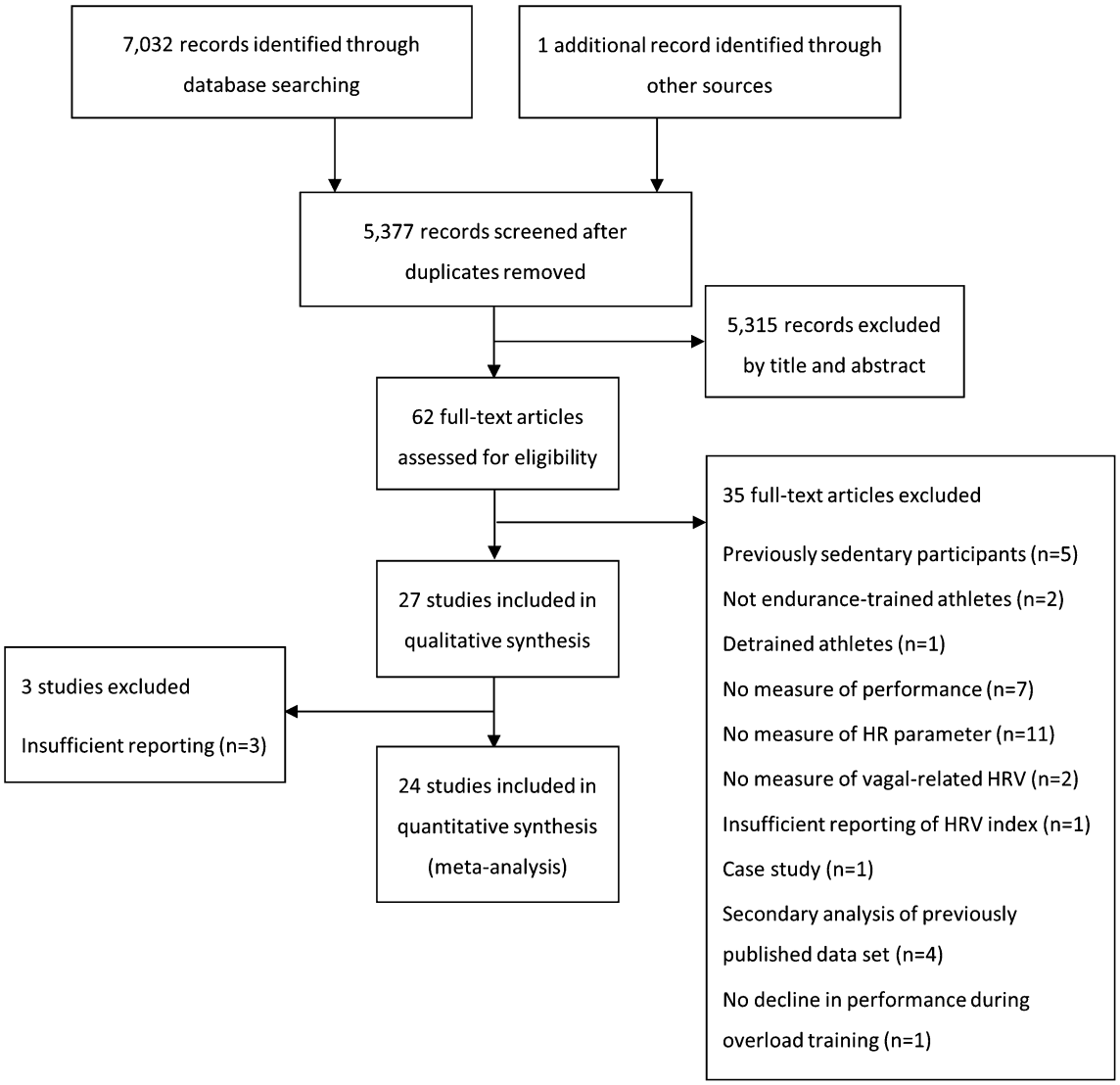


Fig. 1 Literature search flow chart. *HR* heart rate, *HRV* heart rate variability, *n* number of studies

and outcome measures was assessed as unclear in all studies. However, it should be noted that endurance-trained athletes accustomed to periods of varying training load cannot be blinded to periods of light and intensified training.

Attrition bias within the identified studies was assessed as either unclear or low risk. Five studies were assessed at high risk of a reporting bias [34, 36, 40–42]. Three studies mentioned a vagal-related HRV index in their study methodology, but did not report the effect of training on those indices [36, 40, 42]. Dupuy et al. [41] mentioned standard deviation of instantaneous beat-to-beat *R–R* interval variability (SD1) in their study methodology, and reported it as a resting measure, but did not report the effect of training on post-exercise assessment. Hedelin et al. [34] assessed HFP in a supine and a 70 ° upright tilted position, but only reported supine data.

3.3 Participants

Overall, the included studies reported on 355 athletes who underwent a training intervention designed to increase or decrease their performance and had their autonomic HR regulation assessed. Of these studies, 20 utilized male participants only [1, 18, 19, 30, 31, 35, 37–39, 41, 43, 45–53], while six utilized a mixed sex cohort [32–34, 40, 42, 44], and one did not report sex [36]).

A total of 12 studies included trained athletes competing at international [34, 50], national [30, 46, 47, 49, 52], or state/province/regional [1, 38, 40–42] level, while 15

Table 1 Summary of included studies with training interventions leading to increased exercise performance

Study	*n*	Athletes	Length of intervention	Performance parameter assessed	SMD (post-pre)	HR parameter details			
						HR parameter assessed (index)	SMD (post-pre)	Recording length	Recording posture (time of day[a])/ exercise mode/exercise intensity
Capostagno et al. 2014 [45]	M: 7	Recreational level cyclists	2 weeks	40 km TT	0.30	Post-exercise HRR	0.25	60 s	Seated/cycling/ ~90 % max HR
Da Silva et al. 2014 [46]	M: 6	National level runners	7 weeks	5 km TT	1.40	Resting HRV (RMSSD)	0.05	5 min	Seated (at time of laboratory test)
						Resting HRV (HFP)	0.04		
						Resting HRV (SD1)	−0.08		
Wallace et al. 2014 [36]	6	Recreational level runners	15 weeks	1500 m TT	0.82	Resting HRV (SD1)	−0.32	3 min	Standing (morning waking)
Boullosa et al. 2013 [30]	M: 8	National level soccer players	8 weeks	YYIRT1	0.21	Resting HRV (RMSSD)	0.26	3 h	Supine (sleeping)
						Resting HRV (HFP)	0.75		
						Resting HRV (SD1)	0.28		
Buchheit et al. 2013 [49]	M: 18	National level ARF players	2 weeks	YYIRT2	2.27	Post-exercise HRV (SD1)	1.76	3 min	Seated/running/13 km/h (~88 % max HR)
Buchheit et al. 2013 [50]	M: 20	International level soccer players	12 days	YYIRT1	0.83	Resting HRV (SD1)	0.51	3 min	Seated (morning waking)
Oliveira et al. 2013 [47]	M: 10	National level futsal players	3 weeks	YYIRT1	0.71	Resting HRV (RMSSD)	0.80	5 min	Seated (at time of laboratory test; spontaneous breathing)
						Resting HRV (HFP)	0.86		
Vesterinen et al. 2013 [48]	M: 25	Recreational level runners	28 weeks	MAS	0.57	Resting HRV (RMSSD)	0.76	4 h (sleep)	Supine (sleep)
						Resting HRV (HFP)	0.29		
Buchheit et al. 2012 [1]	M: 33	Regional level soccer players	35 weeks	MAS	0.38	Post-exercise HRV (RMSSD)	0.38	3 min	Seated/running/9 km/h (~74 % max HR)
						Post-exercise HRR	−0.09	60 s	
Buchheit et al. 2011 [52]	M: 15	National level soccer players	8 days	YYIRT1	0.45	Post-exercise HRV (SD1)	0.38	3 min	Seated/running/9 km/h (~68 % max HR)
						Post-exercise HRR	−0.04	60 s	

Table 1 continued

Study	*n*	Athletes	Length of intervention	Performance parameter assessed	SMD (post-pre)	HR parameter details			
						HR parameter assessed (index)	SMD (post-pre)	Recording length	Recording posture (time of day[a])/ exercise mode/exercise intensity
Buchheit et al. 2010 [31]	M: 11	Recreational level runners	8 weeks	MAS	0.79	Resting HRV (RMSSD)	0.57	5 min	Supine (morning waking)
				10 km TT	0.46	Post-exercise HRV (RMSSD)	0.75	3 min	Standing/running/60 % MAS (~78 % max HR[b])
						Post-exercise HRR	1.22	60 s	
Lamberts et al. 2009 [37]	M: 14	Recreational level cyclists	4 weeks	40 km TT	0.68	Post-exercise HRR (40 km TT)	1.20	60 s	Seated/cycling/max exercise (~98 % max HR)
				MAP	0.39	Post-exercise HRR (HIT)	0.88		Seated/cycling/~94 % max HR
Buchheit et al. 2008 [38] GRS	M: 8	Regional level handball players	9 weeks	V_{IFT}	0.92	Post-exercise HRV (RMSSD)	0.75	5 min	Seated/running/60 % V_{IFT} (~79 % max HR)
						Post-exercise HRV (HFP)	0.56		
						Post-exercise HRR	0.33	60 s	
Buchheit et al. 2008 [38] GHIT	M: 7	Regional level handball players	9 weeks	V_{IFT}	1.05	Post-exercise HRV (RMSSD)	1.26	5 min	Seated/running/60 % V_{IFT} (~79 % max HR)
						Post-exercise HRV (HFP)	1.29		
						Post-exercise HRR	1.21	60 s	
Kiviniemi et al. 2007 [35] GnHRV	M: 8	Recreational level runners	4 weeks	MAS	0.48	Resting HRV (sit; HFP)	0.00	5 min	Seated (morning waking; spontaneous breathing)
						Resting HRV (stand; HFP)	1.33		Standing (morning waking; spontaneous breathing)
Kiviniemi et al. 2007 [35] GHRV	M: 9	Recreational level runners	4 weeks	MAS	0.90	Resting HRV (sit; HFP)	0.64	5 min	Seated (morning waking; spontaneous breathing)
						Resting HRV (stand; HFP)	0.57		Standing (morning waking; spontaneous breathing)
Carter et al. 2003 [32]	M: 12 F: 12	Recreational level runners	12 weeks	3.2 km TT	0.31	Resting HRV (HFP)	0.73	10 min	Supine (at time of laboratory test; controlled breathing @ 15/min)
Laffite et al. 2003 [51]	M: 7	Recreational level runners	8 weeks	V50	0.40	HR acceleration	1.34	9 min 23 s[c]	Upright/running/~91 % max HR

Table 1 continued

Study	*n*	Athletes	Length of intervention	Performance parameter assessed	SMD (post-pre)	HR parameter details			
						HR parameter assessed (index)	SMD (post-pre)	Recording length	Recording posture (time of day[a])/exercise mode/exercise intensity
Hedelin et al. 2000 [33]	M: 8 F: 9	Recreational level cross-country skiers	35 weeks	GXT TTE	0.50	Resting HRV (HFP)	0.20	5 min	Supine (at time of laboratory test; controlled breathing at 12/min)

ARF Australian Rules football, *F* female, *GHIT* group randomized to perform high intensity training intervention, *GHRV* group randomized to perform training guided by daily HRV assessment, *GnHRV* group randomized to perform a predetermined training program without the use of daily HRV assessment, *GRS* group randomized to perform repeated sprint training intervention, *GXT TTE* time to exhaustion during a graded exercise test, *HFP* high-frequency power from spectral analysis, *HIT* high-intensity training, *HR* heart rate, *HRR* heart rate recovery, *HRV* heart rate variability, *max HR* maximum heart rate, *M* male, *MAP* maximal aerobic power, *MAS* maximal aerobic speed, *n* sample size, *RMSSD* root-mean-square difference of successive normal *R–R* intervals from time-domain analysis, *SD1* standard deviation of instantaneous beat-to-beat *R–R* interval variability from Poincare plots, *SMD* standardized mean difference, *TT* time trial, V_{IFT} peak velocity reached during 30–15 intermittent fitness test, *V50* speed at one half of the difference between maximal aerobic speed and the individual anaerobic threshold speed, *YYIRT1* Yo-Yo intermittent recovery test level 1, *YYIRT2* Yo-Yo intermittent recovery test level 2

[a] Applicable only to measures of resting HRV

[b] Age-predicted maximum heart rate [208 − (0.7 × age)]

[c] Measured during TTE at 93 % maximal aerobic speed

included trained but recreational athletes [18, 19, 31–33, 35–37, 39, 43–45, 48, 51, 53].

3.4 Study Outcomes

A total of 17 studies induced improvements in performance following a training intervention [1, 30–33, 35–38, 45–52]. Within these studies, Kiviniemi at al. [35] and Buchheit et al. [38] had two separate cohorts complete different training interventions, and the outcome of these interventions on performance differed between groups; thus, these groups were treated independently for analysis. The studies of Kiviniemi at al. [35] and Lamberts et al. [37] reported two independent outcomes within a single measure of autonomic HR regulation. That is, Kiviniemi at al. [35] assessed resting HFP in both sitting and standing postures, while Lamberts et al. [37] reported HRR following two different maximal performance tests: a graded cycling exercise test to determine maximal aerobic power, and a 40-km cycling time trial. Since a consensus has not yet been established on which posture should be utilized in the assessment of resting HRV, nor is it known which performance test allows for superior measurement of post-exercise HR parameters, both measures were included for analysis.

Within these 17 studies, ten investigated resting HRV [30–33, 35, 36, 46–48, 50]. Of the four studies that conducted their assessments upon waking in the morning [31, 35, 36, 50], one was assessed in a supine posture [31], two in a seated posture [35, 50], and two in a standing posture [35, 36] (Kiviniemi at al. [35] assessed resting HFP in both sitting and standing postures). Two studies conducted their assessments during overnight sleep in a supine posture [30, 48], while four studies conducted their assessments at the time of laboratory visits [32, 33, 46, 47]: two were assessed in a seated posture [32, 33] and two in a standing posture [46, 47].

Ten studies induced reductions in performance following a training intervention [18, 19, 34, 39–44, 53]. Chalencon et al. [40] conducted two separate overreaching interventions leading to decreased performance, and both interventions were treated independently within this review. The studies of Dupuy et al. [41], Le Meur et al. [39], and Bellenger et al. [19] reported two independent outcomes within a single measure of autonomic HR regulation. Dupuy et al. [41] reported changes in resting HRV (in RMSSD, HFP, and SD1) under two conditions of sleep: during a full 4-h period, and during the isolated slow-wave sleep periods of this 4-h sleep. These authors also reported post-exercise HRV (in both RMSSD and HFP) and HRR following two different maximal performance tests: a graded running exercise test to determine maximal aerobic speed, and a time to exhaustion test at 85 % of maximal

Table 2 Summary of included studies with training interventions leading to decreased exercise performance

Study	*n*	Athletes	Length of intervention	Performance parameter assessed	SMD (post-pre)	HR parameter details			
						HR parameter assessed (index)	SMD (post-pre)	Recording length	Recording posture (time of day[a])/exercise mode/exercise intensity
Bellenger et al. 2015 [19]	M: 11	Recreational level cyclists/triathletes	2 weeks	Work done during 5 min TT	−0.37	HR acceleration (cycling)	−0.48	5 min 30 s	Seated/cycling/100 W
						HR acceleration (running)	−0.43	5 min 30 s	Upright/running/8 km/h
Thomson et al. 2015 [53]	M: 11	Recreational level cyclists/triathletes	2 weeks	Work done during 5 min TT	−0.33	Post-exercise HRR	0.82	60 s	Supine/cycling/maximal exercise[b]
Le Meur et al. 2013 [39]	M: 16	Recreational level triathletes	3 weeks	GXT distance covered	−0.62	Resting HRV (RMSSD)	0.39	4 min	Supine (morning waking)
						Resting HRV (HFP)	0.14		
						Resting HRV (RMSSD)	0.34		Standing (morning waking)
						Resting HRV (HFP)	0.49		
Nelson et al. 2013 [18]	M: 8	Recreational level cyclists/triathletes	2 weeks	Work done during 5 min TT	−0.48	HR acceleration	−0.65	5 min 30 s	Seated/cycling/100 W
						Post-exercise HRR	0.46	60 s	Supine/cycling/maximal exercise[b]
Chalencon et al. 2012 [40] OR1	M: 6 F: 4	Regional level swimmers	4 weeks	400 m freestyle performance	−0.06	Resting HRV (HFP)	−0.34	Overnight	Supine (sleeping)
Chalencon et al. 2012 [40] OR2	M: 6 F: 4	Regional level swimmers	5 weeks	400 m freestyle performance	−0.14	Resting HRV (HFP)	−0.26	Overnight	Supine (sleeping)
Dupuy et al. 2012 [41]	M: 11	Regional level endurance athletes	2 weeks	MAS	−0.15	Resting HRV (4hS; RMSSD)	0.17	4 h	Supine (sleeping)
						Resting HRV (4hS; HFP)	0.49		
						Resting HRV (4hS; SD1)	0.17		
						Resting HRV (SWS; RMSSD)	0.01	10 min	
						Resting HRV (SWS; HFP)	−0.05		
						Resting HRV (SWS; SD1)	−0.09		
				TTE @ 85 % MAS	−0.78	Post-exercise HRV (MAS; RMSSD)	0.59	5 min	Seated/running/maximal exercise
						Post-exercise HRV (MAS; HFP)	0.66		
						Post-exercise HRV (TTE; RMSSD)	0.68		
						Post-exercise HRV (TTE; HFP)	0.33		
						Post-exercise HRR (MAS)	0.08	60 s	
						Post-exercise HRR (TTE)	0.46		
Baumert et al. 2006 [44]	M: 5 F: 5	Recreational level runners/triathletes	13 days	MAP	−7 %[c]	Resting HRV (RMSSD)	−24 %[c]	NR	Supine (at time of laboratory test)
						Resting HRV (HFP)	−46 %[c]		
Garet et al. 2004 [42]	M: 4 F: 3	Regional level swimmers	3 weeks	400 m freestyle performance	0.11	Resting HRV (HFP)	−29 %[c]	6 h	Supine (sleeping)

Table 2 continued

Study	*n*	Athletes	Length of intervention	Performance parameter assessed	SMD (post-pre)	HR parameter details			
						HR parameter assessed (index)	SMD (post-pre)	Recording length	Recording posture (time of day[a])/exercise mode/exercise intensity
Bosquet et al. 2003 [43]	M: 9	Recreational level runners/triathletes	3 days	MAS	−0.29	Resting HRV (HFP)	20 %[c]	Overnight	Supine (sleeping)
Hedelin et al. 2000 [34]	M: 6 F: 3	International level canoeists	6 days	GXT TTE	−1.00	Resting HRV (HFP)	−0.31	NR	Supine (at time of laboratory test; controlled breathing @ 12/min)

F female, *GXT* graded exercise test, *GXT TTE* time to exhaustion during a graded exercise test, *HFP* high-frequency power from spectral analysis, *HR* heart rate, *HRR* heart rate recovery, *HRV* heart rate variability, *M* male, *MAP* maximal aerobic power, *MAS* maximal aerobic speed, *n* sample size, *NR* not reported, *OR 1* overreaching training period 1, *OR 2* overreaching training period 2, *RMSSD* root-mean-square difference of successive normal *R–R* intervals from time-domain analysis, *SD1* standard deviation of instantaneous beat-to-beat *R–R* interval variability from Poincare plots, *SMD* standardized mean difference, *SWS HRV* recorded during periods of slow wave sleep, *TT* time trial, *TTE* time to exhaustion, *4hS* HRV recorded during 4 h of continuous sleep

[a] Applicable only to measures of resting HRV

[b] 5-min time trial to complete as much work in kilojoules as possible

[c] SMD not able to be calculated

aerobic speed. Given that it could not be objectively determined which condition of sleep or which performance test was most sensitive for allowing changes in autonomic HR regulation to be detected, all measures were included for analysis. Le Meur et al. [39] reported changes in resting HRV (in both RMSSD and HFP) in both supine and standing postures during an orthostatic test, and both measures were included for analysis. Bellenger et al. [19] reported changes in rHRI during two different modes of exercise—cycling and running—and both measures were included for further analysis.

Within these ten studies, seven investigated resting HRV [34, 39–44]. Le Meur et al. [39] assessed resting HRV in supine and standing postures upon waking in the morning. Four studies conducted their assessments during overnight sleep in a supine posture [40–43], and two conducted their assessments in a supine posture at the time at which laboratory visits were scheduled [34, 44].

Outcome measures of interest from these studies are shown in Tables 1 and 2.

3.5 Meta-Analysis

Tables 3 and 4 show a summary of within-study comparisons and methods used to calculate the individual study standard error of mean difference.

Four measures of autonomic HR regulation were identified by this review: resting HRV, post-exercise HRR, post-exercise HRV, and HR acceleration.

3.5.1 Resting Heart Rate Variability (HRV)

Figure 2a and b shows the effect of training interventions on resting HRV. A small increase in pooled vagal-related indices of HRV (SMD ± 95 % CI = 0.49 ± 0.15; $P < 0.001$) occurred following training leading to an increase in performance, which was significantly affected by statistical heterogeneity ($P = 0.003$; $I^2 = 54$ %). Index-specific analyses showed small increases in resting RMSSD (0.58 ± 0.22; $P < 0.001$) and HFP (0.55 ± 0.24; $P < 0.001$), and a small but statistically non-significant increase in SD1 (0.23 ± 0.33; $P = 0.16$). Statistical heterogeneity also affected the analysis of HFP ($P = 0.02$, $I^2 = 56$ %).

In those studies inducing a decrease in exercise performance, a statistically non-significant change occurred in pooled indices of vagal-related HRV (0.13 ± 0.16; $P = 0.12$) that was again affected by statistical heterogeneity ($P = 0.06$; $I^2 = 41$ %). A small increase in resting RMSSD (0.26 ± 0.21; $P = 0.01$) was found, but HFP (0.04 ± 0.29; $P = 0.77$) and SD1 (0.04 ± 0.35; $P = 0.82$) were not changed. HFP was significantly affected by statistical heterogeneity ($P = 0.01$; $I^2 = 64$ %).

Table 3 Available data and results for studies leading to increased exercise performance included in meta-analysis

Study	*n*	Information available	HR parameter (index)	SMD (post-pre)	SE (post-pre)	Correlation used
Capostagno et al. 2014 [45]	7	Treatment-specific summaries, mean values provided by authors	Post-exercise HRR	0.25	0.13	0.99
Da Silva et al. 2014 [46]	6	Treatment-specific summaries	Resting HRV (RMSSD)	0.05	0.50	0.58[a]
			Resting HRV (HFP)	0.04	0.47	0.62[a]
			Resting HRV (SD1)	−0.08	0.50	0.58[a]
Wallace et al. 2014 [36]	7	Treatment-specific summaries	Resting HRV (SD1)	−0.32	0.37	0.58[a]
Boullosa et al. 2013 [30]	8	Treatment-specific summaries, 95 % CI provided by authors	Resting HRV (RMSSD)	0.26	0.19	0.93
			Resting HRV (HFP)	0.75	0.37	0.93
			Resting HRV (SD1)	0.28	0.19	0.93
Buchheit et al. 2013 [49]	18	Treatment-specific summaries, *p* value (*t* test)	Post-exercise HRV (SD1)	1.76	0.44	−0.76
Buchheit et al. 2013 [50]	20	Treatment-specific summaries	Resting HRV (SD1)	0.51	0.20	0.58[a]
Oliveira et al. 2013 [47]	10	Treatment-specific summaries, 95 % CI	Resting HRV (RMSSD)	0.80	0.15	0.91
			Resting HRV (HFP)	0.86	0.16	0.94
Vesterinen et al. 2013 [48]	25	Treatment-specific summaries, *p* value (repeated measures ANOVA pairwise comparison), 95 % CI provided by authors	Resting HRV (RMSSD)	0.76	0.20	0.58
			Resting HRV (HFP)	0.29	0.11	0.83
Buchheit et al. 2012 [1]	33	Treatment-specific summaries	Post-exercise HRV (RMSSD)	0.38	0.23	0.11[a]
			Post-exercise HRR	−0.09	0.12	0.72[a]
Buchheit et al. 2011 [52]	15	Treatment-specific summaries	Post-exercise HRV (SD1)	0.60	0.48	−0.76[a]
			Post-exercise HRR	0.04	0.20	0.72[a]
Buchheit et al. 2010 [31]	11	Treatment-specific summaries, *p* value (*t* test)	Resting HRV (RMSSD)	0.57	0.13	0.72
			Post-exercise HRV (RMSSD)	0.75	0.40	0.11[a]
			Post-exercise HRR	1.22	0.26	0.72[a]
Lamberts et al. 2009 [37]	14	Treatment-specific summaries, *p* values (*t* test)	Post-exercise HRR (40-km TT)	1.20	0.16	0.90
			Post-exercise HRR (HIT)	0.88	0.20	0.72
Buchheit et al. 2008 [38] GRS	8	Treatment-specific summaries	Post-exercise HRV (RMSSD)	0.75	0.48	0.11[a]
			Post-exercise HRV (HFP)	0.56	0.60	−0.42[a]
			Post-exercise HRR	0.33	0.28	0.72[a]
Buchheit et al. 2008 [38] GHIT	7	Treatment-specific summaries, *p* values (two-way repeated measures ANOVA pairwise comparison)	Post-exercise HRV (RMSSD)	1.26	0.51	0.11
			Post-exercise HRV (HFP)	1.29	0.64	−0.42
			Post-exercise HRR	1.21	0.20	0.86
Kiviniemi et al. 2007 [35] GnHRV	8	Treatment-specific summaries, *p* value (*t* test)	Resting HRV (sit; HFP)	0.00	0.31	0.62[a]
			Resting HRV (stand; HFP)	1.33	0.32	0.80
Kiviniemi et al. 2007 [35] GHRV	9	Treatment specific summaries, *p* values (*t* test)	Resting HRV (sit; HFP)	0.64	0.29	0.62
			Resting HRV (stand; HFP)	0.57	0.25	0.76
Carter et al. 2003 [32]	24	Treatment-specific summaries	Resting HRV (HFP)	0.73	0.23	0.62[a]

Table 3 continued

Study	*n*	Information available	HR parameter (index)	SMD (post-pre)	SE (post-pre)	Correlation used
Laffite et al. 2003 [51]	7	Treatment-specific summaries *p* value (repeated measures ANOVA pairwise comparison)	HR acceleration	1.34	0.55	0.15
Hedelin et al. 2000 [33]	19	Treatment-specific summaries	Resting HRV (HFP)	0.20	0.20	0.62[a]

ANOVA analysis of variance, *CI* confidence interval, *GHIT* group randomized to perform HIT intervention, *GRS* group randomized to perform repeated sprint training intervention, *GHRV* group randomized to perform training guided by daily HRV assessment, *GnHRV* group randomized to perform a predetermined training program without the use of daily HRV assessment, *HR* heart rate, *HFP* high-frequency power from spectral analysis, *HIT* high-intensity training, *HR* heart rate, *HRR* heart rate recovery, *HRV* heart rate variability, *n* sample size, *RMSSD* root-mean-square difference of successive normal *R–R* intervals from time-domain analysis, *SD1* standard deviation of instantaneous beat-to-beat *R–R* interval variability from Poincare plots, *SE* standard error of standardized mean difference, *SMD* standardized mean difference, *TT* time trial

[a] Correlation assumed

Sub-group analyses for the combinations of posture and recording time of day for studies leading to increases and decreases in performance are shown in Table 5.

3.5.2 Post-Exercise Heart Rate Recovery (HRR)

A moderate increase in post-exercise HRR was found following improved exercise performance (0.63 ± 0.40; $P = 0.002$), but post-exercise HRR was also significantly affected by statistical heterogeneity ($P < 0.001$; $I^2 = 91$ %).

A small increase in post-exercise HRR was also found (0.46 ± 0.20; $P < 0.001$) in those studies inducing a decrease in performance. Figure 3 depicts the effect of training on HRR.

3.5.3 Post-Exercise HRV

A moderate increase in pooled indices of post-exercise HRV (0.83 ± 0.36; $P < 0.001$) was found in studies leading to increased performance. RMSSD (0.60 ± 0.34; $P < 0.001$), HFP (0.90 ± 0.85; $P < 0.04$), and SD1 (1.20 ± 1.13; $P = 0.04$) all showed moderate increases (Fig. 4a).

Studies inducing a decrease in performance demonstrated a small increase in pooled post-exercise HRV (0.58 ± 0.46; $P = 0.01$). Measures of RMSSD (0.64 ± 0.60; $P = 0.04$) increased moderately, while small and non-significant changes in HFP (0.49 ± 0.77; $P = 0.18$) were evident (Fig. 4b).

3.5.4 HR Acceleration

Figure 5 highlights the training-induced changes in HR acceleration. Only one study [51] assessed the effect of training interventions leading to increased exercise performance on HR acceleration, and this study reported an increase (1.34 ± 1.08) in this parameter. A small decrease in this parameter occurred in those studies inducing decreased exercise performance (-0.48 ± 0.21; $P < 0.001$).

4 Discussion

This systematic literature review sought to determine the effect of training interventions leading to decreased and/or increased exercise performance on measures of autonomic HR regulation. Four measures of autonomic HR regulation were identified: resting HRV, post-exercise HRR, post-exercise HRV and HR acceleration. These will be discussed separately.

4.1 Resting HRV

4.1.1 Training Leading to Increased Exercise Performance

Resting measures of RMSSD and HFP increased following training interventions that led to increased performance (0.58 ± 0.22 and 0.55 ± 0.24, respectively), highlighting an increase in cardiac parasympathetic modulation as a positive training adaptation. Studies of SD1 demonstrated a small increase (0.23 ± 0.33).

Interestingly, the analysis of HFP was significantly affected by statistical heterogeneity, but the analysis of RMSSD was not. Statistical heterogeneity in this review suggests that training leading to increased performance does not lead to a uniform change in HFP, which may be due to limitations inherent in its assessment. While

Table 4 Available data and results for studies leading to decreased exercise performance included in meta-analysis

Study	*n*	Information available	HR parameter (index)	SMD (post-pre)	SE (post-pre)	Correlation used
Bellenger et al. 2015 [19]	10	Treatment-specific summaries, 95 % CI provided by authors	HR acceleration (cycling)	−0.48	0.13	0.92
			HR acceleration (running)	−0.43	0.22	0.75
Thomson et al. 2015 [53]	11	Treatment-specific summaries, 95 % CI provided by authors	Post-exercise HRR	0.82	0.32	0.42
Nelson et al. 2014 [18]	8	Treatment-specific summaries, 95 % CI provided by authors	HR acceleration	−0.65	0.55	−0.18
			Post-exercise HRR	0.46	0.12	0.94
Le Meur et al. 2013 [39]	16	Individual specific data provided by authors	Resting HRV (supine; RMSSD)	0.39	0.21	0.66
			Resting HRV (supine; HFP)	0.14	0.20	0.70
			Resting HRV (stand; RMSSD)	0.34	0.17	0.80
			Resting HRV (stand; HFP)	0.49	0.13	0.87
Chalencon et al. 2012 [40] OR1	10	Treatment-specific summaries	Resting HRV (HFP)	−0.34	0.27	0.70[a]
Chalencon et al. 2012 [40] OR2	10	Treatment-specific summaries	Resting HRV (HFP)	−0.26	0.26	0.70[a]
Dupuy et al. 2012 [41]	11	Treatment-specific summaries	Resting HRV (4hS; RMSSD)	0.17	0.25	0.66[a]
			Resting HRV (4hS; HFP)	0.49	0.37	0.70[a]
			Resting HRV (4hS; SD1)	0.17	0.25	0.66[a]
			Resting HRV (SWS; RMSSD)	0.01	0.25	0.66[a]
			Resting HRV (SWS; HFP)	−0.05	0.23	0.70[a]
			Resting HRV (SWS; SD1)	−0.09	0.25	0.66[a]
			Post-exercise HRV (MAS; RMSSD)	0.59	0.46	0.11[a]
			Post-exercise HRV (MAS; HFP)	0.66	0.53	−0.42[a]
			Post-exercise HRV (TTE; RMSSD)	0.68	0.41	0.11[a]
			Post-exercise HRV (TTE; HFP)	0.33	0.51	−0.42[a]
			Post-exercise HRR (MAS)	0.08	0.33	0.42[a]
			Post-exercise HRR (TTE)	0.46	0.32	0.42[a]
Hedelin et al. 2000 [34]	9	Treatment-specific summaries	Resting HRV (HFP)	−0.31	0.30	0.70[a]

CI confidence interval, *HR* heart rate, *HFP* high-frequency power from spectral analysis, *HRR* heart rate recovery, *HRV* heart rate variability, *MAS* maximal aerobic speed, *n* sample size, *OR 1* overreaching training period 1, *OR 2* overreaching training period 2, *RMSSD* root-mean-square difference of successive normal *R–R* intervals from time-domain analysis, *SD1* standard deviation of instantaneous beat-to-beat *R–R* interval variability from Poincare plots, *SE* standard error of SMD, *SMD* standardized mean difference, *SWS HRV* recorded during periods of slow wave sleep, *TTE* time to exhaustion, *4hS* HRV recorded during 4 h of continuous sleep

[a] Correlation assumed

assessments of HFP and RMSSD demonstrate similar day-to-day variability during short-term (5–10 min) recordings [88], HFP assessment has a heightened sensitivity to respiration rates (which has been shown to affect measures of resting HRV [89]). Thus, while the overall effect of a positive training adaptation on HFP is significant, the

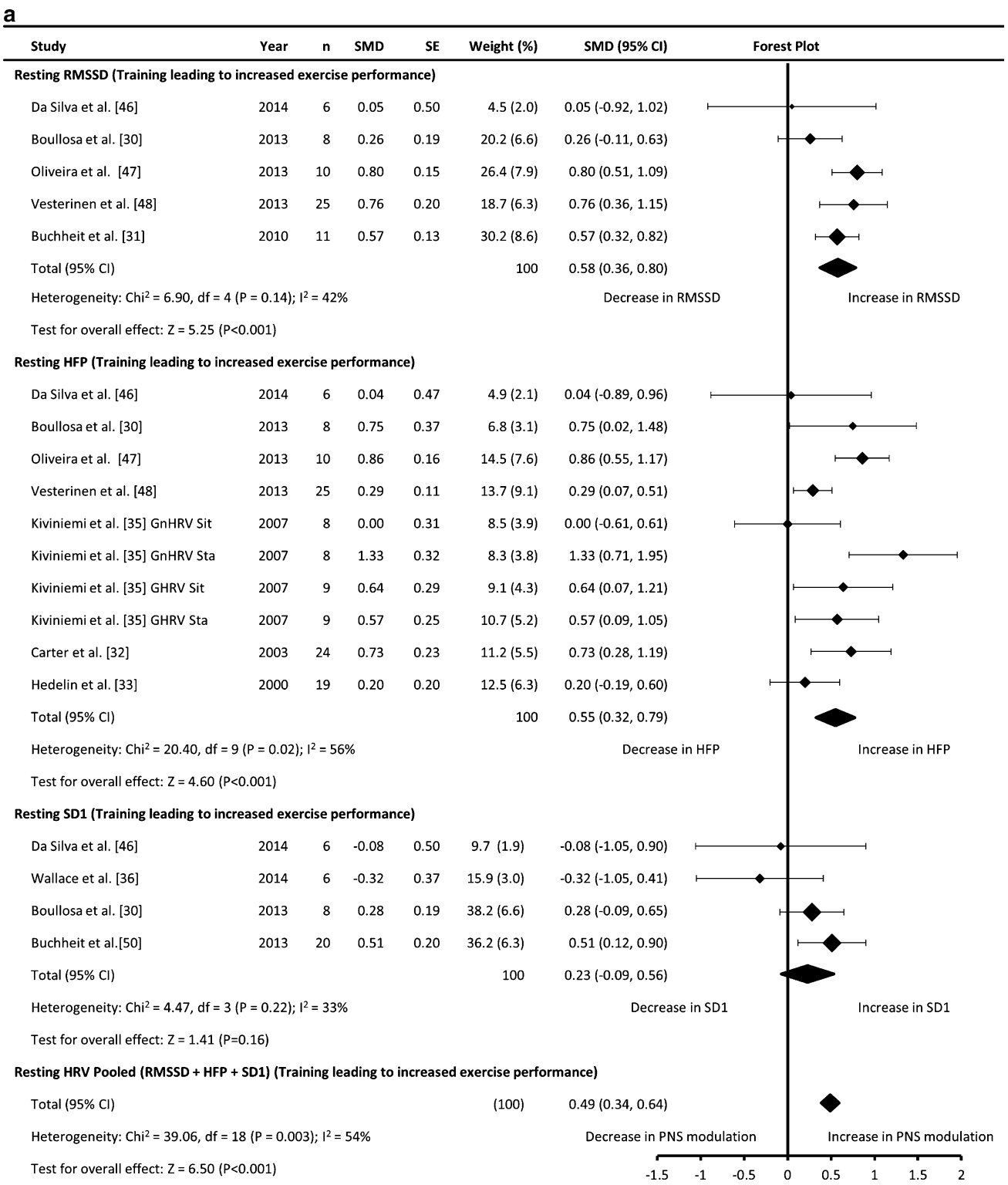


Fig. 2 Effect of training leading to **a** increased and **b** decreased exercise performance on resting heart rate variability. *CI* confidence interval, *GHRV* group randomized to perform training guided by daily HRV assessment, *GnHRV* group randomized to perform a predetermined training program without the use of daily HRV assessment, *HFP* high-frequency power, *n* number of participants, *HRV* heart rate variability, *OR 1* overreaching training period 1, *OR 2* overreaching training period 2, *PNS* parasympathetic nervous system, *RMSSD* the root-mean-square difference of successive normal *R–R* intervals, *SD1* standard deviation of instantaneous beat-to-beat *R–R* interval variability, *SE* standard error, *Sit* sitting, *SMD* standardized mean difference, *Sta* standing, *Sup* supine, *SWS* slow-wave sleep, *4hS* 4-h sleep. Effects were quantified as trivial (<0.2), small (0.2–0.6), moderate (0.61–1.2), large (1.21–2.0), and very large (>2.0)

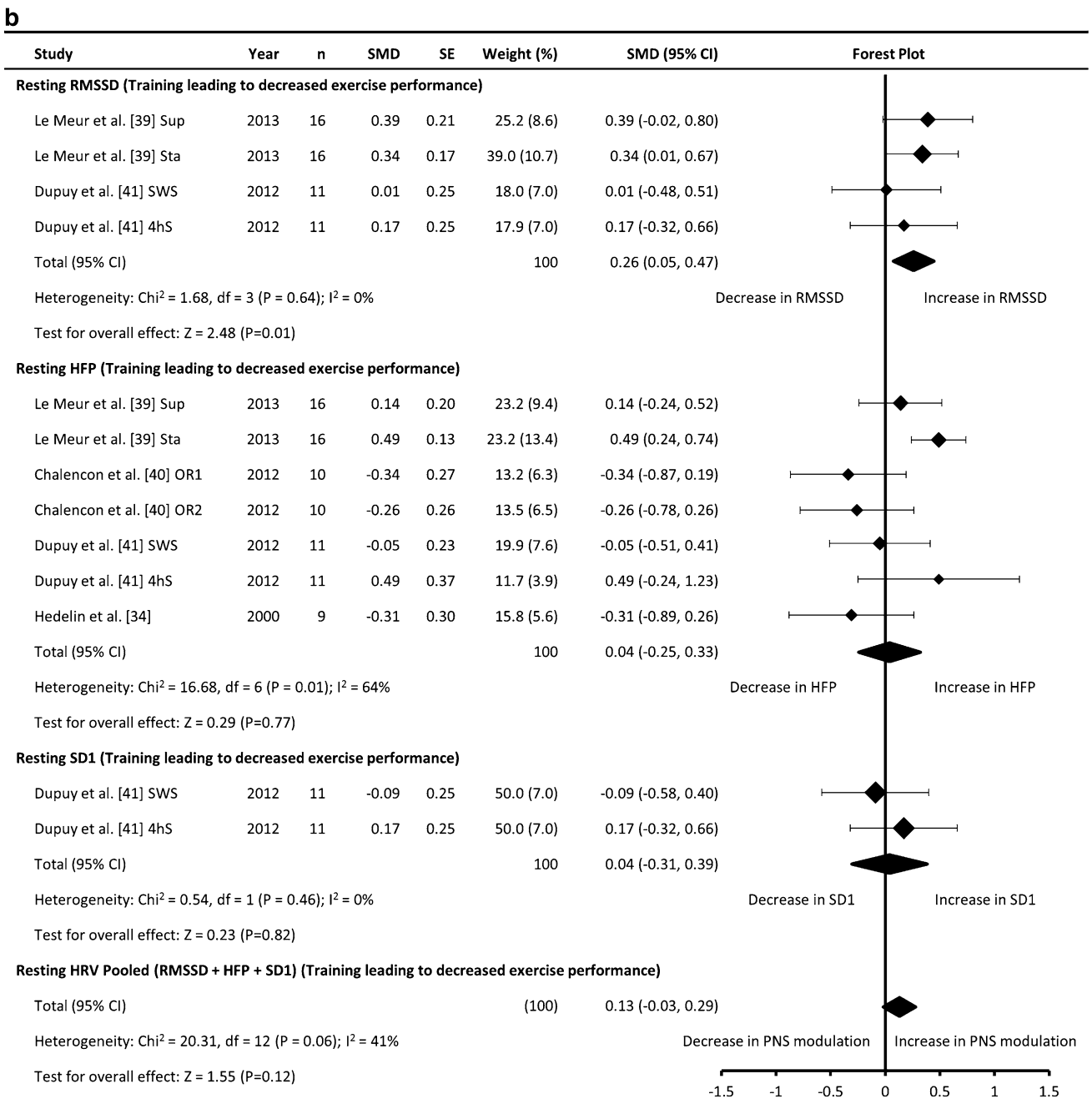


Fig. 2 continued

findings of this review may suggest that RMSSD is a more reliable marker of training status. This finding is in agreement with the recent views of Buchheit [28] and Plews et al. [29], which suggest that RMSSD be utilized as the preferred index of HRV for the monitoring of athletic training status.

Sub-group analyses of the postural and recording time of day combinations may also explain some of the statistical heterogeneity in the analysis of HFP. These sub-analyses demonstrate decreased heterogeneity when HFP is assessed during overnight sleep [0.38 ± 0.37; $I^2 = 30\ \%$ ($P = 0.23$)]; however, heterogeneity remains in supine assessments at the time of laboratory assessments [0.45 ± 0.52; $I^2 = 67\ \%$ ($P = 0.08$)] and during seated assessments upon morning waking [0.33 ± 0.63; $I^2 = 56\ \%$ ($P = 0.13$)] and at the time of laboratory assessments [0.57 ± 0.77; $I^2 = 63\ \%$ ($P = 0.10$)].

Although some statistical heterogeneity in HFP (and indeed in other indices of HRV) may theoretically be attributed to methodological issues such as assessment

Table 5 Sub-group analyses of the effect of training leading to increased or decreased exercise performance on resting heart rate variability

Posture	HRV index	Time of day		
		Morning waking	Sleeping	At time of laboratory test
Studies reporting increased exercise performance				
Supine	RMSSD	0.57 (0.32–0.82) [31]	0.51 (0.02–1.00) $P = 0.04$; $I^2 = 70$ % ($P = 0.07$) [30[a], 48[a]]	NA
	HFP	NA	0.38 (0.02–0.75) $P = 0.04$; $I^2 = 30$ % ($P = 0.23$) [30[b], 48[b]]	0.45 (−0.07 to 0.97) $P = 0.09$; $I^2 = 67$ % ($P = 0.08$) [32, 33]
	SD1	NA	0.28 (−0.09 to 0.65) [30[c]]	NA
	Pooled	0.57 (0.32–0.82) [31]	0.40 (0.20–0.60) $P < 0.001$; $I^2 = 33$ % ($P = 0.20$) [30[a,b,c], 48[a,b]]	0.45 (−0.07 to 0.97) $P = 0.09$; $I^2 = 67$ % ($P = 0.08$) [32, 33]
Seated	RMSSD	NA	NA	0.58 (−0.10 to 1.25) $P = 0.09$; $I^2 = 52$ % ($P = 0.15$) [46[a], 47[a]]
	HFP	0.33 (−0.30 to 0.96) $P = 0.30$; $I^2 = 56$ % ($P = 0.13$) [35[a], 35[b]]	NA	0.57 (−0.20 to 1.34) $P = 0.15$; $I^2 = 63$ % ($P = 0.10$) [46[b], 47[b]]
	SD1	0.51 (0.12–0.90) [50]	NA	−0.08 (−1.06 to 0.90) [46[c]]
	Pooled	0.42 (0.09–0.75) $P = 0.01$; $I^2 = 23$ % ($P = 0.27$) [35[a,b], 50]	NA	0.59 (0.25–0.92) $P < 0.001$; $I^2 = 47$ % ($P = 0.11$) [46[a,b,c], 47[a,b]]
Standing	RMSSD	NA	NA	NA
	HFP	0.92 (0.18–1.67) $P = 0.01$; $I^2 = 71$ % ($P = 0.06$) [35[a], 35[b]]	NA	NA
	SD1	−0.32 (−1.05 to 0.41) [36]	NA	NA
	Pooled	0.54 (−0.30 to 1.38) $P = 0.20$; $I^2 = 82$ % ($P = 0.003$) [35[a,b], 36]	NA	NA
Studies reporting decreased exercise performance				
Supine	RMSSD	0.39 (−0.02 to 0.80) [39[a]]	0.09 (−0.26 to 0.44) $P = 0.61$; $I^2 = 0$ % ($P = 0.65$) [41[a], 41[b]]	NA
	HFP	0.14 (−0.25 to 0.53) [39[b]]	−0.10 (−0.40 to 0.20) $P = 0.52$; $I^2 = 20$ % ($P = 0.29$); [40[a], 40[b], 41[c], 41[d]]	−0.31 (−0.90 to 0.28) [34]
	SD1	NA	0.04 (−0.31 to 0.39) $P = 0.82$; $I^2 = 0$ % ($P = 0.46$) [41[e], 41[f]]	NA
	Pooled	0.26 (−0.02 to 0.54) $P = 0.07$; $I^2 = 0$ % ($P = 0.39$) [39[a,b]]	−0.01 (−0.19 to 0.17) $P = 0.87$; $I^2 = 0$ % ($P = 0.61$) [40[a,b], 41[a,b,c,d,e,f]]	−0.31 (−0.90 to 0.28) [34]
Seated	No studies identified			
Standing	RMSSD	0.34 (0.01–0.67) [39[c]]	NA	NA
	HFP	0.49 (0.24–0.74) [39[d]]	NA	NA
	SD1	NA	NA	NA
	Pooled	0.43 (0.23–0.64) ($P < 0.001$); $I^2 = 0$ % ($P = 0.48$) [39[c,d]]	NA	NA

Data are SMD (95 % CI), statistical significance; I^2 heterogeneity statistic (statistical significance of heterogeneity) [study reference number]

HFP high-frequency power from spectral analysis, *HRV* heart rate variability, *NA* not applicable, *RMSSD* root-mean-square difference of successive normal *R–R* intervals from time-domain analysis, *SD1* standard deviation of instantaneous beat-to-beat *R–R* interval variability from Poincare plots, *SMD* standardized mean difference

[a,b,c,d,e,f] Represent multiple HRV measures within a single study

posture and the time of day at which the recording was taken, the small number of studies that could be included in the sub-group analyses within this review demand that these findings be interpreted with caution. Indeed, this review indicates that more research is required to fully deduce the impact of these methodological considerations on resting HRV.

The overall increase in resting parasympathetic HR modulation demonstrated by RMSSD and HFP suggest that these vagal-related measures of resting HRV may be

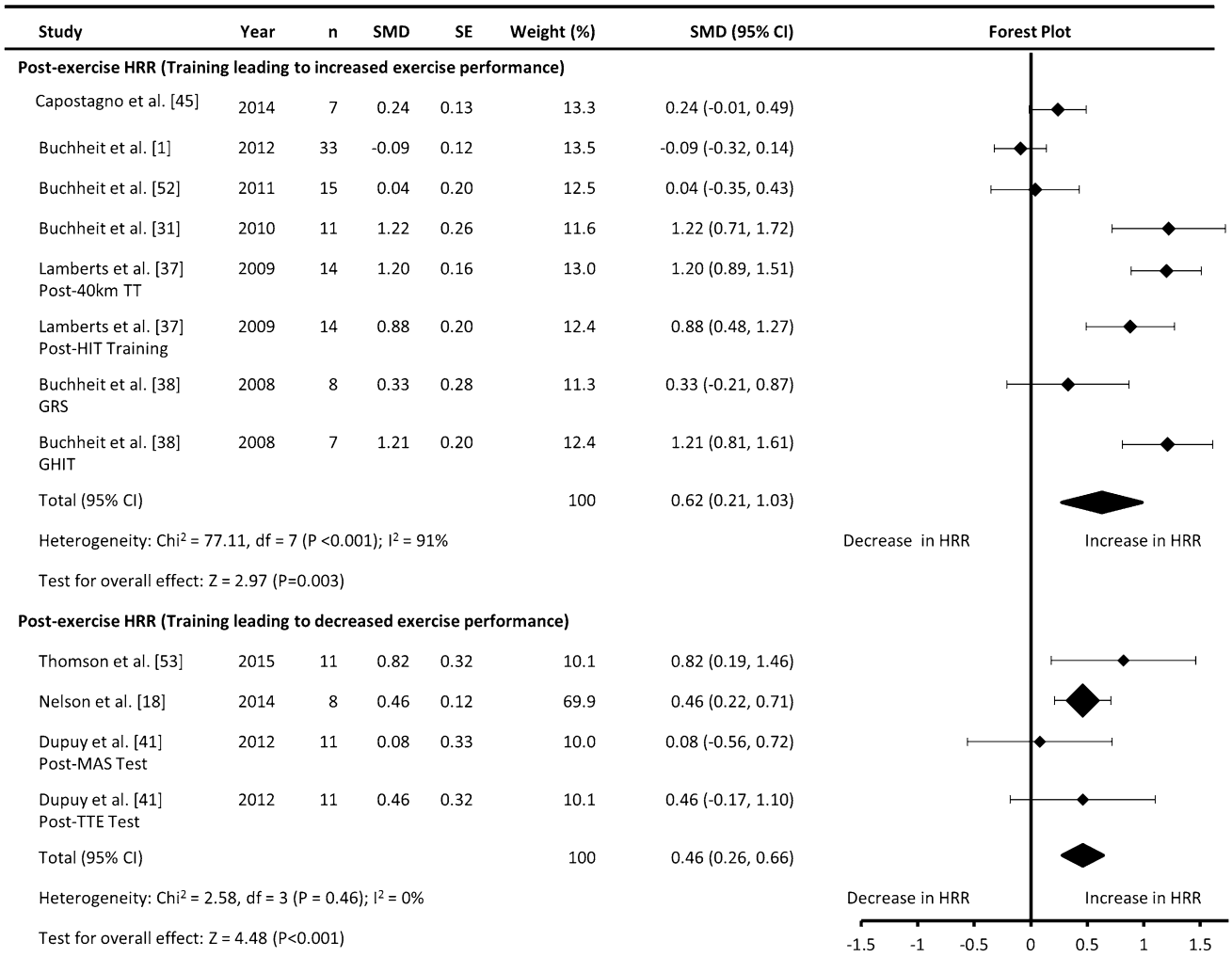


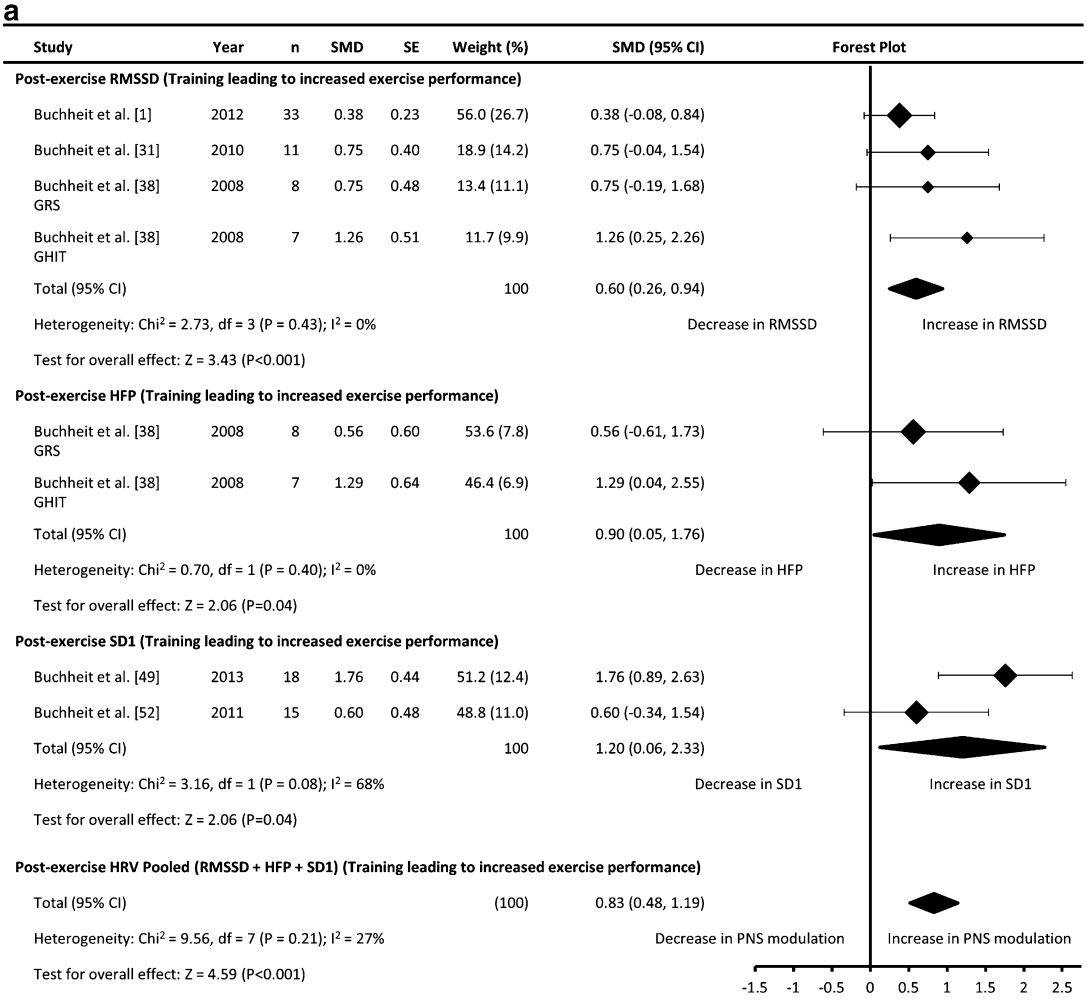
Fig. 3 Effect of training leading to increased and decreased exercise performance on post-exercise heart rate recovery. *CI* confidence interval, *GHIT* group randomized to perform HIT intervention, *GRS* group randomized to perform repeated sprint training intervention, *HIT* high-intensity training, *HRR* heart rate recovery, *MAS* maximal aerobic speed, *n* number of participants, *SE* standard error, *SMD* standardized mean difference, *TT* time trial, *TTE* time to exhaustion. Effects were quantified as trivial (<0.2), small (0.2–0.6), moderate (0.61–1.2), large (1.21–2.0), and very large (>2.0)

sensitive markers for detecting and monitoring positive adaptations to training in athletic populations. This increase in parasympathetic modulation is the result of the classical physiological adaptations shown following endurance training interventions. These adaptations have been described extensively [5, 90, 91], but briefly, endurance training causes a volume load on the heart, which in turn stimulates an increase in left ventricular internal dimension and wall thickness, and in end-diastolic volume (due to increased plasma volume and decreased peripheral resistance), ultimately leading to an increase in stroke volume. An enhanced stroke volume allows for a decrease in HR to maintain cardiac output (at least at rest and during sub-maximal exercise) while decreasing the metabolic load on the heart and creating a more efficient time-pressure relationship. The decrease in HR at rest is due in part to an increase in parasympathetic modulation, which is consequently reflected in an increase in vagal-related indices of HRV.

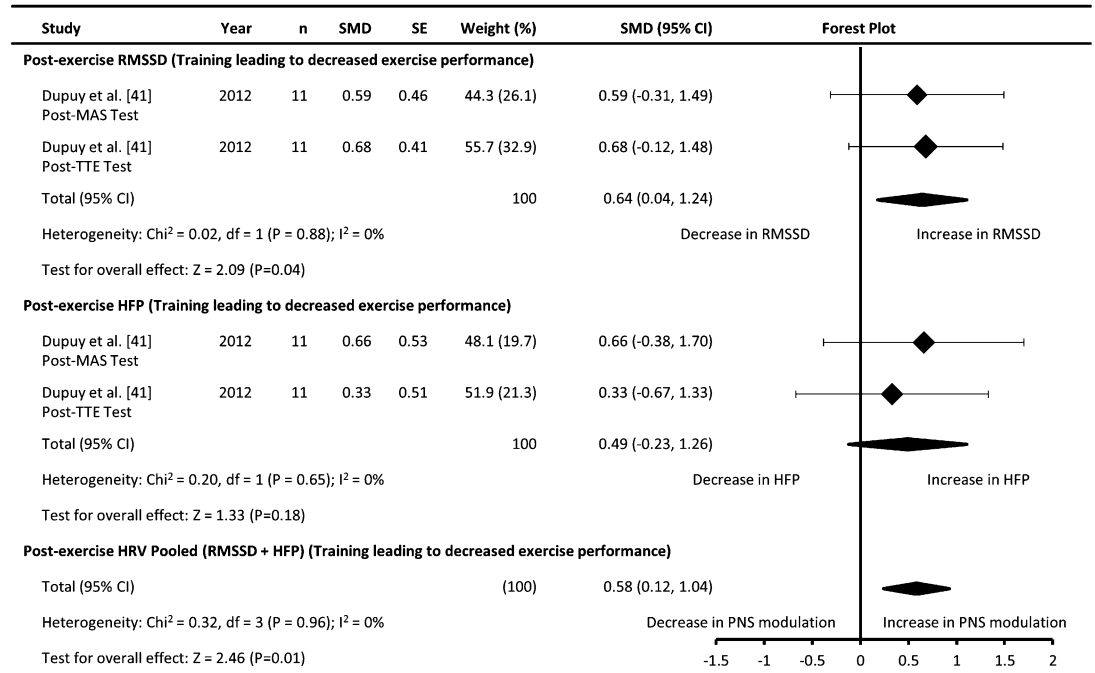
4.1.2 Training Leading to Decreased Exercise Performance

This review identified seven studies designed to induce a state of overreaching and evaluate its effect on resting HRV; however, due to the inclusion criteria set, only four studies were eligible for inclusion in meta-analysis. These studies showed a small increase in RMSSD (0.26 ± 0.21), but trivial changes in HFP and SD1 (0.04 ± 0.29 and 0.04 ± 0.35, respectively). The analysis of HFP was again

a


Study Year n SMD SE Weight (%) SMD (95% CI) Forest Plot
Post-exercise RMSSD (Training leading to increased exercise performance)
Buchheit et al. [1] 2012 33 0.38 0.23 56.0 (26.7) 0.38 (-0.08, 0.84)
Buchheit et al. [31] 2010 11 0.75 0.40 18.9 (14.2) 0.75 (-0.04, 1.54)
Buchheit et al. [38] GRS 2008 8 0.75 0.48 13.4 (11.1) 0.75 (-0.19, 1.68)
Buchheit et al. [38] GHIT 2008 7 1.26 0.51 11.7 (9.9) 1.26 (0.25, 2.26)
Total (95% CI) 100 0.60 (0.26, 0.94)
Heterogeneity: Chi² = 2.73, df = 3 (P = 0.43); I² = 0%
Test for overall effect: Z = 3.43 (P<0.001)
Decrease in RMSSD
Increase in RMSSD
Post-exercise HFP (Training leading to increased exercise performance)
Buchheit et al. [38] GRS 2008 8 0.56 0.60 53.6 (7.8) 0.56 (-0.61, 1.73)
Buchheit et al. [38] GHIT 2008 7 1.29 0.64 46.4 (6.9) 1.29 (0.04, 2.55)
Total (95% CI) 100 0.90 (0.05, 1.76)
Heterogeneity: Chi² = 0.70, df = 1 (P = 0.40); I² = 0%
Test for overall effect: Z = 2.06 (P=0.04)
Decrease in HFP
Increase in HFP
Post-exercise SD1 (Training leading to increased exercise performance)
Buchheit et al. [49] 2013 18 1.76 0.44 51.2 (12.4) 1.76 (0.89, 2.63)
Buchheit et al. [52] 2011 15 0.60 0.48 48.8 (11.0) 0.60 (-0.34, 1.54)
Total (95% CI) 100 1.20 (0.06, 2.33)
Heterogeneity: Chi² = 3.16, df = 1 (P = 0.08); I² = 68%
Test for overall effect: Z = 2.06 (P=0.04)
Decrease in SD1
Increase in SD1
Post-exercise HRV Pooled (RMSSD + HFP + SD1) (Training leading to increased exercise performance)
Total (95% CI) (100) 0.83 (0.48, 1.19)
Heterogeneity: Chi² = 9.56, df = 7 (P = 0.21); I² = 27%
Test for overall effect: Z = 4.59 (P<0.001)
Decrease in PNS modulation
Increase in PNS modulation
-1.5 -1 -0.5 0 0.5 1 1.5 2 2.5

b


Study Year n SMD SE Weight (%) SMD (95% CI) Forest Plot
Post-exercise RMSSD (Training leading to decreased exercise performance)
Dupuy et al. [41] Post-MAS Test 2012 11 0.59 0.46 44.3 (26.1) 0.59 (-0.31, 1.49)
Dupuy et al. [41] Post-TTE Test 2012 11 0.68 0.41 55.7 (32.9) 0.68 (-0.12, 1.48)
Total (95% CI) 100 0.64 (0.04, 1.24)
Heterogeneity: Chi² = 0.02, df = 1 (P = 0.88); I² = 0%
Test for overall effect: Z = 2.09 (P=0.04)
Decrease in RMSSD
Increase in RMSSD
Post-exercise HFP (Training leading to decreased exercise performance)
Dupuy et al. [41] Post-MAS Test 2012 11 0.66 0.53 48.1 (19.7) 0.66 (-0.38, 1.70)
Dupuy et al. [41] Post-TTE Test 2012 11 0.33 0.51 51.9 (21.3) 0.33 (-0.67, 1.33)
Total (95% CI) 100 0.49 (-0.23, 1.26)
Heterogeneity: Chi² = 0.20, df = 1 (P = 0.65); I² = 0%
Test for overall effect: Z = 1.33 (P=0.18)
Decrease in HFP
Increase in HFP
Post-exercise HRV Pooled (RMSSD + HFP) (Training leading to decreased exercise performance)
Total (95% CI) (100) 0.58 (0.12, 1.04)
Heterogeneity: Chi² = 0.32, df = 3 (P = 0.96); I² = 0%
Test for overall effect: Z = 2.46 (P=0.01)
Decrease in PNS modulation
Increase in PNS modulation
-1.5 -1 -0.5 0 0.5 1 1.5 2

◀**Fig. 4** Effect of training leading to **a** increased and **b** decreased exercise performance on post-exercise heart rate variability. *CI* confidence interval, *GHIT* group randomized to perform high-intensity training intervention, *GRS* group randomized to perform repeated sprint training intervention, *HFP* high-frequency power, *HRV* heart rate variability, *MAS* maximal aerobic speed, *n* number of participants, *PNS* parasympathetic nervous system, *RMSSD* the root-mean-square difference of successive normal *R–R* intervals, *SD1* standard deviation of instantaneous beat-to-beat *R–R* interval variability, *SE* standard error, *SMD* standardized mean difference, *TTE* time to exhaustion. Effects were quantified as trivial (<0.2), small (0.2–0.6), moderate (0.61–1.2), large (1.21–2.0), and very large (>2.0)

affected by statistical heterogeneity, and thus these results also suggest that RMSSD may be a more sensitive marker of training status than HFP. However, it should be noted that only two studies (each with two data points) were included in the analysis of RMSSD, and consequently this interpretation may be biased by such a small sample size with multiple comparisons from the same studies.

The presence of statistical heterogeneity within the analysis of HFP is likely caused by the apparent disagreement in the directional change of HFP amongst the identified studies; Dupuy et al. [41] and Le Meur et al. [39] showed increases, but Hedelin et al. [34] and Chalencon et al. [40] showed decreases. Such disagreement extends beyond those studies included in meta-analysis, with three studies included only in qualitative synthesis also showing conflicting effects. Baumert et al. [44] and Garet et al. [42] showed decreases in HFP (and also RMSSD) following 2–3 weeks of overload training leading to reductions in exercise performance, while Bosquet et al. [43] found increased HFP following 3 weeks of overload training.

Taken together, the results of these seven studies highlight that HFP can increase or decrease in association with overreaching-induced reductions in exercise performance (which may also apply to measures of RMSSD given the small number of data points analysed in this review). The recent studies of Plews et al. [67, 86] and Le Meur et al. [39] offer two potential explanations for the disagreement between these studies. First, Plews et al. [67] showed that the distribution of training intensity affects the directional change in vagal-related indices of HRV, with extended periods of training at high intensities (i.e., at a power output greater than that associated with the individual anaerobic threshold) inducing decreases in parasympathetic modulation, and training at low intensities (i.e., at a power output less than that associated with the individual aerobic threshold) resulting in increases in parasympathetic modulation. This may explain the results of Bosquet et al. [43] and Baumert et al. [44], since Bosquet et al. [43] showed an increase in HFP in response to a 100 % increase in the time spent in long-duration low-intensity running, which may closely approximate the intensity associated with the aerobic threshold, while Baumert et al. [44] showed a decrease in RMSSD and HFP in response to training at 85–90 % of maximum HR, which may be considered an intensity close to or above the anaerobic threshold. However, the results of Hedelin et al. [34] and Le Meur et al. [39] cannot be explained by the findings of Plews et al. [67], with Hedelin et al. [34] reporting a decrease in HFP despite the increase in training load eliciting a 25 % increase in the amount of time spent above the anaerobic threshold, and a 67 % increase in the amount of time spent below this threshold. Le Meur et al. [39] reported increases in RMSSD and HFP

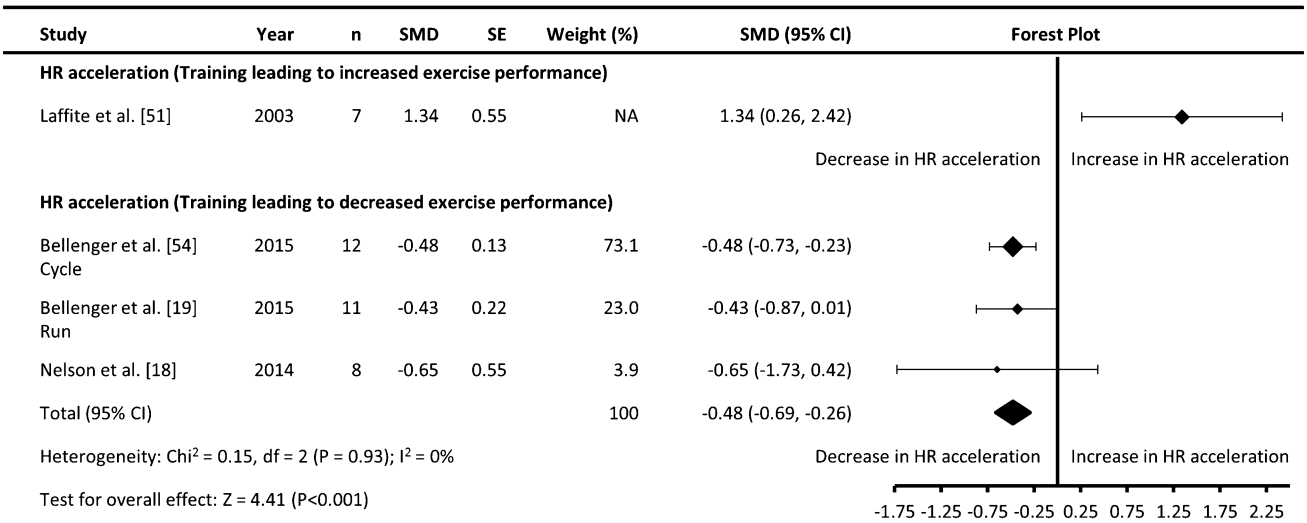


Fig. 5 Effect of training leading to increased and decreased exercise performance on heart rate acceleration. *CI* confidence interval, *HR* heart rate, *NA* not applicable, *n* number of participants, *SE* standard error, *SMD* standardized mean difference. Effects were quantified as trivial (<0.2), small (0.2–0.6), moderate (0.61–1.2), large (1.21–2.0), and very large (>2.0)

despite similar percentage increases (~45 %) in the amount of time spent above and below the anaerobic and aerobic thresholds, respectively, and a 65 % increase in the amount of time spent between these thresholds. Chalencon et al. [40], Dupuy et al. [41], and Garet et al. [42] did not report exercise intensity distribution sufficiently to draw conclusions on this potential moderator variable.

Additionally, Le Meur at al. [39] and Plews at al. [82, 86] recently found that calculating weekly or 7-day rolling averages of HRV (i.e., average of HRV recordings taken each day during their interventions) resulted in enhanced sensitivity for detecting changes in training status compared with isolated measures of HRV (i.e., the HRV recordings taken on the days pre- and post-intervention). These studies showed greater effect sizes, and effect sizes that more closely approximated the concurrent change in performance, when utilizing average measures compared with isolated measures [39, 86]. Plews et al. [86] also found that changes in average HRV correlated more strongly with changes in performance compared with changes in isolated measures, demonstrating a heightened sensitivity of average HRV values for tracking performance changes. The greater effect size and heightened sensitivity of average HRV is likely due to the diminished influence of large day-to-day variations in measures of resting HRV, thereby increasing the signal-to-noise ratio of this parameter [28]. Such variability (~12.5 % co-efficient of variation for vagal-related indices [88]) may be caused by any number of variables that affect the measurement of HRV, including sleep quality and quantity [35], loud startling noises and the presence of light [10], psychological stressors [92], environmental conditions [93], exercise-induced changes in blood plasma volume [67], and the presence of residual (acute) fatigue from previous training sessions [94]. Effectively, average measures of HRV are able to smooth the noise in isolated values of HRV, allowing for improved accuracy and more meaningful assessment of training status. Given these recent findings, the disagreement in overreaching-induced directional change in resting HFP may be due to the studies reviewed using isolated measures of HRV.

If the above hypothesis is correct, then the single study by Le Meur et al. [39] utilizing weekly average measures of HRV would provide the best current evidence for the effect of heavy training on resting indices of HRV, and this study showed an increase in parasympathetic modulation at rest (as assessed by RMSSD and HFP: SMD = 0.62 and 0.91, respectively) in functionally overreached athletes. These authors refer to this increase as parasympathetic hyperactivity and suggest that its presence during maximal exercise may limit the ability to fully engage the sympathetic nervous system, which is supported by the reduction in maximal HR found in their study, and in many other studies of overreaching (see Bosquet et al. [9] and Achten and Jeukendrup [95] for reviews), ultimately leading to a reduction in maximal cardiac output and reduced performance. Unfortunately, the mechanism by which a parasympathetic hyperactivity manifests during heavy training is not well understood at present.

With the limited research on measures of weekly or 7-day rolling average HRV in studies of overreaching, more research is required to confirm the findings of Le Meur et al. [39] and elucidate the mechanism by which potential parasympathetic hyperactivity occurs in the functionally overreached state.

We also need to acknowledge here that the recording posture and/or time of day may explain some of the heterogeneity and lack of agreement in resting HRV in studies leading to decreased performance; however, the small number of studies in these sub-analyses means that caution should be taken when interpreting their findings. This review may offer some support for standing measures (0.43 ± 0.20) over supine measures (0.26 ± 0.28) of resting HRV during morning waking assessments, although potential bias from the single study in these sub-analyses [39] warrants additional research to confirm these results. By extension, these morning waking sub-analyses may also offer some support for this recording over supine recordings taken during periods of sleep (-0.01 ± 0.18) and at the time of laboratory visits (-0.31 ± 0.59).

4.2 Post-Exercise HRR

4.2.1 Training Leading to Increased Exercise Performance

A moderate (0.63 ± 0.40) increase in post-exercise HRR was found in studies inducing improvements in performance, highlighting it as a marker of positive training adaptation. However, the analysis of post-exercise HRR was significantly affected by statistical heterogeneity, which may be caused by the intensity of the exercise preceding the measurement of HRR, since this has previously been shown to affect the reliability of this parameter's assessment [88, 96]. The studies identified by this review demonstrated a wide range in exercise intensity utilized (~68–98 % of maximum HR; Tables 1 and 2).

Since HRR is the result of coordinated interaction between parasympathetic re-activation and sympathetic withdrawal [3], the improvement in HRR following training interventions inducing improvements in performance may be due to an increase in parasympathetic and/or a decrease in sympathetic modulation of HR. Such change in autonomic activity is supported by cross-sectional research, where a faster HRR was demonstrated in athletes than in non-athletes, and these athletes had greater levels of parasympathetic HR modulation as assessed by resting HRV [97, 98]. An increase in parasympathetic modulation in this context is

supported by the increase in resting parasympathetic modulation discussed in Sect. 4.1.1, and may be considered a positive adaptation to training as it would allow for a faster return to homeostasis following an exercise stress [90].

4.2.2 *Training Leading to Decreased Exercise Performance*

While few studies to date have investigated the effects of overreaching training leading to decreased exercise performance on HRR (only three studies were identified), those that are available indicated that HRR was accelerated under these conditions (0.46 ± 0.20).

It is interesting to note that while the majority of studies identified for inducing increases in performance assessed HRR following exercise bouts at sub-maximal intensities, all three studies inducing reductions in performance assessed HRR after maximal exercise. Since the aim of continuous monitoring of HR parameters is to predict training status (for which the gold standard assessment is maximal performance), the finding of accelerated HRR in an overreached state in these three studies is essentially redundant, since a measure of performance (time to complete a set distance, time to exhaustion, maximal aerobic power, or speed) was also measured. Additionally, it may be contraindicated to have an athlete exercise at maximal intensities if they are at risk of developing non-functional overreaching or overtraining, since this will only exacerbate the condition. Future studies should therefore investigate the effect of overreaching on HRR derived from sub-maximal intensities to ascertain whether it is truly a practically applicable measure of fatigue status.

The faster HRR in an overreached state does seem to be paradoxical considering that this review also showed a faster HRR when athletes experienced improvements in performance. However, this finding does align with the potential parasympathetic hyperactivity shown by Le Meur et al. [39] using weekly average values of resting HRV. Given the interaction of parasympathetic re-activation (responsible for the early and rapid deceleration in HR following exercise) and sympathetic withdrawal (responsible for a relatively slower deceleration) to the decrease in post-exercise HRR [14, 99], it is known that excessive sympathetic stimulation during exercise causes a slower HRR when withdrawn, while lower sympathetic stimulation causes a faster HRR [3]. Thus, it may be hypothesized that training interventions inducing a parasympathetic hyperactivity (and/or a decrease in sympathetic stimulation) result in a faster HRR. The three studies identified by this review all measured HRR at the cessation of maximal exercise, and their interventions induced a decrease in maximal HR (SMD = −0.52), indicating that parasympathetic and/or sympathetic modulation were indeed heightened and/or reduced, respectively, potentially leading to a faster HRR in the fatigued state. In further support of this hypothesis, there is recent evidence of a correlation between decreased catecholamine excretion at maximal exertion (indicative of decreased sympathetic modulation) and increased HRR in overreached athletes [100].

The increase in HRR in an overreached state makes the interpretation of changes in this parameter following changes in training load difficult. Since this review suggests that HRR is increased when athletes experience increases and decreases in performance, supplementary markers of training load and tolerance of this training load (e.g., subjective questionnaires) may be required to put these changes into context. For example, if training load is increased substantially and athletes report negative tolerance to such an increase, then an increase in HRR likely reflects a negative response to training (potentially a functionally overreached state leading to reduced performance) and may indicate the need for training load to be reduced to facilitate subsequent positive adaptions to training.

4.3 Post-Exercise HRV

4.3.1 *Training Leading to Increased Exercise Performance*

Training interventions leading to increased performance resulted in moderate increases in post-exercise RMSSD (0.60 ± 0.34), HFP (0.90 ± 0.85), and SD1 (1.20 ± 1.13), demonstrating an increase in post-exercise parasympathetic HR modulation as a positive adaptation to training. This finding is in line with the changes in resting HRV and post-exercise HRR identified in this review, which also demonstrate an increase in parasympathetic modulation (and/or a decrease in sympathetic activity in the case of HRR). Again, the increase in post-exercise vagal-related indices of HRV is likely due to classical cardiovascular adaptations discussed in Sect. 4.1.1; however, in a similar sense to post-exercise HRR, these adaptations indicate a heightened ability to return to homeostasis following an exercise stressor.

4.3.2 *Training Leading to Decreased Exercise Performance*

Only one study was identified for investigating the effect of overreaching interventions leading to decreased performance on post-exercise HRV. However, this study assessed both RMSSD and HFP following two independent exercise tasks, and thus multiple comparisons were made. The results of this study show a moderate increase in RMSSD (0.64 ± 0.60), and a small increase in HFP (0.49 ± 0.77),

leading to a small increase in pooled vagal-related indices of HRV (0.58 ± 0.46). These results suggest that training leading to decreased performance results in an increase in post-exercise parasympathetic HR modulation. While this interpretation should be considered with care since this analysis includes only a single study with potential bias from multiple measures, these results may be supported by the analysis of post-exercise HRR which also shows an increase in parasympathetic modulation (and/or a decrease in sympathetic modulation) of HRR under conditions of fatigue. Given the relative lack of research on post-exercise HRV following overreaching interventions, more research on this autonomic HR parameter as an indicator of overreaching is warranted.

4.4 HR Acceleration

This review highlights the relative lack of research on HR acceleration kinetics at the onset of exercise as a marker of autonomic HR regulation and athletic training status. A single study was identified for assessing the effect of positive training adaptation on this parameter, while two studies were identified for quantifying the effect of negative training adaptation.

4.4.1 Training Leading to Increased Exercise Performance

A large (1.34 ± 1.08) increase in HR acceleration as a positive adaptation to training was found in the single study evaluating this parameter. While caution must be taken when interpreting the results of this parameter, given the limited research, this finding supports the cross-sectional research of Bunc et al. [16], who found faster HR acceleration in trained athletes than in untrained subjects. The identified study also reported a large increase in oxygen uptake acceleration kinetics at the onset of exercise (SMD = 1.81), supporting the hypothesis that improved HR acceleration contributes to a more rapid increase in oxygen delivery to active muscle, which may reduce peripheral muscle fatigue and facilitate improved performance.

Since it has been shown that the rapid increase in HR at the onset of exercise is a result of parasympathetic withdrawal, with sympathetic activation causing a relatively slower increase in HR as exercise intensity increases [6, 15, 101], an increase in HR acceleration may be indicative of an increase in parasympathetic and/or a decrease in sympathetic modulation of the HR response. These mechanisms are supported by the various findings of this review, with an increase in parasympathetic modulation shown by measures of resting and post-exercise HRV (and possibly post-exercise HRR) as a positive training adaptation, and a decrease in sympathetic modulation perhaps demonstrated by post-exercise HRR.

4.4.2 Training Leading to Decreased Exercise Performance

A moderate (−0.48 ± 0.21) decrease in HR acceleration, as measured by rHRI, was found following overreaching interventions, suggesting that this parameter may be a useful marker for identifying the accumulation of training-induced fatigue.

The slowing of rHRI under conditions of fatigue is likely the result of a shift in the balance of parasympathetic and/or sympathetic HR regulation as shown by the various HR parameters assessed in this review (i.e., a potential parasympathetic hyperactivity and/or suppression of sympathetic modulation). Given that relatively little is known about rHRI at present, the exact mechanism is poorly understood and should be the focus of future research.

The small body of literature currently conducted on HR acceleration at the onset of exercise demonstrates that increases in this parameter reflect a positive training adaptation, and decreases reflect an accumulation of training-induced fatigue. However, the results of Bellenger et al. [19] suggest that rHRI may be affected by exercise intensity in a similar sense to HRR, and thus an optimal intensity may be required to most sensitively apply this parameter as a marker of training status. It is also presently unknown whether HR acceleration, in a similar sense to resting HRV, is a more sensitive marker of training status when assessed daily, allowing for a rolling or weekly average to be utilized rather than isolated pre- and post-intervention values, which may be more variable. As such, more research on HR acceleration as a marker of athletic training status is warranted.

5 Limitations

Where applicable, this review attempted to investigate and explain the presence of any statistical heterogeneity in the HR parameters assessed. As mentioned in earlier sections, this heterogeneity may be the result of methodological issues in the assessment of HRV and HRR. However, it should be acknowledged that the presence of heterogeneity in any of the HR parameters assessed in this review may simply be the result of variance in the magnitude of performance change induced by each study, since it may be assumed that those studies inducing a larger change in performance should also elicit a larger change in autonomic HR regulation. The range of performance change shown in Tables 1 and 2 is likely influenced by differences in the intensity versus volume distribution of each training

intervention, and the length of time that it was performed for, in addition to the subtle differences in the caliber of athletes utilized in these studies (i.e., recreational vs. elite).

This literature review may also be limited by the individual studies identified failing to differentiate those athletes who were truly overreached (i.e., experiencing a decline in performance) from those that were only 'acutely fatigued' (i.e., experiencing preserved performance but with high levels of perceived fatigue) [102]. As a result of inter-individual variability, not all athletes within a cohort will necessarily become overreached by the end of a training intervention. However, many studies reported the mean effect of the intervention on performance but not the number of individuals who experienced a meaningful decline in performance. Thus, it was difficult to determine whether the HR response in those athletes experiencing acute fatigue differed from the response in those athletes who were truly overreached. If it were indeed varied, this response may attenuate the true effect of overreaching on autonomic HR regulation, and decrease its sensitivity for predicting athletic training status. Future overreaching studies should therefore account for such inter-athlete variability, potentially by performing sub-group analyses on acutely fatigued and overreached athletes, excluding acutely fatigued athletes from analyses (provided the study's aim was to investigate the effect of overreaching on autonomic HR regulation), or at the very least reporting how many athletes were truly overreached.

Additionally, this review could not accurately differentiate between the training states of functional overreaching, non-functional overreaching, or overtraining. From a practical view, since a state of functional overreaching may ultimately lead to super-compensatory performance adaptations, coaches and athletes would be utilizing parameters of autonomic HR regulation to identify this state of training before training-induced fatigue gives way to non-functional overreaching or overtraining, as these states may result in longer-term attenuation of performance. However, since this review could not differentiate between these training states, it is difficult to determine whether the HR response in athletes experiencing functional overreaching differs from that of athletes who are experiencing non-functional overreaching or overtraining. Again, this may attenuate the true effect of functional overreaching on autonomic HR regulation and decrease its sensitivity for predicting athletic training status.

6 Conclusion

This review sought to examine the effect of training interventions inducing positive and negative adaptations to training, as characterized by improvements and decrements in exercise performance respectively, on measures of autonomic HR regulation.

With regard to interventions inducing improvements in performance, concurrent increases in measures of resting HRV, post-exercise HRV, post-exercise HRR, and HR acceleration were found. These increases were likely the result of increases in parasympathetic HR modulation (and/or decreases in sympathetic modulation in the case of post-exercise HRR and HR acceleration) facilitated by positive adaptations to athletic training.

Studies leading to reductions in performance suggested that overreaching had little effect on resting HRV, as there appears to be disagreement in the direction of change in vagal-related indices of HRV. This disagreement may be the result of methodological issues that should be investigated further, namely the effect of utilizing isolated values compared with weekly/rolling average values, and also the impact of posture and recording time of day. Additionally, this review highlights that increases in post-exercise HRR occur in association with both increases and decreases in exercise performance, which makes the use of post-exercise HRR as an isolated marker of athletic training status difficult. Consequently, additional monitoring variables, such as quantification of total training load and the rate at which that training load is applied, in addition to the athlete's subjective perception of training tolerance, may be required to contextualize changes in HRR. Surprisingly, only one study investigated the effect of overreaching training on post-exercise HRV, and again found an increase in this marker, also making its interpretation in isolation difficult. Limited studies have investigated the effect of training on HR acceleration, and thus more research is required. However, this review shows a decrease in this HR acceleration in response to overreaching training, and thus it may be a potential indicator of training-induced fatigue.

Compliance with Ethical Standards

Funding No sources of funding were used to assist in the preparation of this review. Rebecca Thomson, Eileen Robertson, and Jonathan Buckley are the recipients of a recent Australian Research Council Linkage grant (LP140101013) that was awarded to investigate the potential for the HR acceleration parameter (maximal rate of HR increase) referred to in this review to track how athletes are tolerating changes in training load. This grant does not relate to the work described in the current manuscript, but is for future work.

Conflicts of interest Clint Bellenger, Joel Fuller, Rebecca Thomson, Kade Davison, and Eileen Robertson declare that they have no conflicts of interest relevant to the content of this review. Jonathan Buckley invented the HR acceleration parameter (maximal rate of HR increase) referred to in this review, and the parameter is patented by the University of South Australia. The University of South Australia is seeking to license the technology for commercial gain, and Jonathan Buckley may benefit financially from any such license.

References

1. Buchheit M, Simpson M, Al Haddad H, et al. Monitoring changes in physical performance with heart rate measures in young soccer players. Eur J Appl Physiol. 2012;112:711–23.
2. Borresen J, Lambert MI. The quantification of training load, the training response and the effect on performance. Sports Med. 2009;39:779–95.
3. Borresen J, Lambert MI. Autonomic control of heart rate during and after exercise: measurements and implications for monitoring training status. Sports Med. 2008;38:633–46.
4. Meeusen R, Duclos M, Foster C, et al. Prevention, diagnosis, and treatment of the overtraining syndrome: Joint Consensus Statement of the European College of Sport Science and the American College of Sports Medicine. Med Sci Sports Exerc. 2013;45:186–205.
5. Aubert AE, Seps B, Beckers F. Heart rate variability in athletes. Sports Med. 2003;33:889–919.
6. Robinson BF, Epstein SE, Beiser GD, et al. Control of heart rate by the autonomic nervous system: Studies in man on the interrelation between baroreceptor mechanisms and exercise. Circ Res. 1966;19:400–11.
7. Pichot V, Busso T, Roche F, et al. Autonomic adaptations to intensive and overload training periods: a laboratory study. Med Sci Sports Exerc. 2002;34:1660–6.
8. Pichot V, Roche F, Gaspoz J, et al. Relation between heart rate variability and training load in middle-distance runners. Med Sci Sports Exerc. 2000;32:1729–36.
9. Bosquet L, Merkari S, Arvisais D, et al. Is heart rate a convenient tool to monitor over- reaching? A systematic review of the literature. Br J Sports Med. 2008;42:709–14.
10. Malik M, Bigger JT, Camm AJ, et al. Heart rate variability: standards of measurement, physiological interpretation, and clinical use. Eur Heart J. 1996;17:354–81.
11. Malpas SC. Neural influences on cardiovascular variability: possibilities and pitfalls. Am J Physiol Heart Circ Physiol. 2002;51:H6–20.
12. Borresen J, Lambert MI. Changes in heart rate recovery in response to acute changes in training load. Eur J Appl Physiol. 2007;101:503–11.
13. Imai K, Sato H, Hori M, et al. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. J Am Coll Cardiol. 1994;24:1529–35.
14. Kannankeril PJ, Le FK, Kadish AH, et al. Parasympathetic effects on heart rate recovery after exercise. J Invest Med. 2004;52:394–401.
15. Victor RG, Seals DR, Mark AL. Differential control of heart rate and sympathetic nerve activity during dynamic exercise: insight from intraneural recordings in humans. J Clin Invest. 1987;79:508–16.
16. Bunc V, Heller J, Leso J. Kinetics of heart rate responses to exercise. J Sports Sci. 1988;6:39–48.
17. Amann M, Calbet JA. Convective oxygen transport and fatigue. J Appl Physiol. 2008;104:861–70.
18. Nelson MJ, Thomson RL, Rogers DK, et al. Maximal rate of increase in heart rate during the rest-exercise transition tracks reductions in exercise performance when training load is increased. J Sci Med Sport. 2014;17:129–33.
19. Bellenger CR, Thomson RL, Howe PRC, et al. Monitoring athletic training status using the maximal rate of heart rate increase. J Sci Med Sport. doi:10.1016/j.jsams.2015.07.006 **[Epub ahead of print]**.
20. Thomson RL, Rogers DK, Howe PRC, et al. Effect of acute exercise-induced fatigue on maximal rate of heart rate increase during submaximal cycling. Res Sports Med. 2015. doi:10.1080/15438627.2015.1076414.
21. Moher D, Liberati A, Tetzlaff J, et al. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. BMJ. 2009;339:332–6.
22. Halson SL, Jeukendrup AE. Does overtraining exist? An analysis of overreaching and overtraining research. Sports Med. 2004;34:967–81.
23. Urhausen A, Kindermann W. Diagnosis of overtraining: what tools do we have? Sports Med. 2002;32:95–102.
24. Higgins JPT, Altman DG, Gøtzsche PC, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. BMJ. 2011;343:889.
25. Cohen J. Statistical power analysis for the behavioral sciences. Hillsdale: Sage Publications; 2010.
26. Elbourne DR, Altman DG, Higgins JP, et al. Meta-analyses involving cross-over trials: methodological issues. Int J Epidemiol. 2002;31:140–9.
27. Higgins JPT. Measuring inconsistency in meta-analyses. BMJ. 2003;327:557–60.
28. Buchheit M. Monitoring training status with HR measures: do all roads lead to Rome? Front Physiol. 2014;5:73.
29. Plews D, Laursen P, Stanley J, et al. Training adaptation and heart rate variability in elite endurance athletes: opening the door to effective monitoring. Sports Med. 2013;43:773–81.
30. Boullosa DA, Abreu L, Nakamura FY, et al. Cardiac autonomic adaptations in elite Spanish soccer players during preseason. Int J Sports Physiol Perform. 2013;8:400–9.
31. Buchheit M, Chivot A, Parouty J, et al. Monitoring endurance running performance using cardiac parasympathetic function. Eur J Appl Physiol. 2010;108:1153–67.
32. Carter JB, Banister EW, Blaber AP. The effect of age and gender on heart rate variability after endurance training. Med Sci Sports Exerc. 2003;35:1333–40.
33. Hedelin R, Wiklund U, Bjerle P, et al. Pre and post-season heart rate variability in adolescent cross-country skiers. Scand J Med Sci Sports. 2000;10:298–303.
34. Hedelin R, Kenttá G, Wiklund U, et al. Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. Med Sci Sports Exerc. 2000;32:1480–4.
35. Kiviniemi A, Hautala A, Kinnunen H, et al. Endurance training guided individually by daily heart rate variability measurements. Eur J Appl Physiol. 2007;101:743–51.
36. Wallace LK, Slattery KM, Coutts AJ. A comparison of methods for quantifying training load: relationships between modelled and actual training responses. Eur J Appl Physiol. 2014;114:11–20.
37. Lamberts R, Swart J, Noakes T, et al. Changes in heart rate recovery after high-intensity training in well-trained cyclists. Eur J Appl Physiol. 2009;105:705–13.
38. Buchheit M, Millet GP, Parisy A, et al. Supramaximal training and postexercise parasympathetic reactivation in adolescents. Med Sci Sports Exerc. 2008;40:362–71.
39. Le Meur Y, Pichon A, Schaal K, et al. Evidence of parasympathetic hyperactivity in functionally overreached athletes. Med Sci Sports Exerc. 2013;45:2061.
40. Chalencon S, Busso T, Lacour J-R, et al. A model for the training effects in swimming demonstrates a strong relationship between parasympathetic activity, performance and index of fatigue. PLoS One. 2012;7:1–10.
41. Dupuy O, Bherer L, Audiffren M, et al. Night and postexercise cardiac autonomic control in functional overreaching. Appl Physiol Nutr Metab. 2013;38:200.
42. Garet M, Tournaire N, Roche F, et al. Individual interdependence between nocturnal ANS activity and performance in swimmers. Med Sci Sports Exerc. 2004;36:2112–8.

43. Bosquet L, Papelier Y, Léger L, et al. Night heart rate variability during overtraining in male endurance athletes. J Sports Med Phys Fitness. 2003;43:506–12.
44. Baumert M, Brechtel L, Lock J, et al. Heart rate variability, blood pressure variability, and baroreflex sensitivity in overtrained athletes. Clin J Sport Med. 2006;16:412–7.
45. Capostagno B, Lambert MI, Lamberts RP. Standardized versus customized high-intensity training: effects on cycling performance. Int J Sports Physiol Perform. 2014;9:292–301.
46. Da Silva DF, Verri SM, Nakamura FY, et al. Longitudinal changes in cardiac autonomic function and aerobic fitness indices in endurance runners: a case study with a high-level team. Eur J Sport Sci. 2014;14:443–51.
47. Oliveira RS, Leicht AS, Bishop D, et al. Seasonal changes in physical performance and heart rate variability in high level futsal players. Int J Sports Med. 2013;34:424–30.
48. Vesterinen V, Hakkinen K, Hynynen E, et al. Heart rate variability in prediction of individual adaptation to endurance training in recreational endurance runners. Scand J Med Sci Sports. 2013;23:171–80.
49. Buchheit M, Racinais S, Bilsborough JC, et al. Monitoring fitness, fatigue and running performance during a pre-season training camp in elite football players. J Sci Med Sport. 2013;16:550–5.
50. Buchheit M, Simpson BM, Garvican-Lewis LA, et al. Wellness, fatigue and physical performance acclimatisation to a 2-week soccer camp at 3600 m (ISA3600). Br J Sports Med. 2013;47(Suppl 1):i100–6.
51. Laffite LP, Mille-Hamard L, Koralsztein JP, et al. The effects of interval training on oxygen pulse and performance in supra-threshold runs. Arch Physiol Biochem. 2003;111:202–10.
52. Buchheit M, Voss SC, Nybo L, et al. Physiological and performance adaptations to an in-season soccer camp in the heat: Associations with heart rate and heart rate variability. Scand J Med Sci Sports. 2011;21:e477–85.
53. Thomson RL, Bellenger CR, Howe PRC, et al. Improved heart rate recovery despite reduced exercise performance following heavy training: a within-subject analysis. J Sci Med Sport. 2015. doi:10.1016/j.jsams.2015.02.010.
54. Heydari M, Boutcher YN, Boutcher SH. High-intensity intermittent exercise and cardiovascular and autonomic function. Clin Auton Res. 2013;23:57–65.
55. Mourot L, Bouhaddi M, Perrey S, et al. Decrease in heart rate variability with overtraining: assessment by the Poincaré plot analysis. Clin Physiol Funct Imaging. 2004;24:10–8.
56. Kiviniemi AM, Hautala AJ, Kinnunen H, et al. Daily exercise prescription on the basis of HR variability among men and women. Med Sci Sports Exerc. 2010;42:1355–63.
57. Hautala AJ, Mäkikallio TH, Kiviniemi A, et al. Heart rate dynamics after controlled training followed by a home-based exercise program. Eur J Appl Physiol. 2004;92:289–97.
58. Gamelin FX, Berthoin S, Sayah H, et al. Effect of training and detraining on heart rate variability in healthy young men. Int J Sports Med. 2007;28:564–70.
59. Tian Y, He ZH, Zhao JX, et al. Heart rate variability threshold values for early-warning nonfunctional overreaching in elite female wrestlers. J Strength Cond Res. 2013;27:1511–9.
60. Sartor F, Vailati E, Valsecchi V, et al. Heart rate variability reflects training load and psychophysiological status in young elite gymnasts. J Strength Cond Res. 2013;27:2782–90.
61. Houmard JA, Kirwan JP, Flynn MG, et al. Effects of reduced training on submaximal and maximal running responses. Int J Sports Med. 1989;10:30–3.
62. Atlaoui D, Pichot V, Lacoste L, et al. Heart rate variability, training variation and performance in elite swimmers. Int J Sports Med. 2007;28:394–400.
63. Botek M, McKune AJ, Krejci J, et al. Change in performance in response to training load adjustment based on autonomic activity. Int J Sports Med. 2014;35:482–8.
64. Iellamo F, Pigozzi F, Spataro A, et al. Autonomic and psychological adaptations in Olympic rowers. J Sports Med Phys Fitness. 2006;46:598–604.
65. Iellamo F, Legramante JM, Pigozzi F, et al. Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. Circulation. 2002;105:2719–24.
66. Cornolo J, Fouillot J-P, Schmitt L, et al. Interactions between exposure to hypoxia and the training-induced autonomic adaptations in a "live high–train low" session. Eur J Appl Physiol. 2006;96:389–96.
67. Plews DJ, Laursen PB, Kilding AE, et al. Heart-rate variability and training-intensity distribution in elite rowers. Int J Sports Physiol Perform. 2014;9:1026–32.
68. Binnie MJ, Dawson B, Arnot MA, et al. Effect of sand versus grass training surfaces during an 8-week pre-season conditioning programme in team sport athletes. J Sports Sci. 2014;32:1001–12.
69. Boushel R, Ara I, Gnaiger E, et al. Low-intensity training increases peak arm VO_2 by enhancing both convective and diffusive O_2 delivery. Acta Physiol. 2014;211:122–34.
70. Buchheit M, Rabbani A. The 30–15 Intermittent Fitness Test versus the Yo–Yo Intermittent Recovery Test Level 1: relationship and sensitivity to training. Int J Sports Physiol Perform. 2014;9:522–4.
71. Buchheit M, Morgan W, Wallace J, et al. Physiological, psychometric, and performance effects of the Christmas break in Australian football. Int J Sports Physiol Perform. 2015;10:120–3.
72. Castagna C, Impellizzeri FM, Chaouachi A, et al. Preseason variations in aerobic fitness and performance in elite-standard soccer players: a team study. J Strength Cond Res. 2013;27:2959–65.
73. Diaz Gomez MM, Bocanegra Jaramillo OL, Teixeira RR, et al. Salivary surrogates of plasma nitrite and catecholamines during a 21-week training season in swimmers. PLoS One. 2013;8:e64043.
74. Faude O, Steffen A, Kellmann M, et al. The effect of short-term interval training during the competitive season on physical fitness and signs of fatigue: a crossover trial in high-level youth football players. Int J Sports Physiol Perform. 2014;9:936–44.
75. Gunnarsson TP, Christensen PM, Thomassen M, et al. Effect of intensified training on muscle ion kinetics, fatigue development, and repeated short-term performance in endurance-trained cyclists. Am J Physiol Regul Integr Comp Physiol. 2013;305:R811–21.
76. Kennedy MD, Tamminen KA, Holt NL. Factors that influence fatigue status in Canadian university swimmers. J Sports Sci. 2013;31:554–64.
77. Manzi V, Bovenzi A, Franco Impellizzeri M, et al. Individual training-load and aerobic-fitness variables in premiership soccer players during the precompetitive season. J Strength Cond Res. 2013;27:631–6.
78. Racinais S, Buchheit M, Bilsborough J, et al. Physiological and performance responses to a training camp in the heat in professional Australian football players. Int J Sports Physiol Perform. 2014;9:598–603.
79. Laursen PB, Blanchard MA, Jenkins DG. Acute high-intensity interval training improves Tvent and peak power output in highly trained males. Can J Appl Physiol. 2002;27:336–48.
80. Achten J, Halson SL, Moseley L, et al. Higher dietary carbohydrate content during intensified running training results in

better maintenance of performance and mood state. J Appl Physiol. 2004;96:1331–40.
81. Neal CM, Hunter AM, Galloway SDR. A 6-month analysis of training-intensity distribution and physiological adaptation in Ironman triathletes. J Sports Sci. 2011;29:1515–23.
82. Plews D, Laursen P, Kilding A, et al. Heart rate variability in elite triathletes, is variation in variability the key to effective training? A case comparison. J Appl Physiol. 2012;112:3729–41.
83. Dupuy O, Lussier M, Fraser S, et al. Effect of overreaching on cognitive performance and related cardiac autonomic control. Scand J Med Sci Sports. 2014;24:234–42.
84. Lamberts RP, Swart J, Capostagno B, et al. Heart rate recovery as a guide to monitor fatigue and predict changes in performance parameters. Scand J Med Sci Sports. 2010;20:449–57.
85. Baumert M, Brechtel L, Lock J, et al. Changes in heart rate variability of athletes during a training camp. Biomed Tech. 2006;51:201–4.
86. Plews DJ, Laursen PB, Kilding AE, et al. Evaluating training adaptation with heart-rate measures: a methodological comparison. Int J Sports Physiol Perform. 2013;8:688.
87. Hug B, Heyer L, Naef N, et al. Tapering for marathon and cardiac autonomic function. Int J Sports Med. 2014;35:676–83.
88. Al Haddad H, Laursen P, Chollet D, et al. Reliability of resting and postexercise heart rate measures. Int J Sports Med. 2011;32:598–605.
89. Brown TE, Beightol LA, Koh J, et al. Important influence of respiration on human *R–R* interval power spectra is largely ignored. J Appl Physiol. 1993;75:2310–7.
90. Kenney WL. Physiology of sport and exercise. In: Wilmore JH, editor. 5th ed. Champaign: Human Kinetics; 2012.
91. Fagard R, Aubert A, Lysens R, et al. Noninvasive assessment of seasonal variations in cardiac structure and function in cyclists. Circulation. 1983;67:896.
92. Dishman RK, Nakamura Y, Garcia ME, et al. Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. Int J Psychophysiol. 2000;37:121–33.
93. Kristal-Boneh E, Froom P, Harari G, et al. Summer–winter differences in 24 h variability of heart rate. J Cardiovasc Risk. 2000;7:141–6.
94. Mourot L, Bouhaddi M, Tordi N, et al. Short and long-term effects of a single bout of exercise on heart rate variability: comparison between constant and interval training exercises. Eur J Appl Physiol. 2004;92:508–17.
95. Achten J, Jeukendrup AE. Heart rate monitoring: applications and limitations. Sports Med. 2003;33:517–38.
96. Lamberts RP, Lambert MI. Day-to-day variation in heart rate at different levels of submaximal exertion: implications for monitoring training. J Strength Cond Res. 2009;23:1005–10.
97. Du N, Bai S, Oguri K, et al. Heart rate recovery after exercise and neural regulation Of heart rate variability in 30–40 year old female marathon runners. J Sport Sci Med. 2005;4:9.
98. Danieli A, Lusa L, Potocnik N, et al. Resting heart rate variability and heart rate recovery after submaximal exercise. Clin Auton Res. 2014;24:53–61.
99. Pierpont GL, Voth EJ. Assessing autonomic function by analysis of heart rate recovery from exercise in healthy subjects. Am J Cardiol. 2004;94:64–8.
100. Aubry A, Hausswirth C, Louis J, et al. The development of functional overreaching is associated with a faster heart rate recovery in endurance athletes. PLoS One. 2015;10(10):e0139754. doi:10.1371/journal.pone.0139754.
101. Rowell LB, O'Leary DS. Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes. J Appl Physiol. 1990;69:407–18.
102. Le Meur Y, Louis J, Aubry A, et al. Maximal exercise limitation in functionally overreached triathletes: role of cardiac adrenergic stimulation. J Appl Physiol. 2014;117(3):214–22.