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REVIEW ARTICLE

# Cardiac Parasympathetic Reactivation Following Exercise: Implications for Training Prescription

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**Abstract** The objective of exercise training is to initiate desirable physiological adaptations that ultimately enhance physical work capacity. Optimal training prescription requires an individualized approach, with an appropriate balance of training stimulus and recovery and optimal periodization. Recovery from exercise involves integrated physiological responses. The cardiovascular system plays a fundamental role in facilitating many of these responses, including thermoregulation and delivery/removal of nutrients and waste products. As a marker of cardiovascular recovery, cardiac parasympathetic reactivation following a training session is highly individualized. It appears to parallel the acute/intermediate recovery of the thermoregulatory and vascular systems, as described by the supercompensation theory. The physiological mechanisms underlying cardiac parasympathetic reactivation are not completely understood. However, changes in cardiac autonomic activity may provide a proxy measure of the changes in autonomic input into organs and (by default) the blood flow requirements to restore homeostasis. Metaboreflex stimulation (e.g. muscle and blood acidosis) is likely a key determinant of parasympathetic reactivation in the short term (0–90 min post-exercise), whereas baroreflex stimulation (e.g. exercise-induced changes in plasma volume) probably mediates parasympathetic reactivation in the intermediate term (1–48 h post-exercise). Cardiac parasympathetic reactivation does not appear to coincide with the recovery of all physiological systems (e.g. energy stores or the neuromuscular system). However, this may reflect the limited data currently available on parasympathetic reactivation following strength/resistance-based exercise of variable intensity. In this review, we quantitatively analyse post-exercise cardiac parasympathetic reactivation in athletes and healthy individuals following aerobic exercise, with respect to exercise intensity and duration, and fitness/training status. Our results demonstrate that the time required for complete cardiac autonomic recovery after a single aerobic-based training session is up to 24 h following low-intensity exercise, 24–48 h following threshold-intensity exercise and at least 48 h following high-intensity exercise. Based on limited data, exercise duration is unlikely to be the greatest determinant of cardiac parasympathetic reactivation. Cardiac autonomic recovery occurs more rapidly in individuals with greater aerobic fitness. Our data lend support to the concept that in conjunction with daily training logs, data on cardiac parasympathetic activity are useful for individualizing training programmes. In the final sections of this review, we provide recommendations for structuring training microcycles with reference to cardiac parasympathetic recovery kinetics. Ultimately, coaches should

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structure training programmes tailored to the unique recovery kinetics of each individual.

## 1 Introduction

Coaches and athletes face the perpetual challenge of balancing training stimulus with recovery. The objective of exercise training is to initiate desirable metabolic, cardiovascular and neuromuscular adaptations to enhance physical work capacity [1]. With the exception of specific training blocks—where the objective is to induce an overreaching response [2]—the most appropriate time to schedule (subsequent) intensive training sessions may be during periods of increased capacity to deviate from resting homeostasis (i.e. at the supercompensation peak) [3]. Conversely, if athletes train without appropriate recovery, this may hinder their training adaptations and performance gains [4]. Therefore, to prescribe/schedule effective training sessions, it is important to monitor recovery [5].

Recovery is a process of restoration to a former or better condition. In the context of exercise physiology, recovery involves the integrated response of many systems that return the body to homeostasis or a higher level of homeostasis through acute adaptations that temporally allow for increased exercise capacity to deviate from resting homeostasis (i.e. supercompensation). During recovery, metabolites accumulated in skeletal muscle during exercise, such as $H^+$ and adenosine triphosphate (ATP) degradation products [6], are removed or recycled. The body temperature [7] and fluid balance [8] return to resting levels, and neuroendocrine-immune responses are activated to restore homeostasis [9]. The autonomic nervous system regulates these physiological processes to varying degrees by altering the secretory activity of glands and the tone of smooth muscle and cardiac muscle. Information from internal organs also influences some of these physiological processes [10, 11]. The cardiovascular system plays a fundamental role during the recovery process. It facilitates many of these physiological processes, including thermoregulation and delivery/removal of nutrients and waste products [12]. Consequently, while not providing a direct measure of autonomic input into all organs, changes in cardiac autonomic activity likely reflect exercise-induced changes in cardiac performance [13] and haemodynamics [14, 15]. These alterations in cardiovascular regulation represent a deviation from homeostasis [15, 16], reflect the redistribution of cardiac output between various body compartments (i.e. skeletal muscle, the brain, internal organs) [17] and trigger autonomic mechanisms that affect the heart and blood vessels to restore homeostasis [17, 18]. Therefore, the time course of cardiac autonomic recovery reflects restoration of cardiovascular homeostasis, which is an important component of overall recovery.

After intense exercise, specific changes in cardiac parasympathetic activity can be assessed quickly and non-invasively by measuring heart rate variability (HRV) [19]. These changes appear to track the time course of restoration of homeostasis [16]. Immediately following exercise, cardiac parasympathetic activity can also be assessed by quantification of heart rate recovery (HRR) [20, 21]. The immediate 60–300 s of heart rate data following exercise are used to assess HRR, depending on the selected method [20]. Conversely, recovery of HRV requires assessment of 180–300 s of heart rate data, which can be collected at various timepoints during the post-exercise recovery period (i.e. within the first 15 min to 72 h following exercise). The distinct determinants of HRR and HRV require further research [20]. However, HRR is generally considered a marker of parasympathetic tone [20, 22] and is reflective of sympathetic withdrawal, which affects the heart rate [23]. By contrast, HRV provides information on modulation of the heart rate [22]. Despite the differences in the underlying determinants of HRR and HRV, HRR cannot be used to track the time course of cardiac parasympathetic reactivation unless exercise is repeated periodically during recovery. Therefore, because the aim of this review was to evaluate cardiac parasympathetic reactivation following a single exercise, our analysis necessitated the use of HRV. The hemodynamic state after exercise represents an interaction between the effects of exercise and homeostatic mechanisms (including feedback from mechanoreceptors in the muscle [24] and baroreceptors in the carotid body [25]). These factors influence the heart and blood vessels to maintain arterial pressure and control the heart rate [17, 26]. It is beyond the scope of this review to detail the complexities of cardiovascular control, so we refer readers to other reviews on that topic [17, 25, 27, 28]. However, for the purpose of this review, both mechano- and baroreceptor feedback are integrated with commands from the brain, contributing to changes in the cardiac autonomic state assessed by HRV following exercise.

Cardiac parasympathetic activity is initially decreased within a few minutes to hours after exercise [29–34]. During the acute recovery period (0–90 min post-exercise), a drop in blood pressure reduces afferent input from baroreceptors. This results in a decrease in cardiac parasympathetic activity and an increase in sympathetic nerve activity [25]. Concurrently, accumulated metabolites (e.g. $H^+$, inorganic phosphate) within the active skeletal muscle stimulate group III and IV afferent nerves [24] and chemoreceptors in the carotid body [35]. This stimulation then evokes a reflex increase in muscle sympathetic nerve activity, known as the metaboreflex. Exercise-induced release of epinephrine into the systemic circulation may

induce further sympathetic excitation, which may in turn suppress cardiac parasympathetic reactivation [36]. As a result, the time required for HRV to return to resting levels is prolonged [37, 38]. Post-exercise parasympathetic activity (as inferred from HRR) correlates significantly with post-exercise plasma epinephrine levels [39], blood lactate concentrations [40], blood acidosis [31, 41] and arterial oxygenation [42]. Somewhat surprisingly, post-exercise sympathetic activity (as assessed by blood pressure variability) does not correlate with post-exercise HRR [40]. A summary of data from three studies [33, 43, 44] demonstrated an almost perfect relationship ($r = -0.93$, 90 % interval [CI] −0.98 to −0.78) between differences in post-exercise parasympathetic activity and differences in post-exercise blood lactate concentrations (see Fig. S1 in the Electronic Supplementary Material) [45]. Furthermore, compared with peripheral chemoreceptors (which are activated by a fall in $O_2$), central chemoreceptors (which respond primarily to the fall in blood pH and hypercapnia), are likely a stronger determinant of post-exercise parasympathetic reactivation [46]. Therefore, acute post-exercise parasympathetic reactivation probably largely depends on accumulation of stress metabolites in the blood and skeletal muscle [31, 40, 41, 45, 46]. However, it is important to note that the apparent dose–response relationship of blood acidosis [31, 41] might be limited over time. Once the metaboreflex is activated at high levels and cardiac parasympathetic activity has been almost abolished, further increases in stress metabolite concentrations might not further affect post-exercise parasympathetic reactivation. This 'ceiling effect' represents a limitation of HRV as a proxy measure of overall recovery (particularly following high-intensity exercise) because the longer the exercise duration, the greater the depletion of muscle glycogen stores and neuromuscular fatigue.

With adequate recovery, cardiac parasympathetic activity can return to pre-exercise levels within 24–72 h [14, 33, 47–50] or can 'rebound' above these levels [16, 51]. Intermediate recovery (1–48 h post-exercise) of cardiac parasympathetic activity is most likely dependent on exercise-induced changes in plasma volume, and resultant arterial-baroreflex stimulation [51, 52]. An increase in blood volume (i.e. hypervolaemia) likely elicits a baroreflex-mediated increase in cardiac parasympathetic activity [53–55], which reaches its maximal level during moderate hypervolaemia [55] (see Fig. S1 in the Electronic Supplementary Material). Conversely, a mild to moderate reduction in central blood volume (i.e. hypovolaemia) deactivates cardiopulmonary baroreflex receptors. This response induces sympathetically mediated peripheral vasoconstriction to maintain sufficient arterial blood pressure during reduced cardiac output [55]. Buchheit et al. [51] observed a very large correlation between changes in cardiac parasympathetic activity and plasma volume ($r = 0.85$, 90 % CI 0.53–0.96) 48 h following supramaximal exercise. Such a correlation does not imply causality. Nevertheless, these data suggest that exercise-induced changes in plasma volume, coupled with modified baroreceptor activation, may in part regulate cardiac parasympathetic reactivation during the days following exercise. Restoration of plasma volume to pre-exercise levels likely signifies the return of the cardiovascular system to homeostasis, but not necessarily restoration of other systems (i.e. neuromuscular [56] or muscle glycogen resynthesis). However, plasma volume can sometimes increase above pre-exercise levels in the hours or days after exercise. In this situation, cardiac parasympathetic activity also 'rebounds' above pre-exercise levels [51]. Increased plasma volume augments stroke volume, diastolic function and maximal oxygen uptake ($\dot{V}O_{2max}$) [57, 58]. Attainment and maintenance of elevated diastolic filling is required for improvement of maximal cardiac function [59–61]. Therefore, training during such periods of plasma volume expansion and rebound in cardiac parasympathetic activity is likely optimal for attaining cardiovascular adaptations and performance gains [3]. In support of this idea, recent investigations described later in this review (see Sect. 5), have demonstrated that high-intensity training during periods of elevated parasympathetic activity leads to positive cardiorespiratory adaptations [62, 63].

In addition to increased plasma volume [51], parasympathetic reactivation following aerobic-based exercise appears to parallel the acute/intermediate recovery response, as described by the supercompensation theory [3]. Changes in cardiac autonomic system function following exercise arguably provide a global marker of the body's homeostatic state, indirectly reflecting an athlete's overall recovery status and readiness to train/perform [64]. The time course and amplitude of the post-exercise cardiac parasympathetic response is primarily dependent on the training load, as determined by exercise intensity [14, 29, 31, 33, 34, 47] and ratings of perceived exertion (RPE) [65]. Factors including exercise duration [29, 34], age [66], gender [63], baseline physical fitness [67], training status [22], psychology [68, 69], central fatigue [70] and post-exercise fluid intake [71] also influence cardiac parasympathetic activity. Thus, changes in cardiac parasympathetic activity are useful for monitoring aspects of recovery that are dependent on cardiovascular function. By contrast, changes in cardiac parasympathetic activity are less useful for monitoring other aspects of recovery such as restoration of muscle and liver glycogen, or repair of damaged muscle tissue [56, 72]. Considering the factors that influence assessment of cardiac parasympathetic activity after exercise (likely contributing

to its moderate level of reliability [73]), evaluation of meaningful changes in the cardiac parasympathetic response requires evaluation of the typical measurement error and the smallest worthwhile change [74].

In summary, we contend that appropriate and optimal training programming and prescription require integration and interpretation of all variables related to the athlete's current physiological and psychological state, with consideration of their training background. However, it is currently impractical (and in some instances impossible) to measure the state of all physiological systems during recovery from exercise. The simplicity of HRV measurement, and the information that it provides on an athlete's (at least cardiovascular) recovery status [64], suggest that daily HRV assessment may assist in setting individualized training prescription/programmes [62, 63].

The purpose of this review was to highlight the value of assessing cardiac parasympathetic reactivation in the hours following a single bout of exercise to derive individual recommendations for training (at least for cardiovascular outcomes). Because of differences in experimental protocols throughout the literature, we adopted a novel method to characterize post-exercise parasympathetic reactivation. This approach involved interpolating available data from published research and adjusting these data for the effects of exercise intensity and fitness level. The quantitative analysis part of this review focused specifically on cardiac parasympathetic reactivation following 'typical' training sessions for athletes. The cardiac parasympathetic response to 'extreme' exercise (e.g. a 75-km cross-country skiing race [16]) and resistance-based exercise (e.g. see references [72, 75–77]) was also reviewed. Finally, while individualized prescription is obviously the ultimate goal for every coach, we have provided, as a starting point, practical recommendations for weekly training programming, based on group-averaged parasympathetic reactivation. Owing to insufficient data, we have not provided any recommendations for programming strength/resistance training.

## 2 Mediators of Cardiac Parasympathetic Reactivation

To examine and quantify the time course of cardiac parasympathetic recovery following a single exercise bout (representative of a typical training session), we adopted the following approach.

### 2.1 Methodology

#### *2.1.1 Study Selection*

We used PubMed and Google Scholar to search for investigations of recovery from exercise and HRV published in the English language, up to and including July 2012. We also examined reference lists in review articles and original research articles. The focus of this review was cardiac parasympathetic recovery following a single bout of exercise. Studies were included in the descriptive sections of the review (see Sects. 1, 3, 4) if they provided physiological data relating to cardiac parasympathetic reactivation following a single bout of exercise. More specific criteria were adopted for the quantitative analysis (see Sect. 2). Studies with HRV assessed from the end of exercise up until 72 h post-exercise were included. Studies were excluded (i) if no baseline/pre-exercise HRV data were provided; and (ii) if data for fewer than three post-exercise timepoints were provided. A summary of the eight qualifying studies for quantitative analysis is provided in Table 1.

#### *2.1.2 Data Analysis*

Cardiac parasympathetic activity is commonly inferred from vagal-related indices of HRV calculated in two ways: (ii) by time domains (e.g. the natural logarithm of the square root of the mean squared differences of successive R–R intervals [Ln rMSSD]); and (ii) by frequency domains (e.g. the natural logarithm of the power spectral density in the high-frequency (>0.15 Hz) range [Ln HF]) [19, 78]. If multiple indices were provided, we chose the temporal index (Ln rMSSD) because it is more reliable than spectral indices [73] (i.e. Ln HF), particularly under ambulatory conditions [79]. Because of the effect of posture [44], we extracted HRV data (mean and standard deviation) determined during short-duration (1–15 min) supine or seated posture HRV assessments from each study. When specific values were not provided, figures were visually inspected for relevant data. From the included studies, we examined the time course of changes in HRV during acute recovery (up to 90 min post-exercise) and intermediate recovery (up to 48 h post-exercise). For each study, the HRV at each timepoint provided during the 48 h post-exercise recovery period was converted into a percentage of the baseline level and tabulated in spreadsheet software (Excel® 2010; Microsoft Corporation). Because of differences in study design, the timing of post-exercise HRV assessment for each study included in the quantitative analysis was inconsistent. A total of 18 different timepoints were included for acute recovery, whereas 28 different timepoints were included for intermediate recovery. The level of HRV for each of the 18 or 28 timepoints was then interpolated from the data available in each individual study. Interpolation involved plotting the time course of HRV recovery for each individual study, and using the 'trendline' function. The simplest polynomial based trendline was selected on the basis of the coefficient of

**Table 1** Characteristics of the study groups qualifying for inclusion in the quantitative analysis

| Study | Sample size; fitness level | $\dot{V}O_{2max}$ (mL/kg/min) | Exercise | Exercise duration (min) | Exercise intensity classification | HRV recording method | HRV index | HRV measurement timing |
|---|---|---|---|---|---|---|---|---|
| Furlan et al. [47] | 10; inactive | NA | Treadmill test to exhaustion + 4–6 runs = 30 min | 30 | HI | 15 min supine (respiration rate not specified) | Ln HF | Pre-exercise and 1, 24 and 48 h post-exercise |
| Terziotti et al. [121] | 12; inactive | NA | 20 min @ 50 % of AT (AT = 248 W) | 20 | LI | 10 min seated (controlled respiration, rate not provided) | Ln HF | Pre-exercise and 15, 60 and 180 min post-exercise |
| | | | 20 min @ 80 % of AT (AT = 248 W) | 20 | Th | | | |
| Mourot et al. [49] | 10; moderately trained | 61.3 | CST (VT1 duration so total energy = HIT) | 50 | LI | 5 min supine (spontaneous respiration, always >0.16 Hz) | Ln HF | Pre-exercise and 1, 24 and 48 h post-exercise |
| | | | HIT (4 min @ VT1, 1 min @ >VT2) | 45 | Th | | | |
| Parekh and Lee [122] | 13; inactive | 58.3 | 300 kcal @ 50 % of $\dot{V}O_2R$ | 25 | LI | 5 min supine (spontaneous respiration) | Ln HF | Pre-exercise and 10, 15, 20, 25 and 30 min post-exercise |
| | | | 300 kcal @ 80 % of $\dot{V}O_2R$ | 20 | Th | | | |
| Niewiadomski et al. [123] | 8; inactive | 53 | 2 × 30-s Windgate tests[a] | 15 | HI | 5 min supine (spontaneous respiration) | Ln rMSSD | 48 and 24 h pre-exercise and 1, 24 and 48 h post-exercise |
| | | | 30 min cycling @ 85 % of $HR_{max}$ | 30 | Th | | | |
| Seiler et al. [29] | 9; highly trained | 72 | Above VT2 (6 × 3 min @ 96 % of $\dot{V}O_{2max}$) | 60 | HI | 5 min supine (spontaneous respiration) | Ln rMSSD | Pre-exercise and 0, 10, 25, 55, 85, 115, 175 and 235 min post-exercise |
| | | | Below VT1 60 min (61 % of $\dot{V}O_{2max}$) | 60 | LI | | | |
| | | | Below VT1 120 min | 120 | LI | | | |
| | 8; moderately trained | 60 | Threshold (84 % of $\dot{V}O_{2max}$) | 60 | Th | | | |
| | | | Above VT2 (6 × 3 min @ 95 % of $\dot{V}O_{2max}$) | 60 | HI | | | |
| Kaikkonen et al. [124] | 8; moderately trained | 60 | 7 × 3 min @ 85 % of $v\dot{V}O_{2max}$ (2 min reco) | 45 | Th | 1−2 min seated (spontaneous respiration) | Ln HF | Pre-exercise and 0, 8, 18 and 28 min post-exercise |
| | | | 7 × 3 min @ 93 % of $v\dot{V}O_{2max}$ (2 min reco) | 45 | HI | | | |
| | | | 21 min @ 80 % of $v\dot{V}O_{2max}$ | 31 | Th | | | |
| | | | 21 min @ 85 % of $v\dot{V}O_{2max}$ | 31 | Th | | | |

**Table 1** continued

| Study | Sample size; fitness level | $\dot{V}O_{2max}$ (mL/kg/min) | Exercise | Exercise duration (min) | Exercise intensity classification | HRV recording method | HRV index | HRV measurement timing |
|---|---|---|---|---|---|---|---|---|
| Stanley et al. [30] | 18; moderately trained | 63.9 | 60 min including 8 × 4 min @ 80 % of PPO | 60 | Th | 10 min supine (spontaneous respiration) | Ln rMSSD | Pre-exercise and 10, 45, 70, 100, 130 and 190 min post-exercise |

*AT* anaerobic threshold, *CST* constant-intensity training, *HI* high-intensity training, *HIT* high-intensity interval training, $HR_{max}$ maximal heart rate, *HRV* heart rate variability, *LI* low-intensity training, *Ln HF* natural logarithm of the high-frequency spectral power, *Ln rMSSD* natural logarithm of the square root of the mean squared differences of successive R–R intervals, *NA* not available, *PPO* peak power output, *reco* recovery period, *Th* threshold intensity training, $\dot{V}O_{2max}$ maximal oxygen uptake, $\dot{V}O_2R$ oxygen uptake reserve, *VT1* first ventilatory threshold, *VT2* second ventilatory threshold, $v\dot{V}O_{2max}$ velocity corresponding to $\dot{V}O_{2max}$

[a] We included the Windgate exercise component of the study because although a Windgate test differs from the predominantly aerobic high-intensity exercises in the other studies, the overall testing protocol (i.e. two Windgate tests separated by 3 min of active recovery) is similar to sprint-interval training sessions [125], which are an important training component for many sports

determination ($r^2$) and visual inspection. Interpolation yielded $r^2$ values ranging from 0.88 to 1. If a single polynomial function did not fit, the data were split into two sections and the data were interpolated using a combination of functions. For example, a simple polynomial function typically fitted the data up to 60–100 min, after which a linear function was sometimes more appropriate. The mean time course of recovery of HRV after exercise was obtained by averaging the interpolated data for each individual study at each timepoint for HRV assessment after exercise. No weighting factor was assigned to each individual study prior to calculating the mean time course of recovery of HRV after exercise. For clarity, we have only presented the interpolated data at 1, 24 and 48 h post-exercise for the intermediate recovery response.

We examined the effect of exercise intensity on parasympathetic recovery by categorizing the exercise performed in each study, using a three-zone model [80]. We selected the three-zone model because the first ventilatory threshold appears to define a 'binary' threshold for parasympathetic recovery. Specifically, parasympathetic recovery is prolonged following exercise at intensities above the first ventilatory threshold, compared with exercise performed below it. There is no further delay in parasympathetic recovery following exercise at intensities above the first ventilatory threshold [29]. We examined the effect of athlete fitness/training status on the time course of parasympathetic recovery by categorizing the subjects in each study as 'inactive', 'moderately trained' or 'highly trained', on the basis of the information provided (e.g. maximal oxygen uptake [$\dot{V}O_{2max}$] or training status description). The classification of fitness/training status for each study is displayed in Table 1. We then performed an analysis of covariance (ANCOVA) [Minitab 14.0; Minitab Ltd.] to examine the effect of exercise intensity when adjusted for athlete training status/fitness, and the effect of athlete training status/fitness when adjusted for exercise intensity. The adjusted values (i.e. least squares means) were then plotted against recovery time, and standardized differences were calculated to compare within-condition changes from pre-exercise. To facilitate comparison between studies, whenever possible, we calculated standardized differences [or effect sizes [81]) and interpreted these differences using Hopkins' categorization criteria [82].

The training sessions reported in the studies that qualified for inclusion in our quantitative analysis were of similar duration (narrow range). This restricted our ability to categorize data on the basis of exercise duration. As an alternative approach, we calculated correlation coefficients for HRV at 90-min post-exercise versus the logarithm of exercise duration for all data combined. Using partial correlations, we then adjusted the correlations for both exercise intensity and athlete fitness/training status. When calculating these correlations, we log transformed the exercise duration data (i) to reduce bias arising from non-uniformity error; and (ii) to ensure that the relationship between exercise intensity and time was linear [83]. Because there were insufficient data, we could not calculate correlation coefficients for 48 h post exercise.

The magnitude of within- and between-trial differences between variables was assessed by calculating the effect size, using the pooled standard deviation [81]. The threshold values for effect size statistics were $\leq$0.2, trivial; >0.2, small; >0.6, moderate; >1.2, large; >2.0, very large; $\geq$4.0, extremely large [82]. To interpret the magnitude of correlation ($r$) between exercise duration and post-exercise HRV recovery, we adopted the following criteria: $\leq$0.1, trivial; >0.1 to 0.3, small; >0.3 to 0.5, moderate; >0.5 to 0.7, large; >0.7 to 0.9, very large; >0.9 to 1, almost perfect. If the 90 % CI overlapped small positive and negative values, the magnitude of the correlation was deemed

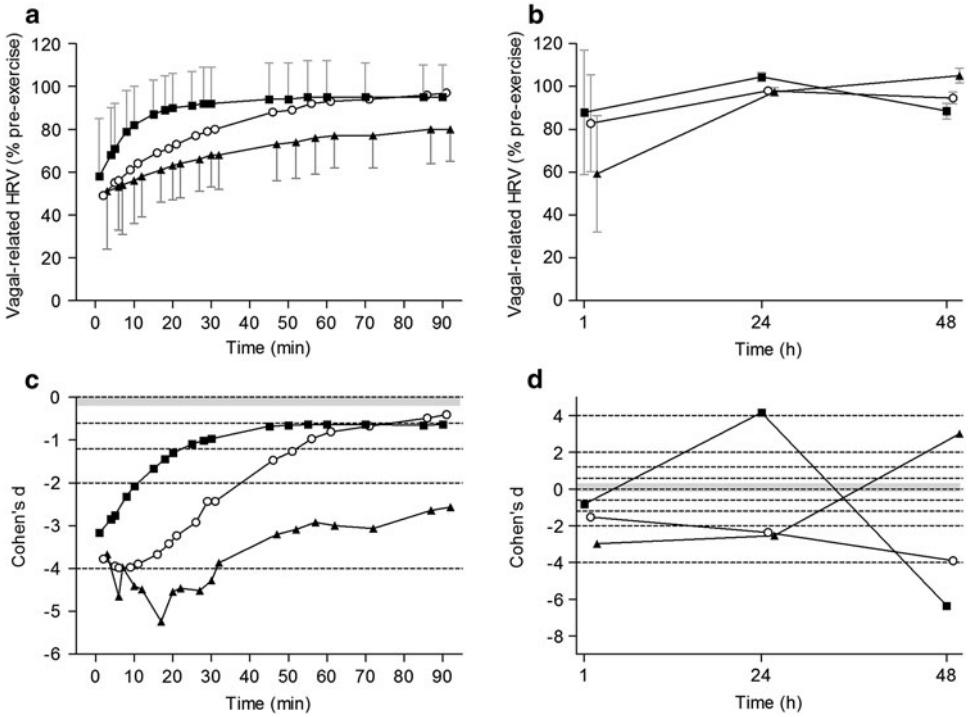


**Fig. 1** Influences of low-intensity exercise (blood lactate <2 mmol/L, maximal heart rate [$HR_{max}$] <82 %, maximal oxygen uptake [$\dot{V}O_{2max}$] <70 %), threshold-intensity exercise (blood lactate <2 to 4 mmol/L, $HR_{max}$ <82 to 89 %, $\dot{V}O_{2max}$ <70 to 82 %) and high-intensity exercise (blood lactate >4 mmol/L, $HR_{max}$ >90 %, $\dot{V}O_{2max}$ >82 %), adjusted for athlete fitness/training status: **a** and **b** influences on mean (±standard deviation) cardiac parasympathetic reactivation, and **c** and **d** influences on standardized differences, **a** and **c** during the acute recovery period (up to 90 min), and **b** and **d** during the intermediate recovery period (1–48 h). The interpolation for the acute recovery period was based on 19 different exercise conditions from eight studies, and the interpolation for the intermediate recovery period was based on seven different exercise conditions from four studies (Table 1). The *dashed lines* represent threshold values for effect size statistics, with the *shaded area* representing a trivial difference. *HRV* heart rate variability

'unclear'; otherwise, the magnitude of the correlation was deemed to be the observed magnitude [82].

## 2.2 Results

### *2.2.1 Effect of Exercise Intensity*

The effect of exercise intensity on acute recovery of cardiac parasympathetic activity (90 min post-exercise) is shown in Fig. 1a. Cardiac parasympathetic activity does not appear to return to pre-exercise values within this timeframe. Exercise at all intensities elicits a very large to extremely large reduction in cardiac parasympathetic activity in the initial 10 min following exercise (Fig. 1c). Recovery of cardiac parasympathetic activity is most rapid following low-intensity exercise (~116 %/h during the first 15 min). After this type of exercise, the post-exercise suppression of cardiac parasympathetic activity is reduced from very large to large ~15 min after exercise, and from large to moderate ~25 min after exercise (Fig. 1c). Recovery of cardiac parasympathetic activity is slower following threshold-intensity exercise (~80 %/h during the first 15 min). After this type of exercise, the post-exercise suppression of cardiac parasympathetic activity is reduced from very large to large ~40 min after exercise, from large to moderate ~50 min after exercise, and from moderate to small ~80 min after exercise (Fig. 1c). Recovery of cardiac parasympathetic activity occurs most slowly after high-intensity exercise (~40 %/h during the first 15 min). After this type of exercise, the post-exercise suppression of cardiac parasympathetic activity is extremely large until ~30 min after exercise, and remains very large until at least 90 min after exercise (Fig. 1c).

The effect of exercise intensity on intermediate recovery of cardiac parasympathetic activity (48 h post-exercise) is shown in Fig. 1b. The suppression of cardiac parasympathetic activity is moderate 1 h after low-intensity exercise, large 1 h after threshold-intensity exercise, and very large 1 h after high-intensity exercise (Fig. 1d). Cardiac parasympathetic recovery to pre-exercise levels occurs within 24 h following low-intensity exercise. Of note, the extremely large difference in cardiac parasympathetic activity at 24 h post-exercise is indicative of a rebound in HRV above pre-exercise levels (Fig. 1d). However, 48 h after low-

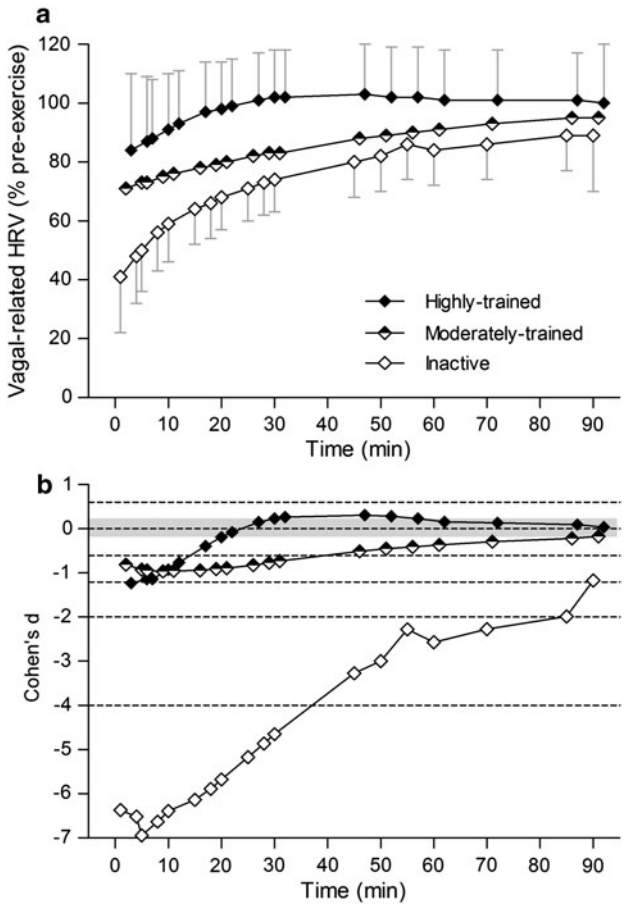


**Fig. 2** Influences of athlete fitness/training status, adjusted for exercise intensity, during the acute recovery period (up to 90 min): **a** influences on mean cardiac parasympathetic activity (±standard deviation), and **b** influences on standardized differences. The interpolation was based on 19 different exercise conditions from eight studies (Table 1). The *dashed lines* represent threshold values for effect size statistics, with the *shaded area* representing a trivial difference. *HRV* heart rate variability

intensity exercise, cardiac parasympathetic activity has returned to below pre-exercise levels (an extremely large effect; Fig. 1c). By contrast, cardiac parasympathetic activity follows a different time course during intermediate recovery from threshold- and high-intensity exercise. After threshold-intensity exercise, the post-exercise suppression of cardiac parasympathetic activity remains very large, even at 48 h (Fig. 1d). After high-intensity exercise, the post-exercise suppression of cardiac parasympathetic activity is stable at 24 h but then rebounds above pre-exercise levels within 48 h (a very large effect; Fig. 1d).

### 2.2.2 *Effect of Fitness/Training Status*

The effect of athlete fitness/training status adjusted for exercise intensity on cardiac parasympathetic recovery is illustrated in Fig. 2. Cardiac parasympathetic activity is reduced immediately following exercise in individuals at all fitness levels (Fig. 2a). However, post-exercise suppression of cardiac parasympathetic activity is only moderate for elite and endurance-trained athletes, whereas it is extremely large for inactive (untrained) individuals (Fig. 2b). Recovery to pre-exercise levels is most rapid in highly trained athletes: post-exercise suppression of cardiac parasympathetic activity is reduced to small or trivial after ~15 min (Fig. 2b). Moderately trained athletes require a slightly longer time for recovery: post-exercise suppression of cardiac parasympathetic activity is reduced to small or trivial after ~40 min (Fig. 2b). In inactive individuals, post-exercise suppression of cardiac parasympathetic activity remains large even after 90 min of recovery (Fig. 2b).

### 2.2.3 *Effect of Exercise Duration*

When considering data from all studies pooled together (adjusted for both exercise intensity and athlete fitness/training status), there was no clear relationship ($r = -0.35$, 90 % confidence interval −0.67 to 0.07, $n = 17$) between exercise duration and cardiac parasympathetic recovery at 90 min post-exercise.

## 2.3 Summary

Our quantitative analysis confirms that exercise intensity is the key determinant of cardiac parasympathetic reactivation (Fig. 2). Specifically, the first ventilatory threshold appears to define a 'binary' threshold for cardiac parasympathetic reactivation following exercise [29]. We acknowledge that wide variation exists in the same individual performing exercise intensity corresponding to the first ventilatory threshold, because of (i) the method used to identify it, and/or (ii) the protocol used [84]. Consequently, it is important to consider potential error arising from incorrect assessment/reporting of the actual intensity performed in the studies that were included in our current analysis. The fitness/training status of the individual also influences the magnitude of perturbation from homeostasis, and the rate of cardiac autonomic recovery following a single exercise session [16, 29]. Our results suggest that fitter or more highly trained individuals are more resilient to exercise stress, as demonstrated by less perturbation of cardiac parasympathetic activity from pre-exercise activity. This resilience could reflect more rapid autonomic restoration to baseline levels compared with less fit/trained individuals (Fig. 2). Alternatively, it could result from less blood metabolite accumulation at the same relative exercise intensity [29] and therefore less stimulation of metaboreflex pathways. This analysis supports the notion that

individuals with a greater $\dot{V}O_{2max}$ show faster cardiorespiratory recovery after exercise [16, 85, 86]. However, it remains unclear whether this difference is due to more efficient clearance of metabolites in fitter/more well trained individuals (in the absence of any difference in metaboreflex stimulation) [87].

The effect of exercise duration on the cardiac parasympathetic reactivation within the first 90 min post-exercise was unclear. It is important to note that the data available for inclusion limited the present analysis. Typically, the high-intensity training sessions available for inclusion in the analysis were of shorter duration compared with low-intensity training sessions. Nevertheless, our analysis confirms the findings from two previous studies [29, 65]. In the first study [65], exercise of similar duration but of different intensity (and vice versa) was compared. Cardiac parasympathetic reactivation (at least up until 15 min post-exercise) was influenced more by exercise intensity than by duration [65]. In the second study [29], parasympathetic recovery was prolonged following exercise at intensities above the first ventilatory threshold compared with exercise performed below it—with no further delay in parasympathetic recovery observed following exercise of higher intensity and/or longer duration. Therefore, more studies comparing exercise of similar intensity but of varied duration are required (with longer post-exercise autonomic assessment) before we can make definitive conclusions (i.e. clear versus unclear) on the influence of exercise duration on cardiac parasympathetic reactivation kinetics. In general, following a single bout of exercise, while cardiac parasympathetic activity recovers to near pre-exercise levels within 90 min following low- and threshold-intensity exercise (Fig. 1a), complete recovery requires up to 24 h following low-intensity exercise, 24–48 h following threshold-intensity exercise, and at least 48 h following high-intensity exercise (Fig. 1b). Following low-intensity exercise, cardiac parasympathetic activity begins to decrease after 24 h of recovery (Fig. 1b), indicating the commencement of involution [3]. Therefore, although only speculative, these data suggest that the relatively small and transient changes in cardiac parasympathetic activity after low-intensity exercise may not be sufficient to induce substantial positive cardiorespiratory and metabolic adaptations [59]. Alternatively, because (at least) autonomic supercompensation following low-intensity training may occur within 24 h (Fig. 1b), and since cardiac parasympathetic reactivation is delayed by the build-up of metabolites, inclusion of low-intensity training subsequent to a high-intensity session may accelerate metabolite breakdown [88]. Athletes who train twice daily may also benefit from the accelerated recovery (metabolic recovery, as reflected by autonomic recovery) afforded by a low-intensity training session (Fig. 1a).

## 3 Cardiac Parasympathetic Reactivation Following 'Extreme' Exercise

We chose to focus the quantitative analysis part of the review on cardiac parasympathetic reactivation following 'typical' training sessions. Some studies that were excluded because of the 'extreme' nature of the exercise still provided interesting information on cardiac parasympathetic recovery. However, it is important to consider the effect of post-exercise hypotension [14, 15] and altered cardiac function [89] (which can persist for up to 24 h [90]) on the autonomic response following exercise of a prolonged nature. Three groups have investigated cardiac parasympathetic reactivation following ultra-endurance events. These events included a 46-km trail run at an altitude of 2,500 m (mean exercise duration 370 min, likely low-intensity) [14], a 75-km cross-country skiing race (mean exercise duration 271 min, threshold-intensity exercise) [16], and an Ironman triathlon (mean exercise duration 659 min, likely low-intensity exercise) [50]. The suppression of cardiac parasympathetic activity (on the day preceding versus 30–120 min post-exercise) was very large (effect size $= -2.1$ after the cross-country skiing [16]; effect size $= -2.0$ after the trail running [14]; effect size $= -2.2$ [50] after the Ironman triathlon). The athletes in these studies would have undoubtedly experienced excitement and other pre-event stressors, which would likely have reduced parasympathetic activity [30]. Daily monitoring of elite athletes in the lead up to a competition suggests that parasympathetic activity declines in the days prior to the event, possibly because of physiological changes induced by taper [91]. Therefore, the magnitude of cardiac parasympathetic suppression calculated here may underestimate the real effect of exercise per se. Cardiac parasympathetic activity returned to baseline within approximately 24 h, despite the prolonged nature of ultra-endurance exercise and the very large reduction in cardiac parasympathetic activity. Although insufficient data were available to demonstrate a 'binary' threshold for cardiac parasympathetic reactivation, these data suggest that exercise intensity is the primary determinant of post-exercise cardiac parasympathetic reactivation (see Sect. 2), but they also suggest that cardiac parasympathetic reactivation may be independent of the magnitude of the immediate reduction. Together, therefore, these data suggest that assessment of parasympathetic activity for only 5 min post-exercise is likely insufficient for accurate determination of the complete autonomic recovery time. Interestingly, Hautala et al. [16] observed a further increase in cardiac parasympathetic activity above pre-exercise within 48 h after the skiing race described above, which likely reflected supercompensation [3].

## 4 Cardiac Parasympathetic Reactivation Following Strength/Resistance Training

Heart rate variability data have also been used to monitor cardiac parasympathetic responses to specific strength/resistance training sessions. During recovery from resistance training, the heart rate is elevated, while cardiac parasympathetic activity is reduced [72, 75–77]. Heffernan et al. [75] reported that following a training session comprising three sets of 10 repetitions of eight exercises, 30 min of supine recovery was insufficient for the heart rate (+24 %, effect size = +4.8) and cardiac parasympathetic activity (−27 %, effect size = −1.2) to return to baseline levels. Similarly, Teixeira et al. [77] observed that following a training session comprising three sets of 20 repetitions of six exercises, cardiac parasympathetic activity was reduced by 44 % (effect size = −3.4) at 20 min post-exercise and by 21 % (effect size = −1.3) at 50 min post-exercise. Chen et al. [72] found that both weightlifting performance and cardiac parasympathetic activity decreased in the hours (up to at least 24 h) following a 2-h weightlifting training session. However, both weightlifting performance (i.e. seated shoulder press, effect size = 4.6; front squat, effect size = 3.6) and cardiac parasympathetic activity (effect size = 1.1) subsequently increased above baseline levels during the 48 to 72 h of recovery. Niemala et al. [76] observed that following a training session comprised of three sets of 12 repetitions of four exercises at 80 % of one repetition maximum, cardiac parasympathetic activity was reduced by 15 % (effect size = −1.0) 30 min following exercise, returning to baseline levels by 60 min. Interestingly, the same group observed no substantial reduction in cardiac parasympathetic activity following a 'lighter' resistance training session (three sets of 20 repetitions of four exercises at 30 % of one repetition maximum). These data suggest that performance appeared to recover in parallel with cardiac parasympathetic recovery, while markers of muscle damage (e.g. creatine kinase [56, 72]) and perceived muscle soreness did not follow the same time course [72]. Therefore, further research is necessary to better understand the application of monitoring cardiac parasympathetic activity after strength/resistance-based exercise.

## 5 Individualizing Training Prescription with Cardiac Parasympathetic Activity

An adaptable and flexible individualized training programme is the most practical tool for optimizing responses to exercise training [5]. Ideally, training programming and prescription requires integration and interpretation of all variables related to the athlete's current physiological and psychological state, and their training background. However, it is currently impractical (and in some instances impossible) to measure the state of all physiological systems that influence recovery from exercise. Furthermore, the kinetics of recovery differ between physiological systems and depend upon the training stimulus. For example, following high-intensity aerobic exercise, muscle performance [92] appears to recover faster than muscle glycogen repletion [93]. By contrast, the recovery of muscle force is more prolonged after eccentric strength training than after high-intensity aerobic exercise [94]. As previously outlined, changes in cardiac autonomic activity likely reflect exercise-induced changes in cardiac performance [13] and haemodynamics [14, 15], which are important components of overall recovery. However, our current understanding of the link between cardiac parasympathetic activity and all physiological components of recovery is incomplete. Nevertheless, when integrated with information on perceived fatigue and neuromuscular/musculoskeletal strain derived from training logs [95], cardiac parasympathetic reactivation kinetics may provide useful additional information for better individualization of training prescription/programming. In the following section, we discuss how to integrate data on the recovery of cardiac parasympathetic activity after exercise to customize training prescription/programming.

### 5.1 Training Guided by Cardiac Parasympathetic Reactivation Kinetics

Several studies have used cardiac parasympathetic reactivation data to individualize the programming of training sessions, particularly high-intensity aerobic sessions. As discussed earlier (see Sect. 1), this approach is based on the idea that training adaptation is potentiated during periods of supercompensation. In turn, these periods are likely the optimal time to train for greatest performance gain [3]. Kiviniemi et al. [62] trained one group of moderately fit men ($n = 9$) for 4 weeks on the basis of the following principles. If cardiac parasympathetic activity was similar to or higher than the previous day's value (i.e. 10 days' rolling average), then each individual completed a high-intensity training session. Alternatively, if cardiac parasympathetic activity was lower than the preceding day's value, then each individual rested or completed a low-intensity training session (see Fig. S2 in the Electronic Supplementary Material). Another group of men ($n = 8$) followed a standard training programme (2 × [1 day low-intensity, 2 days high-intensity], 1 day rest) for 4 weeks. The weekly training load (training impulse [TRIMP]) was moderately higher in the standard training group than in the HRV-guided group (effect size = 1.1). Peak oxygen consumption ($\dot{V}O_{2max}$) increased slightly in both the standard training group (+1.6 %, effect size = 0.2) and the HRV-

guided training group (+5.5 %, effect size = 0.4 respectively). By contrast, the maximal running velocity at the end of an incremental test after training was ~2× higher in the HRV-guided training group (+5.8 %, effect size = 0.9) than in the standard training group (+4.0 %, effect size = 0.5) [see Fig. S2 in the Electronic Supplementary Material].

In another study, moderately active men trained for 8 weeks following either a similar HRV-guided protocol ($n = 7$) or standard training ($n = 7$), which was flexible and based on the participant's subjective weekly goals [63]. The standard training group completed more training sessions per week (effect size = 3.8) than the HRV-guided training group did, but the weekly training load (TRIMP) tended to be higher in the HRV-guided training group (effect size = 0.3). $\dot{V}O_{2peak}$ increased to a greater extent in the HRV-guided training group (+7.2 %, effect size = 0.9) than in the standard training group (+4.6 %, effect size = 0.3), whereas the maximal running velocity at the end of an incremental test was similar in the HRV-guided training group (+11.1 %, effect size = 0.6) and the standard training group (+6.6 %, effect size = 0.7) [see Fig. S2 in the Electronic Supplementary Material].

These studies demonstrate that compared with traditional training prescription/programming, prescription of training based on individual changes in cardiac parasympathetic activity is equally—if not more—effective for improving aerobic exercise performance. These findings also support the notion that optimization of responses to exercise training requires an individualized training programme [5]. The effectiveness of HRV-guided training may be linked to the concept that the kinetics of cardiac parasympathetic recovery following exercise reflects acute/intermediate training responses, as described by the supercompensation theory. Reduced cardiac parasympathetic activity is not necessarily related to reduced exercise performance [96]. However, although prescription of reduced training intensity or rest on days when cardiac parasympathetic activity is reduced appears to be effective for long-term improvement in aerobic performance and cardiac parasympathetic activity in inactive/moderately trained individuals [62, 63], whether this applies to highly trained/elite athletes requires further research. Furthermore, adding low-intensity training sessions may accelerate recovery of cardiac parasympathetic activity following high-intensity sessions [29, 88]—possibly reflecting improved metabolic recovery, as discussed in Sect. 2.3. Gender specificity must also be considered, because cardiac parasympathetic reactivation following high-intensity training may be delayed in females [63, 97]. Females may require a longer period of recovery between high-intensity sessions. Therefore, to optimize training efficiency and training specificity, training prescription requires assessment of individual responses.

### 5.2 Individual Responses

Following a typical high-intensity interval training session for cyclists, the overall profile/pattern of recovery (an initial decrease immediately post-exercise, followed by a progressive increase in cardiac parasympathetic activity) appears to be similar in different individuals (Fig. 3a). Nevertheless, the specific cardiac parasympathetic reactivation profile differs between individuals (Fig. 3a) [30]. Complimentary to the work of Hautala et al. [16], who noted that greater cardiorespiratory fitness was related to more rapid recovery, we observed a large correlation ($r = 0.78$, 90 % confidence interval 0.21–0.95) between individual levels of cardiac parasympathetic recovery 125 min after exercise and $\dot{V}O_{2peak}$ values, albeit in a limited sample of seven athletes [30].

As athletes rarely perform a single training session in isolation, the compounding effect of subsequent training sessions further complicates an individual's cardiac parasympathetic recovery kinetics over a couple of days [32, 63]. The several-load summation theory suggests that athletes can perform a number of training sessions while they are not fully recovered. Under these conditions, the supercompensation effect occurs following the entire training cycle rather than after a single training session [2]. The analysis we have presented in this review suggests that cardiac parasympathetic activity requires 24–48 h to recover to pre-exercise levels after a high-intensity training session (see Sect. 3, Fig. 2b). Therefore, cardiac parasympathetic activity is likely to decrease over consecutive days of high-intensity training sessions separated by 24 h. Data from our recent study demonstrate the individual recovery response to five consecutive days of cycling training (Fig. 3b) [32]. Cardiac parasympathetic activity decreased following the first high-intensity training session in some athletes (A, B, E, H and I). However, after the second high-intensity session, cardiac parasympathetic activity returned to normal in three athletes (A, C and D), while it remained low in two other athletes (E and I) [Fig. 3b]. After the third high-intensity session, cardiac parasympathetic activity increased in all athletes (and was most pronounced in athlete H). Conversely, in some athletes (C, D, F and G), cardiac parasympathetic activity increased after the first high-intensity training session, followed by varied responses during the subsequent days (Fig. 3b). The individual variability in cardiac parasympathetic response can be explained in part by variations in training intensity from day to day. Throughout the three consecutive days of high-intensity training, in those

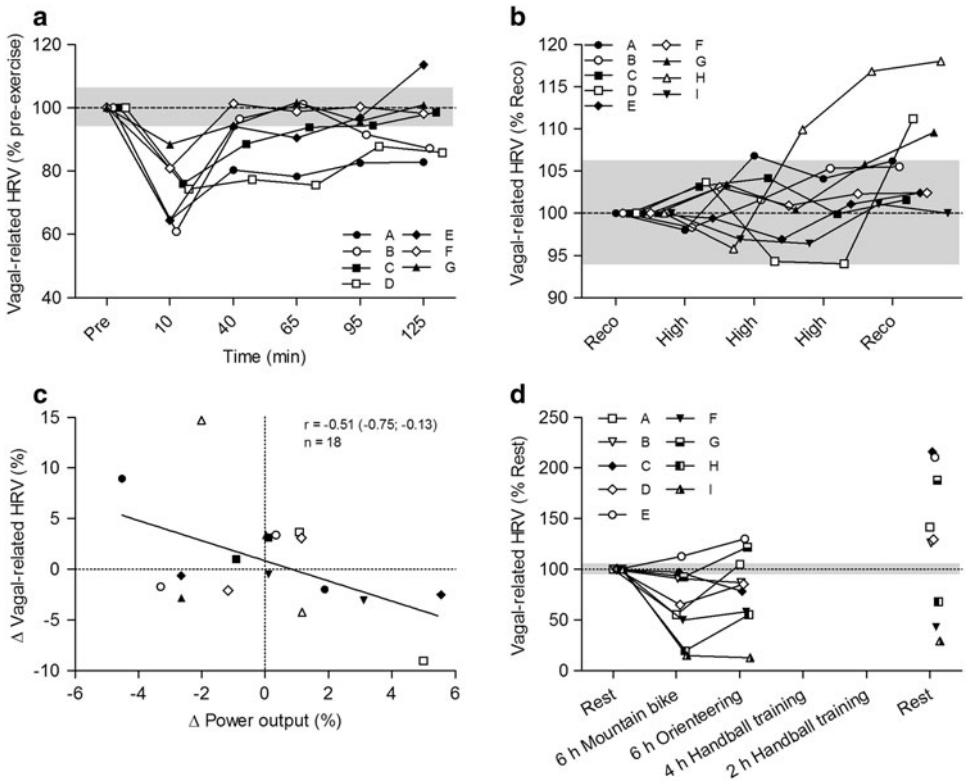


**Fig. 3** Individual cardiac parasympathetic responses to training. **a** Individual time course of cardiac parasympathetic reactivation following a single high-intensity training session (8 × 4 min at 80 % of peak power output, with 1 min active recovery) from immediately prior to exercise (Pre) through to 125 min post-exercise [30]. **b** Individual cardiac parasympathetic responses to 5 days of cycling training. The heart rate variability (HRV) values correspond to the measurements taken on the morning after a low-intensity training session or rest (Reco), or after a high-intensity training session (High) [32]. **c** Relationship (and 90 % confidence interval) between changes in cardiac parasympathetic activity from the preceding day and changes in mean high-intensity training session power output from the preceding day [32]. The different symbols represent individual athletes (i.e. there are two datapoints for each of nine athletes). **d** Individual cardiac parasympathetic responses of elite handball players during six consecutive days of training. The HRV values correspond to the measurements taken on the morning after a rest day, mountain biking, orienteering (running) or handball-specific training. The *shaded areas* represent the coefficient of variation for HRV indices under resting conditions [73]

athletes who trained at a higher relative intensity, HRV was often lower the following day (Fig. 3c). Although the large correlation does not imply causality, these data support our analysis in Sect. 2.2.1, highlighting the fact that cardiac parasympathetic reactivation at ≥24 h after exercise is dependent upon the intensity of the previous training session.

Team sport athletes often adopt a variety of different training modalities (i.e. speed, strength and high-intensity interval training sessions) to supplement specific game-based skill/tactical sessions. Pre-season data from a professional handball team illustrate both the cumulative effect of repeated low-to-moderate exercise sessions on HRV and the individual nature of cardiac parasympathetic responses to a variety of different training sessions (Buchheit, unpublished observations) [Fig. 3d]. Interestingly, among the nine players, only six (players A, B, C, D, E and G) showed a positive response to the training block (i.e. increased cardiac parasympathetic activity exceeding the coefficient of variation for HRV indices). The three 'non-responders' who showed a blunted cardiac parasympathetic response were the older and fitter players of the team, who also reported RPE values markedly lower than the team average during the whole camp. These basic observations suggest that the training load for these three players was possibly insufficient to induce HRV adaptations (and, in turn, possibly cardiorespiratory adaptations). The magnitude of improvement following the training block was also inversely related to age, training background and fitness. Specifically, the younger players who had lower fitness showed the greatest rebound in cardiac parasympathetic activity, which is consistent with the general training responses [98–100]. These data highlight the importance of considering the personal kinetics of cardiac parasympathetic recovery, training intensity and fitness when prescribing training.

### 5.3 Structuring a Microcycle

Manipulation of the acute responses to each training session is important not only for optimization of daily and/or weekly training periodization but also for mid- to long-term physiological and performance adaptations. The following section provides recommendations to help the practitioner understand how to individualize a training programme on the basis of the time course of cardiac parasympathetic reactivation. Recommendations on how to structure training within a weekly microcycle to improve cardiovascular fitness, induce overload or recover are provided in Table 2. For each weekly microcycle, a programme is suggested for highly trained, moderately trained and inactive individuals, with longer recovery allocated for individuals with lower fitness. Although speculative in nature, the timing of training sessions is based on the content of this review (group-averaged cardiac parasympathetic reactivation data). Ultimately, the practitioner will structure a unique programme for each individual athlete on the basis of their specific cardiac parasympathetic reactivation profile. The structure of each microcycle that we present is based on the three-zone model [29] and includes high-, threshold- and low-intensity aerobic/endurance-based training sessions [with specific examples for swimming, cycling, and running (see Table S1 in the Electronic Supplementary Material)]. We have not presented any guidelines for strength/resistance training, because there are currently insufficient data available on the time course of cardiac parasympathetic recovery following this type of exercise (see Sect. 5). To highlight the importance of appropriate timing between training sessions to achieve a desired training outcome, for all three microcycles (i.e. cardiovascular fitness, overload or recovery), we set a constant number of sessions per week for each athlete category, irrespective of the goal of the microcycle, i.e. 10 sessions per week for highly trained athletes, eight sessions per week for moderately trained athletes and four sessions per week for inactive individuals. Importantly, we have supplemented recovery periods of complete rest (between high- and threshold-intensity sessions) with low-intensity training to accelerate recovery [88], because cardiac parasympathetic supercompensation (and likely metabolic and cardiovascular recovery) from such sessions typically occurs within 24 h (Fig. 1b).

We propose that training to improve cardiovascular fitness should be structured such that the expected supercompensatory responses (Fig. 1a and b) are maximized and compounded within the weekly microcycle. Therefore, on the basis of our quantitative analysis (Figs. 1b and 2), at least 48 h of recovery should separate high- and threshold-intensity training sessions, depending on the athlete's fitness level (72 h for inactive individuals) [Table 2]. The rationale for this approach is to match high- and threshold-intensity training with the peak of the HRV supercompensation response. Because highly trained athletes are more resilient to exercise (Fig. 2), we have scheduled high- and threshold-intensity training sessions on two consecutive days to ensure sufficient exercise stimulus prior to a rest day (Table 2).

Overload training involves athletes performing several consecutive training sessions while they are not fully recovered. The supercompensation effect is expected to occur during a subsequent recovery microcycle that would typically follow the overload period. Therefore, to induce a training overload, based on our quantitative analysis, where 24 to >48 h is required (Fig. 1b), we have programmed key high- and threshold-intensity training sessions separated by only 12–48 h (Table 2). The timing of sessions ensures that athletes are sufficiently recovered to complete subsequent training sessions at the required intensity, but does not allow time for supercompensation. It is important to note that despite differences in the desired cardiac parasympathetic activity, the weekly total of high- and threshold-intensity sessions is identical to the microcycle intended to improve cardiovascular fitness (Table 2).

Recovery requires a reduced training stimulus (including added rest) to allow the body to adapt and regenerate. Typically, a recovery microcycle follows a period of overload training or is introduced when an athlete is tired or shows signs of overtraining [101]. Therefore, the recovery microcycle we have outlined is structured on the assumption that the athlete begins the week in a state of overload (i.e. they present with reduced cardiac parasympathetic activity). The amount of rest and the frequency of low-intensity training sessions are increased to accelerate recovery [88]. The frequency of high- and/or threshold-intensity training sessions is reduced, and the sessions are separated by at least 48 h of recovery to allow supercompensation (Fig. 1b). Our quantitative analysis suggests that because of their faster recovery kinetics (Fig. 2), highly and moderately trained athletes require less rest and low-intensity training for cardiac parasympathetic recovery (particularly at the beginning of the week when cardiac parasympathetic activity is expected to be lowest). This notion is reflected in the programming (Table 2).

The examples we have presented provide a general guide for structuring the weekly microcycle (with specific outcome goals in mind), based on the effects of training intensity, duration and athlete fitness/training status. The volume/duration of each training session needs to be appropriate to the individual (for examples, see Table S1 in the Electronic Supplementary Material). As we have briefly discussed earlier (in Sect. 5), the type/mode of training (i.e. strength/resistance versus aerobic endurance) influences the time required for recovery (both overall and for

**Table 2** Examples of weekly microcycles designed to induce specific training adaptations (prescription for male adults)[a]

| | Monday | Tuesday | Wednesday | Thursday | Friday | Saturday | Sunday | Weekly totals | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Sessions | HI | Th | LI |
| Microcycle to improve cardiovascular fitness[b] | | | | | | | | | | | |
| Highly trained | | | | | | | | | | | |
| AM | LI | LI | Rest | LI | HI | Th | Rest | 10 | 3 | 1 | 6 |
| PM | HI | LI | HI | LI | Rest | LI | Rest | | | | |
| Moderately trained | | | | | | | | | | | |
| AM | Rest | LI | Rest | HI | LI | Th | Rest | 8 | 2 | 1 | 5 |
| PM | HI | LI | LI | Rest | Rest | LI | Rest | | | | |
| Inactive | | | | | | | | | | | |
| AM | Rest | Rest | LI | Rest | Rest | LI | Rest | 4 | 2 | 0 | 2 |
| PM | HI | Rest | Rest | HI | Rest | Rest | Rest | | | | |
| Microcycle to induce overload[c] | | | | | | | | | | | |
| Highly trained | | | | | | | | | | | |
| AM | LI | LI | HI | LI | LI | Th | Rest | 10 | 3 | 1 | 6 |
| PM | HI | LI | Rest | HI | Rest | LI | Rest | | | | |
| Moderately trained | | | | | | | | | | | |
| AM | Rest | LI | LI | LI | Th | LI | Rest | 8 | 2 | 1 | 5 |
| PM | HI | Rest | HI | Rest | Rest | LI | Rest | | | | |
| Inactive | | | | | | | | | | | |
| AM | LI | Rest | Rest | HI | Rest | LI | Rest | 4 | 2 | 0 | 2 |
| PM | Rest | HI | Rest | Rest | Rest | Rest | Rest | | | | |
| Microcycle for recovery[d] | | | | | | | | | | | |
| Highly trained | | | | | | | | | | | |
| AM | LI | HI | LI | LI | LI | LI | Rest | 10 | 2 | 1 | 7 |
| PM | LI | LI | Rest | Th | Rest | HI | Rest | | | | |
| Moderately trained | | | | | | | | | | | |
| AM | Rest | LI | Rest | LI | HI | Rest | Rest | 8 | 2 | 0 | 6 |
| PM | LI | HI | LI | Rest | Rest | LI | Rest | | | | |
| Inactive | | | | | | | | | | | |
| AM | Rest | Rest | Rest | Rest | HI | Rest | Rest | 4 | 1 | 0 | 3 |
| PM | Rest | LI | LI | Rest | Rest | LI | Rest | | | | |

*AM* morning, *HI* high-intensity training, *LI* low-intensity training, *PM* afternoon or evening, *Th* threshold-intensity training

[a] Individualized training programmes have been optimized on the basis of the idea that training adaptation is potentiated during periods of (cardiac autonomic) supercompensation, as determined by cardiac parasympathetic reactivation kinetics. For inactive and/moderately trained individuals, a single high-intensity training session may be sufficient to induce a supercompensation response. However, in highly trained athletes, who recover more rapidly from intensive training, several consecutive intensive training sessions with incomplete recovery (i.e. overload microcycles) may be required to induce a worthwhile supercompensation response

[b] On the basis of the examples of specific training sessions described in Table S1 in the Electronic Supplementary Material, the expected weekly volume of training completed during a microcycle designed to improve cardiovascular fitness in highly trained athletes would be 38–54 km (12–17 h) for swimmers, 550–1,000 km (17.5–32.5 h) for cyclists and 100–140 km (7.5–12.5 h) for runners; for moderately trained athletes, it would be 23–31 km (7–11 h) for swimmers, 320–490 km (9.5–15.5 h) for cyclists and 60–92 km (4.75–8.75 h) for runners; and for inactive individuals, it would be 3–8 km (1.6–3.3 h) for swimmers, 34–90 km (1.6–4 h) for cyclists and 10–18 km (1.6–3 h) for runners

[c] On the basis of the examples of specific training sessions described in Table S1 in the Electronic Supplementary Material, the expected weekly volume of training completed during a microcycle designed to induce overload in highly trained athletes would be 38–54 km (12–17 h) for swimmers, 550–1,000 km (17.5–32.5 h) for cyclists and 100–140 km (7.5–12.5 h) for runners; for moderately trained athletes, it would be 23–31 km (7–11 h) for swimmers, 320–490 km (9.5–15.5 h) for cyclists and 60–92 km (4.75–8.75 h) for runners; and for inactive individuals, it would be 3–8 km (1.6–3.3 h) for swimmers, 34–90 km (1.6–4 h) for cyclists and 10–18 km (1.6–3 h) for runners

[d] On the basis of the examples of specific training sessions described in Table S1 in the Electronic Supplementary Material, the expected weekly volume of training completed during a microcycle designed for recovery in highly trained athletes would be 36–53 km (11.5–16.5 h) for swimmers, 380–675 km (13.75–22 h) for cyclists and 84–122 km (6.6–10.5 h) for runners; for moderately trained athletes, it would be 22–30 km (6.5–10.5 h) for swimmers, 290–410 km (9–14 h) for cyclists and 56–86 km (4.5–8.75 h) for runners; and for inactive individuals, it would be 2.5–7 km (1.5–3 h) for swimmers, 31–85 km (1.5–4 h) for cyclists and 9–17 km (1.5–2.5 h) for runners. Highly and moderately trained athletes may substitute a single long slow distance training session for two recovery training sessions (see Table S1 in the Electronic Supplementary Material) on days when two LI sessions are scheduled

individual physiological systems). Other factors relevant to the individual that will likely influence the cardiac parasympathetic response to exercise but are not discussed in detail in this review include the following: increasing age [66], female gender [63, 97], mental stress (i.e. from social and work/study commitments) [68, 70], central fatigue [70] and environmental conditions (e.g. altitude/hypoxia [14, 46, 102–105], cold [16, 106] or heat [56, 107, 108]). When designing these programmes, it is important to consider these factors on balance and adjust the recovery duration accordingly.

## 6 Conclusions

Understanding the time required for recovery following a training session is essential for optimizing daily and weekly periodization, in addition to mid- to long-term physiological and performance adaptation. Although there are infinite ways to structure a training programme [88], it is becoming increasingly evident that individualized training prescription and programming is most effective for attaining optimal physiological outcomes [5].

Cardiac parasympathetic reactivation following a training session is unique to an individual and appears to track the acute/intermediate (at least cardiovascular) recovery response described by the supercompensation theory [3]. The physiological mechanisms underlying the cardiac parasympathetic response are not completely understood. Nevertheless, the accumulation of stress metabolites is likely a key determinant of acute cardiac parasympathetic reactivation [31, 40, 41, 45, 46], while exercise-induced changes in plasma volume likely influence intermediate recovery [51, 52]. Our quantitative analysis reveals that complete cardiac parasympathetic reactivation requires up to 24 h after low-intensity aerobic exercise (Fig. 1), 24–48 h after threshold-intensity exercise and least 48 h after high-intensity exercise (Fig. 1b). Individuals with greater fitness are more resilient to training stress and require less time to recover, as indicated by smaller changes and more rapid recovery of cardiac parasympathetic activity after exercise (Fig. 2). On the basis of the limited evidence currently available, the influence of exercise duration on the time course of cardiac parasympathetic recovery is unclear. However, exercise duration is unlikely to be the greatest determinant of cardiac parasympathetic recovery (see Sect. 2.2.3). Supercompensatory responses (i.e. enhanced cardiac function and $\dot{V}O_{2max}$ resulting from increased plasma volume [57, 58]) may occur within 48 h of high-intensity training (Fig. 1b). Training during such periods of supercompensation is likely optimal for the greatest performance gain (i.e. metabolic, cardiovascular), as training is potentiated [3]. Therefore, monitoring of changes in the level of cardiac parasympathetic activity following exercise provides a good starting point for programming training sessions that aim to induce metabolic and cardiorespiratory adaptations.

Cardiac parasympathetic reactivation does not parallel the recovery kinetics of all physiological systems (i.e. the neuromuscular system). Currently, data on the cardiac parasympathetic reactivation following strength/resistance-based exercise of varied intensity are limited (see Sect. 5). Variables such as muscle soreness, which cannot be tracked by cardiac parasympathetic activity, may be monitored using training logs [95, 109]. The duration of recovery indicated by cardiac parasympathetic reactivation must also be adjusted on the basis of the level of (perceived) neuromuscular and musculoskeletal strain induced by a training session independent of exercise intensity. This is of particular importance for athletes training twice daily and for team sport athletes, where low-intensity training can place high demands on the neuromuscular system [110–112]. The time course of cardiac parasympathetic reactivation is also influenced by multiple variables (related to the athlete profile and training environment), which are not discussed in detail in this review. Accumulating evidence suggests that cardiac parasympathetic recovery kinetics integrate multiple factors that are pertinent to assessing an athlete's state of recovery. Therefore, we propose that in conjunction with daily training logs, individual cardiac parasympathetic activity provides a strong platform for designing customized training programmes to optimize metabolic and cardiorespiratory adaptation.

To implement such a monitoring system, coaches and athletes should measure cardiac parasympathetic activity and include training logs in their daily routine. We suggest recording HRV in a seated position for 5 min immediately after waking in the morning, as this reduces external confounding factors, some of which are listed below. Athletes can choose their own timing and routine (e.g. whether they empty their bladder prior to recording, etc.), as long as their routine is consistent. Additionally, variables that are known to influence HRV recordings (i.e. the temperature of the room, the luminosity of the room, noise etc.) should be standardized, within reason. Above all, the routine should be designed such that it is not a burden on the athlete, so that they will maintain their routine in the long term. We encourage practitioners to use a single HRV index to assess cardiac parasympathetic activity. Research suggests that Ln rMSSD provides the most reliable [73] and practically applicable measure for day-to-day monitoring, particularly under ambulatory conditions [79]. Training logs can be simple (i.e. with a rating scale of 1–7) but should record sleep quality, stress, fatigue, muscle soreness, perceived exercise duration and exercise intensity. These data provide information on central and peripheral factors relating to an

athlete's readiness to perform [95, 109, 113]. The use of HRV data to predict an athlete's freshness/readiness to perform or overtraining, particularly in elite athletes, is beyond the scope of this review but is an area gaining more attention [32, 64, 91, 114, 115]. Once athletes have established a feasible monitoring routine, they can begin to monitor the HRV response (in conjunction with training logs) following different types of training sessions (i.e. high-intensity sessions of short versus long duration) to understand their individual recovery time. They can then use these data to design/better individualize weekly training microcycles and can also gain feedback on how the effective the training structure is for achieving the desired response.

The reliability of the intermediate HRV response (i.e. 48 h post-exercise) following identical training sessions requires clarification. Additionally, better understanding of the cumulative effects of multiple training sessions (e.g. cycling in the morning and afternoon; team-sport specific sessions with a ball in the morning and strength/resistance training in the afternoon) on cardiac parasympathetic reactivation kinetics is required. The effect of a given training stimulus on exercise performance is fundamental to planning training sessions and attaining peak performance for targeted competition. Increasing evidence suggests that individual changes in cardiac parasympathetic activity may predict improvements in performance [96, 116–119] and fitness [96, 120]. Future research should investigate the underlying links between the recovery process (i.e. psychological/central fatigue) and cardiac parasympathetic activity, and specifically examine whether cardiac parasympathetic activity actively affects or simply reflects the recovery process.

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