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Effects of varying training load on heart rate variability and running performance among an Olympic rugby sevens team

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ABSTRACT

Objectives: To evaluate weekly heart rate variability (HRV) responses to varying training load among an Olympic rugby sevens team and to assess whether HRV responses informed on training adaptation.
Design: Retrospective.
Methods: Natural logarithm of the root mean square of successive differences (LnRMSSD), psychometrics and training load from a rugby sevens team (n = 12 males) over a 3-week period were retrospectively analyzed. Week 1 served as baseline while weeks 2 and 3 consisted of peak training loads from the 2016 Olympic preparatory period. Maximum aerobic speed (MAS) was evaluated at the beginning of weeks 1 and 3.
Results: LnRMSSD ($p = 0.68$), its coefficient of variation (LnRMSSDcv) ($p = 0.07$) and psychometrics (all $p > 0.05$) did not significantly change across time. Effect sizes (ES) showed a small increase in LnRMSSDcv after the first week of intensified training (ES = 0.38) followed by a moderate reduction in week 3 (ES = −0.91). Individuals with a smaller LnRMSSDcv during the first week of intensified training showed more favorable changes in MAS ($r = -0.74$, $p = 0.01$), though individual changes only ranged from −1.5 to 2.9%.
Conclusions: In week 3, players accomplished greater external training loads with minimal impact on internal load while wellness was preserved. Concurrently, players demonstrated less fluctuations in LnRMSSD, interpreted as an improved ability to maintain cardiac-autonomic homeostasis despite increments in training load. Monitoring the magnitude of daily fluctuations in LnRMSSD in response to varying training loads may aid in the evaluation of training adaptations among elite rugby players.

1. Introduction

The physical demands of rugby sevens include high-intensity intermittent running, physical collisions such as rucking and tackling and the expression of both technical and tactical abilities.[1] Among other physical qualities, players require well developed cardiovascular fitness to compete at a high level.[2] For example, elite players have been reported to cover ~1.6 km during a 14-min match with an average running speed of 6.4 km h^{-1} and average playing intensity >80% of maximum heart rate.[3] Moreover, the intensive running demands unique to rugby sevens competition have been suggested to contribute to an increased injury risk compared with other rugby codes.[4]

While elite rugby seven players display small inter-player variability in physical and performance characteristics,[5] training responses among players tend to be individual.[6] In appreciation of the non-uniformity in training adaptation among players, it's been suggested that practitioners monitor variables that reflect an individual's capacity to respond to a given training stimulus.[7] In turn, considerable research into various methods for monitoring fatigue and adaptation in players has been performed.[8] However, the variable(s) that provide the best indication of adaptation, particularly among elite team-sport players, still need to be determined.[8]

An individual's homeostatic stress response to training may reflect the magnitude of the imposed training stimulus and is hypothesized to contribute to individual variation in adaptation.[6] The physiological expression of stress is governed largely by the autonomic nervous system which can be assessed easily and non-invasively via heart rate variability (HRV).[9] Monitoring changes in cardiac-autonomic activity, inferred from vagally-mediated (i.e., parasympathetic) HRV, shows promise for reflecting responses and

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adaptation to training programs.[10] Assessed weekly, both average cardiac-vagal activity or its daily fluctuation (quantified by the coefficient of variation, CV) are sensitive to training load[11–13] and running performance.[14–16] Reductions and greater daily fluctuation in vagally-mediated HRV have been observed in individuals exhibiting fatigue and responding poorly to training.[12,16,17] Thus, HRV is an attractive tool to coaches and sports scientists for monitoring players throughout training for evaluating individual adaptation.

The usefulness of HRV for reflecting responses and adaptation to training among elite rugby sevens players has yet to be investigated. This research is needed because rugby sevens players are exposed to rigorous training, competition and international travel schedules which may put them at-risk of fatigue accumulation or injury. Therefore, the aims of this retrospective study were to evaluate weekly HRV responses to varying training load among an Olympic rugby sevens team and to assess whether HRV responses informed on training adaptation (i.e., running performance and perceptual responses). We hypothesize that minimal daily fluctuation in HRV in response to an increase in training load may indicate that loads are being well-tolerated with adequate autonomic recovery between daily training sessions, preventing decrements in or enabling improved running performance.[16]

2. Methods

De-identified data from all members (n = 12 males; height = 184.9 ± 7.1 cm; weight = 90.7 ± 7.0 kg; sum of 8 skinfolds = 63.0 ± 14.3 mm) of an Olympic rugby sevens team were used for this analysis. Ethics approval for retrospective analysis of the pre-existing data was provided by the Institutional Review Board. Consent was obtained from players after being informed that their data would be used for research and their identity would remain anonymous.

This study features a 3-week training period from the preparatory phase of a rugby sevens team in their lead up to the 2016 Olympics. This period was selected for analysis because it involved one week of low training load (baseline) followed by a two-week repeated microcycle sequence consisting of peak training load values from the preparatory period (~8 weeks prior to the Olympics). Additionally, maximum aerobic speed (MAS) was tested at baseline (i.e., beginning of week 1) and again mid-way through the intensified, repeated microcycles (i.e., beginning of week 3). The observation period did not include travel or competitions which are factors known to influence physiological and perceptual recovery status markers. These characteristics (i.e., varying weekly loads, repeated performance testing and lack of travel or competition) enabled us to address the aims of the study described above.

The training structure for each week is presented in Table 1. Weekly training load values in the two weeks before baseline did not exceed baseline values, indicating that the intensified microcycles (i.e., weeks 2 and 3) involved loads that had not been experienced by the players for at least three weeks. The microcycle structure for weeks 2 and 3 were identical (Table 1). Speed sessions were ~40 min in duration and consisted of warm-up (10–15 min), drills (10 min) and multiple sprint efforts (6.9 ± 1.6 sprints/session) for both acceleration and maximal velocity running. Resistance training sessions were ~60 min, performed thrice weekly and involved both strength and power development (work >80% of the 1 repetition maximum). Rugby sessions were ~70 min and involved technical and tactical work and game simulations.

On Monday of weeks 1 and 3 at the same time and location, running performance was evaluated via 1200 m time trial for the determination of MAS. Previous research showed that MAS is strongly related to distance covered during professional rugby competition.[18] The MAS test was performed on a rugby field measured 100 m in length and thus required completion of 12 continuous field-length repetitions. Players were encouraged to complete the test as fast as possible. The time to completion in seconds was divided by the distance in meters to calculate m s^{-1}.

Subjective measures of wellbeing were acquired daily throughout the observation period via wellness questionnaire adapted from Gastin et al.[19] On a 1–10 likert-type scale, players rated their perceived level of sleep quality, energy level, muscle soreness, recovery and mood. Questionnaires were completed via smartphone daily by 8:00 am. The weekly mean for each wellness variable was calculated intra-individually for analysis. Wellness data were used to facilitate interpretation of weekly HRV responses.[10]

Players recorded their resting-HRV seated for 60 s[20] daily after waking and before food or fluid ingestion while remaining motionless and breathing spontaneously. HRV was recorded with a Bluetooth chest-strap (H7 Polar Electro, Kempele, Finland) paired with a freely available smartphone application (Elite HRV, Asheville, North Carolina, USA). The vagally-mediated HRV parameter used for analysis was the logarithm of the root-mean square of successive differences (LnRMSSD) as this is the preferred parameter for player-monitoring in field-settings.[10] Both the weekly mean of LnRMSSD (LnRMSSDm) and it's CV (LnRMSSDcv) were calculated intra-individually for analysis.[11] Overall compliance with daily HRV recordings was 87 ± 10%.

While the Polar H7 has been shown to accurately obtain RR intervals,[21] agreement between the Elite HRV application and electrocardiography (ECG) for determining ultra-short LnRMSSD has not been investigated. Therefore, we compared simultaneous recordings of LnRMSSD derived from the Elite HRV application and ECG (Biopac MP100, Colletta, CA, USA) in supine, seated and standing positions among 10 collegiate athletes. We replicated the comparison and statistical procedures used previously.[22] Differences between supine, seated and standing measures were not significant ($p = 0.80$, 0.52 and 0.49, respectively) and the standardized differences (effect size, ES) were considered trivial (ES ≤0.03 for each). The correlations between the application and ECG were near perfect ($r = 0.99$ for each position). Additionally, upper and lower limits of agreement were tight (upper and lower limits = 0.03 to −0.03 for supine, 0.08 to −0.10 for seated and 0.13 to −10 for standing).

During field-based training sessions, players wore a GPS device (10 Hz Viper Pod, STATSports, Newry, Ireland) for the quantification of external training load. Similar GPS devices using sampling rates of 10 Hz have been shown to be valid and reliable for quantifying running-based movement.[23] The devices were secured in a pocket on the compression shirt, positioned between the scapulae. Recorded movement variables included total meter distance covered (TD), meter distance covered at high speed (HS) (≥18 km h^{-1}) and number of accelerations (ACC) and decelerations (DEC) (≥3 m s^{-2}).[1] Internal training load was quantified via the session rating of perceived exertion (sRPE) method. Within 30 min following any training session, players rated their perceived level of exertion using the CR-10 Borg scale which was subsequently multiplied by the session duration in minutes.[24] The weekly sum for each training load variable was calculated intra-individually for analysis.

Analyses were carried out using Excel 2016 (Microsoft Corp., Redmond, Washington, USA) and JMP Pro 13 (SAS Institute Inc., Cary, North Carolina, USA). Data are reported as mean ± SD. $P < 0.05$ was the threshold for statistical significance. Shapiro–Wilk tests revealed that all variables met the assumption of normality ($p > 0.05$). Linear mixed models were used to evaluate weekly variation in LnRMSSD (mean and CV), psychometric and training load parameters. Tukey's Honest Significant Difference (HSD) test was

Table 1
Microcycle training structure for each week of training.

Week	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
1	Off	RT	Speed, RT, rugby	Rugby, RT	Rugby	Off	Off
2 & 3	Speed, RT, rugby	Rugby, RT	Off	Rugby, RT	Rugby, RT	Off	Off

Off = no training; RT = resistance training; Speed = speed training; Rugby = rugby training.

used for post-hoc analyses of significant effects. For each model, time (i.e., week) was included as a fixed within-subjects repeated measure and player identification was included as a random effect. ES were used to evaluate the magnitude of changes between weeks for each variable.[25] ES were interpreted using the following qualitative thresholds: <0.20 was trivial; 0.20–0.59 was small; 0.60–1.19 was moderate; 1.20–1.99 was large; and >2.0 was very large.[26]

MAS values were compared via paired t-test and ES. Partial correlations, adjusting for baseline MAS, were used to determine whether changes in running performance from baseline to beginning of week 3 (MAS week 3 – MAS baseline = ΔMAS) were related with LnRMSSD-derived variables. Since the second MAS test was performed on Monday of week 3 (i.e., before week 3 training loads), only weeks 1 and 2 LnRMSSD values were analyzed. Correlation coefficients were interpreted using the following qualitative thresholds: 0–0.29 was small, 0.30–0.49 was moderate, 0.50–0.69 was large, 0.70–0.89 was very large, and 0.90–1.00 was near perfect.[26] The smallest worthwhile change (SWC) in MAS was determined as ± 0.2 of the between-player standard deviation from baseline values.[25]

3. Results

HRV, psychometric and training load values along with comparison statistics are displayed in Table 2. LnRMSSDm ($p = 0.68$), LnRMSSDcv ($p = 0.07$) and psychometrics (all $p > 0.05$) did not significantly change across weeks. ES analysis showed that LnRMSSDcv demonstrated a small increase from weeks 1 to 2 followed by a moderate reduction from weeks 2 to 3 (Table 2). Individual LnRMSSDm and LnRMSSDcv values from weeks 1 to 3 are displayed in Fig. 1a. Significant effects were observed for all training load variables (all $p < 0.05$). Post-hoc analyses revealed large to very large increases in each training load parameter from baseline to week 2 while TD and HS increased moderately from weeks 2 to 3.

Two players did not complete a MAS test and therefore only 10 players' data was used for related analyses. The difference between baseline MAS ($4.56 \pm 0.13\ m\ s^{-1}$) and week 3 MAS ($4.57 \pm 0.12\ m\ s^{-1}$) was trivial and non-significant ($p = 0.59$, ES = 0.08). Partial correlation analyses, adjusting for baseline MAS, revealed that the LnRMSSDm of weeks 1 and 2, LnRMSSDcv of week 1 and the changes in LnRMSSDm and LnRMSSDcv from weeks 1 to 2 were not related to ΔMAS (r ranged from −0.37 to 0.26, all $p > 0.05$). On the other hand, week 2 LnRMSSDcv significantly related with ΔMAS ($r = -0.74$, $p = 0.01$) (Fig. 1b). Players were grouped according to whether their MAS improved (Group 1, n = 4), did not change (Group 2, n = 2), or decreased (Group 3, n = 4) according to SWC thresholds. Group values for ΔMAS and week 2 LnRMSSDcv are displayed in Fig. 1c.

4. Discussion

The purpose of this study was to evaluate weekly changes in HRV in response to varying training load and to determine whether HRV parameters informed on individual training adaptation among an Olympic rugby sevens team. The main finding was that the first exposure to the intensified microcycle provoked a small increase in LnRMSSDcv. Subsequently, players demonstrated a moderate reduction in LnRMSSDcv, interpreted to reflect an improved ability to maintain cardiac-autonomic homeostasis during the second exposure to increased training load. Additionally, week 2 LnRMSSDcv values were inversely associated with individual changes in running performance, independent of baseline MAS.

Previous studies in collegiate soccer players[11] and short-distance swimmers[12] reported moderate increases in LnRMSSDcv during 1–2 weeks of intensified training, concurrent with decrements in psychometric parameters. We observed only a small increase in LnRMSSDcv in response to the first week of intensified training while psychometric parameters were maintained. Thus, the elite players from the current study demonstrated better tolerance to increased training load relative to collegiate players.[11,12] We found no change in LnRMSSDm at the team level after an increase in training load. Moreover, individual analysis demonstrated that only 4 of 12 players experienced a change in LnRMSSDm that exceeded the SWC threshold commonly used for LnRMSSD.[12,17] The unchanged LnRMSSDm observed in the current study contrasts with results from collegiate players who demonstrated small – moderate reductions in LnRMSSDm following 1–2 weeks of intensified training.[11,12] However, the finding of no change in LnRMSSDm is in agreement with that of professional futsal players who showed no changes (LnRMSSDm ranged from 3.8 ± 0.5 to 4.0 ± 0.3) across a 5-week preseason.[27] Evaluated daily, Thorpe et al. found no significant ($p > 0.05$) changes in LnRMSSD at the team-level among professional soccer players during in-season microcycles.[28] Nakamura et al. reported only small daily changes in LnRMSSD across a 1-week training camp among national rugby players.[20] Thus, it seems that at the team-level, LnRMSSDm is subject to less variation, while LnRMSSDcv may be a more sensitive training-response marker among elite-level players. Nevertheless, it is likely that LnRMSSDm is still meaningful at the individual level when monitored over chronic training periods in elite players as persistent reductions in LnRMSSDm exceeding the SWC may reflect a more severe level of maladaptation.[17] We speculate that the imposed load and frequency of training during the current observation period, despite being peak loads from within 12 weeks of the Olympics, were not high enough to provoke LnRMSSDm changes at the team level due to the elite status and training history of the players.

Previous studies in collegiate players have reported a return of LnRMSSDcv to baseline[12] or below baseline[11] in response to training load reduction. In the present study, week 3 LnRMSSDcv demonstrated a moderate reduction despite significant increments in TD and HS, though sRPE did not change significantly. A decrease in the internal to external training load ratio has been suggested to reveal a positive adaptation.[29] The sRPE (i.e., internal load) to HS (i.e., external load) ratio decreased (−13%, ES = −0.60) from week 2 to 3, indicating that greater external workloads were accomplished in week 3 with minimal impact on perceived training load. We speculate that the players performed greater external workloads in week 3 because they had become familiarized with the training structure and content of week 2. Adaptation to the first exposure of the intensified microcycle may explain why less perturbation in cardiac-autonomic homeostasis (i.e., reduced LnRMSSDcv) was observed upon the second exposure in week 3. A previous investigation demonstrated small to moderate reductions in LnRMSSDcv among professional futsal players after exposure to sRPE loads that

Table 2
Cardiac-autonomic, psychometric and training load values and comparison statistics.

	Week 1	Week 2	Week 3	Comparison statistics (P, ES)		
				Weeks 1 vs. 2	Weeks 2 vs. 3	Weeks 1 vs. 3
LnRMSSDm	4.41 ± 0.36	4.43 ± 0.33	4.45 ± 0.39	NS, 0.06	NS, 0.06	NS, 0.11
LnRMSSDcv (%)	6.32 ± 2.58	7.22 ± 2.13	5.38 ± 1.90	NS, 0.38	NS, −0.91	NS, −0.41
Sleep (au)	7.59 ± 0.74	7.50 ± 0.92	7.76 ± 0.77	NS, −0.11	NS, 0.31	NS, 0.23
Energy (au)	7.11 ± 0.82	7.40 ± 0.83	7.44 ± 0.83	NS, 0.35	NS, 0.05	NS, 0.40
Soreness (au)	6.95 ± 0.90	7.12 ± 0.96	7.17 ± 1.01	NS, 0.18	NS, 0.05	NS, 0.23
Recovery (au)	6.69 ± 0.76	6.85 ± 0.77	6.95 ± 0.80	NS, 0.21	NS, 0.13	NS, 0.33
Mood (au)	8.23 ± 0.88	8.36 ± 0.89	8.26 ± 1.04	NS, 0.15	NS, −0.10	NS, 0.03
sRPE (au)	2455.6 ± 262.8	3515.1 ± 743.1	3639.9 ± 307.6	<0.05, 1.90	NS, 0.22	<0.05, 4.13
Total dist (m)	12065.1 ± 1138.5	27250.2 ± 3693.9	30381.8 ± 1855.1	<0.05, 5.56	<0.05, 1.07	<0.05, 11.90
High speed (m)	1949.8 ± 882.5	4857.2 ± 857.4	5788.8 ± 825.3	<0.05, 3.34	<0.05, 1.11	<0.05, 4.49
High accel	102.8 ± 34.0	236.3 ± 46.7	232.9 ± 41.1	<0.05, 3.27	NS, −0.08	<0.05, 3.45
High decel	72.8 ± 16.2	190.0 ± 36.5	200.8 ± 39.0	<0.05, 4.15	NS, 0.29	<0.05, 4.29

LnRMSSDm = weekly mean natural logarithm of the root-mean square of successive differences; LnRMSSDcv = weekly coefficient of variation of the natural logarithm of the root-mean square of successive differences; sRPE = session rating of perceived exertion; Dist = distance; Accel = accelerations; Decel = decelerations; ES = effect size; NS = not significant (p > 0.05).

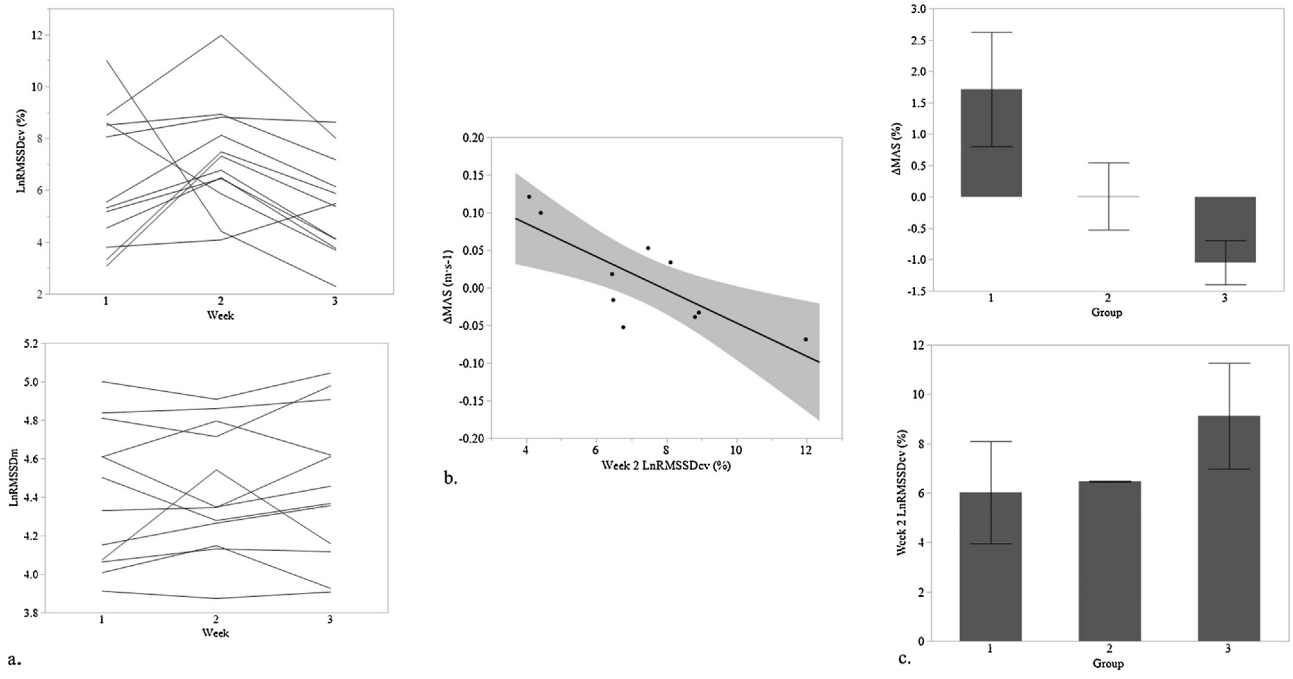

Fig. 1. (a) Individual values for the weekly mean (LnRMSSDm) and coefficient of variation (LnRMSSDcv) of the natural logarithm of the root mean square of successive differences. LnRMSSDm responses were inconsistent whereas 9 of 12 players showed some increase in LnRMSSDcv from weeks 1 to 2 and 10 of 12 players showed some decrease in LnRMSSDcv from weeks 2 to 3. (b) Scatterplot of the coefficient of variation of the natural logarithm of the root mean square of successive differences (LnRMSSDcv) from week 2 and the change in maximal aerobic speed (ΔMAS). The 95% confidence interval is represented by the shaded area. (c) Comparison of the coefficient of variation of the natural logarithm of the root mean square of successive differences (LnRMSSDcv) from week 2 and changes in maximum aerobic speed (ΔMAS%) among players who improved (Group 1, n = 4), did not change (Group 2, n = 2) or decreased (Group 3, n = 4) MAS based on the smallest worthwhile performance change threshold.[25] There is considerable overlap in MAS responses among individuals with LnRMSSDcv near the group average (e.g., LnRMSSDcv values between 6–8%). LnRMSSDcv values <6% and >8% were more sensitive to changes in MAS.

elicited greater LnRMSSDcv values in previous weeks.[27] Accordingly, the moderate reduction in LnRMSSDcv observed in the current study at week 3 is interpreted as a positive coping response by the players. This assertion was further supported by the preservation of wellness parameters in week 3 relative to baseline, which typically decrease with intensified training.[11,12,19]

After adjusting for baseline MAS, week 2 LnRMSSDcv was inversely associated with ΔMAS. This indicates that players who experienced less daily fluctuations in LnRMSSD in response to the initial spike in training load demonstrated more favorable MAS responses and vice-versa. A previous study among collegiate female soccer players found that reductions in LnRMSSDcv midway through a 5-week off-season training program were inversely associated with improvements in intermittent running performance from pre- to post-training.[16] The lack of association between LnRMSSDm and changes in running performance contrasts previous investigations that found large relationships between changes in LnRMSSDm and aerobic fitness among recreational runners[30] and collegiate soccer players.[16] However, it should be noted that the individual changes in MAS observed in the current study were small (ΔMAS range −1.5 to 2.9%) due to the brief observation period. Moreover, it is difficult to determine if the MAS changes were a result of changes in fitness or fatigue. Thus, additional research is needed involving longer periods of training that may cause larger MAS changes to further determine associations

between LnRMSSD parameters and changes in running performance among elite team-sport players.

This study is limited by the brief observation period and small sample size. The lack of laboratory measures of fitness and physiological responses (e.g., neuroendocrine, muscle damage and inflammatory markers) and use of a smartphone application for HRV measurements are also limitations. Future research among elite team-sport players over longitudinal training periods is needed with various laboratory-based markers of performance (e.g., neuromuscular and psychomotor) to develop a further understanding of LnRMSSD responses to training and their associations with performance adaptation.

5. Conclusion

The current findings provide some support for the hypothesis that individuals experiencing less fluctuations in LnRMSSD during intensified training are responding more favorably to the stimulus. Individuals who exhibited a smaller LnRMSSDcv during the initial spike in training load (i.e., week 2) displayed more favorable changes in running performance and vice-versa. However, this correlation should be interpreted with caution given that the individual changes in MAS were quite small. In week 3, players accomplished greater external training loads with minimal impact on internal training load while wellness markers were preserved. Concurrently, the players displayed moderate reductions in LnRMSSDcv, possibly reflecting an improved ability to maintain cardiac-autonomic homeostasis during the repeated microcycle.

Practical implications

- When evaluated as a group, LnRMSSDcv may be a more sensitive training response marker than LnRMSSDm during training load variations among elite players.
- LnRMSSDcv did not display a linear dose–response relationship with training load. Rather, LnRMSSDcv seems to reflect an adaptive physiological response to the imposed training stimulus which may be useful for identifying individuals responding undesirably to training.
- Elite rugby players presenting large day-to-day fluctuations in LnRMSSD in response to training load variation should be monitored closely for performance decrements, particularly when nearing important competitions.

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